Sarcoptic mange and the demography of the red fox, Vulpes vulpes

ELEANOR SARAH DEVENISH-NELSON

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By

Eleanor Sarah Devenish-Nelson

School of Biological and Biomedical Sciences

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Abstract

Vertebrate species are managed for many reasons, including their role as economically important predators or as carriers of disease. Successful management depends on the ability to predict the outcome of management actions on a species' population dynamics. However, uncertainty in the models used to make such predictions can arise from multiple sources, including sampling error in vital rates, intraspecific demographic variation and unknown interspecific interactions. The red fox *Vulpes vulpes* provides a useful model organism for exploring such uncertainty, because management of this important predator and disease host is often ineffective, despite substantial sampling effort.

By explicitly accounting for sampling error in survival and fecundity, confidence intervals for population growth rates were derived from published point estimates of red fox demographic data. Uncertainty in population growth rates was found to be high, requiring a quadrupling of sampling effort to halve the confidence intervals. Given the often poor justification for the choice of distribution used to model litter size, the influence of probability distributions on population model outcomes was tested. In this first comprehensive evaluation, estimates of quasi-extinction and disease control probabilities for three Canid species were found to be robust to litter size distribution choice.

Demographic analyses of the red fox revealed a medium to fast life history speed and significant survival and fecundity contributions from juveniles to population growth. Intraspecific variation was detected within these spectra of demographic metrics: the first such demonstration for carnivores. Simulated data substitution between fox populations revealed that geographic proximity and similar levels of anthropogenic disturbance did not infer demographic similarity. Considering the sampling effort expended on the red fox, the species appears well-studied; yet, substantial limitations in data collection were identified.

Compartment modelling of a sarcoptic mange outbreak in an urban fox population in Bristol, UK, revealed that disease transmission was frequency-dependent, consistent with contact rates being determined by social interactions rather than by population density. Individual-based modelling suggested that indirect transmission, genetic resistance and long-distance recolonisation were required to replicate the observed rapid spread of mange and subsequent population recovery. Thus, this first attempt to model mange dynamics in this canid provided novel insight into previously uncertain epidemiological and behavioural processes in the transmission of sarcoptic mange in the red fox.

Declaration

The material contained within this thesis has not previously been submitted for a degree at Durham University or any other university. The research reported within this thesis has been conducted by the author unless indicated otherwise.

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Chapter 1 General introduction

The broad themes addressed in this thesis concern demography and disease ecology of the red fox *Vulpes vulpes*. Here, I will begin by introducing the wider importance of these areas of study. I will go on to explain why the fox represents a useful case study, detailing key characteristics of its life history, demography and sociality that relate to the focus of the thesis. I will also introduce the Bristol fox population, which is the focus of the disease ecology chapters of this thesis. I will then summarise relevant aspects of population dynamics, including inter- and intra-specific life history variation and data uncertainty. Next, I introduce the disease that is the subject of this thesis, sarcoptic mange *Sarcoptes scabiei*, with a summary of the current knowledge of the impacts and dynamics of this disease in wild canids. Following this introduction to mange, I review important aspects of disease dynamics, including assumptions of disease transmission and the implications of sociality for disease spread. I will conclude by outlining my thesis aims and structure.

1.1 Background

Vertebrate species are managed for many reasons, including their roles as resources, invasive species, economically-injurious predators and carriers of disease (Hoffman *et al.* 2011). Vertebrates are also managed as integral components of biodiversity, being valued for both their rarity (Mace *et al.* 2007) and commonness (Gaston & Fuller 2008). For any management objective, success depends on the ability to predict the outcome of management actions on a species' population dynamics. However, the models used to make such predictions are often prone to uncertainty arising from multiple sources, including sampling error, intraspecific variation and poorly-understood interspecific interactions. Management decisions are frequently based on incomplete demographic information (Slade *et al.* 1998) and, of necessity, often utilise data substituted between populations, or gained from studies of similar, more common species (Pech *et al.* 1997, Githiru *et al.* 2007). Carnivores in particular, are important as predators and disease hosts (Baker *et al.* 2008) and their management is often the focus of contention due to their charismatic nature and source of human-

wildlife conflict (Gittleman *et al.* 2001). Further, data limitations due to the challenges of studying these typically elusive mammals, makes estimating demographic rates difficult (Gese 2001). Thus, using a widespread carnivore to explore sources of uncertainty for population modelling is of wide relevance to vertebrate management.

It is particularly useful for the management of vertebrate populations to determine how vital rates vary and which vital rates make the greatest contribution to population growth. Demographic rates, including survival and fecundity, are shaped in part by environmental and demographic stochasticity (Benton & Grant 1999). Consequently, understanding how vital rates respond to different selection pressures, such as harvest, disease or climate (Bieber & Ruf 2005, Milner *et al.* 2007, Jones *et al.* 2008b), is of direct relevance for management. Demographic rates are also determined by a species' life history strategy; thus, it is useful to understand how different strategies influence a species' response to perturbation (Heppell *et al.* 2000). Well-studied species provide meaningful insights into the importance of interspecific variation in the contribution of vital rates to population growth and the influence of life history strategy (Gaillard *et al.* 1998, Sæther & Bakke 2000, Coulson *et al.* 2005). Increasingly, there is evidence of significant demographic differences between populations (Nilsen *et al.* 2009, Johnson *et al.* 2010, Servanty *et al.* 2011). In this context, investigating intraspecific variation in population dynamics is potentially informative of the different selection pressures acting on a species' life history.

The red fox *Vulpes vulpes* (Linnaeus 1758) has many attributes that make it a useful model species to explore a range of theoretical and applied ecological questions. This carnivore is highly adaptable to diverse habitats and is the most widespread extant terrestrial carnivore species (Schipper *et al.* 2008). Foxes are often locally abundant and heavily managed, being of ecological and economic importance as predators (Baker *et al.* 2002, Baker *et al.* 2008, Saunders *et al.* 2010) and disease hosts (Chautan *et al.* 2000, Deplazes *et al.* 2004, Soulsbury *et al.* 2007). Agricultural and environmental damage due to foxes in Australia is valued at over AUS$200 million per annum, with the cost of bait control in New South Wales estimated at AUS$7.3 million per year (Saunders & McLeod 2007); in Europe, between 1978 and 1994, the cost of rabies

vaccine baits was approximately $US83 million (Stohr & Meslin 1996). However, much of this management effort is often inadequate (Gentle *et al.* 2007, Saunders *et al.* 2010). Variation in management outcomes may be in part due to gaps in our knowledge of fox population dynamics (Saunders & McLeod 2007), despite this carnivore being subject to much sampling effort (e.g. Storm *et al.* 1976, Baker *et al.* 2001b). In particular, the response of different fox populations to intrinsic and extrinsic pressures such as density-dependent processes (Berry & Kirkwood 2010), anthropogenic mortality (Baker *et al.* 2002, Aebischer *et al.* 2003) and disease (Chautan *et al.* 2000) is still poorly understood. It is therefore useful to determine not only the status of our current understanding of fox demography, but also to consider whether fox populations exhibit intraspecific demographic variation.

Disease plays an important role in vertebrate management, often being difficult to control, generating controversy and incurring considerable economic costs (Carter *et al.* 2009). The transmission of disease to endangered populations and emerging zoonoses in particular poses a significant problem for wildlife managers (Daszak *et al.* 2000, Breed *et al.* 2009). Host-pathogen relationships are one type of interspecific interaction that can have a significant role in shaping a hosts' life history (Jones *et al.* 2008b) and population dynamics (Tompkins *et al.* 2002). Thus, a comprehensive understanding of demographic processes in the absence of infection will improve the prediction of disease spread. Describing disease transmission in social species, such as the fox (Cavallini 1996), is especially challenging, because variation in inter- and intra-group encounters affects the rate that disease spreads, potentially resulting in non-linear disease dynamics (Altizer *et al.* 2003b). Further, elucidation of disease dynamics in wild populations are hampered by data limitations; difficulties in observing disease outbreaks often result in a high uncertainty associated with prevalence data (Jenelle *et al.* 2007, McClintock *et al.* 2010).

Sarcoptic mange *Sarcoptes scabiei* is a potentially devastating disease that affects a wide range of rare and abundant mammalian species (Pence & Ueckermann 2002), as well as being of considerable economic importance for domestic species (Walton *et al.* 2004). Mange has the highest incidence of arthropod diseases in carnivores that are

threatened by disease, as listed by the IUCN's Red List (Pedersen *et al.* 2007). Although mange is an important disease, fundamental epidemiological and ecological aspects, such as disease transmission, host-parasite interactions and immunobiology, remain poorly understood (Arlian 1989, Bornstein *et al.* 2001, Walton *et al.* 2004), limiting the management of this disease. Foxes are particularly susceptible to mange: populations throughout the world have been severely depleted due to mange outbreaks and the disease often remains at low levels for years (Storm *et al.* 1976, Lindström *et al.* 1991, Soulsbury *et al.* 2007). Yet, there is no clear understanding of the mechanisms driving the long-term dynamics of mange in foxes and other canids. In this context, a well-studied fox population that experienced a mange outbreak provides an opportunity to explore theoretical hypotheses relating to the transmission and persistence of mange. Insight into the dynamics of mange in foxes is of direct application to management and increases our understanding of important ecological and evolutionary processes of disease transmission in a social carnivore.

1.2 The red fox, *Vulpes vulpes*

The red fox is a small canid occurring naturally throughout Eurasia, North America and North Africa and introduced to Australia (Long 2003). Foxes are found in a wide range of habitats, including cities, farmland, forests, coastal dunes, tundra, prairie and deserts (Storm *et al.* 1976, Harris 1977, Englund 1980, Mulder 1985, Heydon & Reynolds 2000, Dell'Arte & Leonardi 2005), revealing the ability of this species to adapt to its surroundings. Across the fox's global distribution, population density varies widely (0.08 to 37 individuals km^{-2}, Appendix 1); several reasons have been proposed to explain population differences in density, including prey availability, habitat type (Webbon *et al.* 2004), level of culling (Heydon *et al.* 2000) and extent of seasonality (Bartoń & Zalewski 2007). The following sections summarise relevant aspects of red fox demography and social organisation, and introduce the Bristol red fox population.

For the purpose of this thesis, fox is used to refer to the red fox. Fox age classes are defined as cubs (< 6 months), subadults (6-12 months) and adults (> 12 months) and juvenile is used to refer to all individuals under one year (Harris & Trewhella 1988).

1.2.1 Demography

Life history rates are fundamental biological parameters that are the foundation for all demographic models and determine a populations' dynamics. Survival rates of foxes vary between age classes and populations. The mean life expectancy in both hunted and non-hunted populations is between one and three years (Yoneda & Maekawa 1982, Harris & Smith 1987). Hunting is often the highest cause of mortality in many controlled fox populations (Phillips *et al.* 1972, Tullar & Berchielli 1981, Reynolds & Tapper 1995), while in uncontrolled populations, road accidents typically result in the majority of deaths (Harris & Smith 1987, Gosselink *et al.* 2007). Juveniles are especially vulnerable to mortality. In the first four weeks up to 20% of cubs die (Harris 1977). Dispersal occurs predominantly among juveniles in their first autumn, with a higher proportion of males than females dispersing (Harris & Trewhella 1988). Inexperience and dispersal exposes juveniles to the risks of road accidents, antagonistic contacts, and increases their susceptibility to hunting and disease (Storm *et al.* 1976, Harris 1977, Yoneda & Maekawa 1982, Lindström 1989). Relative mortality of dispersers varies among populations. In a rural US population, mortality of dispersers was higher than non-dispersing individuals (Gosselink *et al.* 2007), while Soulsbury *et al.* (2008a) found that mortality did not differ significantly between dispersers and non-dispersers in an urban UK population. Adult mortality is typically lower than that of juveniles (Storm *et al.* 1976, Harris & Smith 1987). When social stage is accounted for, mortality of subordinate adults is higher than that of dominants; few subordinates survive to attain territories because dominants live, on average, twice as long (Baker *et al.* 1998).

Productivity differs according to age class and among fox populations. Females are physically able to reproduce in their first winter (Harris & Smith 1987). Typically only one litter is produced per year (Englund 1970), with mean litter size ranging from 3.1 to 8.0 (Appendix 2). Mean litter size was found not to be correlated with latitude (Lord 1960) and often varies little between populations (Lloyd *et al.* 1976). Younger vixens are more likely than older individuals to produce smaller litters or fail to breed (Englund 1970, Harris 1979). The incidence of non-breeding females varies widely among age classes and populations (Zabel & Taggart 1989, Marlow *et al.* 2000),

ranging from 0% to 90% (Appendix 2). The causes of non-breeding females are multiple; as well as physiological reasons (Harris 1979), breeding females are determined partly by resource availability and social factors related to density-dependence (Macdonald 1979, Zabel & Taggart 1989, Iossa *et al.* 2009). In this context, using the proportion of non-breeding females as a measure of the influence of density-dependence on productivity is a better predictor than other measures such as litter size and neonatal loss (Harris 1977). That both survival and productivity rates exhibit inter-population differences alludes to the potential existence of population-specific demographic tactics in this species.

1.2.2 Sociality

The influence of sociality on population dynamics is important for predicting the success of management actions. Social processes such as territoriality can limit population size and non-territorial animals can buffer populations against the loss of reproductive individuals (Cooper *et al.* 2009, Eccard *et al.* 2011, Penteriani *et al.* 2011). A large proportion of carnivores exhibit some degree of sociality, with canids being the most social (Gittleman 1989). The causal mechanisms for the evolution of groups in carnivores have been extensively reviewed (see Macdonald 1983, Bekoff *et al.* 1984, Gittleman 1989) and include predator defence, food exploitation, alloparental care and cooperative foraging. The costs and benefits of dispersal and philopatry, relating to the attainment of dominance and breeding opportunities, are particularly important in explaining group living in foxes (Baker *et al.* 1998, Soulsbury *et al.* 2008a), since foxes do not cooperatively forage or display group defence against predators. Foxes are one of a number of carnivore species including Eurasian badgers *Meles meles* (Woodroffe & Macdonald 1995), Ethiopian wolves *Canis simensis* (Sillero-Zubiri *et al.* 1996), and striped hyaenas *Hyaena hyaena* (Wagner *et al.* 2008), that forage alone but share all or part of a common territory. Macdonald (1983) termed this “spatial grouping”. Individuals forming spatial groups have home ranges that fall within the same territory boundary (Macdonald *et al.* 1981, Poulle *et al.* 1994, White *et al.* 1996), thus potentially benefiting from alloparental care and shared boundary defence (Macdonald 1983). The size of fox groups varies widely between populations, from

monogamous pairs to medium-sized groups (Newsome 1995, Cavallini 1996). Contact between individuals, such as inter-group interactions, establishes the degree of sociality in a species (Bekoff *et al.* 1984). Unlike many canids where direct interactions are frequent, both inter- and intra-group direct contacts are atypical for foxes (White & Harris 1994, Baker & Harris 2000), although this low level of social interaction is thought to be sufficient to maintain social cohesion (Poulle *et al.* 1994, White & Harris 1994).

1.2.3 The Bristol red fox population

The increase in the UK urban fox population during the 20th century has been attributed to a combination of factors, including an increase in scavenged food and post-war changes in urban environments (Harris & Rayner 1986a, 1986b, 1986c). Fox densities in Bristol, UK, are among the highest in the world (Harris 1981). Prior to a sarcoptic mange outbreak in 1994, adult density was exceptionally high at 37 km^{-2} (Baker *et al.* 2000). The outbreak reached a peak in the autumn/winter of 1995 and as a result the population in the city declined by over 95% (Soulsbury *et al.* 2007). In Bristol, fox social groups typically consist of a dominant pair, several philopatric subordinates and related offspring (Baker *et al.* 1998); pre-mange, group size had reached a peak of 6.57 individuals per group, which declined to 1.67 in the winter of 1995, before the eventual collapse of group formation and loss of all groups from the study area in 1996 (Baker *et al.* 2000). Since the outbreak, population recovery has been slow and mange has remained at low levels in the Bristol foxes (Soulsbury *et al.* 2007, S. Harris *pers. comm*.). Monitoring of the population has been continuous since 1977 (Baker *et al.* 2001b, Whiteside *et al.* 2011), therefore providing a valuable long-term dataset of demographic and social parameters (Harris & Smith 1987, Trewhella *et al.* 1988, White & Harris 1994, Baker *et al.* 1998, Baker & Harris 2000, Soulsbury *et al.* 2008a, Iossa *et al.* 2009, Soulsbury *et al.* 2011, Whiteside *et al.* 2011). That this data set also contains prevalence data during and after a mange outbreak is of enormous importance for the development and validation of disease models.

1.3 Population dynamics

Population growth is an important focus of wildlife biologists because of its fundamental importance for both conservation and management (Mills 2007). Meaningful information on the population growth rate and vital rate contributions can be determined from projection models of populations (Caswell 2001), such as the Leslie matrix (e.g. Ezard *et al.* 2010, Salguero-Gómez & de Kroon 2010), which are constructed relatively simply using life-history data. Further, matrix models can be structured to incorporate stage (or age) classes, one of the leading sources of variation in a populations' demographics (Benton *et al.* 2006). With the application of perturbation analyses to projection models, the relative and absolute stage contributions to population growth can be identified (Benton & Grant 1999). Thus, matrix models form the basis of many population viability analyses (Morris & Doak 2002) and also provide useful information for addressing questions of ecological and evolutionary interest, including linking fitness to life-history (Pelletier *et al.* 2007), identifying life history trade-offs (Gaillard & Yoccoz 2003), and determining the effects of climate (Coulson *et al.* 2001) and harvesting regimes (Ginsberg & Milner-Gulland 1994). In this context, is useful to gain a comprehensive understanding of variation in a species' dynamics across its range. The following sections consider variation in life history strategy and the contribution of vital rates to population growth and discuss how demographic modelling is affected by data uncertainty, in light of the current knowledge of fox population dynamics.

1.3.1 Life history variation

The information generated by projection models is useful for categorising species or populations according to life history strategy. One example is the fast-slow continuum (Heppell *et al.* 2000, Oli & Dobson 2003, Gaillard *et al.* 2005), a measure of how species resolve the evolutionary trade-off between reproduction and survival (Bielby *et al.* 2007). Life history theory predicts that contributions from the fecundity of younger age classes to population growth should be larger for mammals that mature early and are short-lived, so-called 'fast' mammals, whereas adult survival is more important for those long-lived 'slow' mammals that mature late (Heppell *et al.* 2000).

In relation to other carnivores, foxes are expected to fall towards the former category because of their early age of first reproduction, short life expectancy, and fairly large litter sizes. Elasticity analyses, which determine the proportional contribution of demographic parameters to population growth, have shown that juvenile foxes make the largest contribution to population growth (McLeod & Saunders 2001), although this study focused on a limited number of populations and failed to incorporate stochasticity in vital rates. Thus, it remains unknown whether these patterns are robust to the inclusion of variation. Indeed, predictions of life history contributions from deterministic analyses can vary unexpectedly when accounting for uncertainty in demographic rates, being of direct consequence to management (Wisdom *et al.* 2000, Johnson *et al.* 2010). Further, it is unclear if the apparent consistency of age-specific contributions to population growth translates into similar consistency in life history speed, because there are few estimates of life history speed for foxes (but see Oli & Dobson 2003). Given that defining a species' position on the fast-slow continuum provides a measure of a species' response to perturbations and adaptability to the local environment, classifying fox populations according to life history speed is of relevance for refining future fox management.

1.3.2 Intraspecific variation

Insight into intraspecific demographic variation increases our understanding of the evolution of life-history strategies. Recently, modelling has revealed inter-population demographic variation in large herbivores, as a response to differing selection pressures such as hunting and climate (Nilsen *et al.* 2009, Johnson *et al.* 2010, Servanty *et al.* 2011). This is in contrast to theory that predicts limited variation in demographic tactics, since the majority of demographic variation is accounted for by phylogeny and body mass (Gaillard *et al.* 2005). For example, substantial differences in vital rate contributions were found between populations of Sierra Nevada bighorn sheep *Ovis canadensis sierra* (Johnson *et al.* 2010) and roe deer *Capreolus capreolus* life-history speed slowed down in populations experiencing increasing environmental severity (Nilsen *et al.* 2009). Studying intraspecific variation removes the effects of phylogeny on life history variation (Frederiksen *et al.* 2005), but inter-population differences

have, to date, been overlooked for carnivores. Foxes exhibit plasticity in adapting to a wide variety of habitats (Storm *et al.* 1976, Harris 1977, Englund 1980, Mulder 1985, Dell'Arte & Leonardi 2005) and are subject to a wide range of climatic and management conditions. Life history rates are sensitive to environmental (Soulsbury *et al.* 2008b) and anthropogenic pressure (Lloyd *et al.* 1976, Chautan *et al.* 2000). Given the notable sampling effort expended on the fox and its wide distribution, this species presents an ideal opportunity to explore inter-population variation in the demography of a carnivore.

Inter-population variation in life history is of consequence for management (Johnson *et al.* 2010). One such example is that of management decisions based on surrogate data, a practice in demographic modelling that is necessitated by the often limited availability of demographic data (Schtickzelle *et al.* 2005, Githiru *et al.* 2007). The extent to which surrogate data might affect model outcomes, such as estimates of the population growth rate, has received little attention (but see Caro *et al.* 2005). Demographic data have been substituted previously from one fox population to simulate another, in order to address management concerns (e.g. Pech *et al.* 1997); thus, in light of possible intraspecific differences, it is useful to determine whether there is sufficient similarity to justify substitution of data between fox populations.

1.3.3 Uncertainty in population modelling

An issue of widespread concern in population modelling is how to account for uncertainty in demographic data, which can lead to uncertainty in model predictions (Beissinger & Westphal 1998, Doak *et al.* 2005, Bakker *et al.* 2009). Life history data are widely collected by field biologists, yet there is significant disparity in how the data are recorded, calculated and presented, as well as being limited by logistical constraints. Uncertainty in vital rates arises not only from sampling error, but also from variation due to environmental and demographic stochasticity, known as process error (Bolker 2008). Often, analyses of demographic processes focus on mean values rather than the intraspecific variation in a trait (Bolnick *et al.* 2011). Ignoring uncertainty in vital rate estimates could lead to misguided inference of the relative importance of life history

rates (Caswell 2001), which could be especially problematic for small populations, for which the effects of variability in vital rates are more pronounced (Melbourne & Hastings 2008). To incorporate uncertainty when the variance of a parameter is known, vital rates are drawn from probability distributions (Hilborn & Mangel 1997, Morris & Doak 2002, Mills 2007). For many demographic rates, however, the effects of distribution shape on model outcomes are either contradictory (e.g. survival, Fieberg & Ellner 2001, Kaye & Pyke 2003) or remain to be determined (e.g. litter size, Kendall & Wittmann 2010). Methods exist to separate process error from sampling error, such as discounting the total variance by the estimated sampling error (Kendall 1998, White 2000). Incorporating uncertainty is particularly challenging when previously published parameters do not explicitly report measures of variance or studies have not been of sufficient duration to account for environmental variation. To date, the focus of incorporating variation into demographic models has mostly been on process error (e.g. Akçakaya 2002, Kendall & Fox 2002), with fewer studies explicitly accounting for sampling error (Holmes 2001, Bakker *et al.* 2009).

Data uncertainty is a concern in species where demographic data have been collected from mortality data, for example as in foxes, due to the assumptions and biases associated with using such data (Caughley 1977). As a result of using mortality data such as standing age distributions, vital rates are often presented as point estimates. Hence, the uncertainty in these rates is not reflected in subsequent estimates of demographic descriptors. For instance, the intrinsic rate of increase has been determined for only a few fox populations globally, with typically stable growth (Hone 1999, McLeod & Saunders 2001, Korytin 2002), but measures of confidence are lacking for these estimates. Quantifying the influence of parameter uncertainty for model predictions and determining measure of confidence in demographic descriptors is of direct application to management.

1.4 Sarcoptic mange, *Sarcoptes scabiei*

Sarcoptic mange is a disease of widespread importance, affecting over 100 domestic and wild mammal species (Pence & Ueckermann 2002), including both threatened and abundant wild mammalian populations (see Table 1.1 for examples). Importantly, the transmission of this disease has the potential to 'spill-over' between domestic and wild mammals (Leon-Vizcaino *et al.* 1999, Daszak *et al.* 2000, Gortazar *et al.* 2007). The origin of sarcoptic mange in wild animals almost certainly stems from domesticated species, which are presumed to have caught the disease from humans (Fain 1978). In domesticated pigs, the annual economic loss due to sarcoptic mange was estimated at over AUS$500,000 in South Australia (Dobson & Cargill 1979) and US$84-115 per individual sow in North Carolina (Arends *et al.* 1990). While sarcoptic mange has not been implicated in the extinction of any wild species, it probably caused the extirpation of the fox population on Bornholm island, Denmark (Bornstein *et al.* 2001). Recent occurrences of sarcoptic mange in global fox populations are summarised in Table 1.2. Recent European outbreaks can be traced to the spread of mange through fox populations in continental Europe during the 1960s (Simpson 2002). The disease is now widespread in Britain, although the prevalence varies regionally (Soulsbury *et al.* 2007). The following sections provide an overview of sarcoptic mange in wild canid populations, highlighting areas of uncertainty and of importance for modelling this diseases' dynamics.

In this thesis, mange refers to sarcoptic mange. An epizootic is defined as a phase of rapid disease spread when many individuals are infected simultaneously, while enzootic refers to a disease phase where infection is constantly present in a population, but only a small number of individuals are affected at any one time (Collinge & Ray 2006).

Table 1.1. Examples of sarcoptic mange in wild mammalian populations.

Species	*Country*	*Epizootic/ enzootic*	*Mortality (%) or number of records*	*Possible vector*	*Threatened*	*Reference*
Marsupialia						
Common wombat *Vombatus ursinus*	Australia	Enzootic	35%		No	1
Koala *Phascolarctos cinereus*	Australia	Enzootic	2%	Wombat	No	2
Primate						
Mountain gorilla *Gorilla beringei*	Uganda	-	5 individuals	Humans	Yes	3
Carnivora						
Coyote *Canis latrans*	USA	Epizootic	20 - 100%		No	4
Iberian wolf *Canis lupus signatus*	Spain	-	20%	Red fox	Yes	5
Pampas fox *Pseudalopex gymnocercus*	Bolivia	Enzootic	13 - 25%		-	6
Raccoon dog *Nyctereutes procyonoides*	Japan	-	3 Individuals		No	7
Fisher *Martes pennanti*	USA	-	1 individual	Porcupine	No	8
Cheetah *Acinonyx jubatus*	Kenya	Enzootic	12·8%	Thompsons gazelle	Yes	9
Eurasian lynx *Lynx lynx*	Switzerland	-	3 Individuals	Red fox	Yes	10
Raccoon *Procyon lotor*	USA	Enzootic	3 individuals		No	11
Black bear *Ursus americanus*	USA	-	3 individuals		No	12
Artiodactyla						
Alpine chamois *Rupicapra rupicapra*	Italy	Epizootic	41 - 70%		No	13
Cantabrian chamois *Rupicapra pyrenaica parva*	Spain	Epizootic	1 – 21%		No	14
Spanish ibex *Capra pyrenaica hispanica*	Spain	Epizootic	81%	Domestic goats	Yes	15
Barbary sheep *Ammotragus lervia*	Spain	Epizootic	86%	Domestic goat	Yes	16
Bighorn sheep *Ovis canadensis*	USA	Epizootic	>80%	Domestic sheep	No	17
Red deer *Cervus elaphus*	Spain	Enzootic	80 individuals	Chamois	No	18
Thompsons gazelle *Eudorcas thomsonii*	Kenya	Enzootic	0·81%		No	9
Erinaceomorpha						
African pygmy hedgehog *Atelerix albiventris*	Nigeria	-	3.6 %		No	19
Rodentia						
Fox squirrel *Sciurus niger rufiventer*	USA	Epizootic	30 – 50%		No	20
Marsh rabbit *Sylvilagus palustris*	USA	-	7 individuals		No	21
Common porcupine *Erethizon dorsatum*	USA	-	2 individuals		No	22

[1](Hartley & English 2005);[2](Obendorf 1983); [3](Graczyk *et al.* 2001); [4](Pence *et al.* 1983);[5] (Oleaga *et al.* 2011); [6](Deem *et al.* 2002); [7](Ninomiya & Ogata 2005); [8](O'Meara *et al.* 1960); [9](Gakuya *et al.* 2012); [10](Ryser-Degiorgis *et al.* 2002);[11]Fitzgerald et al., 2004); [12](Schmitt *et al.* 1987); [13](Rossi *et al.* 2007); [14](Fernandez-Moran *et al.* 1997); [15](Leon-Vizcaino *et al.* 1999);[16](Gonzalez-Candela *et al.* 2004); [17](Jessup *et al.* 1991 cited in Woodroffe, 1999); [18](Oleaga *et al.* 2008);[19](Okaeme & Osakwe 1985); [20](Allen 1942); [21](Stringer *et al.* 1969); [22](Payne & O'Meara 1958).

Table 1.2. Mortality and prevalence of sarcoptic mange in red fox *Vulpes vulpes* populations.

Country	***Epizootic/ Enzootic***	***Mortality***	***Prevalence***	***Reference***
Bristol, UK	Epizootic	>95 %		1
Surrey, UK	Enzootic		14 Individuals	2
Sweden	Epizootic	21-100 %		3
Norway	Epizootic		6.6 % to 30 %	4
Denmark	Epizootic	>70%		5
Italy	Enzootic		25.3 %	6
Spain	Enzootic		3.16 %	7
Slovakia	Enzootic	24.4 %		8
Hungary	Enzootic	21 %		9
USA	Epizootic		>50 %	10
USA	Epizootic		11 – 59 %	11
USA	Epizootic	45 % (urban)		12
Australia	Enzootic		14 %	13
Japan	Enzootic		7 Individuals	14

[1](Soulsbury *et al.* 2007); [2](Bates 2003); [3](Danell & Hornfeldt 1987); [4](Davidson *et al.* 2008); [5](Forchhammer & Asferg 2000); [6](Balestrieri *et al.* 2006); [7](Gortazar *et al.* 1998); [8](Kočišová *et al.* 2006); [9](Sreter *et al.* 2003); [10](Trainer & Hale 1969); [11](Tullar & Berchielli 1981); [12](Gosselink *et al.* 2007); [13](Marlow *et al.* 2000); [14](Tsukada *et al.* 1999)

1.4.1 Life history

Sarcoptic mange is caused by *Sarcoptes scabiei* (Linnaeus 1758), a burrowing mite (Acari: Astigmata, Sarcoptidae) that consumes tissue fluid and living cells (Arlian 1989). The mites' morphology and life history are described in detail in several studies (see Fain 1978, Arlian 1989, Bornstein *et al.* 2001, Pence & Ueckermann 2002, Walton *et al.* 2004). The life cycle of a fertilised female lasts between four and six weeks, with 3-4 eggs being laid daily that hatch three days later; development of all five nymphal stages is complete in roughly two weeks (Bornstein *et al.* 2001). Transmission occurs through larval stages and possibly by mature females (Walton *et al.* 2004). Under optimal ambient conditions of high humidity and low temperature, all life stages can survive up to several weeks off the host (Arlian 1989).

1.4.2 Clinical symptoms and immunology

Clinical signs of mange have been extensively reviewed and are similar for most mammal species (see Arlian 1989). Once in the skin, mites release a secretion into the tissue that causes hypersensitivity and an itching reaction in the host (Pence & Ueckermann 2002). The latent period (the time for clinical signs to become apparent) in canids is 10 to 30 days, dependent on the mite load and individual hypersensitivity (Bornstein *et al.* 2001). High host densities of mites are common and up to 5000 individuals per square centimetre are reported on foxes (Little *et al.* 1998). In foxes, hyperkeratosis (the characteristic crusty skin of mange) is noticeable one to two months after initial infection; the average time from first capture and diagnosis to death is 3.7 months (Newman *et al.* 2002). Although mange is not always fatal, death is frequently caused by starvation, dehydration, hypothermia and secondary bacterial infections (Bornstein *et al.* 2001). The progression of the disease is typically classified by visible development: in foxes, class I refers to infected individuals displaying no sign of hyperkeratotic mange and class II denotes the presence of hyperkeratotic mange (Newman *et al.* 2002). Behavioural changes in infected canids are less well documented than pathological symptoms, although infected foxes have been observed utilising smaller than normal ranges (Overskaug 1994).

The immune response to mange is complex, with evidence of both cell-mediated (activation of specialised cells such as T-lymphcytes) and humoral (secretion of antibodies) responses (Arlian 1989). Empirical evidence of either acquired or genetic resistance to mange remains uncertain. In captive canids, there is experimental evidence both for and against acquired immunity (Arlian *et al.* 1994, Little *et al.* 1998). The inconsistency in these results could be related to the quantity of mites given during re-infection, or may indicate that immunity is acquired for low-grade levels of infection (Little *et al.* 1998). In some populations, foxes and coyotes *Canis latrans* have been observed to recover from mange, although it is unclear whether these individuals can subsequently became re-infected (Storm *et al.* 1976, Pence & Windberg 1994, Chronert *et al.* 2007). Long-term adaptation to mange in wild canid populations is supported in a serological study of a Danish fox population (Davidson *et al.* 2008).

1.4.3 Transmission

Transmission of mange mites is thought to occur through both direct and indirect contact (Pence & Ueckermann 2002). The disease is transmitted directly by contact between individuals such as allogrooming, suckling and aggressive interactions. Dispersing individuals are implicated in transporting mange mites over long-distances, although there is little empirical evidence for this process (Lindström 1992, Pence & Windberg 1994). Indirectly, the mite can be transmitted through fomites (Arlian 1989), inanimate objects such as bedding material capable of transferring an infectious agent. In Russian fox populations, mange was thought to be transmitted via the sharing of dens (Gerasimov 1958). However, empirical data regarding the substances involved and subsequent contact rates with these fomites are lacking for other canid populations.

1.5 Disease dynamics

Host-parasite interactions are one of a number of interspecific interactions that can affect a populations' dynamics (Tompkins *et al.* 2002). In contrast to many interspecific interactions, such as predator-prey interactions, parasites obtain their nutrients from one or few hosts, as opposed to predators that consume many prey throughout their life. Parasites frequently cause morbidity, rather than mortality, the effects of which are often noted at an individual level more than a population-level. Compared to other interspecific interactions, including predation and competition, the extent that parasites shape host population dynamics has until relatively recently been neglected by ecologists (Dobson & Hudson 1986). Disease is a potentially important influence on life history, exerting selection pressure on survival and reproduction and altering life history speed (Jones *et al.* 2008b). Thus, understanding the dynamics of a disease is important for species management. Disease dynamics are traditionally described by deterministic, continuous time models that classify individuals according to their infection status and follow the rate of change for each disease compartment (Anderson & May 1992). These models, such as S(E)IR (Susceptible-(Exposed)-Infectious-Recovered/Removed) models, can provide meaningful estimates of epidemiological parameters, such as transmission coefficients, that characterise

particular disease systems (Keeling & Rohani 2008). The transmission coefficient, β, is a key determinant of R_0, the basic reproductive number, defined as the average number of secondary infections produced by an infected individual in a totally susceptible population (Hethcote 2000). R_0 is a key parameter for determining the probability of establishment, prevalence and threshold of an epidemic and is species- and often population-specific (Keeling & Rohani 2008). A disease is likely to invade a population when $R_0 > 1$ (Anderson & May 1992). Understanding the effects of disease on a hosts' population dynamics is of applied importance, for example for determining the probability of disease-induced extinction (McCallum *et al.* 2009) or the effects of predator control on disease spread (Packer *et al.* 2003) and for identifying population stages with disproportionate disease risk (Klepac *et al.* 2009). The following sections provide an overview of the processes involved in describing disease transmission, including the effects of sociality, and discuss current knowledge of the dynamics of mange in wild canid populations.

1.5.1 Disease transmission

The estimation of epidemiological parameters in wild populations is notoriously challenging (McCallum *et al.* 2001). Difficulties in disease detection, such as reduced capture rates due to disease-induced behavioural changes, misclassification or the often opportunistic sampling associated with data collection during disease outbreaks, can result in large uncertainty in parameter estimates (Conner *et al.* 2000, Jenelle *et al.* 2007, McClintock *et al.* 2010). For example, the transmission coefficient, β, requires knowledge of the frequency of contacts between susceptible and infected individuals, and the number of contacts that result in infection (Begon *et al.* 2002). These data are rarely available for wild populations and thus β is often estimated by fitting parameters to data on disease prevalence or incidence (Barlow 1995). Few mange outbreaks in wild populations have been studied sufficiently to enable elucidation of long-term temporal dynamics; to date, the only comprehensive simulation of mange dynamics has been conducted for coyotes (Leung & Grenfell 2003). The basic reproductive ratio, R_0 and the transmission coefficient, β, do not appear to be determined in the published literature for canid species.

Correctly determining transmission mechanisms is important for predicting disease persistence and defining host-density disease thresholds (McCallum *et al.* 2001). Typically, directly transmitted diseases are modelled by one of two mechanisms. The first, density-dependent transmission, assumes that contact rates with infected individuals are linearly proportional to the density of the population (Begon *et al.* 2002). However, contact rates do not always increase simultaneously with density; in this instance, the second mechanism, frequency-dependent transmission, may be more appropriate. Frequency-dependent transmission is characterised by constant contact rates, with transmission increasing with the proportion of infected individuals in the population (Begon *et al.* 2002). Frequency-dependent transmission is generally assumed to apply to sexually transmitted or vector-borne diseases, due to contact rates being independent of population size (McCallum *et al.* 2001). Despite these definitions, there is much uncertainty when describing transmission modes (McCallum *et al.* 2001, Begon *et al.* 2002, Lloyd-Smith *et al.* 2005a) and recent studies question the assumptions associated with density-and frequency-dependent transmission (Caley & Ramsey 2001, Begon *et al.* 2003, Smith *et al.* 2009c, Beeton & McCallum 2011). The relationship between mange prevalence and density in fox populations is varied. Mange spread rapidly in the high-density fox population in Bristol (Baker *et al.* 2000). Gortazar *et al.* (1998) found higher mange prevalence in low than in high density fox populations, and Lindström and Morner (1985) reported a lower rate of disease spread in high fox density habitats than in low density habitats. Given the lack of a consistent relationship of mange with population density, it is useful to explore the mechanism of transmission in this disease.

1.5.2 Sociality and disease transmission

Sociality shapes an individual's risk of infection by altering either its underlying immunity, or contact with infected individuals, due to the influence of social processes, such as group size and structure (see Mooring & Hart 1992, Cote & Poulin 1995, Altizer *et al.* 2003b, Nunn *et al.* 2008, Rifkin *et al.* 2012). In group-living species, contact rates vary according to the inter- and intra-group interactions that reflect a population's level of social organisation (Bekoff *et al.* 1984), resulting in the potential for certain

group members to make a disproportionate contribution to disease transmission. Due to the non-linear relationship of social contact rates with density, classic assumptions of the transmission mechanisms used in disease modelling might not hold true (McCallum *et al.* 2001). Sterner and Smith (2006) proposed that due to variability in encounters arising from the territorial nature of foxes and changes in density, a combination of density- and frequency-dependent transmission functions might best explain disease transmission. That social parameters are well-recorded for the Bristol fox population (Baker *et al.* 2001b), provides an opportunity to explore the influence of contact rates on mange transmission.

Low inter-group contact rates, such as those observed in foxes (White & Harris 1994) and badgers (Böhm *et al.* 2008), lend support to the premise that group-structuring reduces disease spread (Loehle 1995, Carter *et al.* 2007). The role of stable territorial structures in inhibiting the spread of bovine Tb in badgers has been emphasised by studies describing increased disease spread due to culling-induced behavioural changes (sometimes referred to as 'social perturbation'; Rogers *et al.* 1998, Carter *et al.* 2007, McDonald *et al.* 2008). Adjustments in territory size may be expected with behavioural changes during disease epizootics and disease may be sustained by the movement of dispersing animals into empty territories. However, in two fox populations in Bristol and Sweden, existing groups expanded their territories into empty territories during mange outbreaks, as opposed to new groups forming (Lindström 1992, Baker *et al.* 2000). Territory expansion could thus reduce the spread of disease by limiting the opportunity for infected newcomers to colonise (Baker *et al.* 2000), but this hypothesis requires validation.

Recent studies highlight the importance of incorporating social processes into disease models (Haydon *et al.* 2002, Shirley *et al.* 2003, Hosseini *et al.* 2004, Harris *et al.* 2008, Wasserberg *et al.* 2009, Craft *et al.* 2011). While simple deterministic models are useful for exploratory purposes (Smith *et al.* 2009a), social interactions and spatial processes such as dispersal are hard to capture in compartment models. By contrast, social behaviour and spatial processes can easily be incorporated in individual-based models (Haydon *et al.* 2002, Rushton *et al.* 2006, Nunn *et al.* 2008, Kramer-Schadt *et*

al. 2009), due to the non-analytical framework of these modelling approaches (Grimm & Railsback 2005). A stochastic individual-based model incorporating social interactions was better able to predict the observed patterns of mange in coyotes than traditional deterministic epidemiological models (Leung & Grenfell 2003). It is therefore of interest to determine whether a similar approach is suitable for modelling mange in foxes.

1.5.3 Disease cycles and resistance

Mange outbreaks often exhibit cycles. Epizootics can occur at intervals of 40 to 50 years, followed by mange persistence for up to 20 years at enzootic levels (Lindström *et al.* 1994, Pence & Windberg 1994). Rapid evolution of immunity to disease (Bonneaud *et al.* 2011, Robinson *et al.* 2012) promotes host-parasite coexistence, allowing populations to recover and disease to persist under enzootic conditions. Thus, the possible immunity to low-grade mange infections described previously could reflect an evolutionary adaptation to the parasite. Yet, the effects of mange epizootics vary widely between species and populations, causing significant declines in some populations (Soulsbury *et al.* 2007), while having little effect on others (Pence & Windberg 1994). The contrasting effects of mange outbreaks and the contradictory empirical evidence for immunity (see above), suggest inter-and intra-specific genetic variability in resistance. Rapid evolutionary dynamics also act as a selection pressure on the virulence of a parasite (Altizer *et al.* 2003a), which is consistent with the theory that mutant strains of mange cause epizootics in novel populations (Pence & Ueckermann 2002). Leung and Grenfell (2003) found support for inherited resistance when simulating epidemiological patterns and population recovery of mange in coyotes; this suggests that understanding the evolution of immunity is likely to be necessary for understanding patterns of mange transmission. Long-term enzootic disease persistence in the Bristol fox population (Soulsbury *et al.* 2007) is indicative of a degree of immunity; however, empirical evidence is lacking and simulations are needed to explore the potential role of resistance.

1.6 Thesis aims and structure

In this thesis, the demography and disease dynamics of the red fox will be explored using a suite of modelling techniques. The initial aim of the study, to model sarcoptic mange dynamics in an urban red fox population, prompted the need for a further understanding of the demography of this species. First, fundamental demographic properties of fox dynamics will be examined, and in doing so, two issues pertinent to ecological modelling will be addressed: data uncertainty and intraspecific demographic variation. Finally, an outbreak of mange in the Bristol fox population is modelled, using both epidemiological and individual-based approaches to explore disease transmission in this social carnivore. Collectively, the chapters in this thesis will provide new insight into demography and disease ecology that can be applied to the management of this species.

Following this introduction, chapter 2 presents a method for incorporating the uncertainty of vital rate point estimates into matrix models. The approach is illustrated using published data for three fox populations. The consequences of failing to provide measures of uncertainty in the population growth rate are highlighted and guidance is provided on the sample sizes needed to reduce uncertainty in this rate.

In chapter 3, the suitability of probability distributions used to model intraspecific litter size variation in population models is considered. Here, probability distributions are fitted to empirical litter size frequencies for terrestrial carnivore species. The robustness of population model predictions of quasi-extinction and disease control to distribution choice is determined for three canid species, including the fox.

In chapter 4, a review of the current knowledge of global fox demography is undertaken. Matrix models constructed from previously published data are used to investigate population-level variation in demographic tactics, including life history speed and vital rate contributions to population growth. The consequence of substituting data between populations is then illustrated for population growth rate estimates.

In chapter 5, the ability of deterministic SEI (Susceptible-Exposed-Infected) compartment models to describe age-specific heterogeneities of mange prevalence in the Bristol fox population is established. Assumptions of disease transmission pathways in this social species are tested and the basic reproductive number, R_0, is estimated for the most parsimonious model.

In chapter 6, an individual-based model is developed to describe the Bristol fox population during the high density conditions prior to the outbreak of mange. A pattern-orientated approach is used to evaluate whether the model captures emergent social and demographic properties at the individual and population level. The influence of sociality is examined in relation to management issues, including disease and population control.

In chapter 7, the dynamics of mange in the Bristol fox population is explored further with the stochastic individual-based modelling approach developed in chapter 6. The recovery of the population after an epizootic and the persistence of mange at enzootic levels are explored through the addition of indirect transmission and genetic resistance. The implications of sociality for disease transmission are examined.

Chapter 2 Uncertainty in population growth rates: the red fox *Vulpes vulpes* as an example

2.1 Introduction

Demographic modelling is widely used in conservation and management (Mills 2007, Milner-Gulland & Rowcliffe 2007). As modelling techniques have become increasingly sophisticated, a growing literature has dealt with the importance of acknowledging process error (or environmentally-driven variation in demographic parameters) in model analyses (Tenhumberg *et al.* 2008, de Valpine 2009, Salguero-Gómez & de Kroon 2010). By contrast, assessments of the implications of observation error (arising from sampling limitations) for model precision are often lacking (but see Doak *et al.* 2005, Fiske *et al.* 2008), perhaps due to a widespread acknowledgement of the ubiquity of sampling constraints (Beissinger & Westphal 1998). Here, methods are discussed to infer accuracy of vital rate estimates, even where parameter uncertainty has not been reported explicitly. It is shown that acknowledging limits to precision can be an important element of demographic inference, with implications for data collection protocols.

Age- or stage-structured (Leslie or Lefkovitch) matrix population models are conceptually clear and relatively easily parameterised, with well-characterised properties; as such, the use of matrix models is particularly widespread in ecology (Ezard *et al.* 2010, Salguero-Gómez & de Kroon 2010). Studies utilising matrix population modelling rely on data from a variety of sources. Frequently, the studies' authors have also collected the demographic data used to parameterise the transition matrix. In these cases, sample variance is used to establish vital rate distributions and resampling techniques are available to determine the consequences of that uncertainty for estimates of population growth (e.g. Kalisz & McPeek 1992, Wisdom *et al.* 2000). In spite of this, many authors routinely publish point estimates of asymptotic population growth (λ), without accompanying metrics of precision such as standard errors or confidence intervals (furthermore, this practice is not limited to relatively low-ranking journals; Table 2.1).

When modellers use data that were not collected specifically for the purposes of demographic insight, further problems arise. Hunting records are a common source of such data, even though they are associated with a number of important assumptions that limit their use and compel caution in their interpretation (Caughley 1977). Even accepting these limitations, hunting data are often reported inconsistently and, in particular, are frequently presented without estimates of accompanying uncertainty. In these situations, likelihood approaches provide a convenient method to infer the distribution and extent of uncertainty around the best estimate for the parameter of interest. Hitherto, likelihood methods have largely been neglected for exposing the uncertainty associated with the output of projection matrices.

In this chapter, techniques are presented for inferring, retrospectively, the uncertainty of demographic parameters due to observation error in demographic data. Following others (e.g. McCarthy 2007) Bernoulli processes, such as survival or probability of breeding, are distinguished from Poisson processes, such as litter size. This approach is illustrated with reference to the red fox *Vulpes vulpes*, the most widely distributed extant wild terrestrial mammalian species (Schipper *et al.* 2008), extensively studied throughout its geographic range due to its ecological, economic, and cultural importance (e.g. see Heydon & Reynolds 2000, Saunders *et al.* 2010). The fox is widely hunted, making the species a rich source of demographic data. Comparisons of fox population growth rates in different parts of the world have been used to classify the species along the "fast-slow" life history continuum (Oli & Dobson 2003) and have also been used to make inferences about the species' response to different environmental and management pressures (McLeod & Saunders 2001). Determining the confidence that can be placed in these assessments is, therefore, crucial for a number of applications.

Here, it is illustrated how likelihood profiles can be determined for fox demographic parameters and use resampling techniques to assess confidence in resultant estimates of population growth. These results highlight the need for caution in generalising about differences in the dynamics of populations. The utility of this resampling approach is illustrated to provide information about required sampling effort.

2.2 Methods

2.2.1 Literature review of published demographic rates

A literature review of published demographic studies was conducted to determine the number of studies that failed to include an accompanying measure of uncertainty of the estimated population growth rate. A Web of Science (http://apps.isiknowledge.com) search was conducted from January 2008 to May 2010 using the search terms “population growth” AND “matrix model” AND “demography”. The results were separated by taxa, and further distinguished by those that used previously published data to estimate matrix transition elements. The impact factor of the journal was also recorded for each result. The number of studies using published demographic data was recorded, as were those studies published in a journal with a 5-year impact factor of four or higher (based on Web of Science, Journal Citation Reports).

2.2.2 Likelihood profiles for demographic parameters

Age-specific survival and proportion of breeding females are Bernoulli processes, in the sense that each female can be considered a “trial” with a binomial outcome (live or die, breed or fail to breed). Taking the example of survival, hunting data often yield numbers of individuals in different age classes. If the data are assumed to have been collected at a time when the population approximated its stable age distribution, survival of individuals of age x can be inferred from the relative number of individuals in age classes x and x+1 (f_x and f_{x+1}, respectively). The point estimate of survival, P_x, is given by $P_x = f_{x+1} / f_x$. Occasionally, $f_{x+1} > f_x$, or the population is known to have been growing at some rate (r) during the period of data collection; Caughley (1977, pp. 90-96) presents methods to deal with both of these situations. Very often, sample sizes for older age classes are sufficiently restricted that it is useful to truncate the age distribution and create composite classes for all age classes beyond a given age. In these cases, the point estimate of survival is given by $P_{x^*} = f_{x>x^*} / (f_x + f_{x>x^*})$, where x^* is the final age class.

In the previous formulae, the number of trials is represented by the denominator of the point estimate equation, whilst the number of "events" (or successes) is given by the numerator. However, the point estimate for survival is only an estimate. It is often more interesting to consider the relative probability with which any other true parameter value could have yielded the same outcome, i.e. the same number of events from the same number of trials. Assuming a uniform prior probability for any putative survival rate, the likelihood of any given survival rate, P_x, is given by:

$$L(P_x \mid f_x, f_{x+1}) = \binom{f_x}{f_{x+1}} P_x^{f_{x+1}} (1 - P_x)^{f_x - f_{x+1}}. \quad (1)$$

This likelihood distribution is easily evaluated using the "dbinom(events,trials,P_x)" function in R 2.12.0 (R Development Core Team 2010). Given data, for example, on the proportion of shot females that show signs of breeding, the same approach can be used to determine the likelihood profile for the probability of breeding, B_x. If there is prior information about the focal parameter, then it can easily be incorporated using a Bayesian approach (see McCarthy 2007).

When estimating age-dependent, per-capita, fecundity rates it is assumed that only information on the number of females of age x that bred is available, denoted N_x, and the total number of offspring that they produced, denoted Y_x. Here, it is assumed that the number of offspring a female produces, given that she has produced at least one offspring, is distributed according to a shifted Poisson distribution. The point estimate for average litter size for breeding females in age class x is simply $m_x = Y_x / N_x$. The likelihood that the true mean litter size is m_x, is:

$$L(m_x \mid N_x, Y_x) = \frac{(\mu N_x)^y e^{-\mu N_x}}{y!}, \quad (2)$$

where $\mu = m_x - 1$ and $y = Y_x - N_x$. These adjustments are necessary to shift the Poisson distribution of litter sizes one interval to the right, removing the possibility of zero

litter sizes for females that breed. This likelihood distribution is also easily determined in R using the "dpois(y, μN_x)" function.

2.2.3 Confidence intervals for population growth estimates: the red fox as an example

Published demographic data were extracted for three red fox populations of management interest: a culled Australian population (Coman 1988, McIlroy *et al.* 2001, Saunders *et al.* 2002), a non-culled Australian population (Marlow *et al.* 2000), and combined data from culled USA populations (Storm *et al.* 1976, Tullar & Berchielli 1981, Nelson & Chapman 1982, Tullar & Berchielli 1982, Allen 1984). Female-only, post-breeding "birth-pulse" models were constructed of the form $\boldsymbol{N}_{t+1} = \boldsymbol{A}.\boldsymbol{N}_t$, where $\boldsymbol{N}_t$ is a vector of numbers of females in each age class at time t and $\boldsymbol{A}$ is the transition matrix. The transition matrix was based on four age classes (juveniles, 0+; yearlings, 1+; young adults, 2+; and older adults, ≥ 3 years) and took the form:

$$\boldsymbol{A}_t = \begin{bmatrix} F_1 & F_2 & F_3 & F_{4*} \\ P_1 & 0 & 0 & 0 \\ 0 & P_2 & 0 & 0 \\ 0 & 0 & P_3 & P_{4*} \end{bmatrix}. \qquad (3)$$

To avoid small sample size issues among older age classes, only four age classes were used; it is unusual for individuals to survive past 4 years (Tullar & Berchielli 1981, Soulsbury *et al.* 2008a).

Deterministic growth, λ_i, of population i, was determined from the dominant eigenvalue of $\boldsymbol{A}_i$ using point estimates of each matrix element for survival, calculated as detailed above. Fecundity matrix elements (F_x) were determined from the proportion of breeding females (B_x), the average age-specific litter size (m_x) and a generalised birth sex ratio of 1:1 (Vos & Wenzel 2001), so that $F_x = 0.5P_xB_xm_x$.

Confidence intervals were determined using a resampling (or parametric bootstrap) approach (Wisdom *et al.* 2000). Specifically, λ_i was determined from 10,000 replicate projection matrices, with each element drawn from its corresponding likelihood

distribution; confidence intervals for λ_i were taken as the range encompassing the central 95% of λ_i estimates.

2.2.4 Implications for sample size

To illustrate an additional benefit of the resampling approach for quantifying uncertainty, a "generic" fox population (*sensu* Marboutin *et al.* 2003) was created from the focal studies. Generic demographic parameters were calculated by summing "events" and "trials" across the three studies; thus, parameters were weighted by the size of studies. The stable stage distribution (SSD) was calculated from the right eigenvector of the generic projection matrix, $\boldsymbol{A}_g$. The effect of different sample sizes on the level of confidence that could be placed in estimates of population growth, λ_g was then investigated. Specifically, for a given sample size, *S*, it was assumed that the number of females available for demographic analysis was proportioned among age classes according to the SSD. Those *S* individuals were selected randomly, resampling with replacement, and calculated all matrix elements according to the fates of the selected individuals (whether they lived or died, bred or failed to breed and, if they bred, the number of offspring they produced, drawn from the relevant likelihood distribution). From this resampled matrix, $\lambda_{g,S,j}$ was determined, where *S* was the sample size and $j = 1, 2 \ldots 10^4$ resampled matrices. The process was repeated for a range of sample sizes from 50 to 4,500 females, reflecting the range of sample sizes available for published studies of foxes (minimum 42, Allen 1984, maximum 1701, Harris & Smith 1987). Resultant 95% confidence intervals for estimates of $\lambda_{g,S}$ were plotted against sample size.

2.3 Results

2.3.1 Literature review

A total of 109 studies across a range of taxa provided estimates of the population growth rate. The literature review suggests that failing to provide an accompanying measure of uncertainty in the asymptotic growth rate is a widespread practice (Table 2.1). Further, this practice was not restricted to studies using published demographic rates, or to low-ranking journals.

Table 2.1. Results of a literature review showing the percentage of studies that failed to include an accompanying measure of uncertainty of the estimated population growth rate. Sample sizes in parentheses.

Taxon	***Studies without confidence estimates***	***Studies using published vital rates***	***Studies using published rates with no confidence estimates***	***Studies with 5-year impact factor ≥ 4***	***Studies without confidence estimates with 5-year impact factor ≥ 4***
Birds	58 (19)	37 (19)	57 (7)	21 (19)	27 (11)
Fish	70 (10)	60 (10)	67 (7)	50 (10)	43 (7)
Herptiles	17 (6)	0 (6)	0 (0)	17 (6)	0 (1)
Insects	43 (7)	43 (7)	67 (3)	57 (7)	33 (3)
Mammals	38 (26)	23 (26)	50 (6)	54 (26)	50 (10)
Plants	31 (35)	9 (35)	100 (3)	17 (35)	73 (11)
Other	50 (6)	17 (6)	100 (1)	45 (6)	0 (3)
Total	42 (109)	24 (109)	65 (26)	45 (109)	43 (46)

Table 2.2. Demographic data used to define projection matrices for three independent fox populations and a "generic" population based on data from the three other populations. Sample sizes in parentheses.

Parameter	***Notation***	***Australia[§]*** ***(hunted)***	***Australia[†]*** ***(non hunted)***	***USA[‡]***	***Generic [§† ‡]***
Age distribution	f_0	518	51	1992	2561
	f_1	143	20	817	980
	f_2	88	13	216	317
	f_3	67	14	168	249
	f_{4*}	32	3	62	97
Survival f_{x+1}/f_x	P_1	0.28	0.39	0.41	0.38
	P_2	0.62	0.65	0.26	0.32
	P_3	0.53	0.92	0.60	0.79
	P_{4*}	0.32	0.18	0.27	0.28
Probability of breeding	B_1	0.77 (200)	1.00 (19)	0.68 (82)	0.76 (301)
	B_2	0.88 (64)	1.00(13)	0.92 (36)	0.90 (113)
	B_3	0.88 (34)	1.00 (9)	0.91 (22)	0.91 (65)
	B_{4*}	0.94 (54)	1.00 (3)	0.97 (34)	0.96 (91)
Mean litter size	m_1	3.22 (154)	3.50 (19)	4.52 (73)	3.75 (246)
	m_2	4.00 (56)	3.91 (13)	5.07 (35)	4.33 (104)
	m_3	4.80 (30)	3.09 (9)	5.83 (21)	4.57 (60)
	m_{4*}	4.80 (51)	3.76 (3)	5.91 (33)	4.82 (87)
Fecundity $0.5P_x B_x m_x$	F_1	0.34	0.69	0.63	0.55
	F_2	1.08	1.27	0.61	0.63
	F_3	1.13	1.43	1.59	1.63
	F_{4*}	0.73	0.33	0.77	0.65

[§]Coman, 1988; McIlroy *et al.,* 2001; Saunders *et al.,* 2002; †Marlow *et al.,* 2000; ‡ Storm *et al.*, 1976; Tullar & Berchielli 1981; Nelson & Chapman 1982; Tullar & Berchielli 1982; Allen 1984.

2.3.2 Red fox demographic parameters

Demographic parameters for the three focal populations are summarised in Table 2.2. Also shown are the parameters for the generic population, derived by combining data from the three studies.

2.3.3 Likelihood profiles for demographic parameters

The width (or, equivalently, uncertainty) of likelihood distributions is clearly influenced by both sample size and mean survival rate. Uncertainty is greatest for intermediate vital rates (e.g. probabilities closer to 0.5 than to either zero or unity) and when sample size is low (Figure 2.1). Likelihood profiles were determined for each of the demographic parameters: an example for the Australian population is shown in Figure 2.1. The SSD for these populations is heavily skewed towards younger age classes and this is reflected in the sample sizes available for each age class (see Table 2.1); hence, there is a tendency for likelihoods to show wider distributions for all parameters associated with older age classes (Figure 2.2). The exception to this is the final age class, at which the age distribution is truncated, which has the potential for larger sample sizes than the penultimate age class.

2.3.4 Confidence intervals for population growth estimates

Confidence intervals associated with population growth estimates were generally large and all overlapped with $\lambda = 1$ (denoting a stable population) (Figure 2.3). That all the confidence intervals overlapped with unity does not suggest that these are likely to be stable populations, but it does highlight the uncertainty arising from observation error alone. For example, the point estimate of population growth for the relatively intensively studied USA population (with survival data inferred from over 3,000 culled foxes from the combined studies, but see Chapter 4) suggested an annual increase of approximately 8%. By contrast, 95% confidence intervals for that population varied from suggesting a decline of over 1% per annum, to an annual increase of nearly 16%. Ignoring density dependence, this range of outcomes is equivalent to a population that could decline by 10% over seven years, to one that could grow by 100% in just five years.

In each case, the point estimate of λ was slightly higher than the stochastic mean estimate. This is particularly noticeable for the non-hunted Australian population, which has very small sample sizes. This overestimation can be explained by Jensen's inequality, a mathematical property of non-linear functions. Specifically, the overestimation will occur if lambda is a non-linear decelerating function of a given parameter (Fiske *et al.* 2008).

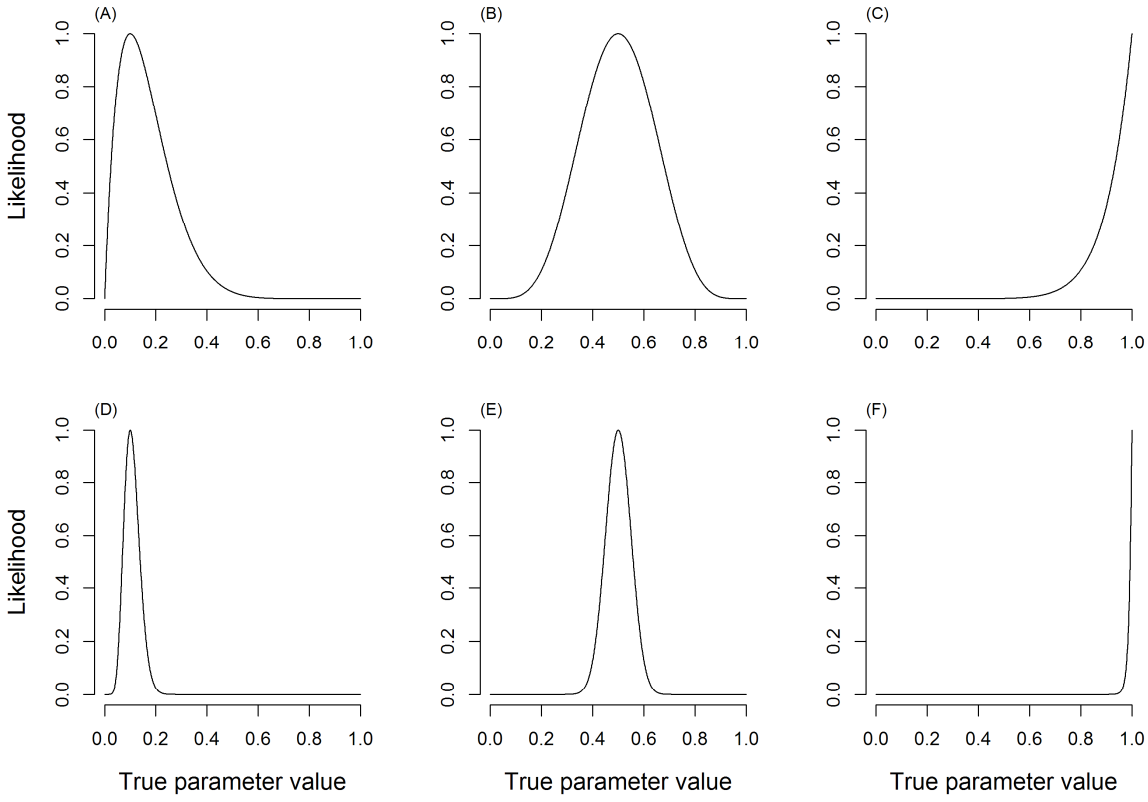

Figure 2.1. Likelihood distributions for vital rates simulated with varying sample sizes. Average survival rates of 0.1 (A & D), 0.5 (B & E), and 1.0 (C & F) are simulated with varying age class sample sizes: (A-C) N = 10, (D-F) N = 100. Likelihoods were rescaled to peak at 1.0.

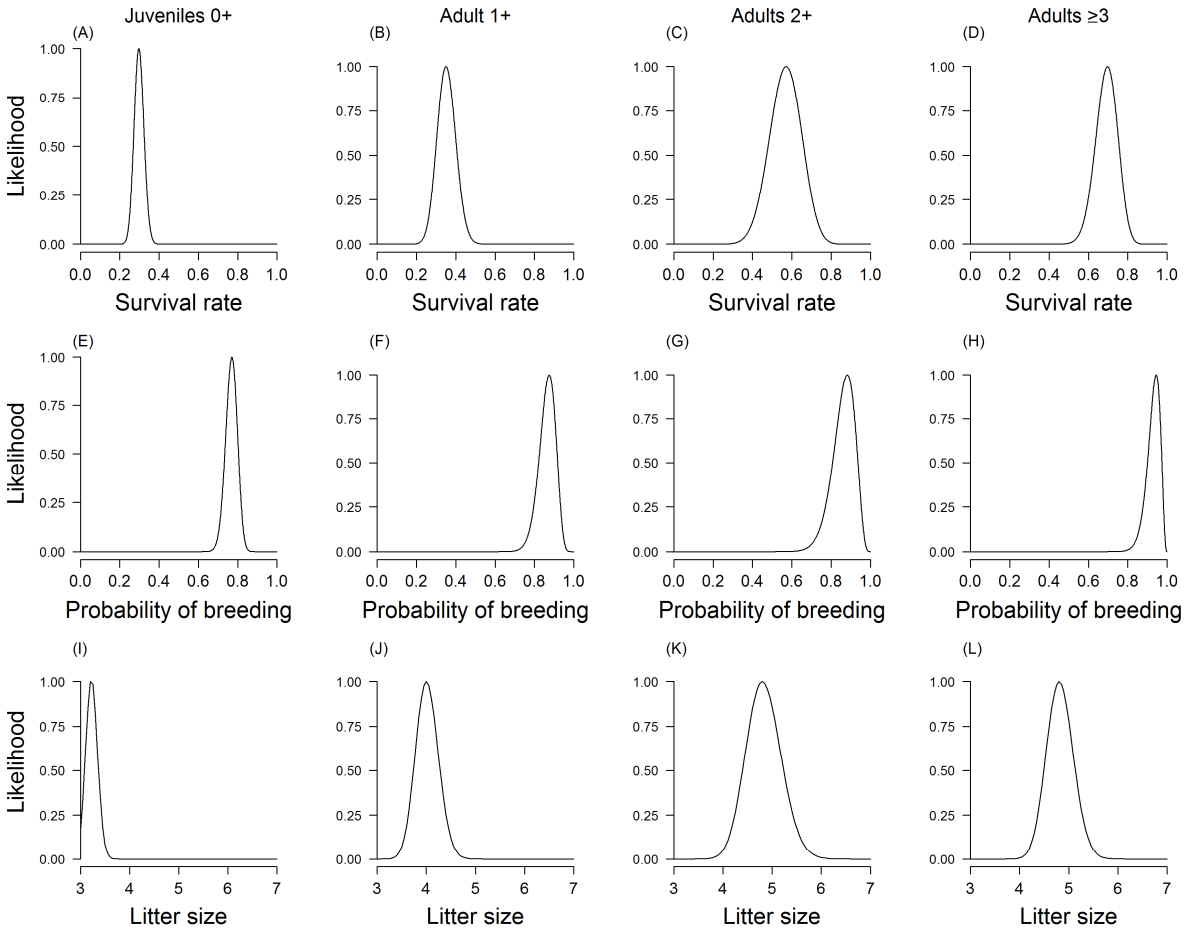

Figure 2.2. Likelihood distributions for demographic parameters of the hunted Australian population. From left to right for age classes 1 to 4: (A-D) survival rates (P_x), (E-H) probability of breeding (B_x) and (I-L) litter size (m_x). Likelihoods were rescaled to peak at 1.0.

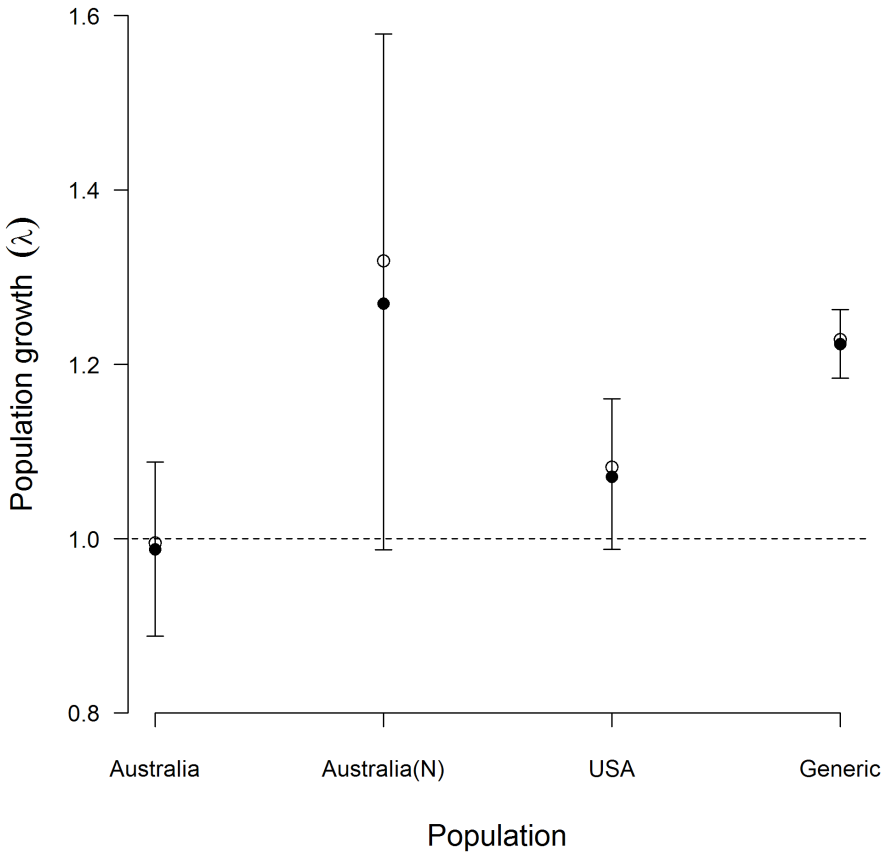

Figure 2.3. Asymptotic population growth rates (λ) for three fox populations and a "generic" population. The figure shows point estimates (determined from the dominant eigenvalue of the population's projection matrix) (open circles), as well as mean (filled circles) and 95% confidence intervals (error bars) determined from 10^4 Monte Carlo resamples from the likelihood distributions of all underlying parameters. The line at λ = 1 indicates stability.

2.3.5 Implications for sampling effort

Confidence intervals around estimates of the generic population's asymptotic growth rate were initially broad but reduced in width at a decreasing rate as sample size increased (Figure 2.4A). In fact, in line with probability theory, the width of the 95% confidence intervals declines with sample size to the half power (Figure 2.4B), indicating that to reduce confidence intervals by half, the sample size needs to be increased fourfold.

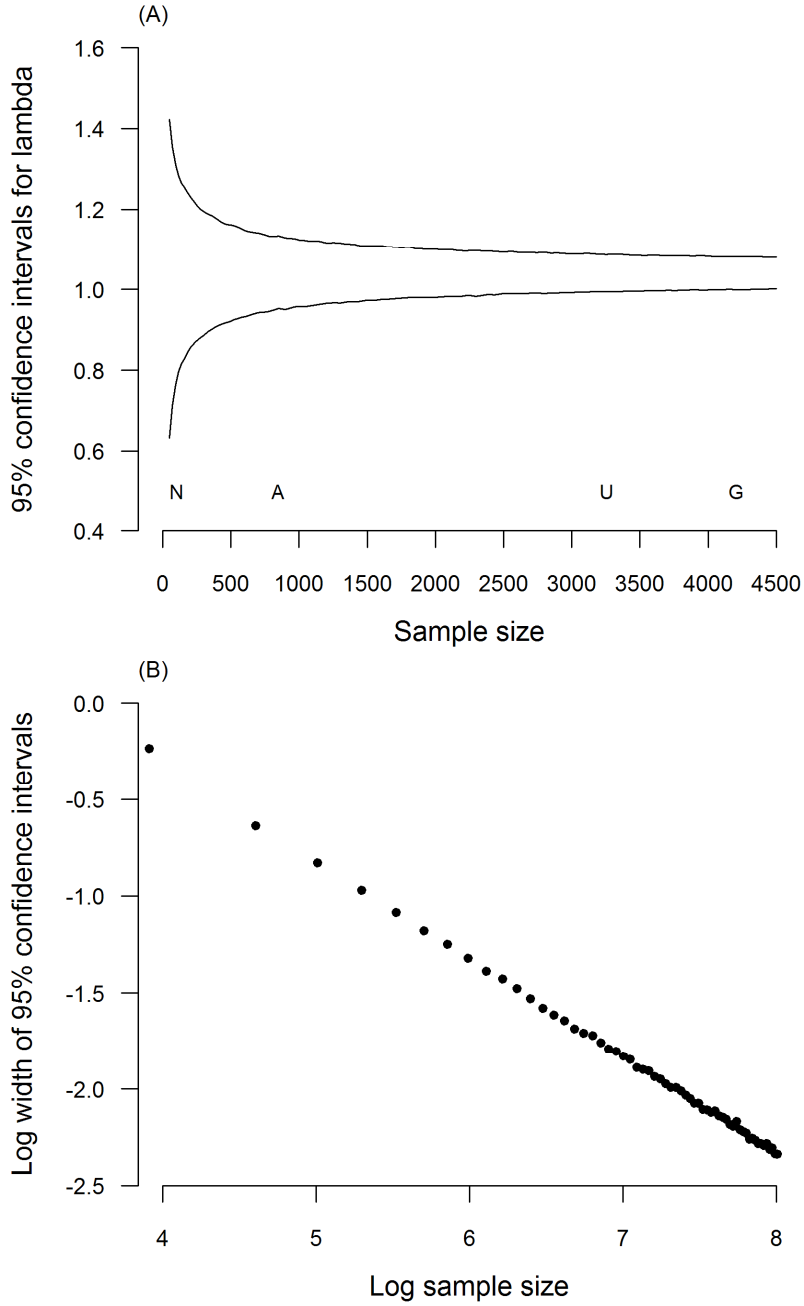

Figure 2.4. Effect of sample size on uncertainty associated with estimated asymptotic population growth (λ). (A) 95% confidence intervals were calculated by resampling with replacement from the individuals available from the generic population (see text for further details). Letters indicate total sample sizes for the age distributions of the focal populations [N, non-hunted Australia = 101; A, Australia = 848; U, USA = 3255; G, Generic = 4204]. (B) Log sample size plotted against log width of the 95% confidence intervals (slope = -0.50).

2.4 Discussion

2.4.1 Accounting for uncertainty: moving beyond point estimates

Matrix modelling (Salguero-Gómez & de Kroon 2010), resampling techniques (Wisdom *et al.* 2000) and likelihood approaches (Hobbs & Hilborn 2006) are increasingly commonly used in ecology. In spite of this, they do not appear to have been combined to provide insight into the limitations imposed on matrix projections by observation error. Here, it has been shown that deriving likelihood distributions from point estimates of demographic parameters is straightforward with freely-available software. Resampling from parameter distributions derived from published studies of fox demography and focusing on estimates of asymptotic growth rate, it has been illustrated that observation error can introduce substantial uncertainty into demographic inference. These results have implications when applying matrix projection models to real world problems and, more generally, for the interpretation of demographic data.

Resampling from measured distributions has been applied to matrix projection models to determine the impacts of a range of sources of variation on the population dynamics of the focal system (e.g. Kalisz & McPeek 1992, Schleuning & Matthies 2008). However, when matrix parameters are derived from data from other sources, accompanying measures of uncertainty are often lacking. This is particularly the case when data are derived from hunting records. Indeed, even in situations in which raw data are available, the review of published studies suggests that generating point estimates of asymptotic growth rate without accompanying estimates of uncertainty is common practice. In either case, the example presented here shows that it should be perfectly possible to infer uncertainty in underlying parameters and, using resampling, to assess how that uncertainty propagates through to insights into population dynamics. This is particularly pertinent, given that demographic models are frequently relied upon to make predictions based on these “uncertain” estimates, such as with regard to sustainable harvesting rates (Marboutin *et al.* 2003) and minimum viable population sizes (Beissinger & Westphal 1998).

Recently, concerns have emerged regarding an over-reliance on stable, asymptotic properties of projection matrices. Ezard *et al.* (2010) noted that anthropogenic impacts frequently perturb populations away from their expected stable stage distributions (SSD), with the result that transient dynamics following a disturbance can depart significantly from the dynamics associated with asymptotic conditions. The consequence is that longer term trajectories can be quite different from those predicted by standard deterministic projection matrix analyses. Ezard *et al.* (2010) recommended a greater focus on transient dynamics and, in particular, a focus on matrix properties decoupled from the assumption of SSDs e.g. by using analyses based on observed stage distributions. Caution is also urged in the interpretation of dynamical parameters derived from standard matrix projection analyses. Indeed, the resampling of likelihoods approach could easily be combined with Ezard *et al.*'s (2010) focus on observed stage distributions.

Although likelihood methods are presented as a useful way to infer uncertainty in point estimates of demographic parameters, this is considered a starting point for more critical analyses of demographic data. For example, McCarthy (2007) presents methods for improving the construction of parameter likelihoods through the establishment of informative priors. In addition, although the shifted Poisson distribution was used to describe litter sizes, further analyses are required to identify the most generally applicable distributions (see chapter 3). In the specific case of litter size, Morris and Doak (2002) have suggested that the stretched beta might be more appropriate. In general, a greater use of online supplementary materials is advocated to provide raw data emerging from studies, in order to aid future analyses of vital rate distributions. In this context it is also important for age distributions to be presented as yearly data (rather than aggregated across years) to improve estimations of vital rate variance, construct periodic models, and incorporate stochasticity.

2.4.2 Applied implications of population growth uncertainty

Foxes are widespread and often abundant and, as a result, they have been extensively studied in a wide range of locations. In spite of this, it remains the case that most fox demographic data are collected through hunting returns. Utilising these minimal data

to their maximum potential is important, not only for foxes, but for other species for which demographic data are collected by similar methods (e.g. Solberg *et al.* 1999, Bischof *et al.* 2008). Increasing our understanding of fox population dynamics is important for designing more efficient management strategies, predicting effects of environmental changes, and understanding evolutionary processes. Although several studies have estimated fox population growth rates (e.g. Pech *et al.* 1997, Hone 1999, Heppell *et al.* 2000, McLeod & Saunders 2001, Oli & Dobson 2003), those results have been presented as point estimates, with no indication of the confidence that could be placed in them. The temptation is, thus, to make comparisons between the growth rates of different populations, potentially attributing those differences to aspects of management or ecological circumstance. In this context, determining confidence intervals about estimates of λ is obviously essential, and these results highlight the need for caution in making comparisons between populations without accounting for uncertainty.

2.4.3 Inference for future data collection

Knowledge of optimal sample sizes has implications for allocating resources (e.g. sampling effort for capture-mark-recapture studies). These results indicate that small initial increases in sample size will yield substantial reductions in uncertainty; however, as sample sizes increase, further effort to collect additional samples yields diminishing returns explained by a simple power law. Doak *et al.* (2005) suggest that it might often be beneficial to increase study duration, rather than sampling intensity. However, smaller sample sizes often lead to bias in demographic inference (Fiske *et al.* 2008). These results suggest that small studies should be avoided but that, as sample size increases, it will be beneficial to devote resources towards determining the mechanistic basis for intrinsic variation, rather than simply to collect more samples; in many systems, this argues in favour of extending study duration to capture the drivers of inter-annual variation, commonly a significant source of variance.

To derive the relationship between sample size and uncertainty in λ, it was assumed that individuals would be sampled approximately in proportion to the SSD. For studies based on mortalities such as shooting or road deaths, this seems to be an appropriate

approach (assuming that the population approximates the SSD; but see Ezard *et al.* 2010). In addition, studies that have considered the best allocation of sampling effort by age or stage (e.g. Gross 2002, Fiske *et al.* 2008) have shown that sampling in proportion to the SSD is the approach likely to yield the least uncertainty in demographic parameters. Certainly, sampling in proportion to the SSD will yield a higher number of juveniles, which typically make the most significant contribution to fox population growth i.e. have the highest elasticities (Harris & Smith 1987, McLeod & Saunders 2001). Owing to the fact that the most important observation errors will arise from inadequate sampling of life stages with the highest elasticities (Caswell 2001), the value (in this case) of sampling in proportion to the SSD is clear. Although it is not possible to define a one-size-fits-all sampling intensity, the simple approach that is presented here should be applicable to a wide range of species. Moreover, the finding that quadrupling the sample size is likely to halve the confidence interval is likely to be very general.

2.5 Conclusion

A brief example has been presented of how more information can be extracted from the type of published data that form a common source for demographic modelling. The results highlight the fact that, even for well-studied species such as the red fox, sampling limitations and inherent variability can limit the precision with which characteristics of population dynamics can be identified. A more widespread use of these straightforward approaches (and related techniques) is recommended, in order to promote a greater awareness of the limitations of many population analyses.

Chapter 3 The effects of litter size variation for models of carnivore extinction risk and management

3.1 Introduction

Demographic variation, resulting from extrinsic and intrinsic sources, fundamentally affects population dynamics and is particularly important when assessing extinction risk for threatened species (Boyce *et al.* 2006, Lee *et al.* 2011). Predictions of population dynamics depend on the ability to attribute sources of stochasticity accurately in population models (Melbourne & Hastings 2008, Ovaskainen & Meerson 2010). Of particular importance is the distinction between demographic stochasticity, the random fate of an individual, and demographic heterogeneity, the individual variation in traits, both of which make important contributions to a populations' total demographic variance (Kendall & Fox 2003, Melbourne & Hastings 2008). Such stochasticity in demographic fates can easily be accounted for by drawing rates from appropriate probability distributions (Akçakaya *et al.* 1999, Morris & Doak 2002). Yet, models often assume that vital rates are homogenous among conspecific individuals, thereby masking the underlying mechanisms by which population dynamics are affected by intraspecific variation (Bolnick *et al.* 2011).

Mean litter (or clutch) size has long been the focus of evolutionary and population biologists concerned with causes of interspecific variation (Blueweiss *et al.* 1978, Böhning-Gaese *et al.* 2000, Jetz *et al.* 2008, Kulesza 2008), correlations with environmental gradients (Lord 1960, Cardillo 2002, Jetz *et al.* 2008, Bywater *et al.* 2010) and optimality in this trait (Lack 1947, Charnov & Krebs 1974, Smith & Fretwell 1974, Sikes & Ylonen 1998). However, intra-population variation in litter size has been largely overlooked (but see Kendall & Wittmann 2010). Limited knowledge of the underlying measures of empirical litter size distributions, such as the degree of dispersion, hinders the accurate representation of the stochasticity of this parameter in population models. When modelling litter size as a separate component, most studies fail to validate their choice of probability distribution (e.g. Rushton *et al.* 2006, Conner *et al.* 2008, Pitt *et al.* 2008, Chapron *et al.* 2009) or use empirical frequencies

(e.g. Ginsberg & Woodroffe 1997, Shirley *et al.* 2003). Demographic stochasticity in offspring number is most commonly modelled with Poisson or normal distributions (Akçakaya 1991, Lacy 1993, Morris & Doak 2002), although there is little theoretical justification for these choices (Kendall & Wittmann 2010). Furthermore, many demographic modelling programmes (e.g. VORTEX, Lacy 1993, and RAMAS, Akçakaya *et al.* 1999) have limited provision for specifying distributions. Unlike survival, which is a Bernoulli process (Akçakaya 1991), choosing a distribution to describe variation in litter sizes in multiparous species can be complex because the biology of reproduction differs substantially among species and is ultimately limited by physiological capacity. Standard probability distributions might lack the flexibility required to account for litter size variation in many species.

In population modelling, the influence of distribution choice has only been considered previously for demographic parameters other than litter size, with a focus on environmental stochasticity. Studies that modelled environmental stochasticity found that population growth rate (λ) estimates were underestimated as a result of inaccurately defined, symmetrical survival distributions (Slade & Levenson 1984) and large differences in λ estimates were found when drawing recruitment rates from different distributions (Nakaoka 1997). Yet, the shape of the distribution may also be important for populations that are susceptible to fluctuations in vital rates as a result of demographic stochasticity, such as small populations. Failing to account for demographic stochasticity in litter size may lead to inaccurate predictions of extinction risk (Kendall & Wittmann 2010). In this context, it is useful to establish whether failing to incorporate an appropriate theoretical distribution for litter size, describing an individual's demographic fate, could lead to erroneous estimates of model outputs.

Here, the fit of specified candidate probability distributions to empirical data on terrestrial carnivore litter size frequencies was examined. The Carnivora exhibit some of the most diverse life history traits of all mammalian orders, as reflected in their broad range of litter sizes (Ewer 1973). While many carnivores are at increasing risk of extinction (Purvis *et al.* 2000), others are predators of economic importance or are important hosts of zoonotic and wildlife diseases such as rabies (Baker *et al.* 2008);

although data collection is often challenging (Gese 2001), both categories of carnivore are frequently the subject of population models (e.g. Smith & Harris 1991, Ginsberg & Woodroffe 1997, Kohlmann *et al.* 2005). Given the importance of carnivore management and the sparseness of much of the data used to model carnivore demography, it is useful to establish whether the choice of distribution used to model demographic stochasticity in litter sizes affects the inferences drawn from models of carnivore population dynamics. To illustrate the applied importance of using appropriate distributions, three previously published population models are replicated to determine the consequences of mis-specifying litter size distributions for inferences regarding extinction probabilities or disease dynamics.

3.2 Methods

3.2.1 Probability distribution fitting

Litter size frequency data were collated for 32 terrestrial multiparous carnivore species, from 64 published studies of 73 wild populations, to reflect the diversity of life history within the order. Each species has a single annual breeding attempt. None of the studies included litters of zero; modelling litter size inherently assumes that an individual has bred. Studies were included regardless of sample size, in order to determine the influence of sampling effort. If studies presented data for multiple conspecific populations or for multiple methods of litter size determination, these were analysed as discrete datasets. For 15 species, data were obtained for between two and ten populations. For three species, data from multiple methods of litter size determination (e.g. placental scars and direct counts) were available. Thus, consideration was also given to whether there was strong support for genuine underlying difference in litter size distributions between conspecific populations or between data determined by different methodologies (for a given population).

Twelve probability distributions were selected based on a review of previous studies. Specifically, four discrete distributions were chosen: the Poisson distribution (Morris & Doak 2002); the generalised Poisson, which has a wide-ranging suitability for describing litter size frequencies (Kendall & Wittmann 2010); the binomial distribution, previously fitted successfully to carnivore litter data (Kendall & Wittmann 2010); and the negative binomial, widely used to describe ecological processes (e.g. Shaw *et al.* 1998). For each discrete distribution, both a “right shifted” and “zero-truncated” form were fitted (Appendix 3), to exclude litter sizes of zero. For zero-truncation, the probability mass function was scaled by the exclusion of predicted zeros. Shifting involved moving the entire distribution one interval to the right. Three continuous probability distributions were chosen: the normal and lognormal distributions are both widely used (Morris & Doak 2002), although log-transformation is not recommended for count data (O'Hara & Kotze 2010); and the stretched beta (two and three parameter forms), as proposed by Morris and Doak (2002). Appendix 3 provides details of how these continuous distributions were converted into discrete forms.

Maximum-likelihood parameters, denoted $\hat{\boldsymbol{\theta}}$, were estimated using the "optim" function in R 2.14.0 (R Development Core Team 2011). Here, the multinomial log-likelihood defined by $\boldsymbol{\theta}$ and given all the data is:

$$LL(\boldsymbol{\theta} \mid \text{data}) = \Gamma(N+1) + \sum_{i=1}^{x_{\max}} \left[N_i \ln P_i(\boldsymbol{\theta}) - \Gamma(N_i+1) \right], \qquad (1)$$

where N is the total number of litters observed, N_i is the number of litters observed of size i, P_i is the predicted litter size probability determined by a given distribution (Appendix 3), x_{max} is the maximum litter size, and Γ(x) is the complete gamma function. The fits for each probability distribution were compared using Akaike's Information Criterion (AIC), a metric of model parsimony that reflects the trade-off between model fit and parameter uncertainty (Burnham & Anderson 2002, Richards 2005). All distributions having a ΔAIC ≤ 6 of the best fitting distribution (i.e. lowest AIC) were considered to have some support (Richards 2008). To check that the best-fitting models were consistent with the data and because of the small sample sizes of the predicted frequencies, goodness-of-fit tests were performed using Fisher's Exact Test. Whether sample size had an effect on the number of parsimonious distributions was assessed using linear regression. Variance-mean ratios (Sokal & Rohlf 1987, p.69) were determined to measure the dispersion of the empirical and fitted distributions.

3.2.2 Intraspecific variation in litter size distributions

In addition to establishing whether interspecific differences exist in the suitability of probability distributions to model litter size, it is also interesting to consider intraspecific variation in describing litter size. Intraspecific variation can be examined through a two-part analysis. First, evidence was sought that distinct probability distributions are required to describe the litter size distributions of conspecific populations. Specifically, is a distinct probability distributions needed to describe the litter size data (hereafter referred to as "a dataset") taken from populations that are separated geographically, or where the data have been determined using different methodologies? Second, if it is established that the same distribution can be applied to

specified datasets, do the same parameter values of the probability distribution function describe the given datasets adequately?

The first component of the analyses determined whether the same probability distribution could be applied to the specified datasets. For a given pair of datasets, the joint AIC value was calculated for each possible probability distribution combination. Specifically, let $M(i,j)$ be a model where probability distribution i is fitted to the first dataset and probability distribution j is fitted to the second dataset. The log-likelihood of this model is then simply the sum of the log-likelihoods of each probability distribution fitted to their specified dataset. Whether the same probability distributions adequately described the datasets was evaluated by determining if any model where $i = j$ was within 6 units of the smallest AIC (over all possible probability distribution combinations). This approach is readily generalised for more than two datasets. Only parsimonious distributions as determined by the initial fitting (see above), for the geographic and methodological datasets, respectively, were included in these analyses.

If at least one probability distribution could adequately describe the specified datasets, the second component of the analyses sought to determine whether the same parameter values could be used to describe each of the datasets. Specifically, let $LL(\hat{\boldsymbol{\theta}} \mid S)$ be the maximum log-likelihood when the probability distribution described by the parameters $\boldsymbol{\theta}$ is fitted to dataset S. The maximum log-likelihood when two datasets are described by distinct parameter sets is $LL_1 = LL(\hat{\boldsymbol{\theta}}_1 \mid S_1) + LL(\hat{\boldsymbol{\theta}}_2 \mid S_2)$; and when the two datasets are described by a probability distribution with the same parameters, the maximum log-likelihood is $LL_0 = LL(\hat{\boldsymbol{\theta}}_3 \mid S_3) + LL(\hat{\boldsymbol{\theta}}_3 \mid S_3)$. A log-likelihood ratio test was then used to determine whether the simpler model (using a single parameter set) provided a more parsimonious description of the combined datasets than its expanded alternative (using two distinct parameter sets). The test statistic is determined by the deviance, defined as $G=2(LL_1-LL_0)$. The distribution of G is approximately chi-squared, with the degrees of freedom (df) equal to the additional number of free parameters

required for the more complex model (Sokal & Rohlf 1987). This approach is also readily generalised for more than two datasets.

The above approaches were used to test for intraspecific differences in the underlying litter size distributions of the red fox *Vulpes vulpes*. Litter size data collected from six geographically distinct populations were used, where data were determined by placental scars. Data for these populations were combined over 4, 3, 4, 5, 6, and 17 year periods, respectively (Appendix 4). Three methodologies used to determine litter size for one red fox population (S. Harris, *unpublished data*) were then compared, using data determined by placental scars, embryo counts and direct counts, combined over a 17 year period.

3.2.3 Carnivore population models

Published stochastic population models of three management scenarios were used to illustrate the broader applied significance of this study. The Canidae were chosen because they provide the widest range of litter sizes within the Carnivora (Ewer 1973). Models were chosen to depict a range of conservation and management scenarios that could be replicated from published data; the intention was to identify whether the choice of distribution used to represent litter sizes influences predicted model outcomes. Here, "outcomes" refers to a major emergent parameter from the models, on which further inference would be based (see below). The emergent parameter of interest varied because the three models were created for different applications. Using the parameters that were estimated by maximum likelihood as described above, 10,000 stochastic replicates of the models were simulated drawing litter sizes from each of the 12 probability distributions. This enabled calculation of 95% confidence intervals around a binomial outcome (Hilborn & Mangel 1997). For each case study, disparities were determined between the outcome values of the 12 model versions. This allowed the evaluation of the effect on each model of employing different litter size distributions, in relation to the degree of empirical support for those distributions.

Table 3.1. Parameter values for the three population models (Kohlmann *et al*. 2005; Smith and Harris 1991; Ginsberg and Woodroffe 1997).

Initial parameter value	***Model 1. Island fox (Urocyon littoralis)***	***Model 2. Red fox (Vulpes vulpes)***	***Model 3. African wild dog (Lycaon pictus)***
Quasi-extinction or disease density threshold	50	87% of initial population	One sex remains
Years	100	3	50
Time step	Annual	Monthly	Annual
Age at first reproduction	2	1	3
Sex ratio at birth	0.5	0.5	0.55
Dispersal age	1	1	-
Dispersal probability	0.01	Female (month 7-12): 0.03, 0.030, 0.136, 0.045, 0.045, 0.030 Male (month 7-12): 0.68, 0.102, 0.182, 0.159, 0.102, 0.057	-
Dispersal survival	0.8	-	-
Annual mortality rate pup	0.31 ± 0.59	-	0.68 ± 0.20
Annual mortality rate juvenile male	0.25 ± 0.60	Monthly: 0.137, 0.045, 0.040, 0.048, 0.036, 0.035, 0.044, 0.044, 0.039, 0.062, 0.032, 0.035	0.20 ± 0.03
Annual mortality rate juvenile female	0.17 ± 0.47	Monthly: 0.129, 0.052, 0.067, 0.037, 0.042, 0.037, 0.044, 0.032, 0.039, 0.025, 0.034, 0.030	0.20 ± 0.03
Annual mortality rate adult male	0.25 ± 0.60	Monthly: 0.035, 0.039, 0.020, 0.028, 0.014, 0.039, 0.036, 0.046, 0.041, 0.121, 0.069, 0.029	0.15 ± 0.03
Annual mortality rate adult female	0.17 ± 0.47	Monthly: 0.041, 0.055, 0.035, 0.025, 0.023, 0.034, 0.044, 0.049, 0.035, 0.062, 0.041, 0.036	0.15 ± 0.03
Probability of breeding	1	0.8	0.58 (dominant pairs only)
Density dependence in breeding (% breeding at carrying capacity)	West subpopulation: 58.38 East subpopulation: 55.03	-	-
Carry capacity	West subpopulation: 300 East subpopulation: 1300	-	20
Initial population size	West subpopulation: 90 East subpopulation: 63	1 male and 1 female per group, additional male or female added with probability of 0.80 and 0.58 additional individual 0.47 probability of being juvenile	20
Disease Introduction	-	September	-
Incubation period	-	1 month	-
Probability of becoming rabid once exposed	-	0.42	-
Disease mortality	-	1	-
Control	-	40% control every 2 months, 3 months after disease introduction	-
Catastrophes	Frequency: 0.2 Reduction in survival: 0.8	-	Mild: Frequency: 0.05 Survival reduction: 0.85 Reproduction reduction: 0.5 Severe: Frequency: 0.03 Survival reduction: 0.5

First, the island fox *Urocyon littoralis* was investigated, which reached near extinction on Santa Catalina Island due to an outbreak of canine distemper virus (Clifford *et al.* 2006). A density-dependent population viability analysis (PVA) was conducted for two subpopulations; the outcome of interest was the probability of quasi-extinction, defined in this model as the probability of the population declining to 50 individuals, due to a disease epidemic. Specifically, an annual, density-dependent, stochastic PVA of the Santa Catalina island fox population was written, based on Kohlmann *et al.* (2005) with initial parameter values taken from their model (Table 3.1). Mean litter size in their model was taken from Coonan *et al.* (1998); here, the empirical litter size frequency data were obtained from Coonan (*unpublished data*). Two subpopulations (east and west) were simulated over a 100-year period, with a catastrophe event occurring at a frequency of 20%, and a severity of an 80% reduction in survival. In this way, the model encapsulates a disease event (e.g. canine distemper virus). Breeding was density-dependent, and varied between both subpopulations. The proportion of females breeding at the carrying capacity for each subpopulation was determined according to equation (1) in Kohlmann *et al.* (2005). Following Miller and Lacy (2005), environmental variation was simulated by drawing age-specific mortality rates at the start of each year from a binomial distribution with a specified mean and standard deviation (Table 3.1) and demographic stochasticity in mortality was modelled with a binomial trial. The PVA (Kohlmann *et al.* 2005) was run in VORTEX, and the same sequence of events was used (Miller & Lacy 2005) to create the model in R 2.14.0 (R Development Core Team 2011) to allow greater flexibility in specifying probability distributions.

Second, the red fox was investigated, a locally abundant carnivore that is the focus of much attention due to its economic importance as a predator and role in the spread of rabies (Chautan *et al.* 2000). A model simulating fox control after a rabies outbreak was replicated to illustrate, as the outcome of interest, the probability of successful disease control. Here, a monthly, stochastic, simulation model of the red fox was constructed, based on Smith and Harris (1991), with initial parameter values taken from their model (Table 3.1). Litter size frequency data from Bristol (S. Harris, *unpublished data*) were used. Breeding was simulated in April (month 1 in this model),

and one female per group was given an opportunity to breed. Age-specific mortality probabilities were drawn from a binomial distribution. During months 7 to 12 juvenile males and females dispersed with set probabilities. The model was run for three years. Rabies was introduced by infecting all foxes within one group at the beginning of September (month 6) in the first year, with a latency period of one month before becoming infectious. Neighbouring individuals were then infected with the following contact probabilities: within group infection 0.9, neighbouring cubs during summer 0.3, if male, to infect neighbouring females during winter 0.9, any other neighbour infection 0.6. The original analysis (Smith & Harris 1991) determined that for successful disease eradication the initial population size needed to be reduced by 87%. This was achieved in their model by implementing a control regime, starting three months after the first detection of rabies, which consisted of a total of four control events, each with 40% fox removal every two months.

Finally, the African wild dog *Lycaon pictus* was investigated, which is restricted throughout much of its range and susceptible to several diseases, including rabies (Vial *et al.* 2006). A density-dependent PVA for small wild dog populations was reproduced to determine quasi-extinction probabilities (the outcome variable), defined here as the probability of only one sex remaining. Specifically, an annual, stochastic PVA of the African wild dog was simulated, based on Ginsberg and Woodroffe (1997), with initial parameter values taken from their model (Table 3.1). Their model was run in VORTEX, and as in Model 1, the same sequence of events was used (Miller & Lacy 2005) to create the model in R 2.14.0 (R Development Core Team 2011). Litter size in their model was input as an empirical distribution and these data were used to fit the 12 probability distributions used in this study. Following Miller and Lacy (2005), environmental variation was simulated by drawing age-specific mortality rates at the start of each year from a binomial distribution with a specified mean and standard deviation (Table 3.1) and demographic stochasticity in mortality was modelled with a binomial trial. A small population of 20 individuals was simulated for 50 years, found from their PVA to be the most susceptible to extinction. Breeding was not density dependent, but at the start of each simulation it was assumed that the population was at carrying capacity (Ginsberg & Woodroffe 1997), and following VORTEX (Miller &

Lacy 2005), truncation was applied above this value by including a separate survival component. Two catastrophes were included, a mild and a severe, to simulate environmental events, or a disease outbreak respectively. Following Vial *et al*. (2006), the effects of including a component Allee effect, which is exhibited through a positive relationship between population size and a measurable component of fitness (Stephens *et al.* 1999), were also considered through a reduction in recruitment. Here, for computational and data requirement reasons African wild dog litter size was modified rather than reducing pup mortality, by decreasing individual litter size by a quantity determined as a function of group size, *sensu* (Vial *et al.* 2006). Specifically, each litter size draw was reduced by a quantity defined as $k(P_t - N)$, where P_t is the carrying capacity, k, estimated to be 0.8 (Vial *et al.* 2006), is the slope of the relationship between pack size (here, population, N) and number of pups recruited to yearling age.

These three investigations illustrate canids with small, medium, and large mean litter sizes, respectively (Appendix 4). The results of all three replicated models were compared with the original model predictions to ensure accurate replication, except for Model 3 with the inclusion of an Allee effect, which the original model did not incorporate. All modelling and analyses were conducted in R 2.14.0 (R Development Core Team 2011).

3.3 Results

3.3.1 Variation in litter size distributions

Variance-mean ratios (mean = 0.40, SD ± 0.40) indicated that empirical distributions tend to be underdispersed and display, on average, weak positive skew (mean coefficient of skewness = 0.07, SD ± 0.31, Appendix 4). While the majority of datasets represented one population (96%), most data were presented from studies over multiple years (97%) (Appendix 4). Best fitting distributions differed substantially between datasets (Table 3.2 and Appendix 5), although all distributions with ΔAIC ≤ 6 provided a good fit to the empirical data (Appendix 6). For 97% of all datasets, several of the 12 candidate distributions (mean = 6.54, SD ± 3.38) could not be discounted based on their AIC values (Appendix 5 and Figure 3.1A-L for examples). The most widely applicable distribution was the discretised normal, with ΔAIC ≤ 6 for 95% of datasets; all other distributions were selected for between 22% and 87% of datasets. The "right shifted" method consistently performed better than zero-truncation (Appendix 5), being on average 1.32 (SD ± 0.16) times more likely to have a ΔAIC ≤ 6. The selection of distributions by AIC also depended on sample size and the sampling method used to determine litter size for each dataset. As expected, there was a negative relationship between sample size and the number of distributions with ΔAIC ≤ 6 (r^2 = 0.35, p < 0.0001, n = 80). The relationship between mean litter size and the number of distributions with ΔAIC ≤ 6 was not significant (r^2 = 0.008, p = 0.43, n = 80). When repeating these analyses with datasets with $n \geq 20$ (where n is the number of litters sampled) to increase statistical power, the relationships between the number of distributions with ΔAIC ≤ 6 with sample size and mean litter size remained the same (r^2 = 0.32, p < 0.0001, n = 61 and r^2 = 0.02, p = 0.31, n = 61, respectively; Figure 3.2).

Table 3.2. Model selection results for fitting probability distributions to carnivore litter size frequencies. The number of datasets tested for each species (denominator, see Appendix 4 for details) indicating the number of datasets that were satisfied by a given distribution (numerator, see Appendix 5 for details). Bold indicates distributions that were most parsimonious for at least one dataset. SP: Shifted Poisson; ZTP: Zero-truncated Poisson; SB: Shifted binomial; ZTB: Zero-truncated binomial; SNB: Shifted negative binomial; ZTNB: Zero-truncated negative binomial; SGP: Shifted generalised Poisson; ZTGP: Zero-truncated generalised Poisson; DN: Discretised normal; DLN: Discretised lognormal; DSB3; Discretised stretched-beta (3 parameter form); DSB2; Discretised stretched-beta (2 parameter form).

Species	***SP***	***ZTP***	***SB***	***ZTB***	***SNB***	***ZTNB***	***SGP***	***ZTGP***	***DN***	***DLN***	***DSB3***	***DSB2***
Canidae												
Vulpes velox	1/1	-	1/1	-	-	-	1/1	-	**1/1**	1/1	1/1	1/1
Vulpes macrotis	-	-	1/2	-	-	-	-	-	**2/2**	1/2	1/2	**2/2**
Vulpes vulpes	**5/12**	2/12	4/12	4/12	2/12	-	4/12	2/12	**11/12**	**3/12**	6/12	7/12
Urocyon littoralis	**1/2**	1/2	1/2	1/2	1/2	1/2	1/2	1/2	**1/2**	2/2	2/2	2/2
Urocyon cinereoargenteus	1/2	1/2	1/2	1/2	1/2	1/2	1/2	1/2	2/2	**2/2**	2/2	**2/2**
Alopex lagopus	-	-	1/3	-	1/3	-	**1/3**	1/3	2/3	**2/3**	3.3	3/3
Canis lupus	**2/2**	2/2	1/2	1/2	1/2	2/2	2/2	2/2	**2/2**	1/2	2/2	2/2
Lycaon pictus	**1/4**	1/4	-	-	1/4	1/4	3/4	**3/4**	**4/4**	2/4	4/4	3/4
Nyctereutes procyonoides	1/1	1/1	1/1	1/1	-	-	1/1	-	**1/1**	1/1	1/1	1/1
Hyaenidae												
Crocuta crocuta	-	-	1/3	1/3	-	-	-	-	**3/3**	3/3	3/3	**2/3**
Procyonidae												
Procyon lotor	1/1	1/1	1/1	1/1	-	-	1/1	1/1	1/1	**1/1**	1/1	1/1
Felidae												
Acinonyx jubatus	-	-	1/1	-	-	-	-	-	1/1	1/1	1/1	**1/1**
Felis concolor	1/3	-	2/3	2/3	-	-	-	-	**3/3**	3/3	3/3	**3/3**
Felis iriomotensis	**1/1**	1/1	-	-	1/1	1/1	1/1	1/1	1/1	1/1	1/1	1/1
Lynx pardinus	-	-	1/1	-	-	-	-	-	1/1	**1/1**	1/1	1/1
Panthera tigris altaica	**1/1**	1/1	-	1/1	1/1	1/1	1/1	1/1	1/1	1/1	1/1	1/1
Panthera onca	**1/1**	1/1	-	1/1	1/1	1/1	1/1	1/1	1/1	1/1	1/1	-
Panthera leo	2/6	-	**3/6**	1/6	-	-	2/6	-	**6/6**	**5/6**	6/6	**6/6**
Panthera pardus	-	-	1/1	-	-	-	-	-	1/1	**1/1**	1/1	1/1
Leopardus pardalis	**1/1**	1/1	1/1	1/1	1/1	1/1	1/1	-	1/1	1/1	1/1	1/1
Ursidae												
Ursus maritimus	-	-	-	-	-	-	-	-	**4/4**	**4/4**	4/4	**4/4**
Ursus arctos	-	-	**2/4**	-	-	-	-	-	**2/4**	3/4	31/4	**4/4**
Ursus americanus	2/7	2/7	6/7	3/7	1/7	-	2/7	1/7	**7/7**	**5/7**	4/7	**5/7**
Mustelidae												
Lutra lutra	**4/7**	2/7	**3/7**	4/7	3/7	1/7	4/7	1/7	**7/7**	7/7	7/7	**4/7**
Lontra canadensis	**2/2**	2/2	**2/2**	2/2	2/2	1/2	2/2	2/2	2/2	2/2	2/2	2/2
Mustela erminea	1/1	1/1	1/1	1/1	-	-	1/1	1/1	-	1/1	1/1	**1/1**
Mustela nigripes	-	-	1/1	-	-	-	-	-	**1/1**	1/1	1/1	1/1
Martes pennanti	-	-	-	-	-	-	-	-	1/1	**1/1**	1/1	-
Martes americana	**1/1**	1/1	1/1	1/1	1/1	1/1	1/1	1/1	1/1	1/1	1/1	1/1
Spilogale putorius	1/1	1/1	1/1	**1/1**	1/1	-	1/1	1/1	1/1	1/1	1/1	1/1
Gulo gulo	-	-	1/1	1/1	-	-	-	-	**1/1**	1/1	1/1	-
Meles meles	-	-	1/2	1/2	-	-	-	-	**1/2**	**2/2**	2/2	2/2

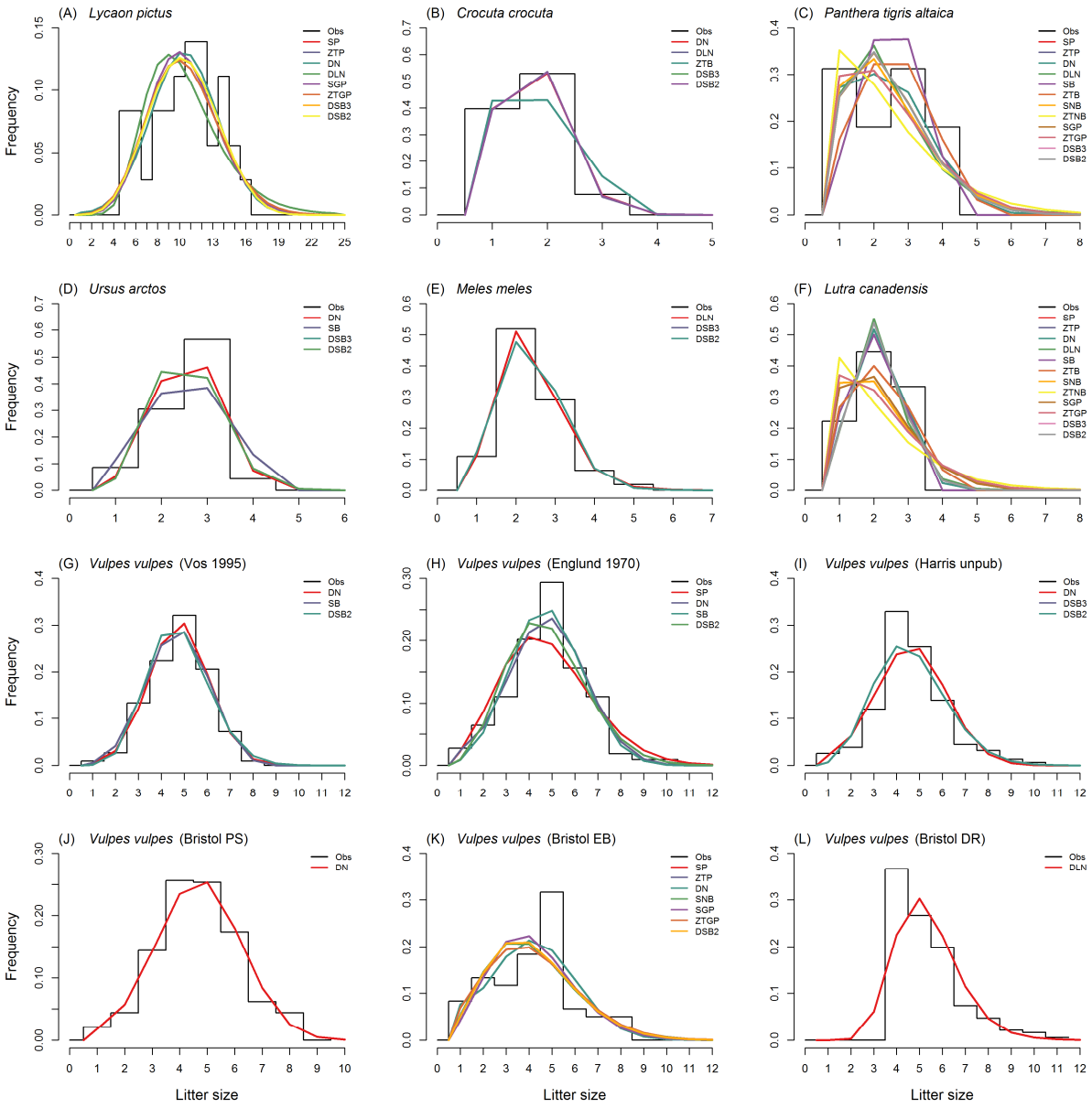

Figure 3.1. Observed litter size frequencies with fitted distributions with ΔAIC ≤ 6. The top two panels show for a range of sample sizes (of litters sampled), mean litter size, and carnivore families. The third panel from the top shows three populations of *Vulpes vulpes* with litter size determined by placental scars and the bottom panel illustrates three different methods for determining litter size of a Bristol population of *Vulpes vulpes* (S. Harris, *unpublished data*). (A) *Lycaon pictus,* n = 36 (Creel *et al.* 2004); (B) *Crocuta crocuta,* n = 53 (Watts & Holekamp 2008); (C) *Panthera tigris altaica*, n = 16 (Kerley *et al.* 2003); (D) *Ursus arctos,* n = 46 (Miller *et al.* 2003); (E) *Meles meles*, n = 110 (Neal & Cheeseman 1996); (F) *Lontra canadensis,* n = 9 (Hamilton & Eadie 1964); (G) *V. vulpes*, n = 112 (Vos 1995); (H) *V. vulpes*, n = 113 (Englund 1970); (I) *V. vulpes*, London, n = 158 (S. Harris, *unpublished data*); (J) *V. vulpes*, placental scars, n = 340; (K) *V. vulpes*, embryos, n = 60; (L) *V. vulpes*, direct counts, n =191. See Appendix 4 for details of datasets. Distribution abbreviations: observed frequencies (Obs); shifted Poisson (SP); ZT Poisson (ZTP); discretised normal (DN); discretised lognormal (DLN); discretised stretched beta – 2 parameter form (DSB2); discretised stretched beta 3 parameter form (DSB3); shifted generalised Poisson (SGP); ZT generalised Poisson (ZTGP); shifted binomial (SB); ZT binomial (ZTB); shifted negative binomial (SNB); ZT negative binomial (ZTNB).

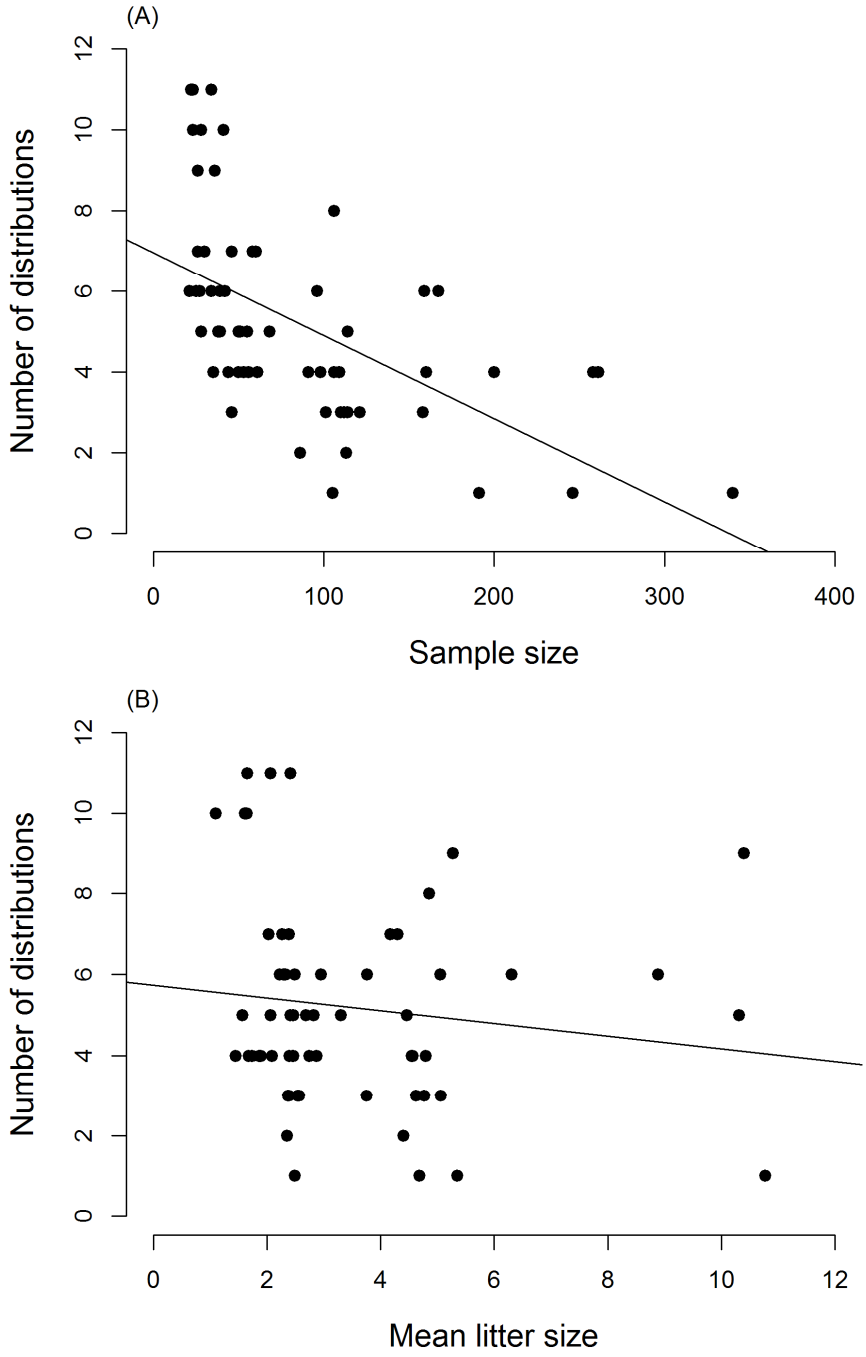

Figure 3.2. Linear regression of the number of probability distributions fitted to litter size frequency data with ΔAIC scores ≤ 6 against (A) sample size (r^2 = 0.28, p < 0.0001) and (B) mean litter size (r^2 = 0.02, p = 0.32). Only datasets with n ≥ 20 (litters sampled) are included.

Table 3.3. Results of model selection to test for intraspecific geographic variation in the best-fitting litter size distributions for six red fox populations. Only models where datasets fitted to probability distribution* combinations had a ΔAIC score ≤ 6 are presented. For details of the datasets, refer to the references in Appendix 4.

Dataset [Reference]								
1[4]	***2***[8]	***3***[8]	***4***[8]	***5***[9]	***6***[11]	***Log-likelihood***	***AIC***	***ΔAIC***
DN	DN	SP	DN	DN	DN	-118.20	260.41	5.69
DN	DN	DN	DN	DN	DN	-115.36	254.72	0.00
DN	DN	DN	SB	DN	DN	-117.32	258.64	3.92
DN	DN	DN	ZTSB	DN	DN	-117.73	259.47	4.75
DN	DN	DLN	DN	DN	DN	-118.08	260.17	5.45
DN	DN	ZTSB	DN	DN	DN	-115.53	255.06	0.34
DN	DN	ZTSB	SB	DN	DN	-117.49	258.98	4.26
DN	DN	ZTSB	ZTSB	DN	DN	-117.91	259.81	5.09
DN	DN	DSB3	DN	DN	DN	-116.10	258.20	3.48
DN	DN	DSB2	DN	DN	DN	-116.92	257.84	3.12
DN	DSB3	DN	DN	DN	DN	-117.17	260.34	5.62
DN	DSB3	ZTSB	DN	DN	DN	-117.34	260.68	5.96
SB	DN	SP	DN	DN	DN	-118.32	260.64	5.92
SB	DN	DN	DN	DN	DN	-115.48	254.95	0.23
SB	DN	DN	SB	DN	DN	-117.43	258.87	4.15
SB	DN	DN	ZTSB	DN	DN	-117.85	259.70	4.98
SB	DN	DLN	DN	DN	DN	-118.20	260.40	5.68
SB	DN	ZTSB	DN	DN	DN	-115.65	255.30	0.58
SB	DN	ZTSB	SB	DN	DN	-117.61	259.21	4.49
SB	DN	ZTSB	ZTSB	DN	DN	-118.02	260.04	5.32
SB	DN	DSB3	DN	DN	DN	-116.22	258.43	3.71
SB	DN	DSB2	DN	DN	DN	-117.04	258.07	3.35
SB	DSB3	DN	DN	DN	DN	-117.28	260.57	5.85
DSB3	DN	DN	DN	DN	DN	-115.75	257.50	2.78
DSB3	DN	ZTSB	DN	DN	DN	-115.92	257.85	3.13
DSB3	DN	DSB2	DN	DN	DN	-117.31	260.62	5.90

*Distribution abbreviations: SP: Shifted Poisson; ZTP: Zero-truncated Poisson; SB: Shifted binomial; ZTSB: Zero-truncated binomial; SNB: Shifted negative binomial; SGP: Shifted generalised Poisson; ZTGP: Zero-truncated generalised Poisson; DN: Discretised normal; DLN: Discretised lognormal; DSB3: Discretised stretched-beta (3 parameter form); DSB2: Discretised stretched-beta (2 parameter form).

Table 3.4. Results of model selection to test for intraspecific methodological variation in the best-fitting litter size distributions for the Bristol red fox population. Only models where datasets fitted to probability distribution* combinations had a ΔAIC score ≤ 6 are presented. For details of the datasets, refer to the references in Appendix 4.

Dataset [Reference]			***Log-***		
1 [11]	***2*** [12]	***3*** [13]	***likelihood***	***AIC***	***ΔAIC***
DN	SP	DLN	-83.63	177.27	2.24
DN	ZTP	DLN	-83.20	176.40	1.38
DN	DN	DLN	-81.51	175.02	0.00
DN	SNB	DLN	-84.21	180.42	5.39
DN	SGP	DLN	-83.63	179.27	4.24
DN	ZTGP	DLN	-83.20	178.40	3.38
DN	DSB2	DLN	-83.66	179.31	4.29

3.3.2 Intraspecific variation in litter size distributions

While there was little support for intraspecific differences between conspecific red fox populations, distinct probability distributions best described litter size data determined by pre- and post-birth methodologies. Model selection results for the specified geographically distinct red fox populations supported models using the same distribution (Table 3.3), suggesting that the focal datasets could be described adequately using the discretised normal. Further, a single parameter set adequately described the discretised normal litter size distribution (G = 119.23, df = 10, p <0.001) for these geographically separated red fox populations. For litter size data of a red fox population determined by different methodologies, a difference in the underlying distributions was inferred by the lack of support for models using the same distributions (Table 3.4). Thus, these methodological datasets were best described by distinct distributions and parameter sets.

3.3.3 Carnivore model outcomes

The demographic modelling showed that the distribution chosen to represent litter size uncertainty in the three canid models has limited impacts, regardless of the fit of the distributions. PVA models for island foxes showed that estimating extinction probability was largely unaffected by the choice of distribution, with less than 1% difference in quasi-extinction probabilities between models that used the best and

worst fitting litter size distributions (Figure 3.3A&B). Similarly, regardless of whether the litter size distributions used in the model provided a good fit to empirical litter size data, there was only a 2% difference in the probability of successful disease control in the rabies model for red foxes (Figure 3.3C&D). Likewise, quasi-extinction probabilities for African wild dogs showed only a 1% difference among models that employed different litter size distributions (Figure 3.3E&F). When litter size was reduced as a function of group size, to simulate an Allee effect, the influence of the distributions was slightly greater (Figure 3.3G&H), with an increase of approximately 4% between quasi-extinction probabilities for the best and worst-fitting distributions. Even in this case, only models employing the worst-fitting distributions differed substantially in their predictions from those of models employing other distributions. Coefficients of variation (CV) were small for all model outcomes (Table 3.5), with the greatest variation in the African wild dog model with an Allee effect; best-fitting distribution (CV = 0.712) was 1.07 times more variable than for the worst fitting model (CV = 0.668). For all the models, the best-fitting distributions were able to describe accurately the variance and skew of the empirical distribution (Figure 3.3A-H).

Table 3.5. Coefficient of variation for model outcomes of quasi-extinction probabilities* and probability of successful disease control†, for 12 probability distributions.

Distribution	***Island fox* (West)***	***Island fox* (East)***	***Red fox†***	***African wild dog without Allee****	***African wild dog with Allee****
SP	1.161	1.354	0.307	1.388	0.713
ZTP	1.116	1.298	0.333	1.407	0.703
SGP	1.102	1.302	0.332	1.409	0.709
ZTGP	1.130	1.339	0.332	1.416	0.712
SB	1.154	1.350	0.293	1.409	0.668
ZTB	1.126	1.335	0.306	1.418	0.689
SNB	1.134	1.330	0.332	1.431	0.712
ZTNB	1.122	1.307	0.332	1.428	0.736
DN	1.161	1.353	0.307	1.399	0.712
DLN	1.143	1.339	0.319	1.424	0.737
DSB2	1.133	1.336	0.307	1.403	0.711
DSB3	1.133	1.319	0.320	1.406	0.710

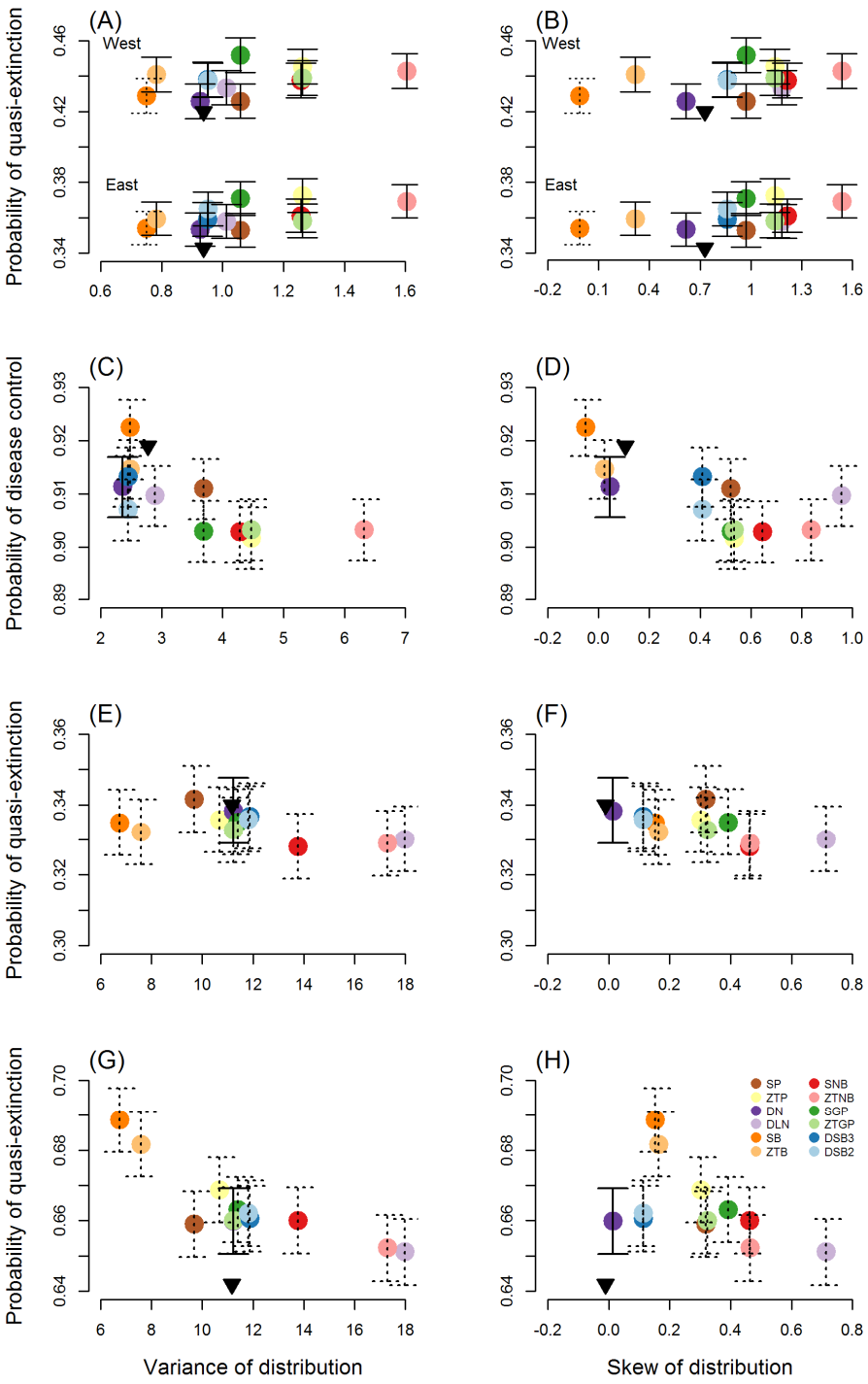

Figure 3.3. Model outcomes for 12 probability distributions against the variance and skew of distributions, showing quasi-extinction probabilities and probability of successful disease control, with 95% confidence intervals. (A & B) Island fox *Urocyon littoralis* PVA: west and east subpopulations; (C & D) red fox *Vulpes vulpes*; (E & F) African wild dog *Lycaon pictus* PVA without an Allee effect; (G & H) African wild dog PVA with an Allee effect included as a decrease in litter size as a function of group size. Solid error bars indicate distributions with ΔAIC ≤ 6. ▼ indicates the estimate from the previously published model, with the empirical litter size variance in the left panels and empirical litter size skew in the right panels (except G & H, for which there is no previous model estimate).

3.4 Discussion

Multiple distributions were shown to be consistent with the data for describing litter size frequencies for a range of carnivore species. However, the outcomes of demographic models appear robust to the choice of litter size distribution. These findings are discussed in light of the biological implications of litter size distribution choice and the applied importance of incorporating suitable probability distributions in demographic models.

3.4.1 Describing litter size variation

Unlike many biological parameters, offspring number is often underdispersed (Gallizzi *et al.* 2008, Mokkonen *et al.* 2011) and positively skewed (Shine & Greer 1991, Beja & Palma 2008). Litter size frequencies are best fitted by probability distributions able to describe the biological constraints on the upper limit of offspring production. While the Poisson distribution is most commonly used for fitting count data in general, it does not allow for underdispersion. In contrast, the generalised Poisson separates the variance from the mean (Kendall & Wittmann 2010), allowing greater flexibility, but at the cost of additional parameters. Of the continuous functions, the discretised normal distribution is the most flexible and is suitable for data characterised by low variance. Small sample sizes increase the uncertainty of the observed parameter estimates, and this uncertainty translates into the selection of multiple distributions (i.e. populations with large sample sizes had fewer distributions with $\Delta AIC \leq 6$).

In a recent model of vertebrate reproductive success, the zero-truncated generalised Poisson was consistently the best-fitting of several parametric distributions fitted to litter size (Kendall & Wittmann 2010). However, that study only included one carnivore population, the lion *Panthera leo*, which was fitted solely by the zero-truncated-binomial. In this study, that distribution performed less well, perhaps because more competitive functions were considered (including shifted discrete distributions and discretised continuous distributions) that were not assessed in the earlier study (Kendall & Wittmann 2010). The better fit of shifted forms over zero-truncation, possibly arising because removing zeros is not a random process and changes the

shape of the distribution, suggests that further work is needed to determine whether there is an underlying probabilistic mechanism in the distribution of litter size.

The lack of evidence for intraspecific variation in underlying litter size distributions for the example presented in this study could indicate that biological limitations on reproduction allow for little variation in this trait within a species. The known biases associated with litter size determination methodologies for red foxes (Allen 1983, Elmeros *et al.* 2003), probably explain the observed differences in litter size distributions, although the results of the management scenarios analysed in this study (see next section) suggest that this finding is unlikely to be of consequence for future modelling efforts. There were insufficient data to allow for comparisons of inter-annual variation in litter size distributions; therefore, given that the majority of datasets in this study were collated over multiple years, the results must be interpreted with caution in light of potential temporal variation.

These analyses assumed that individuals had the same underlying expected reproductive capacity. However, demographic heterogeneity in offspring production is influenced by many factors, including female age, body condition or social status (Woodroffe & Macdonald 1995, Iossa *et al.* 2008), as well as trade-offs between production and pre-weaning mortality (Sibly & Brown 2009), and maternal versus offspring selection pressure on lifetime reproductive success (Wilson *et al.* 2005). The methods in these analyses could be incorporated into population models that address such intrinsic individual variation, as well as those modelling environmental stochasticity.

3.4.2 Applied importance of litter size distributions

Despite inter-specific variability in the consistency of distributions to describe litter size data, it is shown here that model outcomes of applied management scenarios, e.g. extinction risk, may be robust to such variation in litter size. The lack of any apparent effect of litter size distribution choice in carnivore models might be because mammalian litter sizes are generally small due to physiological limitations. Underdispersion will promote sampling of offspring closer around the mean;

therefore, sampling variation will only weakly impact model outcomes. There are indications that the distribution choice could be of potential consequence in limited circumstances. In the case of African wild dog populations that exhibit a component Allee effect, the example presented here illustrates how modelling reproduction using an ill-fitting, underdispersed distribution can result in an overestimation of extinction risk (see Figure 3.3E-H).

Further work is required to determine the potential influence of temporal variation in the underlying litter size distribution on predictions of extinction risk. This is particularly important given that temporal or environmental variability means that combining data over time will inflate estimates of litter size variation, leading to erroneous predictions of extinction risk. In spite of these concerns, the lack of available data meant that pooling data was necessary for here; consequently, these results are indicative only of how mis-specified distributions could affect model predictions. As in Kendall & Wittmann (2010), it is stressed that determining appropriate distributions is a step towards a more mechanistic understanding of litter size variability that could provide insight into a species' response to selective pressures or management actions.

That litter size distributions have limited effects on the outcomes of management models may also reflect the relative contributions of life history traits to population growth. For long-lived species such as carnivores (Heppell *et al.* 2000), the elasticity of adult survival typically contributes more to population growth than fecundity. Indeed, variance in demographic parameters with low elasticities will have little effect on the variance of the population growth rate, due to the near linear relationship between population growth and vital rates (Caswell 2000). Notably, for all three canid populations in the models presented here, the elasticity of survivorship is as high or higher than fecundity (Chapter 4, Ginsberg & Woodroffe 1997, Kohlmann *et al.* 2005), which is consistent with the limited impact of litter size variation observed in the case studies.

3.5 Conclusion

Although this study focused on the Carnivora, these findings should apply to taxa with multiparous females, including other mammals, birds and lizards. While it is hard to determine the exact ecological and physiological mechanisms generating a litter size distribution, insight into the drivers of these empirical distributions could aid our understanding of the adaptation of reproductive strategies to extrinsic and intrinsic population pressures. Recent work demonstrating that female red foxes exhibit sex-biased investment in offspring as a function of body mass and population density suggests that altering litter size composition rather than litter size could be an alternative mechanism for increasing fitness (S. Harris & H. M. Whiteside, *unpublished data*). Ultimately however, applied models for carnivores appear to be robust to choice of litter size distribution, which has positive implications for modelling species with limited data.

Chapter 4 A review of the demography of global red fox, *Vulpes vulpes* populations

4.1 Introduction

Demographic modelling is widely used in conservation and management (Mills *et al.* 1999, Fieberg & Ellner 2001) but data availability frequently imposes significant limitations on modellers (Caro *et al.* 2005). Data are often patchily reported because they have been collected for purposes other than to derive demographic parameters (Baker *et al.* 2004, Imperio *et al.* 2010). Moreover, demographic parameters are often missing for a focal population, requiring modellers to rely on surrogate data from other populations of the same species (Pech *et al.* 1997, Peck *et al.* 2008), or even from similar species (Schtickzelle *et al.* 2005, Githiru *et al.* 2007). Whilst the consequences of these problems can be hard to determine, well-studied species are increasingly being used to gain insights into the consequences of demographic differences between species (Coulson *et al.* 2005) or populations (Nilsen *et al.* 2009, Johnson *et al.* 2010).

The insights gained from recent analyses of multiple populations within a species suggest a high degree of inter-population variability in demography. For example, Nilsen *et al.* (2009) showed population-specific demography of roe deer *Capreolus capreolus* resulting from distinct climatic conditions, predation and harvest levels, and Servanty *et al.* (2011) found variation along the fast-slow continuum among wild boar *Sus scrofa* populations facing different hunting pressure. Similarly, Johnson *et al.* (2010) demonstrated substantial differences in vital rate contributions between populations of Sierra Nevada bighorn sheep *Ovis canadensis sierra* in various phases of population growth. To date, these cross-population comparisons have focused on large herbivores and some bird species (Frederiksen *et al.* 2005, Tavecchia *et al.* 2008). Indeed, Nilsen *et al.* (2009) speculated that the high degree of intraspecific variation in life history speed that they observed in roe deer might be a characteristic of large herbivore dynamics. Here, the presence of similar patterns of intraspecific variability in a widely-studied carnivore is considered.

Red foxes *Vulpes vulpes* are the most widespread, extant, terrestrial mammal (Schipper *et al.* 2008) and are also a species of great economic, cultural, and disease importance (Baker *et al.* 2008). Hence, many years of sampling effort have been devoted to the red fox to gain insight into its life history for both management purposes (Smith & Harris 1991) and studies of sociality (Soulsbury *et al.* 2008a). Despite this intensive effort, successful management of foxes often remains difficult (Saunders *et al.* 2010) and demographic analyses of many fox populations are lacking. Recent deterministic models of red foxes have suggested that demographic traits, particularly age-specific contributions to population growth, are highly consistent across a sample of populations (McLeod & Saunders 2001). However, whether this pattern is robust to the method used to assess contributions to population growth, such as classical perturbation (Caswell 2001) or incorporating variation through life-stage simulation analyses (LSA) (Wisdom *et al.* 2000), is unknown. It is also unclear whether the apparent consistency of age-specific contributions to population growth translates into high consistency of life history speed, because there are only a few estimates of life history speed metrics for foxes (see Oli & Dobson 2003). Foxes are found across many habitats, from tundra to arid environments, and with rural and urban populations (Pils & Martin 1978, Harris & Smith 1987, Lindström 1989, Saunders *et al.* 2002). Given this diversity, with evidence of within population inter-annual variation of body mass and reproductive strategies (Soulsbury *et al.* 2008b, S. Harris & H. M. Whiteside *unpublished data*) and the potential sensitivity of life history rates to anthropogenic pressure (Lloyd *et al.* 1976), differing demographic tactics may be expected between populations.

Here, a comprehensive review of published studies of red fox demography is presented. With 70 years of published studies, collating these extensive data for the first time provides a unique resource for assessing the worldwide variability in the demography of this common and often intensively-managed species. The collated data are used to construct matrix projection models to determine basic demographic descriptors. Given that the fox is a generalist occurring over a wide range of habitat conditions, harvest levels, and population densities, it is predicted that life history speeds of distinct populations of this carnivore will be highly variable, with a gradient

of fast to slow with increasing latitude (Ferguson & Larivière 2002). It is expected that the importance of vital rates with low variation will appear greater when using traditional perturbation analyses than when using LSA, because the latter incorporates observed parameter variability. It is also predicted that as foxes are highly adaptable, modelled population growth rates will be sensitive to substituting the most variable life history rates between fox populations. It is shown that data for relatively few fox populations are adequate for detailed demographic analyses. However, those examined suggest important population-level differences in fox life history, with implications for erroneous management prescriptions when using surrogate data.

4.2 Methods

4.2.1 Data collection, fox life cycle, and matrix element calculation

Life history data from 57 fox populations were collated, totalling 96 papers published since the 1940s. Searches were conducted in Web of Science (http://webofknowledge.com, July 2010) using combinations of the search terms "red fox" OR "*Vulpes vulpes*" AND "demography", "population ecology" OR "life history". Demographic rates from these papers are summarised and, as a measure of data quality, study attributes including sample size, duration, size of study area, and data type were recorded (see Appendix 1). Methods of determining age, litter size and proportion of barren females were classified as well -, adequately-, or poorly-defined (see Appendix 2). This classification included, for example, how post-implantation loss was classified in the description of barren females, or if full descriptions of ageing methods were provided.

From this data review, sufficient age-specific vital rates were obtained for eight populations (studies 1, 3, 26, 27, 38, 41, 51 and 54 in Appendices 1 and 2). To select populations for demographic modelling, only data from study populations were used for which all the required demographic data were available. This meant eliminating some populations where the age-specific data (e.g. litter size or probability of breeding) were incomplete. Only data were used from populations for which age or stage- (i.e. juvenile, adult) specific values were provided for all vital rates. Stage-specific vital rates were deemed acceptable because, typically, the most significant differences exist between juveniles and adults (Figure 4.1). Survival rates were based on standing age distributions; most studies only reported an overall mean number of individuals in each age class, which were used to infer survival estimates. This approach was necessary because most studies were of less than 5 years duration and estimating inter-annual variation from short time periods is unreliable.

The data described above were used to construct density-independent, time-invariant, age-classified matrix models (Caswell 2001). Age-specific models are appropriate for modelling fox population dynamics because attributes such as litter size have been

shown to vary significantly with female age (Harris 1979, McIlroy *et al.* 2001). Populations were assumed to be stable in size (Englund 1970, Nelson & Chapman 1982, Harris & Smith 1987, Marlow *et al.* 2000, Saunders *et al.* 2002). The data had been collected predominantly from hunting returns, reported as standing age distributions, with survival determined from the age frequencies, f_x, for age class x (Caughley 1977, p. 91). As it is unusual for individuals to survive past four years (Pils & Martin 1978, Harris & Smith 1987) four age classes were used in the matrix, $\mathbf{A}_t$, (eqn. 1), where juveniles are age class 0+, and adults are age classes, 1+, 2+ and ≥3 respectively.

$$\boldsymbol{A}_t = \begin{bmatrix} F_1 & F_2 & F_3 & F_{4*} \\ P_1 & 0 & 0 & 0 \\ 0 & P_2 & 0 & 0 \\ 0 & 0 & P_3 & P_{4*} \end{bmatrix}. \qquad (1)$$

Age-specific matrix elements for survival were calculated as (Caswell 2001):

$$P_x = \frac{f_{x+1}}{f_x}, \qquad (2)$$

where P_x is the probability of survival from t to t+l of females in class x. To avoid issues of small sample size in the older classes, and to account for any individuals older than four, a composite final age class was created for all age classes beyond three (≥3). Survival (P_{4*}) was calculated for this age class by $P_{x*} = f_{x>x*} / (f_x + f_{x>x*})$, where x* is the final age class.

Productivity m_x, the expected number of female births per female of age class x, was calculated as:

$$m_x = M_x B_x SR, \qquad (3)$$

where M_x is the proportion of pregnant females, B_x is mean litter size and SR is the sex ratio (Caughley 1977, p. 82). Based on empirical evidence (Vos & Wenzel 2001), a 1:1

birth sex ratio was assumed. Females are able to mate when they are about 10 months old and produce one litter per year thereafter (Englund 1970). Consequently, a post-breeding "birth-pulse" model (Caswell 2001) was formulated. Age-specific matrix elements for fecundity were calculated as:

$$F_x = P_x m_x, \quad (4)$$

where F_x is the expected number of female offspring at time *t+1* per female in class *x* at *t.*

4.2.2 Fast-slow continuum

Life-history 'speed' is determined by how a species resolves the evolutionary trade-off between reproduction and survival, in response to extrinsic mortality and environmental stochasticity (Bielby *et al.* 2007). Oli and Dobson (2003) proposed the ratio of fertility rate to age at first reproduction (F/α) (i.e. the level of reproduction in relation to the onset of reproduction) as a measure of a mammalian species' position on the fast-slow continuum: "fast" species were deemed to have an F/α ratio of > 0.6, whilst "slow" species have an F/α ratio of < 0.15; those in between are considered "medium". Gaillard *et al.* (2005) used generation time as a proxy to determine life-history speed in mammals; fast species typically have a generation time of under two years. Both metrics were used to examine inter-population variation in life history speed of red foxes.

The mean weighted fertility rate was calculated as in Oli and Dobson (2003):

$$F = \frac{\sum_{x=\alpha}^{\omega} w_x F_x}{\sum_{x=\alpha}^{\omega} w_x}, \quad (5)$$

where age at first reproduction, α = 1, age at last reproduction, ω = 4 (consistent with the matrix, eqn. 1), and *w* is the stable age distribution determined from the projection model. Generation time, T_b, was determined according to Gaillard *et al.* (2005):

$$T_b = \sum_x x l_x m_x \lambda^{-x}, \quad (6)$$

where l_x is the proportion of individuals that survive from birth to age x. To calculate confidence intervals for the F/α ratio and T_b, the approach described below was used to conduct resampling for 10,000 matrix replicates.

4.2.3 Perturbation analyses

Perturbation analyses provide a ranking of the relative importance of demographic rates, in the context of their effects on the population growth rate (λ) (Caswell 2001). To decompose contributions to λ by life stage elasticity values (e_{ij}) of λ to the matrix entry a_{ij} (Caswell 2001) were calculated:

$$e_{ij} = \frac{a_{ij}}{\lambda} \frac{\delta\lambda}{\delta a_{ij}}. \quad (7)$$

Traditional perturbation methods do not account for variability and uncertainty in vital rates, potentially masking the true importance of life stages (Mills *et al.* 1999). High uncertainty in vital rate estimation stems from inherent spatiotemporal variation, as well as inevitable sampling and measurement error (Wisdom *et al.* 2000). LSA includes uncertainty in the effects of variance on population growth. Classical elasticity analyses examine the effects of varying vital rates independently about point estimates of their values; in LSA, by contrast, vital rates are varied simultaneously, taking into account interactions in uncertainty in the values of each.

Following previous studies (Wisdom *et al.* 2000) LSA was performed by constructing 10,000 stochastic matrix replicates, using vital rates drawn from appropriate probability distributions. Specifically, best estimates of age-specific survival were derived from standing age distributions using a likelihood approach, assuming that uncertainty around these estimates was beta-distributed (see Figure 2.2, chapter 2). Similarly, the proportion of breeding females of each age-class and age-specific litter sizes were drawn, respectively, from beta and shifted Poisson distributions (chapter 3). Matrix replicates were constructed by resampling from these distributions (Fieberg &

Ellner 2001). To determine the degree of variation in λ explained by each parameter (coefficient of determination, r^2) , λ was regressed against each individual transition element (Wisdom *et al.* 2000). From the matrix replicates, 95% confidence intervals were generated for the mean stochastic estimates of λ for each population. To compare the inferences from the two perturbation methods, the variance of λ explained by each vital rate was determined (Horvitz *et al.* 1997). Following *Coulson et al.* (2005) the square of the elasticity $(e_{ij})^2$ was multiplied with the variance of a given age-specific matrix element $V(a_i)$:

$$\chi_{ij}^{ind} = V(a_{ij})(e_{ij})^2. \quad (8)$$

Using equation (8) the age-specific contributions of survival (χ_{ij}^{P}) and fecundity (χ_{ij}^{F}) to the variance in λ were determined. Hence, it was possible to compare the elasticity variance ratios ($\chi_{ij}^{P} / \chi_{ij}^{F}$) with age-specific ratios based on the contributions of survival r^2 to fecundity r^2 ($r_{P,x}/r_{F,x}$) to λ as determined by the LSA.

4.2.4 Estimating process error

To assess the relative contributions of process and sampling error to observed uncertainty in demographic rates Kendall's (1998) method was used. Only one population had sufficient data with which to apply this technique (Sweden (South), Table 4.1). Age distribution data for this population were available for six consecutive years, and the probability of breeding was available for four of those six years (Englund 1970, 1980). Kendall's method was applied to the survival and breeding probabilities. The contributions of sampling and process error to these vital rates can be estimated by assuming that a beta distribution describes between-year variation in the survival or breeding probability, with the number of survivors and breeders for a given year drawn randomly from the binomial distribution (Kendall 1998). For example, if the probability parameter of interest is π, then the likelihood that the long-term probability is $\bar{\pi}$ and variation in π among years is $\sigma^2(\pi)$, given the data in year *t*, is;

$$L_t(\bar{\pi}, \sigma^2(\pi)) = \binom{N_t}{m_t} \frac{B(m_t + a, N_t - m_t + b)}{B(a,b)}, \quad (9)$$

where N_t is the total number of "trials" (individuals) in year t, m_t is the number of successes (survivors or breeders), B is the beta function, and a and b are the parameters of the beta distribution derived from the mean and variance:

$$a = \overline{\pi}\left[\frac{\overline{\pi}(1-\overline{\pi})}{\sigma^2(\pi)} - 1\right] \quad (10)$$

and

$$b = (1-\overline{\pi})\left[\frac{\overline{\pi}(1-\overline{\pi})}{\sigma^2(\pi)} - 1\right]. \quad (11)$$

The total log-likelihood is the natural logarithm of equation (9) summed across all years of data. Maximum likelihood was then used to find the best parameter estimates for $\overline{\pi}$ and $\sigma^2(\pi)$, with the latter quantifying the variance due to process error.

The relative contributions to uncertainty in λ caused by process and sampling error were estimated as follows. First, to determine the contribution of process error alone, the survival and breeding probabilities for the matrix element replicates were sampled from beta distributions. For both survival or breeding probability, the parameters of the relevant beta distribution were denoted as the mean $\overline{\pi}$ and variance σ^2, both estimated as described above (i.e. with the sampling error removed). The LSA method was then used to determine λ from the matrix replicates. Next, to determine the combined contributions of process and sampling error, the LSA method was used as in the original model. Importantly, however, for each replicate matrix elements were drawn from the beta distributions of the sampling error associated with data from a randomly chosen year.

4.2.5 Data substitution

The consequence of substituting data between populations from the same country was illustrated with two urban UK populations (Bristol and London), one subjected to control measures and the other not, and two USA populations (Midwest and East),

both subject to hunting. Previously, data have been substituted between populations in Australian and the USA (e.g. Pech *et al.* 1997). Consequently, the implications of this intercontinental substitution were also examined. For each case study, matrix components of survival, fecundity, probability of breeding, and litter size were sequentially replaced from one population to another: Bristol data was substituted for the London population, USA (Midwest population) data for the USA (East) population and USA (Midwest population) data for the hunted Australia (Hunted) population. The last example illustrates an alternative approach for data substitution, by using vital rates averaged from all eight populations to substitute into the Australia (Hunted) population. Using the above methods, 95% confidence intervals were generated for the resultant mean stochastic λ estimates for each simulation. All analyses were conducted using R 2.12.0 (R Development Core Team 2010).

4.3 Results

4.3.1 Data review

The review of 57 published demographic studies is summarised in Appendices 1 and 2. This review exposes some significant weaknesses, both in the extent of data coverage and in inconsistent data presentation. For example, 23 of the studies reviewed gave average litter size, but only nine gave age-specific litter sizes (Appendix 2). Whilst age-specific survival was available for 22 populations (Appendix 2), 14 were from populations without corresponding survival rates, restricting demographic modelling to just eight studies (Tables 4.1 and 4.2). In terms of data quality, 31%, 29% and 61% of studies did not adequately define ageing, litter size and probability of breeding, respectively (Appendix 2); in general, these studies gave insufficient details of methodology and definitions. Also, 29% of studies included no details of study attributes such as study area (Appendix 1). Of the eight populations used for the matrix models, none had been studied for more than ten years' duration and age-specific demographic data from all but the Australian populations were collected between the 1960s and mid-1980s (Table 4.1).

Age-specific productivity (m_x) is more variable than survival (P_x) (Figure 4.1). The two parameters show similar patterns with age, with both parameters peaking in young adults (Figure 4.1). Study attributes and vital rates for the eight populations used for analyses are presented in Tables 1 and 2. Coefficients of variation show that fecundity was more variable than survival (mean $CV_F = 0.15$; $CV_S = 0.10$, Table 4.3). These eight populations show a similar relationship to that seen in Figure 4.1 (Table 4.3), with a positive correlation between fecundity and survival in the older age classes (strongest in age ≥ 3 ($r^2 = 0.64$, $p = 0.01$), (Figure 4.2), suggesting that local conditions, rather than trade-offs between recruitment and survival, determine life history properties in foxes.

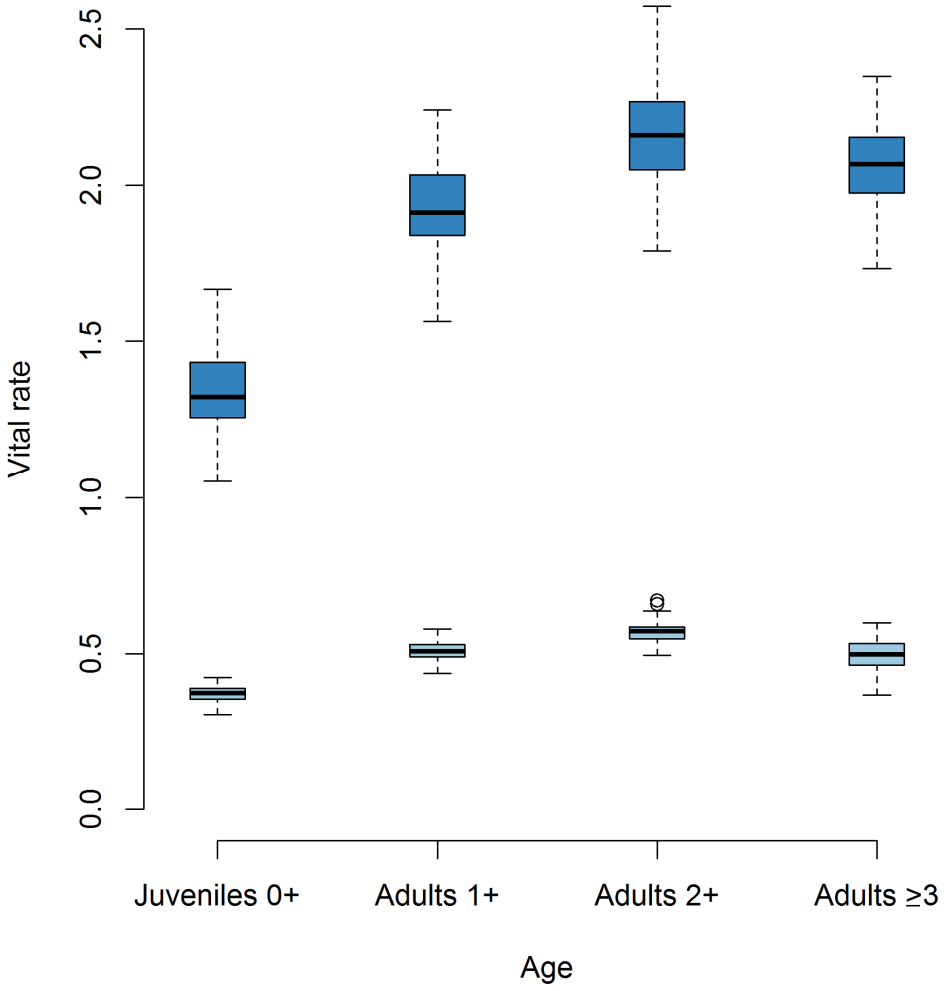

Figure 4.1. Survival (P_x, light blue boxes) and productivity (m_x, dark blue boxes) for global fox populations showing variation and age-specific patterns. Boxes show the sample median, minimum and maximum. Error bars indicate the lower and upper quartiles. Sample sizes of the number of studies used to determine rates are: juveniles 0+ (P_x n =22; m_x n=9); adults 1+ (P_x n=22; m_x n=9); adults 2+ (P_x n=21; m_x n=8); adults ≥3 (P_x n=20; m_x n=8).

Table 4.1. Summary of mean survival rates, P_x, ± standard errors and population attributes for eight fox populations.

	Australia (hunted)	***Australia (non-hunted)***	***UK (Bristol)***	***UK (London)***	***Sweden (North)***	***Sweden (South)***	***USA (Midwest)***	***USA (East)***
P_1	0.30 ± 0.02	0.39 ± 0.07	0.48 ± 0.02	0.42 ± 0.02	0.33 ± 0.02	0.43 ± 0.03	0.33 ± 0.04	0.34 ± 0.05
P_2	0.35 ± 0.05	0.65 ± 0.12	0.54 ± 0.03	0.43 ± 0.03	0.71 ± 0.04	0.53 ± 0.04	0.40 ± 0.07	0.88 ± 0.06
P_3	0.57 ± 0.08	0.92 ± 0.07	0.53 ± 0.03	0.47 ± 0.05	0.50 ± 0.05	0.75 ± 0.05	0.95 ± 0.05	0.57 ± 0.09
P_4*	0.70 ± 0.06	0.18 ± 0.10	0.51 ± 0.03	0.49 ± 0.05	0.59 ± 0.04	0.55 ± 0.04	0.43 ± 0.08	0.53 ± 0.12
Sample size	538	99	1628	1110	1070	827	269	94
Study area (km^2)	200	200	8.9	1618	-	-	83.73	-
Habitat type	Rural	Rural	Urban	Urban	Rural	Rural	Rural	Rural
Study Years	1992; 1994-97	1992	1977-85	1971-77	1966-70	1966-70	1971-75	1976-79
Major source of mortality data	Mixed	Baited	Roadkill	Mixed, shot	Shot	Shot	Mixed	Trapped
Aging method	CA	CA	CA	CA	TE, CA	TE, CA	CA	CA, EW,TE,SM
Level of control**	Intense	No	No	Light/ Average	Light	Intense	Average	Average
Individual density/km^2	-	0.46–0.52	29.5	-	-	-	-	-
Invasive	Yes	Yes	No	No	No	No	No	No
Latitude	-32	-24	51	51	63	59	44	38
References	1	2	3	3	4	4	5	6
Study # in Appendices 1 & 2	51	54	3	1	26	27	38	41

[1]Saunders et al 2002; [2] Marlow *et al* 2000; [3]Harris and Smith 1987: [4]Englund 1980; [5]Pils and Martin 1978; [6]Nelson and Chapman 1982. CA: cementum annuli (of molars or canines); TE: tibia epiphysis closure; EW: eye lens weight; SM: skull measurements; Mixed: Combination of shooting, trapping, gassing, baiting and battues. * see text for explanation. ** determined according to juvenile age ratios (Appendix 2), where an increasing juvenile to adult age ratio is an indication of increasing control (Harris 1977) and if possible, by information provided by each study on the presence or level of hunting.

Table 4.2. Summary of mean fecundity rates, F_x, for eight fox populations.

	Australia (hunted)	***Australia (non-hunted)***	***UK (Bristol)***	***UK (London)***	***Sweden (North)***	***Sweden (South)***	***USA (Midwest)***	***USA (East)***
F_1	0.37	0.686	0.55	0.72	0.29	0.30	0.58	0.40
F_2	0.61	1.271	0.77	1.00	0.79	0.72	0.96	1.46
F_3	1.21	1.426	0.71	1.09	0.79	1.35	2.88	0.89
F_{4*}	1.58	0.332	0.74	0.89	0.83	0.92	0.97	0.81
Sample size	291	47	252	384	161	217	367	94
Method to determine litter size	EM; EM, PS	PS (excluded faded scars)	PS (grade 5 -6)†	PS (grade 5-6)	EM; PS (grade5-6)	EM; PS (grade5-6)	PS (dark), EM	PS
Method to determine barren females	-	PS (excluded faded scars)	FL, FO, FI, LE	NVP	NVP, PPIL	NVP, PPIL	-	NVP
References	1,2	3	4	5	6	6	7	8
Study # in Appendices 1 & 2	51	54	3	1	26	27	38	41

[1]Saunders et al 2002; [2]McIlroy et al 2001; [3]Marlow *et al* 2000; [4]Harris and Smith 1987: [5]Harris 1979; [6]Englund 1980, [7]Pils and Martin 1978; [8]Nelson and Chapman 1982; PS: placental scars; EM: number of embryos; DC: den counts; FL: failure to produce litter; FO: failure to ovulate; FI: failure to implant; LE: lost entire embryos; NVP: no visible signs of pregnancy; PPIL: pre and post implantation loss; - method not given. * see text for explanation. † Placental scar grades refer to the level of fading, with dark scars (5-6) being the most reliable (see Lindström 1981).

Table 4.3. Coefficients of variation for age-specific survival (P_x) and fecundity (F_x) across matrix replicates for eight fox populations (study number refers to study population in Appendices 1 and 2).

		Survival				***Fecundity***			
Study #	***Population***	P_1	P_2	P_3	P_{4*}	F_1	F_2	F_3	F_{4*}
51	Australia (Hunted)	0.08	0.13	0.14	0.08	0.10	0.15	0.18	0.10
54	Australia (Non-hunted)	0.17	0.16	0.10	0.42	0.21	0.21	0.21	0.56
3	UK (Bristol)	0.04	0.05	0.06	0.07	0.07	0.09	0.13	0.12
1	UK (London)	0.05	0.07	0.10	0.10	0.06	0.09	0.12	0.12
26	Sweden (North)	0.02	0.03	0.04	0.03	0.03	0.04	0.05	0.05
27	Sweden (South)	0.06	0.05	0.08	0.06	0.11	0.11	0.11	0.11
38	USA (Midwest): Wisconsin	0.06	0.07	0.06	0.07	0.11	0.11	0.10	0.11
41	USA (East): Maryland	0.11	0.17	0.06	0.18	0.20	0.21	0.16	0.26

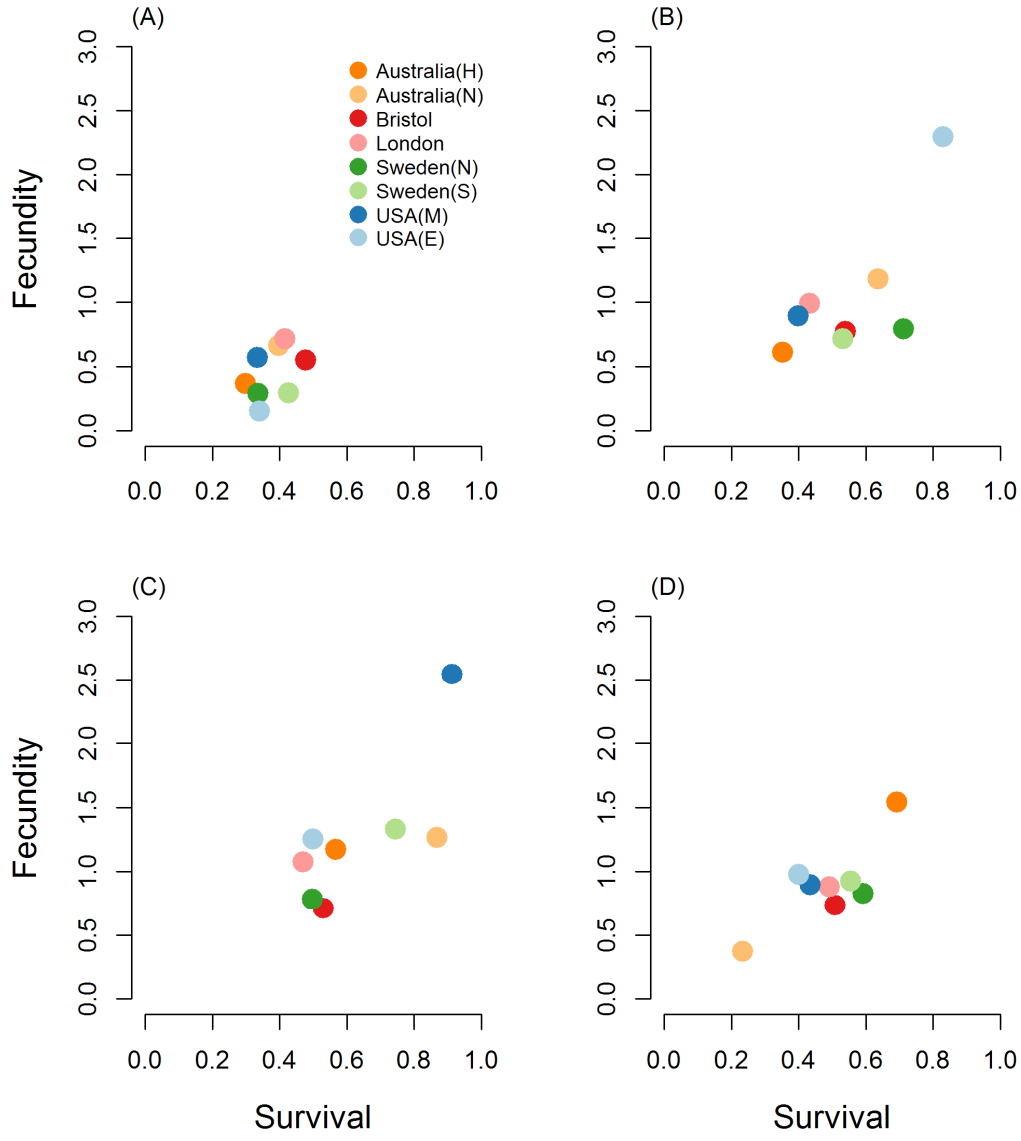

Figure 4.2.Correlation between mean matrix replicates for survival and fecundity for eight fox populations. (A) Juveniles 0+ (r^2 = 0.20, p = 0.23); (B) Adults 1+ (r^2 = 0.51, p = 0.03); (C) Adults 2+ (r^2 = 0.56, p= 0.02); (D) Adults ≥3 (r^2 = 0.64, p = 0.01).

4.3.2 Life history speed

Relative to many other carnivores, red foxes mature early, are fairly short-lived and, as is typical of canids, have larger than average litter sizes; consequently, theory predicts that they should fall towards the fast end of the spectrum (Heppell *et al.* 2000). In fact these analyses show wide variation in the speed of fox populations, from medium to fast species according to the F/α ratio, and slow to fast species according to generation time (Figure 4.3). There is large variation in speed within these classifications; the metrics increased by factors of 3.5 (generation time) and 1.5 (F/α ratio) between the slowest fox population of north Sweden (F/α = 0.53, T_b = 3.13), and the fastest population, London (F/α = 0.81, T_b = 0.90). The Australian hunted population (Australia (Hunted)) has a faster life history than would be expected from its population growth (Figure 4.3). The F/α ratio is positively correlated with λ (r = 0.83, p = 0.01) (Figure 4.3A), and generation time (T_b) is negatively correlated with λ (r= -0.86, p = 0.01) (Figure 4.3B). Unsurprisingly, given that they are determined by the same life-history rates, there is a negative correlation between the F/α ratio and T_b (r = -0.79, p = 0.03) (Figure 4.3C). No correlation was found between life history speed (F/α ratio) and latitude (r = -0.34, p = 0.38). These results suggest that local conditions play a significant role in determining life history rates; for example, good conditions give rise to both high survival and high fecundity, resulting in higher population growth and faster speed.

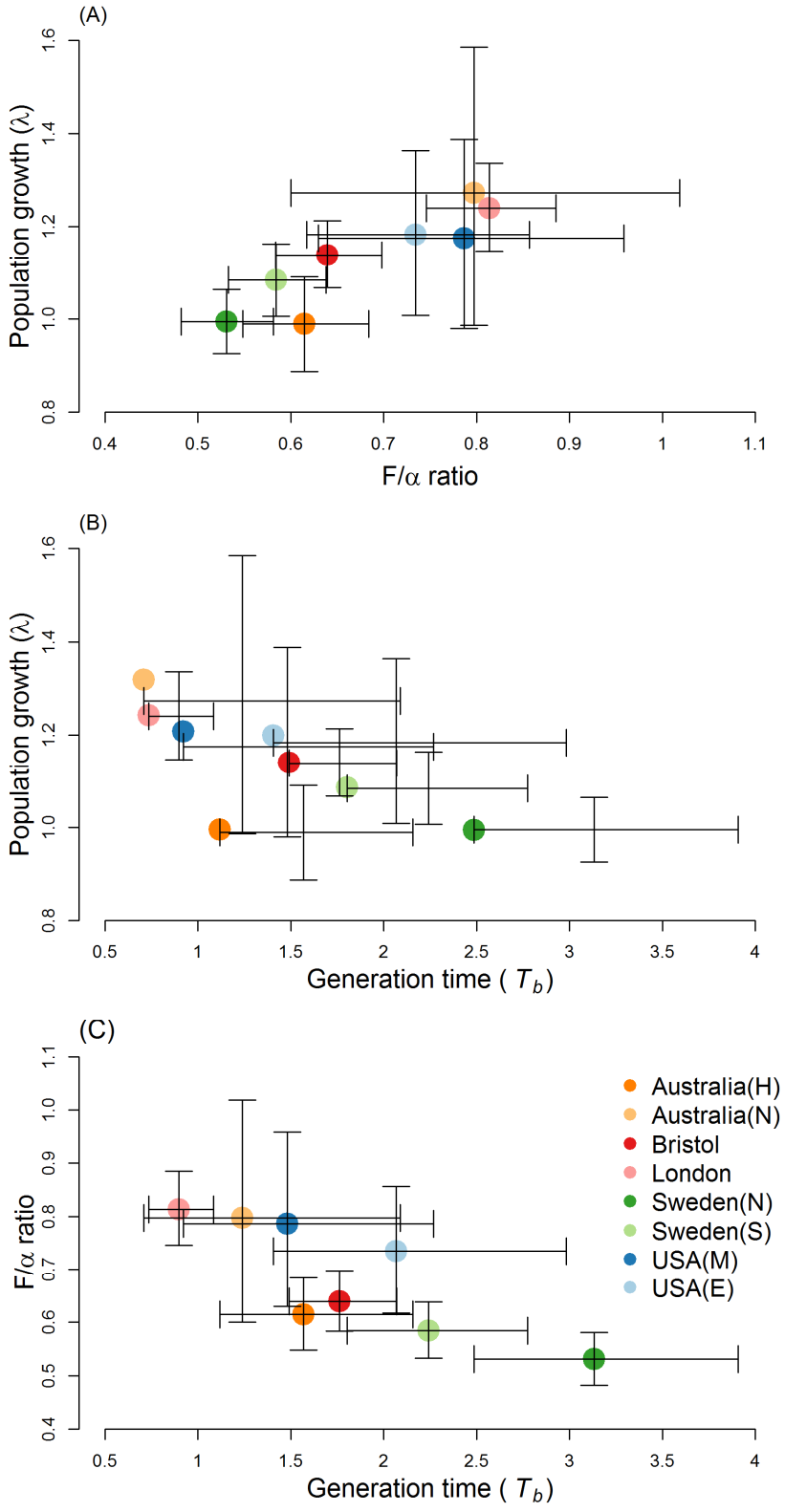

Figure 4.3. The variation in life history metrics and population growth rate between fox populations, and the relationships between these measures, showing 95% confidence intervals. (A) Positive correlation between F/α ratio and population growth rate (λ); and negative correlations between (B) generation time (T_b) and λ; (C) F/α ratio and T_b.

4.3.3 Contribution of vital rates

Life-history theory suggests that relatively early-maturing mammals, such as the fox, should have higher elasticity of fecundity than survival (Heppell *et al.* 2000). Elasticity analysis and LSA reveal two main points: that the youngest age class makes the largest contribution to λ, and that, generally, fecundity is as important as survival (Table 4.4). Despite these patterns, both elasticity and LSA results reveal there is a great deal of inter-population variation in the contribution that vital rates make to λ. For example, there is a threefold difference in fecundity elasticity of the youngest age class (London $e_{F,1}$ = 0.35; Sweden (South) $e_{F,1}$ = 0.10). Life history theory predicts higher sensitivity of λ to fecundity in fast species, to survival in slow species (Heppell *et al.* 2000), and more evenly balanced sensitivity to both parameters in medium species (Oli 2004). Therefore it is expected that, as recruitment drives fast populations, the sensitivity of λ to fecundity should increase as populations get faster (Oli & Dobson 2003). Age-specific variance ratios ($V_{S,x}/V_{F,x}$) show a tendency to decrease across all age classes (strongest in juveniles 0+, r =- 0.75, p = 0.003) with increasing speed (Figure 4.4A), suggesting that fecundity contributions become more important in faster populations. LSA ratios ($r_{P,x}/r_{F,x}$) did not show a significant relationship (strongest in adults 2+, r =- 0.64, p = 0.09) with speed (Figure 4.4B). Evaluating these two ratios (χ_{ij}^{P} / χ_{ij}^{F} and $r_{P,x}/r_{F,x}$) highlights the importance of including variation when estimating the relative contributions of vital rates. When the reduced variability of survival is taken into account, the contribution of survival in slower populations is reduced (Figure 4.4). While it is possible that this reduced variability stems from errors in sampling rather than intrinsic variation, these results are consistent with the prediction of higher variability in the fecundity of this species.

Table 4.4. Age-specific elasticities and coefficients of determination of the LSA for eight fox populations. Elasticities and r^2 are the mean values calculated across all replicates (study number refers to study population in Appendices 1 and 2).

Population	***Elasticity of survival ($e_{P,x}$) and fecundity ($e_{F,x}$)***								***LSA survival r^2 ($r_{P,x}$) and fecundity r^2 ($r_{F,x}$)***							
	$e_{P,1}$	$e_{P,2}$	$e_{P,3}$	$e_{P,4*}$	$e_{F,1}$	$e_{F,2}$	$e_{F,3}$	$e_{F,4*}$	$r_{P,1}$	$r_{P,2}$	$r_{P,3}$	$r_{P,4*}$	$r_{F,1}$	$r_{F,2}$	$r_{F,3}$	$r_{F,4*}$
Australia (Hunted)	0.20	0.14	0.10	0.24	0.12	0.06	0.04	0.10	0.14	0.15	0.08	0.15	0.13	0.14	0.07	0.13
Australia (Non-hunted)	0.28	0.11	0.02	0.01	0.30	0.17	0.09	0.02	0.38	0.08	0.01	0.01	0.41	0.10	0.01	0.01
UK (Bristol)	0.27	0.12	0.06	0.05	0.25	0.15	0.06	0.06	0.23	0.10	0.04	0.03	0.32	0.17	0.07	0.05
UK (London)	0.25	0.09	0.03	0.02	0.35	0.16	0.06	0.03	0.30	0.12	0.03	0.01	0.35	0.14	0.04	0.01
Sweden (North)	0.27	0.12	0.05	0.04	0.25	0.15	0.07	0.05	0.28	0.12	0.04	0.03	0.30	0.14	0.05	0.03
Sweden (South)	0.26	0.16	0.09	0.13	0.11	0.10	0.07	0.09	0.23	0.07	0.09	0.10	0.20	0.11	0.09	0.11
USA (Midwest)	0.27	0.17	0.09	0.09	0.10	0.10	0.09	0.09	0.21	0.17	0.06	0.07	0.18	0.17	0.07	0.08
USA (East)	0.26	0.15	0.05	0.03	0.25	0.11	0.11	0.05	0.26	0.15	0.01	0.02	0.35	0.15	0.03	0.02

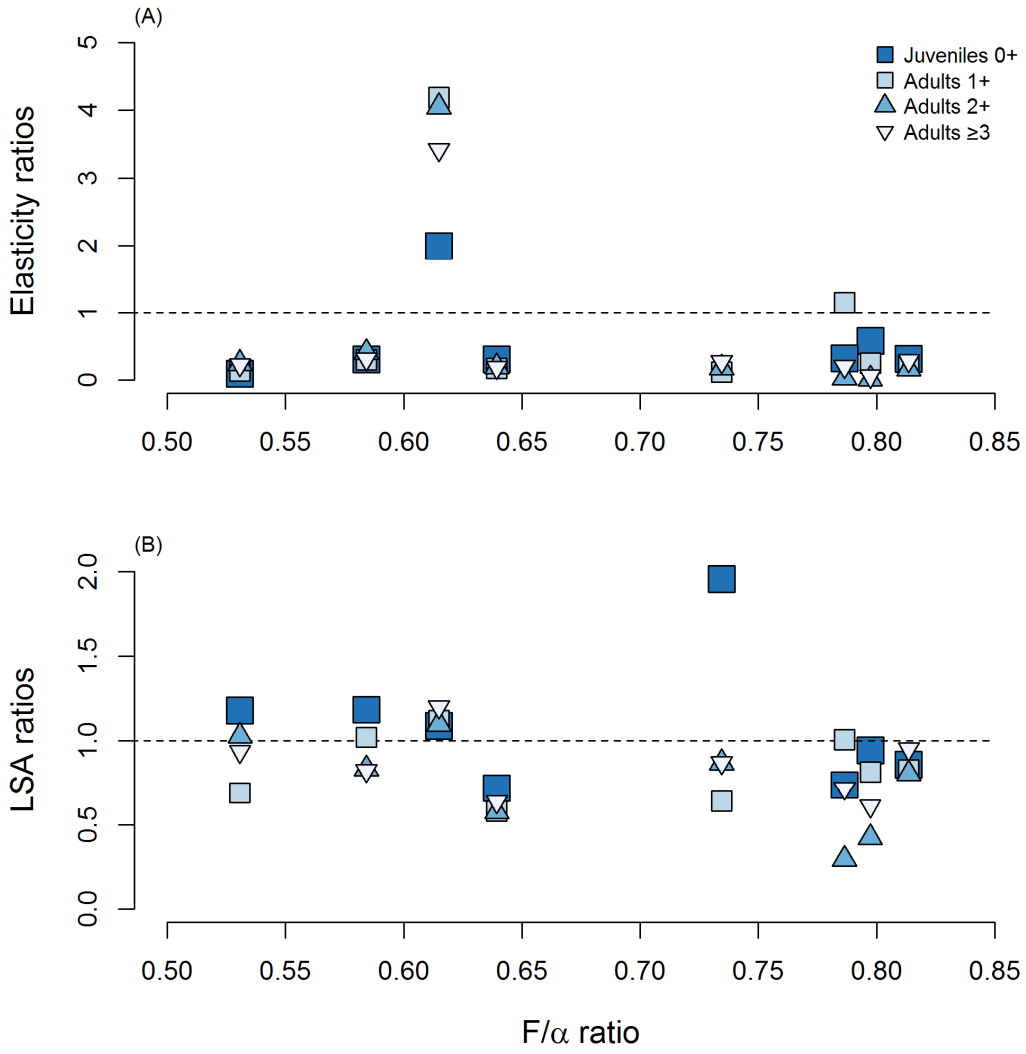

Figure 4.4. Relationship of (A) age-specific variance decomposition ratios ($\chi_{ij}^{P} / \chi_{ij}^{F}$) and (B) life-stage simulation analysis ratios ($r_{P,x}/r_{F,x}$) against the life history speed metric, F/α ratio , for eight for populations, showing the change in contributions with the inclusion of uncertainty.

4.3.4 Process error: an example using a Swedish fox population

The relative contributions of sampling and process error to observed uncertainty in vital rates were determined using Kendall's (1998) method. Sufficient data were available for one study population, the Sweden (South) population. There is good agreement between the mean λ estimates for the Sweden (South) population for all of the three methods used to account for uncertainty in vital rates (Figure 4.5). As expected, the uncertainty in λ is largest when both sources of variance are included (Figure 4.5). Process error and sampling error contributed similar uncertainty to the estimates of λ.

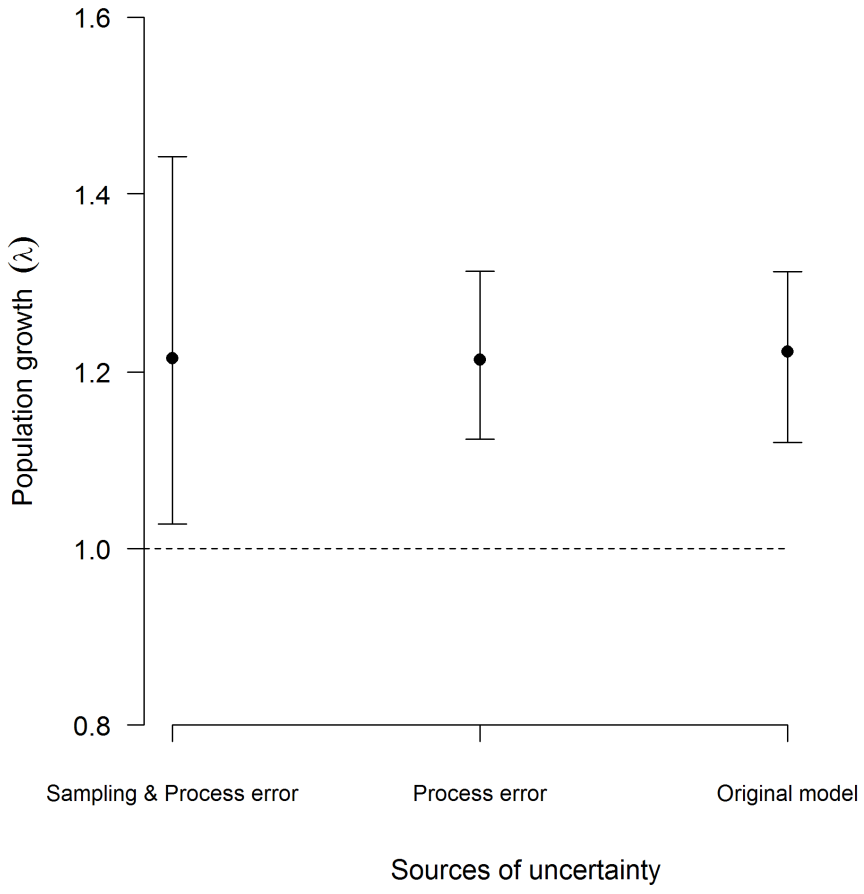

Figure 4.5. Population growth rates for the Sweden (South) population with both process and sampling variance included, sampling error removed, and the estimate from the original model. Error bars are 95% confidence intervals determined from the matrix replicates.

4.3.5 Case studies of data substitution

The importance of accounting for inter-population variation in life history is highlighted by the substitution of vital rate parameters between fox populations; using surrogate data substantially changes the resultant population growth rate estimates (Figure 4.6). The results are particularly striking when substituting Bristol data in the London population, even though both samples come from the same habitat in the same country; surrogate fecundity produces a 23% decrease in λ, whereas substituting survival data increases the λ estimate by 21% (Figure 4.6A). A 23% decrease in λ occurs when only probability of breeding is used, but only a 1% increase in λ when replacing litter size, highlighting that the percentage of breeding females is lower in Bristol, whereas there is no significant difference in litter size between these populations (Harris & Smith 1987). In the USA (Midwest) population breeding probability is higher and more variable than litter size, compared to the USA (East) population. Although the levels of uncertainty in λ are high, differences in mean λ estimates range from a 15% increase with the probability of breeding, to only a 3% decline when litter size is replaced (Figure 4.6B). Many of the age-specific survival and fecundity rates are similar in the Australia (Hunted) and USA (Midwest) populations, leading to smaller differences resulting from data substitution. However, replacing fecundity data produces a 13% increase in λ, and substituting litter size increases λ by 20% (Figure 4.6C), highlighting the dependency of the model outcome on the chosen surrogate parameter. Figure 4.6D illustrates that the population growth rate estimates using the parameter range from the eight populations are closer to the Australia (Hunted) λ estimate than when using surrogate data from just one population, with the exception of when replacing survival data. Noticeably, the Australia (Hunted) population is the only population where survival elasticity was consistently greater than fecundity (Table 4.4), indicating that this population is sensitive to changes in survival rates.

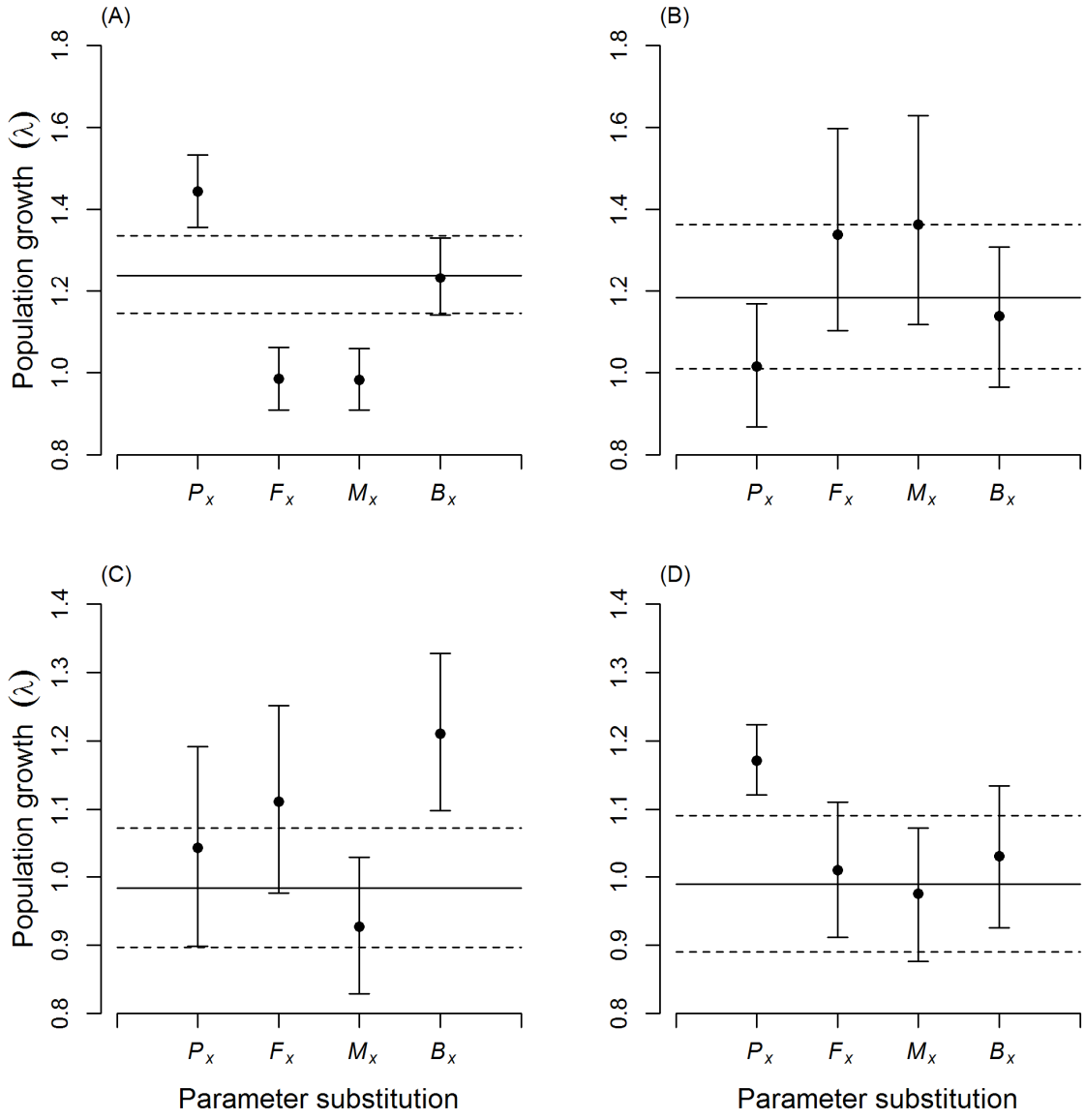

Figure 4.6. Effects of substituting matrix elements and fecundity components on the population growth rate between two urban, and two hunted fox populations, with 95% confidence intervals. (A) London population substituted with the Bristol population vital rates; (B) USA (East) population substituted with the USA (Midwest) population vital rates; (C) Australia (Hunted) population substituted with the USA (Midwest) population vital rates; (D) Australia (Hunted) population substituted with vital rates averaged from all eight populations. The solid line indicates the population growth rate with no data substitution, and the dashed lines indicate the 95 % confidence intervals of this estimate. P_x = survival; F_x = fecundity; M_x = probability of breeding; B_x= litter size.

4.4 Discussion

These analyses highlight the large sampling effort expended on the red fox but, with only eight of 57 studies providing sufficient data for age-specific demographic modelling, also identify shortcomings in current knowledge about interpopulation variability in demography. Recruitment in red fox populations appears to be consistently more variable than, but correlated with, survival across age-classes and populations. Population growth rates were sensitive to changes in both survival and fecundity. These analyses showed large intraspecific variation in demography, in both life history speed and the contribution of vital rates to λ. These results are indicative of the potential role of environmental conditions for determining life history rather than trade-offs between recruitment and survival. Variation in demographic rates between populations illustrated the consequences of data substitution between populations. Inferences gained from population models are likely to be highly sensitive to the practice of data substitution, and this will vary with the vital rate replaced. The outcomes of this study are discussed in the context of four broad issues: emerging recognition of the variation in life history among populations within a species; perturbation analyses and their implications for management; data substitution in demographic modelling; and recommendations for ongoing studies of demography in red foxes and similar species.

4.4.1 Inter-population variation in life history speed

The determination of life-history speed along the fast-slow continuum has been much debated (Oli 2004, Gaillard *et al.* 2005, Bielby *et al.* 2007). Intraspecific studies have used both generation time (Nilsen *et al.* 2009) and the F/α ratio (Bieber & Ruf 2005). It was found that both metrics correlated with λ, suggesting that as Oli and Dobson (2005) found, both are at least partially indicative of a fox population's current trajectory. The calculation of confidence intervals for the most commonly used metrics of the fast-slow continuum was illustrated, and it is suggested that the use of confidence intervals should be routine before making inferences about the extent to which populations differ in life history speed.

Phylogeny and body mass typically account for much of the variation in life history variables (Gaillard *et al.* 2005) and, consequently, within-species variation in demographic tactics is generally expected to be limited. A practical application of defining a population's position on the fast-slow continuum is to provide a measure of the population's response to perturbations and adaptability to the local environment. This 'interpopulation' approach (Nilsen *et al.* 2009) merits further attention for comparing population responses to specific pressures and exploring evidence of trade-offs between recruitment and survival. Recent comparisons show that roe deer do not exhibit this trade-off, slowing down their life history in harsher environments because they cannot increase reproduction when faced with increased mortality in adverse conditions (Nilsen *et al.* 2009). In wild boar, by contrast, the contribution of life history tactics shifted from juvenile to adult survival as conditions changed from poor to good (Bieber & Ruf 2005). Similarly, Servanty *et al.* (2011) found that wild boar increased life history speed by increasing fecundity when facing higher hunting pressure. Tasmanian devils *Sarcophilus harrisii* show increased reproduction in young age classes as a response to disease mortality (Jones *et al.* 2008b). Here, however, these results point towards substantial variation in fox life history speed; although the majority of fox populations that were modelled would be classified as 'fast' by either metric, two of the eight populations (both from Sweden) lay outside that category (one of them substantially). Compared to other hunted fox populations, the Australia (Hunted) population shows surprisingly low λ considering its short generation time. This suggests that is it unable to respond to the hunting pressure by increasing reproduction. However, at the time of data collection the population was experiencing a drought, which had a negative effect on reproduction (McIlroy *et al.* 2001), highlighting the conflicting response to anthropogenic versus climate pressures. Conversely, the faster speed of the London population compared to the non-hunted Bristol population suggests a possible compensatory response to hunting, although the lack of additional data on immigration and density hinders assigning causation to this variation. The population with the slowest life history (by both metrics) is the Sweden (North) population, probably reflecting the harsh winter conditions and food limitations that it experiences (Lindström 1989), although fluctuations in this

population's density may violate assumptions of a stable population size. Slower species are expected in habitats with low productivity but high environmental variation (Ferguson & Larivière 2002). In foxes, the relationship between the environment and life history rates is complex: environmental variability is an important determinant of lifetime productivity (Soulsbury *et al.* 2008b), and body condition, driven partly by climatic conditions, is an important factor affecting both survival (Gosselink *et al.* 2007) and fecundity (Cavallini 1996). Bartoń and Zalewski (2007) found fox density was negatively correlated with an index of seasonality within Eurasia, suggesting that such an index could also be used to explain variation in life history speed between populations. However, using latitude as a proxy for seasonality, no correlation was found in this study. Similarly, previous studies have failed to demonstrate a relationship between litter size and latitude (Lord 1960).

4.4.2 Vital rate contributions and life-history characteristics

That younger age classes are important to growth is unsurprising for a species with a relatively fast life history and is consistent with the observation that juveniles comprise an average of 60% of fox populations (Lloyd *et al.* 1976, Nelson & Chapman 1982, Marlow *et al.* 2000). Although juvenile foxes are particularly susceptible to anthropogenic control (Englund 1970, Pils & Martin 1978), heterogeneity in hunting effort generates source populations (Baker & Harris 2006) and, together with constant immigration from dispersers (Rushton *et al.* 2006, Gentle *et al.* 2007), helps to explain why some populations remain stable or grow despite hunting pressure. While compensatory responses in productivity are thought to occur in areas of high hunting pressure (Harris 1977, Cavallini 1996), these results provide little evidence for this for the populations analysed here (see previous section). Thus, as McLeod and Saunders (2001) conclude, targeting the youngest age class is likely to be the most effective form of management when the aim is to decrease the population.

Traits that have a large impact on λ are predicted to be buffered against variation (Pfister 1998), but demographic analyses of mammals are not always consistent with this theory (e.g. Creel *et al.* 2004, Henden *et al.* 2009). In these analyses, λ was equally sensitive to the contributions of fecundity and survival. Foxes are expected to have

higher contributions to λ from fecundity than survival, but it was found that fecundity is more variable than survival, possibly because fecundity is influenced more than survival by complex factors, which include food limitation, body mass, and social factors (Lindström 1988, Cavallini 1996, Iossa *et al.* 2008). However, when considering demographic contributions in the context of the fast-slow continuum, the equal sensitivity of λ to both rates corresponds to that expected with a medium speed. It was also found that the relative contribution of vital rates varied among populations, especially in the youngest age class, which drive growth. Changes in relative elasticities between demographic rates have been demonstrated as a response to environmental conditions (Bieber & Ruf 2005), with potential management implications if demographic traits are to be targeted based on data from fluctuating conditions. Given that variation is an important factor driving population dynamics, it is advantageous to incorporate as high a degree of realism as possible into models (Mills *et al.* 1999, Wisdom *et al.* 2000). Studies using multiple demographic analyses, such as those in this study, have illustrated how predicted life history contributions can differ with the inclusion of variation (Wisdom *et al.* 2000, Johnson *et al.* 2010); these results reinforce that conclusion.

4.4.3 Representativeness of process error example

Given that process error could only be separated for one population, this analysis raises the question of how representative the Sweden (South) population is of other fox populations. The Sweden (South) population most likely falls towards the higher end of the process error spectrum, coming from an area that is prone to environmental fluctuations, although not as extreme as experienced farther north in Sweden but there were less data available for this population. However, it is known to be subject to high inter-annual variation owing to regulation by prey cycles (Lindström 1989). As many fox populations are likely to experience less environmental variation, the process variation in these populations is expected to be less pronounced. However, these results should be interpreted with caution, given that Doak *et al.* (2005) suggest that studies of less than five years duration are inadequate to quantify

sources of variation, and that sample sizes for the Sweden (South) population were small in some years.

4.4.4 Validity of using substitute demographic parameters

The use of substitute data in demographic modelling is often necessary but requires great caution, even at the intraspecific level. Bristol and London foxes might be expected to share similar properties, being urban populations in relatively close proximity. However, at the time of data collection the London fox population was subject to hunting (Harris 1977), illustrating that geographical proximity of populations is no guarantee of the validity of this approach. Pech *et al.* (1997) used USA data for their model of an Australian population to test the impact on λ of reducing the fecundity of an invasive population. These results illustrate how replacing fecundity, and its component elements, could have led to flawed outcomes. In the case of foxes, recruitment is the most variable life history rate, so should be substituted with great caution. If in doubt, the most comprehensive approach might involve substituting data from across the range of available values, and acknowledging the resultant uncertainty.

Data substitution is often inevitable in situations concerning highly endangered, elusive, or data-deficient species, highlighting the need for long-term research. It occurs in many forms, such as using data from species of the same family (Finkelstein *et al.* 2010), species sharing similar attributes (Schtickzelle *et al.* 2005), or making assumptions about a parameter based on a different (Peck *et al.* 2008) or captive (Martinez-Abrain *et al.* 2011) population. Githiru *et al.* (2007) evaluated the applicability of substituting data from a common species, the white-starred robin *Pogonocichla stellate*, for a critically endangered thrush *Turdus helleri*; both species responded to habitat disturbance with higher fluctuating asymmetry and lower effective population density. The sensitivity of λ estimates to surrogate demographic parameters illustrated by the case studies suggests a finer scale approach is required compared to the broad measures of similarity applied in Githiru *et al.*'s (2007) approach. These results are in agreement with Caro *et al.* (2005) that surrogate data should be used only when similar traits can be identified; following Johnson *et al.*

(2010), caution is urged against substituting data between demographically distinct populations.

4.4.5 Data quality implications and recommendations

As the most widespread terrestrial mammal, the red fox has been subject to extensive study throughout its range. Despite the constraints on studying carnivores, data exist for an impressive number of red fox populations; however, for the amount of sampling effort, surprisingly few populations can be described by a matrix model with all necessary vital rates. Further, demographic data were biased towards collection during the 1970s. The quality of data is also restricted, in some published papers, by unclear methodologies, inconsistent definitions of key parameters, and issues related to basic study attributes. Sampling design is a direct source of bias for parameter estimation, but is often beyond the control of researchers due to funding and logistical limitations. However, it is important to take into account that sample size (Gross 2002), duration (Fieberg & Ellner 2001), and area (Steen & Haydon 2000) can have repercussions for the precision of demographic estimates.

The rarity with which quantifiable study attributes such as habitat, environmental, and anthropogenic variables were reported also limits analysis of the impact of these factors on inter-annual variability in population processes. Covariates, such as hunting effort and those that enable scaling from an urban to rural gradient (e.g. human or road density), are easy to measure and can be important predictors in more powerful models (Mladenoff *et al.* 1995). As with other studies (Wisdom *et al.* 2000, Nilsen *et al.* 2011), quantification of inter-annual variation in vital rates is possible for few of the fox populations studied. This is disappointing, given the importance of stochasticity for populations (Melbourne & Hastings 2008) and the advances in demographic modelling for incorporating variation (Kendall 1998, White 2000, Akçakaya 2002, Udevitz & Gogan 2012). In this regard, the studies in these analyses are limited both by their relatively short durations and by their sample sizes. Further, the seasonal variation that exists in trap capture rates between age and sex classes, which also mirrors the susceptibility to culling (Baker *et al.* 2001a), implies that important classes could be underrepresented at key times of years. These differences are due to behavioural

changes throughout the year, such as vixens being harder to catch when breeding. It is suggested (S. Harris, *pers. comm.*) that best practice for measuring inter-annual variation in key demographic rates is to sample during the dispersal period (October to December in the northern hemisphere). Whilst such samples may be skewed towards dispersing subadults, particularly males, they are the least biased samples in light of the behavioural processes occurring throughout the year. Specifically, samples during this period would show (i) how many cubs survive to independence (the ratio of cubs to adults); (ii) annual proportions of adult vixens that bred from placental scar counts; (iii) mean annual litter sizes (from placental scar counts); (iv) annual variations in both cub and adult sex ratios; and (v) annual variations in adult survival. Presenting data for this specific period separately would facilitate comparisons between populations. Currently, few studies make it clear how sampling effort varied through the year; biases in sampling effort skews samples towards the age and sex classes that were most vulnerable during the main collection period.

Most available data on red foxes are from mortality studies, which have associated assumptions (for a review see Caughley 1977). Ultimately, however, mortality data such as hunting bag returns will remain an important source of information for fox populations. Four particular issues arise when presenting the data from these studies, all of which should be straightforward to remedy. First, studies differ in their definition of age classes. Factors affecting uncertainty in ageing methods and their minimisation have been discussed extensively elsewhere (Allen 1974, Harris 1978). Whether the first year after birth is described as age class zero, or one, leads to confusion in interpreting published age-specific data, as does dividing the first year into shorter periods, such as pre-and post-weaning, or into 3-month segments, although there are biological and ecological arguments justifying this division (Marlow *et al.* 2000). Similarly, the term “juvenile” is not consistently linked to a specific age class; an appropriate definition includes all individuals under the age of one i.e. cubs and subadults (Soulsbury *et al.* 2008b). Second, inconsistent determination of fecundity is a major source of confusion surrounding the conversion of vital rates to matrix elements (Noon & Sauer 1992). The interpretation and definition of techniques to determine litter size have been extensively reviewed (Englund 1970, Harris 1979, Lindström 1981, Allen 1983). It is

unclear whether guidelines for using placental scars to determine litter size (Englund 1970) are widely followed but explicit reference to these guidelines would promote greater confidence in the data obtained from specific studies. Third, of the components driving reproductive output, the proportion of breeding females varies more widely between populations than litter size (e.g. Harris 1979, Zabel & Taggart 1989), often due to complex social factors (Macdonald 1979, Iossa *et al.* 2009). The definition of “barren” females is an area of particular uncertainty and great variability. "Barren" can indicate animals that are unable to reproduce, as well as those that are capable of reproducing but fail to do so in a particular year. In addition, reproductive failure could occur at various points: failure to mate; failure to implant fertilised ova; death of the entire litter during pregnancy; and loss of an entire litter immediately following parturition, due to infanticide or other social factors. It is recommended that, rather than using the ill-defined term “barren”, future studies define the proportion of females experiencing reproductive failure at any given stage, as has been done for Eurasian badgers *Meles meles* (Cresswell *et al.* 1992). Fourth, "hunting" samples vary between countries depending on legal restrictions and local practices. At the moment, for instance, it is unclear how samples taken by driven shoots, night shoots, snaring, leghold traps or digging out of dens differ: data from different collection methods should be presented separately and by time of year to facilitate analyses on the impact of sampling method on demographic parameters. Furthermore, demographic data are often restricted to technical reports (e.g. Whitlock *et al.* 2003), where these are made widely accessible, they might represent a substantial source of more directly useable raw data.

4.5 Conclusion

Demographic analyses of red foxes highlight inter-population differences in life-history. Currently, however, data required to identify the drivers of these demographic patterns are lacking. The difficulties of interpreting models based on uncertain data were reiterated. While it is recognised that, for many species, data are often limited both in quality and quantity, these results caution against data substitution unless exploratory demographic analyses suggest high levels of consistency between

populations. Superficially, the red fox appears well studied. As a result, a good understanding of red fox demography might be assumed. In reality, in spite of the fox's widespread distribution, abundance and economic importance, there are remarkably few usable demographic data from much of its range. Studies of other abundant and widespread species suggest that great insight can be gained by comparing intraspecific demography. Demographic research on the red fox lags behind that on ungulates, for example, studies of which have been used to examine the effects on population dynamics of harvesting regimes (Servanty *et al.* 2011), quantitative trait variation (Pelletier *et al.* 2007), and climate (Coulson *et al.* 2001). Few broad scale models of age-specific survival and fecundity of multiple carnivore populations have been conducted. Here, the range of analyses that can be performed using published data was illustrated. Further long-term research would be necessary to minimise sampling bias and to determine whether apparent inter-population differences are robust to temporal variation. With improvements in reporting standards, much more remains to be learnt about this important and widespread carnivore.

Chapter 5 Transmission mechanisms of sarcoptic mange *Sarcoptes scabiei* in a social carnivore, the red fox *Vulpes vulpes*

5.1 Introduction

Sarcoptic mange, caused by the highly contagious mite *Sarcoptes scabiei*, affects over 100 domestic and wild mammalian species (Pence & Ueckermann 2002). Mange has been identified as a potential emerging disease (Daszak *et al.* 2000) threatening endangered species such as cheetahs *Acinonyx jubatus*, gorillas *Gorilla gorilla* and Iberian ibex *Capra pyrenaica* and posing a risk of cross-species infection for domestic species (Smith *et al.* 2009b). Red foxes *Vulpes vulpes* have been known hosts of mange (*S. scabiei* var. *canis*) since the 1600s (Friedman 1947, cited in Newman *et al.* 2002), and epizootics of mange have caused significant population declines of fox populations worldwide (Gerasimov 1958, Storm *et al.* 1976, Lindström & Morner 1985, Forchhammer & Asferg 2000, Soulsbury *et al.* 2007). Despite extensive work on the clinical aspects of *S. scabiei* (see Arlian 1989), fundamental aspects of mange epidemiology are undefined for many wild mammalian host populations (Bornstein *et al.* 2001), including the basic reproductive number, R_0, the transmission coefficient, β and the infectious period, γ. The basic reproductive number represents the number of secondary cases produced by one infectious individual in an entirely susceptible population and is central for predicting disease establishment in a population. Yet estimates of R_0 have not been determined for canid hosts of mange. The transmission coefficient determines the number of new cases per unit time. However, the transmission pathways that promote the persistence and cycles of mange in wild host populations remain to be fully identified.

Mange often persists in wild canid populations for many years at an enzootic level (Gortazar *et al.* 1998, Gosselink *et al.* 2007) and 30 to 40 year cycles of mange epizootics have been identified in coyotes *Canis latrans* (Pence & Windberg 1994). Whilst both direct and indirect routes have been implicated in mange transmission (Pence & Ueckermann 2002), the few models that exist have focused primarily on

direct mechanisms (see Leung & Grenfell 2003, Lunelli 2010). Consequently, there is a need to refine our understanding of the dynamics of mange in wild canids.

Modelling provides a valuable tool for identifying the population and life history attributes of hosts and pathogens that drive disease prevalence and transmission (Anderson & May 1979). Specifically, model-based parameter estimation provides a mechanistic means for understanding the impact of a pathogen on a host's population dynamics (Dobson & Hudson 1992, Tompkins *et al.* 2002) and can also lead to management recommendations (Hess 1996, Packer *et al.* 2003). Deterministic compartment models, such as those describing the transition between susceptible, infected, and recovered states (Anderson & May 1992), are a widely used epidemiological approach for describing disease patterns (Smith *et al.* 2009a). However, a key limitation of the parameterisation of theoretical wildlife disease models is the often limited availability of empirical data (Barlow 1995).

Because of the difficulties of observing the frequency of individual contacts and those contacts with susceptible individuals that subsequently cause infection, determining disease transmission rates for wild populations is notoriously problematic (McCallum *et al.* 2001). Thus, epidemiological parameters, including β, are frequently estimated by fitting models to empirical data (Smith *et al.* 2009a), such as prevalence data. Prevalence, defined as the proportion of individuals in a sample that are infected, is a commonly collected source of disease data (e.g. Caley & Ramsey 2001, Roche *et al.* 2009). The use of prevalence data in modelling is typically based on the assumption that the observed variation in this measure reflects the true variation in the disease prevalence of the target population. Unfortunately, many sources of uncertainty arise in prevalence data owing, in part, to imperfect detection of disease in wild populations; this can result from limitations in diagnosis, altered behaviour of infected animals, or the often opportunistic or uneven sampling effort (Conner *et al.* 2000, Jenelle *et al.* 2007, McClintock *et al.* 2010). Given concerns about data reliability, ensuring that model predictions are supported by empirical data is a well acknowledged, but poorly addressed issue in disease modelling (Barlow 1995).

For reasons of computational simplicity, disease models often assume that there is no within-population variation in prevalence. Yet heterogeneities in disease prevalence frequently exist between groups of individuals within a population, and can have a significant impact on disease dynamics (Woolhouse *et al.* 1997, Altizer *et al.* 2003b, Lloyd-Smith *et al.* 2005b). In cases where disease prevalence varies due, for example, to the different susceptibilities of age, sex or social classes, this variation can be explicitly modelled by adding stage-specific transmission terms (Bolzoni *et al.* 2007). For example, incorporating stage structure significantly improved the fit of models of phocine distemper in harbour seals *Phoca vitulina*, possibly attributable to the contact behaviour of juveniles and breeding adults during the pupping season (Klepac *et al.* 2009). Clearly, variation in prevalence is of consequence for determining contact rates, and hence patterns of disease transmission.

In epidemiological compartment models, transmission is typically assumed to be density-dependent for all diseases other than sexually transmitted diseases, with the latter being described by frequency-dependent transmission to account for contact rates being independent of population size (McCallum *et al.* 2001, Begon *et al.* 2002). Recent studies, however, have brought these assumptions into question, suggesting that the importance of changes in host contact rates for disease transmission has been underestimated (Begon *et al.* 1999, Caley & Ramsey 2001, Smith *et al.* 2009c). For instance, Begon *et al.* (2003) found that frequency-dependent transmission of cow pox in two species of rodent was supported over density-dependent models. A switch from density-dependent to frequency-dependent transmission has been predicted in social species if contact rates remain constant and territory size but not group size changes, whereas if both properties change simultaneously, transmission may occur along a continuum between the two mechanisms (Smith 2006). In a review of modelling approaches, Sterner and Smith (2006) suggested that a combination of density- and frequency-dependent functions may be necessary to describe rabies dynamics in foxes due to changes in territorial contact rates and group size with density.

Transmission of many diseases, including mange, can also occur indirectly through contact with inanimate substances, such as when scraping under fences or sharing

dens. These substances, known as fomites, are capable of being infected by free-living parasite stages (Anderson & May 1981). This additional transmission pathway often occurs in combination with direct transmission, adding an extra layer of complexity that has only recently been widely incorporated into wildlife disease models, but that has been found to improve model fit (Barlow *et al.* 2002, Roche *et al.* 2009, Rohani *et al.* 2009). For example, combined density-dependent and indirect transmission models were the most parsimonious for chronic wasting disease in mule deer *Odocoileus hemionus*, and the estimated effort required for eradication of this disease was higher than previously predicted given the longer disease persistence in these models (Miller *et al.* 2006). Indeed, diseases with a free-living stage can persist at very low host densities (Anderson & May 1981). Sauvage *et al.* (2003) found that the persistence of hantavirus in bank voles *Clethrionomys glareolus* at low densities could only be captured by models that included indirect transmission. In foxes, den-sharing was an important mode of indirect transmission of mange in Russia (Gerasimov 1958). It is worthwhile, therefore, not only to determine whether the mechanism of direct transmission can be identified, but also to assess the importance of including an alternative transmission pathway for mange dynamics in a social species.

A mange epizootic occurred among Bristol's urban fox population in the mid 1990s, causing a drastic population decline (Baker *et al.* 2000), and the disease has remained enzootic in the study area. This urban population has been monitored continually for over 30 years, providing long-term data on fox demography and mange prevalence (Baker *et al.* 2001b). The population reached exceptionally high densities prior to the epizootic, which may have contributed to the spread of mange. However, since sociality is well established in foxes (Cavallini 1996), it is useful to determine whether mange transmission in this species is density- or frequency-dependent. It is also useful to consider age-specific prevalence as an indication of the influence of life history stage on mange transmission. Further, the low rates of direct inter-group contact (White & Harris 1994, Baker & Harris 2000, Giuggioli *et al.* 2011, Soulsbury *et al.* 2011) imply that an indirect component may be required to describe the observed rapid transmission of mange. The Bristol fox population provides a unique opportunity to explore the disease dynamics and transmission pathways of mange in this social

species. Combining traditional compartment modelling with an information theoretic approach, this study considers whether (i) SEI models can describe the dynamics of mange in the Bristol fox population; (ii) prevalence data support either frequency- or density-dependent transmission; and (iii) indirect transmission improves the fit of models to the data. Epidemiological parameter estimates are reported for parsimonious models. Finally, the results are discussed in the context of modelling disease in a social species.

5.2 Methods

5.2.1 Data

The Bristol fox population experienced a sarcoptic mange epizootic during 1994 to 1996; prevalence peaked in the autumn of 1995 when it was estimated that close to 100% of the population was infected (Baker *et al.* 2000). At the start of the epizooty the total (adult and cubs) fox population density was 58.3 individuals km^{-2}, but this declined by 95% by the end of 1996 (Baker *et al.* 2000). Population recovery has been slow and mange has remained at enzootic levels in this urban population since 1996 (Soulsbury *et al.* 2007). Annual post-breeding population densities (Baker *et al.* 2001b, Whiteside *et al.* 2011), were estimated from capture-mark-recapture data (e.g. Baker *et al.* 2000). Four years with missing estimates (1996-97; 2000-01) were determined by linear interpolation. Population density estimates for 1994 to 2010, presented in Figure 5.1, show the population decline due to the mange epizootic.

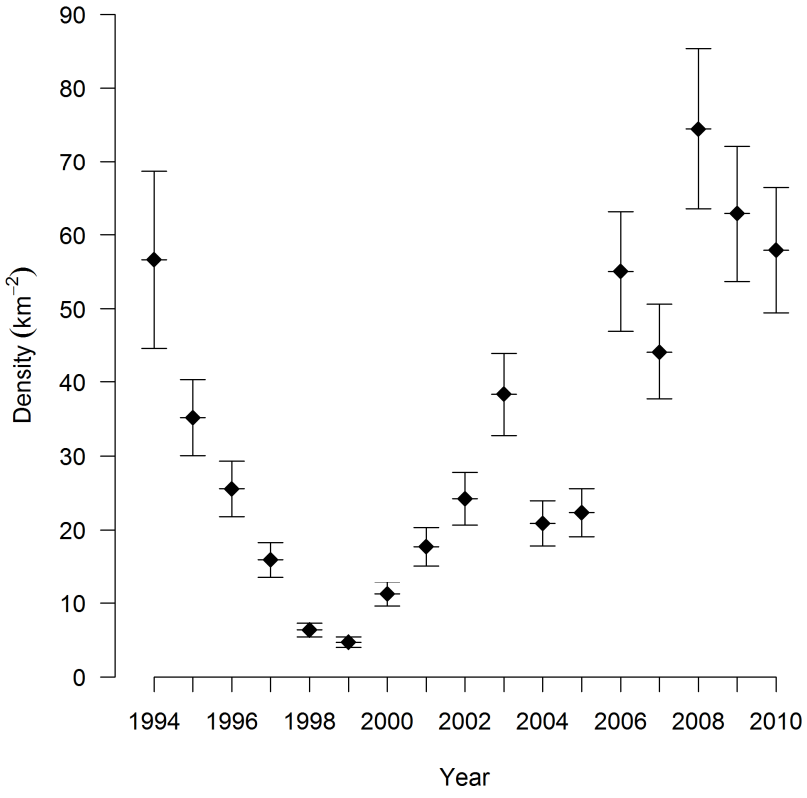

Figure 5.1. Population density estimates to define the post-breeding density $N_k(t)$, here, the total number of adults and cubs, used for the annually varying model. Error bars indicate standard deviations.

The symptoms of mange are not immediately apparent after exposure to the mites. In canids, the observed average latent period is between 10 and 30 days (Bornstein *et al.* 2001). The itching response triggered by the mites eventually leads to hyperkeratosis, the crusty appearance associated with mange; subsequent bacterial infection is the predominant cause of death, although mange is not always fatal. For detailed descriptions of clinical symptoms of mange see Bornstein *et al.* (2001) and Newman *et al.* (2002). On average, the time to death of infected juveniles and adults was 3.5 months during the epizootic (Newman *et al.* 2002).

Prevalence and mortality data used in this analysis were based on data collected through the recapture of radio-collared or marked individuals, and recovery of fox carcasses, from 1994 to 2010 (n=1662 records) (S. Harris *pers. comm.*) from a 14km^2 area of suburban Bristol (see Newman *et al.* 2002, Soulsbury *et al.* 2007 and references therein for descriptions of sampling protocols). Mange diagnosis was classified according to the disease manifestation; class I and class II were defined as no evidence of, and presence of hyperkeratotic mange, respectively (see Newman *et al.* 2002). Due to the small monthly sample sizes, class I and class II data were combined to obtain the number of infected individuals per month. Monthly prevalence was then calculated as the proportion of infected juveniles and adults respectively. To determine uncertainty in the prevalence data 95% confidence intervals were calculated from likelihood profiles (Bolker 2008).

Mean monthly sample sizes for adults (2.61, SD ± 0.79, n = 502) and juveniles (5.53, SD ± 1.30, n =1061) were consistent during the year (Figure 5.2), with the exception of a peak in juvenile capture and mortality records in the summer months, which reflects the newly mobile cubs (Figure 5.2A). Juveniles were sampled (Figure 5.2A), on average, twice as frequently as adults (Figure 5.2B), reflecting the age distribution of the population. Mean sample sizes of infected individuals for monthly prevalence data were low for both age classes (adults 0.63, SD ± 0.28, n = 120; juveniles 0.99, SD ± 0.33, n = 191; Figure 5.2). Age-related patterns in the monthly prevalence of mange (Figure 5.3) suggest some seasonality, particularly in juveniles. Confidence intervals are wide, however, indicating substantial uncertainty in the data.

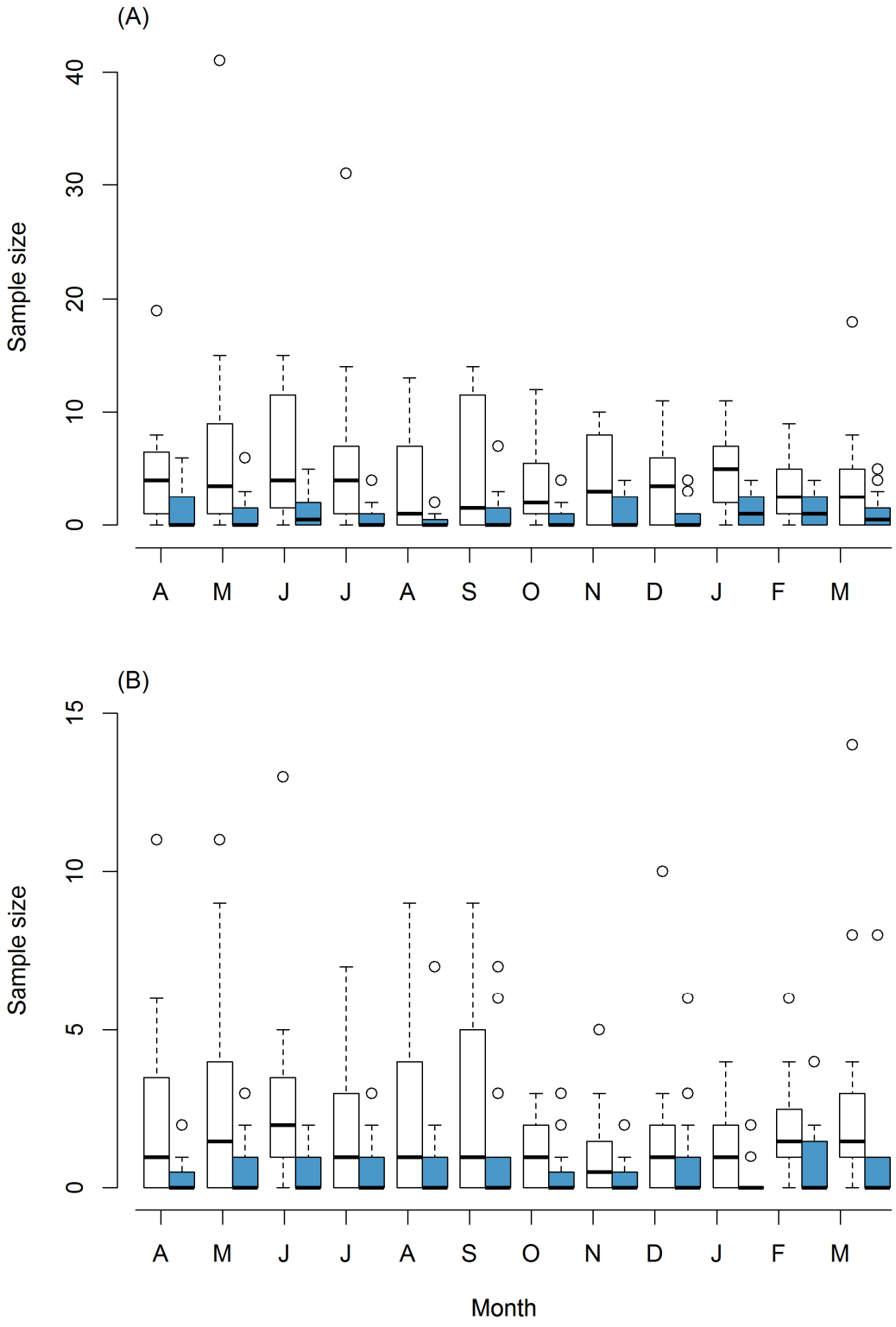

Figure 5.2. Monthly number of individuals of total sampled foxes (no fill) and infected foxes (blue) from 1994 – 2010. (A) Juveniles; (B) adults. Boxes show the sample median, minimum and maximum. Error bars indicate the lower and upper quartiles and outliers are indicated by open circles.

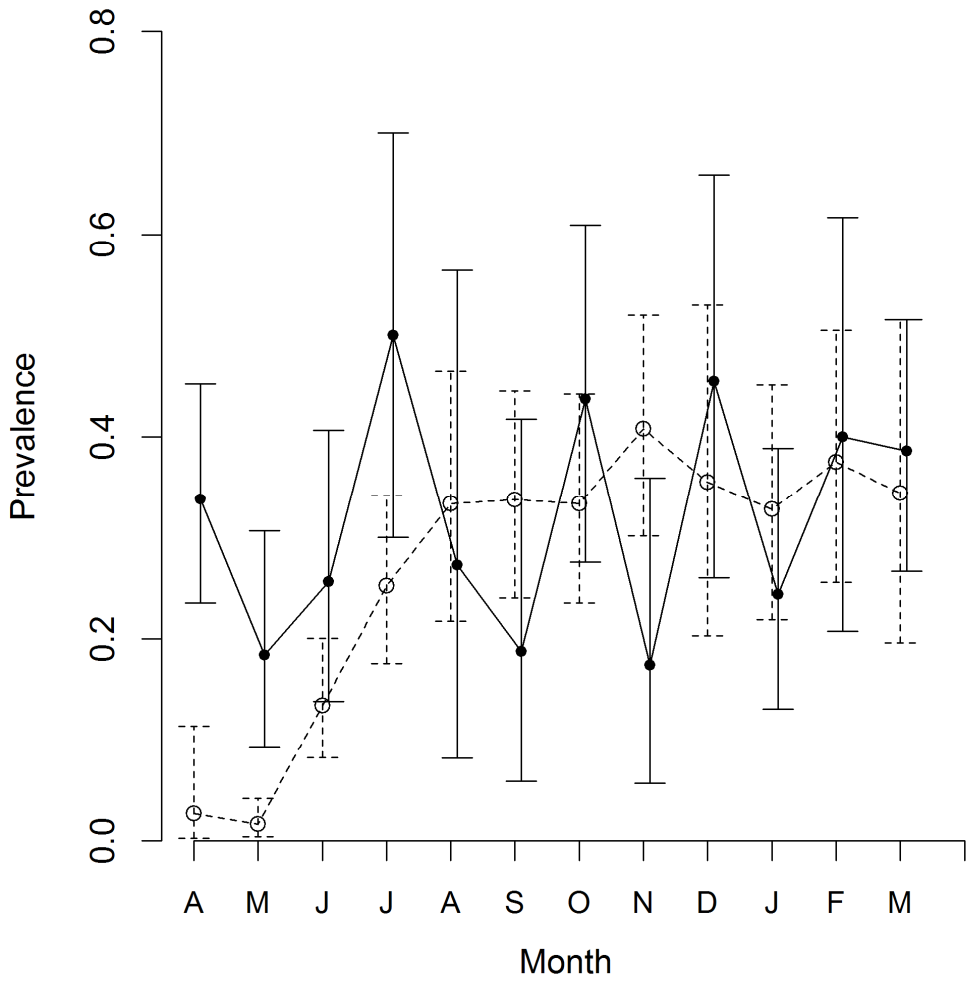

Figure 5.3. Mean monthly prevalence of mange infection for juveniles (dashed line, open circles) and adults (solid line, closed circles) from 1994 – 2010, with 95% confidence intervals.

5.2.2 Modelling mange dynamics

5.2.2.1 Mange as a microparasite

Most definitions of parasites assume that microparasites are small and numerous, reproduce rapidly within the host and once infected, a host will die or recover largely independent of the parasite load (Anderson & May 1992). In comparison, macroparasites are typically assumed to be larger, have an intermediate host and off-host reproduction, and with morbidity and mortality dependent on the parasite burden (Anderson & May 1992). In reality, micro-and macro-parasites lie along a continuum of these epidemiological properties (Anderson & May 1979). *S. scabiei* is conventionally classified as a macroparasite, although it displays several microparasite

attributes: the small mites reproduce directly and rapidly on the host and are able to transfer directly between host individuals. Thus, a microparasite modelling approach was used in this study.

5.2.2.2 SEI models

Compartment modelling was used to estimate the epidemiological parameters, β and *γ*, and to compare different pathways of mange transmission in foxes. Specifically, an SEI model was used in which densities (*N*) of individuals in a given population are categorised into classes according to their disease status as susceptible (*S*), exposed (*E*), and infected (*I*) (i.e. $N = S+E+I$). The exposed class was included to incorporate the time taken between foxes becoming exposed to the mites and becoming infectious. Recovered individuals were assumed to return directly to the susceptible class, because although a low number of foxes in Bristol were observed to recover fully, re-infection of individuals was also observed (S. Harris *pers. comm.*). Two forms of direct transmission were modelled. Density-dependent transmission was the first direct mechanism modelled (M_1). Here, the transmission rate is proportional to the density of susceptible and infected groups within the population (βSI), which results in prevalence increasing linearly with density. The second mode of direct transmission, frequency-dependent (M_2), assumes that the infection rate is dependent on the proportion of infective individuals in the population ($\beta SI/N$). In this case, opportunities for contact between an infectious and susceptible individual are independent of population size (Begon *et al.* 2002). Frequency-dependent transmission accounts for the possibility that the rate of infectious contacts per infected individual might not increase linearly with density, which could arise as a consequence of contact rates being determined by social interactions. Host demography was incorporated into the models with a fixed *per capita* mortality rate (see Table 5.1) and a birth pulse. Foxes breed annually and for modelling purposes it is typically assumed that all cubs are born on April 1st (Harris & Smith 1987). Thus, for convenience, the total population size was reset annually to a post-breeding density (N_k), occurring in March because this process was modelled at the end of the month. In this way, a pulse of new susceptible individuals (S_{bj}) was introduced into the population each year ($S_{bj} = N_k - N$). Because of

the large fluctuations in population density over the data collection period (Iossa *et al.* 2009), two versions of each model were run: the first used a fixed N_k, whilst the second used a post-breeding density, $N_k(t)$, that varied annually based on an independent set of density data. The fixed N_k was set as the combined juvenile and adult population density estimate of the initial conditions (Table 5.1), and $N_k(t)$ was defined as the total population density estimate for year *t*.

Table 5.1. Definition of fitted and fixed parameters used in SEI models. Initial values of fitted parameters were estimated from the literature where possible.

Parameter	***Definition***	***Fixed or fitted* parameter***
β_{jj} , β_{aa}	Age-specific density-dependent transmission (day^{-1})	*
β'_{jj} , β'_{aa}	Age-specific frequency-dependent transmission ($individual^{-1}\ day^{-1}$)	*
β_f	Indirect transmission (day^{-1})	*
γ	Infectious period = $1/\gamma$ (day^{-1})	200 days*
σ	Latent period = $1/\sigma$ (day^{-1})	30 days
α	Disease-induced mortality rate = $1/\alpha$ (day^{-1})	100 days
μ_j	Juvenile‡ *per capita* mortality probability ($year^{-1}$)	0.3†
μ_a	Adult‡ *per capita* mortality probability ($year^{-1}$)	0.5†
ω	*per capita* reproductive rate of mite on infected individuals (day^{-1})	*
ε	Rate of loss of the pathogen in environment = $1/m$ (day^{-1})	10 days
S_{0j}	Initial density of susceptible juveniles (km^{-2})	21
S_{0a}	Initial density of susceptible adults (km^{-2})	36
I_{0j}	Initial density of infected juveniles(km^{-2})	0.01
I_{0a}	Initial density of infected adults (km^{-2})	0.01
F_0	Initial density of fomites	1
K	Fixed post-breeding density	56.65

†Annual probabilities were converted to daily rates by $-\ln(\mu)/360$

‡Juveniles were defined as all individuals under one year, and adults as all individuals older than one year (Harris & Trewhella 1988).

To account for potential age-specific variation in prevalence, the SEI model was extended to include age structure. A "Who Acquires Infection From Whom" (WAIFM) transmission matrix was used (Keeling & Rohani 2008) to denote transmission, β, from one class to another:

$$\beta = \begin{pmatrix} \beta_{jj} & \beta_{ja} \\ \beta_{aj} & \beta_{aa} \end{pmatrix}, \qquad (1)$$

where *j* and *a* represent juveniles and adults respectively. To reduce uncertainty in resultant parameter estimates and to maintain analytical tractability, it was assumed that β_{ja} was equal to β_{jj}, and β_{aj} equalled β_{aa}. Each year, at the time of the birth pulse, juveniles in a given disease state matured into adults of the corresponding disease class. The following ordinary differential equations (ODEs) describe disease dynamics between birth pulses according to the density-dependent SEI model (Figure 5.4):

$$\begin{aligned}
\frac{dS_j}{dt} &= -\mu_j S_j - (\beta_{jj} I_j + \beta_{aj} I_a) S_j + \gamma I_j \\
\frac{dE_j}{dt} &= -\mu_j E_j - \sigma E_j + (\beta_{jj} I_j + \beta_{aj} I_a) S_j \\
\frac{dI_j}{dt} &= -(\alpha + \mu_j) I_j + \sigma E_j - \gamma I_j \\
\frac{dS_a}{dt} &= -\mu_a S_a - (\beta_{aa} I_a + \beta_{ja} I_j) S_a + \gamma I_a \\
\frac{dE_a}{dt} &= -\mu_a E_a - \sigma E_a + (\beta_{aa} I_a + \beta_{ja} I_j) S_a \\
\frac{dI_a}{dt} &= -(\alpha + \mu_a) I_a + \sigma E_a - \gamma I_a,
\end{aligned} \qquad (2)$$

where μ is the age-specific natural death rate of the host, α is the disease-induced mortality rate, σ is the rate of progression from the latent stage once exposed, γ is the infectious period (parameter definitions are specified in Table 5.1) and β denotes the transmission coefficient for age-specific density-dependent transmission according to the WAIFM matrix (eqn.1).

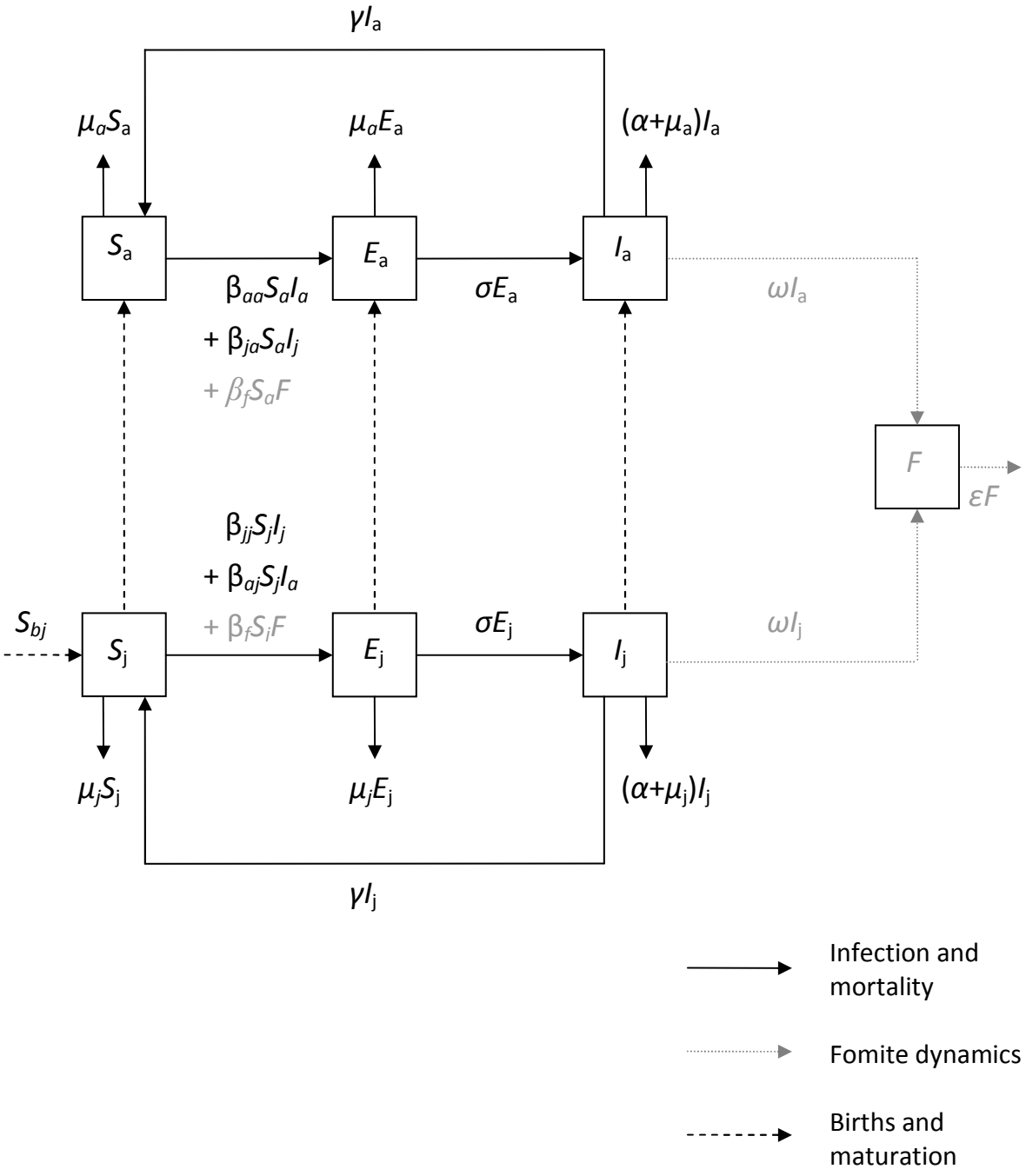

Figure 5.4. SEI compartment model diagram illustrating age-specific density-dependent direct transmission with host demography. Indirect transmission and fomite dynamics are indicated in grey. Parameter descriptions are presented in Table 5.1.

The transmission coefficient, β_f, describes indirect transmission via infection through the contact of susceptible individuals with free-living mites on infected substrates. For analytical tractability, it was assumed that β_f was not age-specific. Indirect transmission was combined with direct transmission e.g. $S_j(\beta_j I_j + \beta_{aa} I_a + \beta_f F)$, to give a total infection rate, given that indirect pathways are unlikely to be the sole transmission mechanism. For density-and frequency-dependent models that incorporated indirect transmission (M_3 & M_4), an additional compartment (F) followed the densities of mites in the environment:

$$\frac{dF}{dt} = \omega(I_j + I_a) - \varepsilon F, \qquad (3)$$

where ω is the rate mites are released into the environment by the total infected individuals, and ε is the death rate of mites on fomites (see Table 5.1). Under average ambient conditions, all life stages can survive an average of 10 days off the host, but this can increase to several weeks if conditions are optimal (Arlian 1989). The rate that mites are released into the environment, ω, is an unknown parameter; ω is dependent on the reproductive rate of the mites and individual parasite loads. Female mites produce 3-4 eggs per day, with an average life expectancy of 5 weeks (Arlian *et al.* 1989), but parasite loads and the rate at which mites are released from the host remain undetermined and so ω was a fitted parameter.

5.2.2.3 Parameter fitting and model selection

The SEI model parameters were fitted to the prevalence data using maximum likelihood. This analysis is based on the assumption that the transmission rate, β, of mange in a population, N, of S susceptible individuals produces I infected individuals per day, given that E individuals were exposed to the mite and became infectious. The probability an individual in the population is infected, p, is given by I/N. Predictions of the model can be compared to empirical observations on the prevalence of infected individuals by considering the process of field data collection as a series of binomial trials. Let the months in the total time series be denoted by [m = 1, 2, 3, …, D]. Within a given month, each individual sampled can be considered as a "trial", with the total

number of individuals sampled in each age class denoted n_x. Assuming that the probability of becoming infected, p_x, is uniform among individuals sampled of age x, the number of infected individuals within an age class, y_x, will follow a binomial distribution. Thus, the likelihood at time m that proportion p_x of either juveniles or adults in the population are infected, given that a random sample of n_x individuals includes y_x infectives, is

$$L(p_x \mid n_x, y_x) = \binom{n_x}{y_x} p_x^{y_x} (1-p_x)^{n_x - y_x}. \qquad (4)$$

Observed variation in the rate of infection can arise as a result of sampling error, including misdiagnosis, or due to the effects of unmeasured factors such as differences in individual susceptibility. If these sources of variation are unaccounted for and result in overdispersed data, then unnecessarily complex models can be selected when using information theoretic approaches because model precision will be overestimated (Anderson *et al.* 1994, Richards 2008). To measure the degree of dispersion in the data, the variance inflation factor, $\tilde{v}$, was estimated by dividing the variation in the observed data (saturated model, where the number of parameters equals the number of observations) by the variation in the most complex binomial model (Richards 2008). If overdispersion is present ($\tilde{v} \geq 2$), a compound distribution can be fitted to the data instead (Richards 2008). For binomial data, an appropriate compound distribution is the beta-binomial distribution. This model assumes that variation in p_x across samples within a given time period is described by the beta distribution:

$$f(p_x; \overline{p}_x, \phi) = \frac{\Gamma(a+b)}{\Gamma(a)\Gamma(b)} p_x^{a-1} (1-p_x)^{b-1}, \qquad (5)$$

where the parameter ϕ quantifies the variation in p_x, $\overline{p}_x$ is the mean probability of success, Γ(x) is the complete gamma function, $a = \overline{p}_x / \phi$, and $b = (1 - \overline{p}_x)/\phi$. Substituting equation (5) into equation (4) gives the compound beta-binomial distribution. If **θ** is the set of model parameters required to calculate $\overline{p}_x$ and the dispersion coefficient ϕ, then the likelihood of **θ** at time m can be calculated as

$$L(\boldsymbol{\theta} \mid n_x, y_x) = \frac{\Gamma(n_x+1)\Gamma(a+b)\Gamma(y_x+a)\Gamma(n_x-y_x+b)}{\Gamma(y_x+1)\Gamma(n_x-y_x+1)\Gamma(a)\Gamma(b)\Gamma(n_x+a+b)}. \quad (6)$$

Equation (6) approximates the binomial distribution as the dispersion parameter, ϕ, approaches zero. The total log-likelihood of the model, defined by **θ** and given all the data, is then the log of equation (6) summed over age classes *j* and *a* over the total time period, *D*:

$$LL(\boldsymbol{\theta} \mid \text{data}) = \sum_{m=1}^{D} \left\{ \sum_{j=1}^{n_j} (\ln L[\boldsymbol{\theta} \mid n_{jm}, y_{jm}]) + \sum_{a=1}^{n_a} (\ln L[\boldsymbol{\theta} \mid n_{am}, y_{am}]) \right\}. \quad (7)$$

To determine if the disease transmission model presented above is consistent with the data, it is useful to compare predicted dynamics with a null model in which disease prevalence is constant in time. A beta-binomial null model (M_H) was fitted which simply assumed that the probability a sampled individual in each age class was diseased was, on average, time-invariant ($p_x = \overline{p}_x$). The ability of SEI models to capture patterns in the prevalence data was determined by comparing the likelihoods of the null model, M_H, and those models that included disease parameters (M_1 to M_4).

In general, because epidemiological ODE models cannot be solved analytically due to their non-linear properties it is necessary to use a discrete approximation. Thus, to obtain prevalence patterns, $p_x(m)$, predicted by each SEI model, the associated system of ODEs (eqn. 2) was solved using the fourth-order Runge-Kutta method, a widely used method of numerical integration that calculates the state variables by evaluating their derivatives at four points along each time-step (Press *et al.* 2007). The set of model parameter values fitted to the monthly age-specific prevalence data for direct transmission were $\boldsymbol{\theta} = \{\beta_{jj}, \beta_{aa}, \gamma, \phi\}$ and $\boldsymbol{\theta} = \{\beta'_{jj}, \beta'_{aa}, \gamma, \phi\}$ for density- and frequency-dependent transmission respectively; for models that include indirect transmission, the models were defined by $\boldsymbol{\theta} = \{\beta_{jj}, \beta_{aa}, \beta_f, \omega, \gamma, \phi\}$ and $\boldsymbol{\theta} = \{\beta'_{jj}, \beta'_{aa}, \beta_f, \omega, \gamma, \phi\}$. Parameter estimates were determined by maximising the total model log-likelihood (eqn. 7) using the "optim" function in R 2.14.0 (R Development Core Team 2011). Where possible, parameter values estimated from the literature were used as initial

starting points (see Table 5.1). To distinguish between the competing models, Akaike's Information Criterion (AIC) was used; to avoid instances where the best AIC model does not have the lowest AIC value due to uncertainty from sampling error, all models with ΔAIC ≤ 6 units were considered to have some level of support (Richards 2008). A bootstrap approach was used to calculate 95% confidence intervals for each parameter of the best fitting model selected by AIC. Specifically, 1000 model replicates were fitted by re-sampling the prevalence data between years, but from the same month.

5.2.2.4 Basic reproductive number

The basic reproductive number, R_0, is used to determine the probability of a disease spreading in a population (Hethcote 2000); a pathogen can invade if $R_0 > 1$ (Anderson & May 1992). R_0 is dependent upon the rate of contact between individuals, the probability of infection given contact, and the duration of infectiousness per individual. R_0 tends to be maximised at intermediate levels of disease-induced mortality because both extremely high and low virulence would cause pathogens to die out rapidly (Walther & Ewald 2004). R_0 was calculated for the most parsimonious model selected by AIC, using the parameter value estimates obtained from maximum likelihood. Because of the heterogeneities in infection rates between age classes of structured SEI models, the total R_0 needs to account for these age-specific contributions; that is, the contribution coming from the number of secondary cases arising in one age group from a case in a second age group, assuming that every individual in the first age group is susceptible. A "next generation matrix" can be derived from the WAIFM matrix and the population age distribution (Diekmann *et al.* 1990), such as for density-dependent transmission:

$$\boldsymbol{A} = \begin{pmatrix} \beta_{jj} n_j / \gamma & \beta_{jk} n_j / \gamma \\ \beta_{aj} n_a / \gamma & \beta_{aa} n_a / \gamma \end{pmatrix}, \qquad (8)$$

where $n_j = (S_{0j}/N)$ and $n_a = (S_{0a}/N)$. This matrix, $\boldsymbol{A}$, provides a weighting of the contribution of each age class to the spread of infection and the overall R_0 is calculated

as the dominant eigenvalue of ***A*** (Keeling & Rohani 2008). For models including indirect transmission, the overall R_0 is equal to $R_0 + R_0^{indirect}$, where $R_0^{indirect}$ is calculated as:

$$R_0^{indirect} = \frac{\omega\beta_f(S_{0j} + S_{0a})}{m(\mu + \alpha + \gamma)}, \quad (9)$$

(Rohani *et al.* 2009). All analyses were conducted in R 2.14.0 (R Development Core Team 2011).

5.3 Results

5.3.1 Transmission mechanisms

Prevalence data were overdispersed (variance inflation factor, $\hat{v}$ = 2.79) and, therefore, all model likelihoods were calculated assuming that monthly observations of disease prevalence were distributed according to the beta-binomial distribution (eqn. 6). SEI models were consistently better than the null model at explaining the prevalence of mange in foxes (see ΔAIC values in Table 5.2). The most parsimonious models (M_{2c} and M_{2v}) indicate strong support for frequency-dependent transmission of mange (Table 5.2) in the Bristol fox population. Including annual variation in density (M_{2v}; Figure 5.5) did not improve the fit of M_{2c} (Table 5.2). The extra parameters required to describe indirect transmission were seldom justified by the extent of improvement in model fit. One frequency-dependent model incorporating indirect transmission (M_{4c}) performed well (Table 5.2), but M_{4c} is an expanded version of M_{2c} and, as such, its higher AIC value suggests that it lacks credibility (Richards 2008).

The most parsimonious model (M_{2c}) captured observed intra-annual patterns well (Figure 5.6), illustrating the rapid transmission and peak mange prevalence seen in the empirical data on both juveniles (Figure 5.6) and adults (Figure 5.6). The low prevalence amongst juveniles in April to May corresponds to the post-birth period. The birth pulse promotes the observed cycles and persistence of mange by periodically introducing new susceptible individuals into the population, while disease-induced mortality is offset by the high transmission rate. That the model prediction does not fall within the confidence interval for juvenile prevalence in May (Figure 5.6) probably reflects that the timing of births was invariant in the model, whilst the actual timing of births varies among individuals and years. The large uncertainty in the empirical data confounds attempts to identify inter-annual patterns in prevalence.

Table 5.2. Model selection results for null and SEI models. The number of parameters (K), log-likelihoods (LL), and AIC values for each model are presented. Parameters are defined in the methods, and Table 5.1.

Model		***Parameters***	***K***	***Log-likelihood***	***AIC***	***ΔAIC***
M_H	Null model	$\overline{p}_j$, $\overline{p}_a$ ϕ	3	-325.92	657.83	26.32
M_{1c}	Density-dependent + fixed density N_k	β_{jj}, β_{aa}, γ, ϕ	4	-322.87	653.74	22.23
M_{1v}	Density-dependent + varying density $N_k(t)$	β_{jj}, β_{aa}, γ, ϕ	4	-320.48	648.97	17.46
M_{2c}	Frequency-dependent + fixed density N_k	β'_{jj}, β'_{aa}, γ, ϕ	4	-311.78	631.51	0.00
M_{2v}	Frequency-dependent + varying density $N_k(t)$	β'_{jj}, β'_{aa}, γ, ϕ	4	-312.00	632.00	0.48
M_{3c}	Density-dependent + Indirect + fixed density N_k	β_{jj}, β_{aa}, γ, ϕ, β_f, ω	6	-326.21	664.42	32.91
M_{3v}	Density-dependent + Indirect + varying density $N_k(t)$	β_{jj}, β_{aa}, γ, ϕ, β_f, ω	6	-323.80	659.59	28.08
M_{4c}	Frequency-dependent + Indirect + fixed density N_k	β'_{jj}, β'_{aa}, γ, ϕ, β_f, ω	6	-311.83	635.66	4.14
M_{4v}	Frequency-dependent + Indirect + varying density $N_k(t)$	β'_{jj}, β'_{aa}, γ, ϕ, β_f, ω	6	-323.85	659.71	28.54

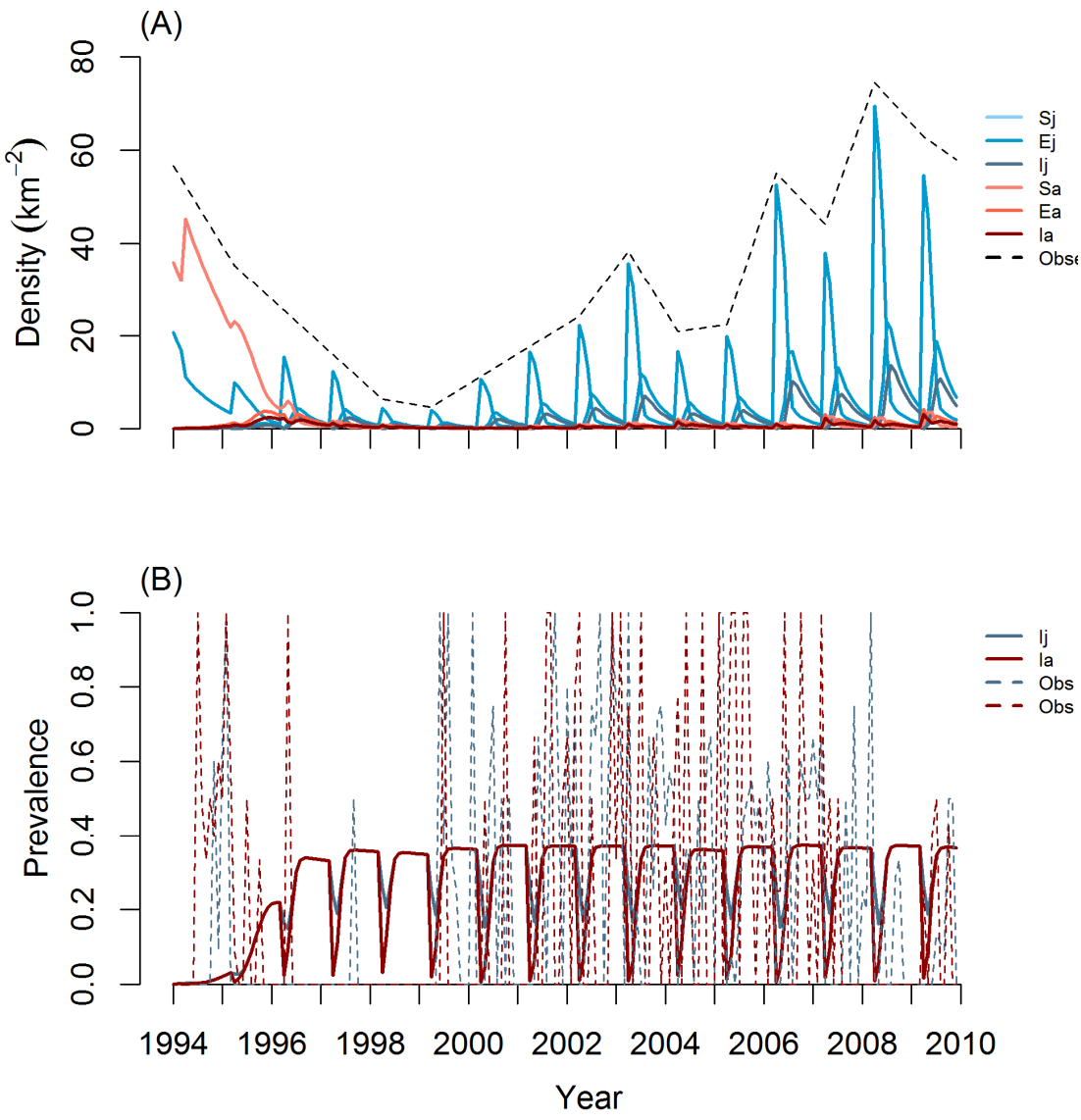

Figure 5.5. The predicted population density (A) and prevalence (B) for the frequency-dependent SEI model with annual variation in density (M_{2c}). Solid lines indicate predicted density for juveniles and adults of susceptible and exposed individuals (S_j, S_a, E_j, E_a) and predicted density and prevalence of infected juveniles and adults (I_j and I_a), against the observed population density and age-specific prevalence data (dashed lines).

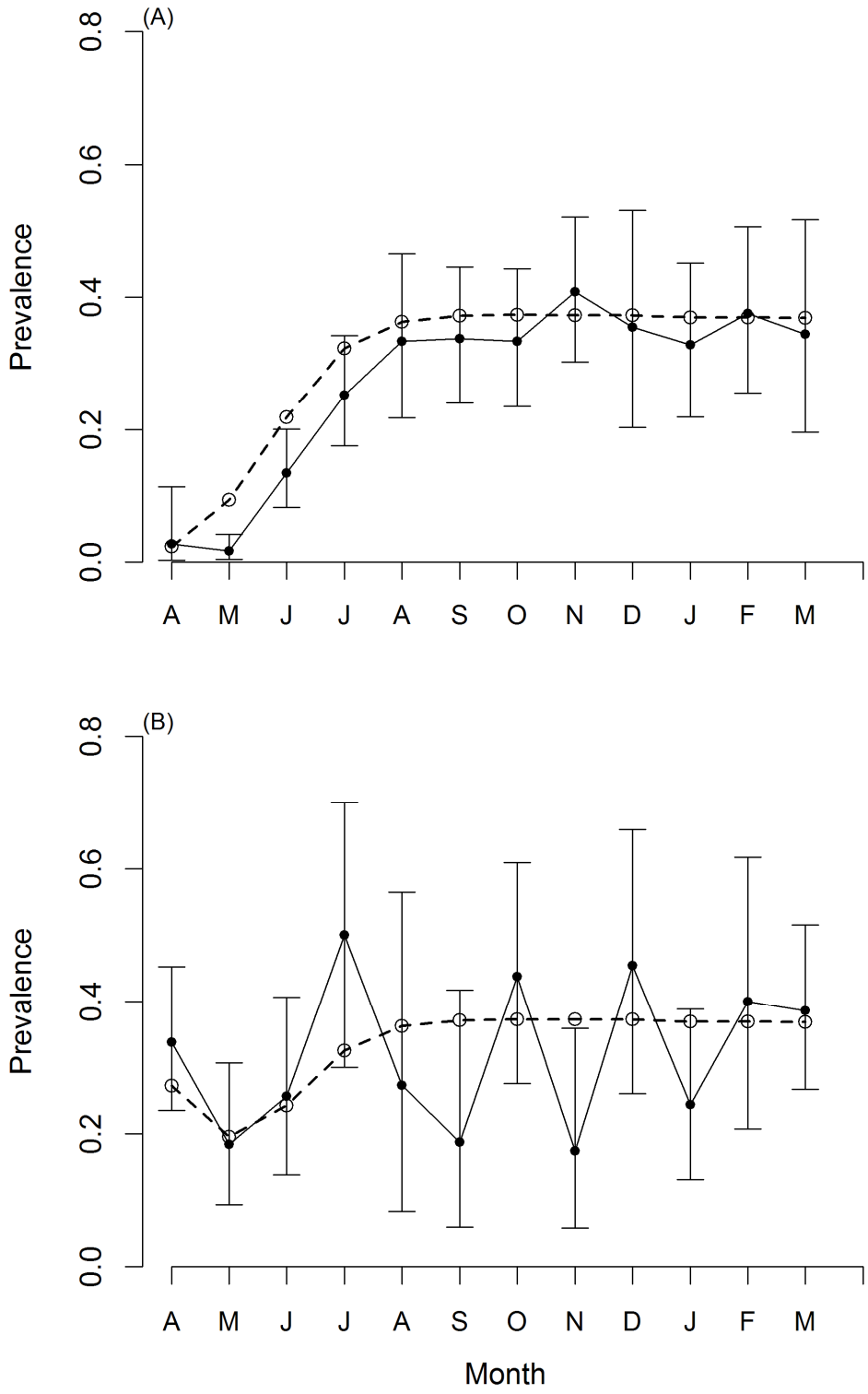

Figure 5.6. The predicted probability of infection (dashed line, open circles) for the frequency-dependent SEI model (M2c), for juveniles (A) and adults (B), against the observed prevalence data (solid line, closed circles see Figure 5.3).

5.3.2 Estimation of epidemiological parameters

Fitted values for epidemiological parameters of sarcoptic mange in foxes are presented in Table 5.3 for the model with the greatest support. The monthly prevalence of mange in foxes is overdispersed (ϕ) with respect to the binomial distribution (Table 5.3). Although the 95% confidence intervals for the transmission coefficients overlap (Table 5.3), juvenile transmission was consistently higher than adult transmission in the bootstrap replicates. The best model estimate of the juvenile transmission rate was four times higher than that of adults (Table 5.3). The best estimate of R_0 and the associated 95% confidence intervals are all greater than one (Table 5.3), consistent with the long-term persistence of mange in the population. The best estimate of the infectious period, γ, corresponds to 30 days (Table 5.3), reflecting the rate at which the infected leave the infectious class. The short infectious period that emerged from model selection would be associated with relatively high survival, which does not match high levels of observed mortality. The discrepancy between the best model estimate and the initial value estimated from the literature may be a reflection of either uncertainty in the prevalence data or the limitations of using traditional compartment modelling for a social species. However, substantial uncertainty in this parameter estimate is evident from the wide confidence intervals.

Table 5.3. Estimated parameter values for the best-fitting model, with bootstrapped 95% confidence intervals. See Table 5.1 for parameter descriptions.

Model	**β'_{ii}**	**β'_{aa}**	***γ***	**ϕ**	***R_0***
M_{2c} Frequency-dependent + N_k	0.228 (0.087–0.378)	0.057 (0.032–0.160)	0.034 (0.012–0.111)	0.249 (0.107–0.404)	3.49 (1.77 – 4.11)

5.4 Discussion

This study generates fundamental epidemiological parameter estimates for mange in red foxes, providing insight into the dynamics of mange in the Bristol fox population. Age-specific heterogeneities in prevalence were detected in the data and analyses suggest that frequency-dependent transmission is the predominant transmission mechanism. These findings are discussed in light of transmission dynamics, variation in disease prevalence, and the implications of sociality for disease transmission.

5.4.1 Mange transmission mechanisms

The SEI models captured the persistence of mange in the Bristol fox population, with the transmission coefficients, β'_{jj} and β'_{aa}, describing the initial speed of transmission. The estimate of R_0 is consistent with mange successfully invading this urban fox population, and is of a similar magnitude to the estimate for mange in chamois *Rupicapra rupicapra* (R_0 = 4.8 - 5.1) (Lunelli 2010). Although R_0 is species and population specific, estimates of R_0 are useful indications of the likelihood of mange persisting in wild populations, especially of those experiencing similar habitat conditions. Thus, estimating R_0 for mange in other fox populations is important, given that habitat, climate and behavioural differences can affect this number (Harvell *et al.* 2002, Lloyd-Smith *et al.* 2005b, Hartemink *et al.* 2009), and that the Bristol population may not be illustrative of this disease in certain fox populations.

Given the high density prior to the epizootic, it may be expected that the fox population in Bristol exceeded a critical host-density threshold, a feature of density-dependent transmission, below which a disease cannot be sustained (McCallum *et al.* 2001). However, modelling suggested that mange dynamics are unlikely to be driven by density and that frequency-dependent transmission is the most probable pathway for mange transmission in the Bristol fox population. This finding implies that contact rates remain constant despite increases in the density of infected individuals and is consistent with the fox social system, in which both inter- and intra-group contact rates are determined by social interactions (Baker & Harris 2000). If social interactions

do not increase with density in fox populations, perhaps owing to territorial behaviour such as olfactory communication (Giuggioli *et al.* 2011), the opportunities for infection are limited. The negligible effect of density on disease prevalence is further supported by the fact that models with varying density did not perform significantly better than those models with constant density. Mange outbreaks in other fox populations have also been found to be unrelated to high density. No clear correlation was found between fox abundance and prevalence in Spain (Gortazar *et al.* 1998) and a slower rate of spread was observed in high rather than low density habitats in Sweden (Lindström & Morner 1985). The persistence of mange at low fox densities in Bristol is also consistent with frequency-dependent transmitted diseases, which can be sustained at lower host densities than density-dependent pathogens (Ryder *et al.* 2007).

The lack of support for indirect transmission could mean either that the role of this pathway is not significant in the Bristol fox population, or that this result is due to model simplifications. Further, a recent study suggests that models of indirect and direct transmission pathways can be indistinguishable when using population-level data, especially when the pathogens' dynamics are fast, i.e. the pathogen has a short off-host survival time (Cortez & Weitz 2013). Understanding of indirect transmission of mange remains inadequate; den sharing was important for the transmission of mange in a Russian population (Gerasimov 1958), yet, this behaviour may be low in the Bristol fox population (S. Harris *pers. comm.*), as reflected in the results here. However, the models did not account for inter-and intra-group encounters, in part due to data limitations. Assuming these encounters were equal may have caused direct contact rates to be overestimated, and thus, increased the importance of direct transmission. While the empirical estimates of contact with fomites, and shedding of mites from infected individuals are hard to quantify, simulations that can incorporate social contacts, such as individual-based models, may help to provide insight into the potential role of other factors for mange transmission.

As with other studies that have compared transmission mechanisms (Caley & Ramsey 2001, Begon *et al.* 2003, Smith *et al.* 2009c), this analysis raises questions about the

traditional assumptions of disease transmission in epidemiological modelling. More complex functions for modelling transmission exist which may be appropriate for social species (Smith 2006), such as either modelling transmission as separate mechanisms between and within groups (de Jong *et al.* 2002) or as a continuum between density-and frequency-dependent (Smith *et al.* 2009c). However, these methods require high-resolution data like that obtained from sero-prevalence studies, which are seldom available for wild populations. Data limitations, combined with complex transmission pathways, are often found to limit substantially the potential to tease apart putative modes of transmission (Caley & Ramsey 2001, Begon *et al.* 2003, Miller *et al.* 2006, Roche *et al.* 2009). Despite the uncertainty in prevalence arising from sampling limitations, the results presented in this chapter highlight that long-term disease data, obtained from a well-studied population such as the Bristol foxes, can make important contributions towards elucidating disease transmission pathways using traditional epidemiological modelling.

5.4.2 Variation in the probability of infection

Age-specific differences in disease susceptibility and transmission are well documented in many species (Bolzoni *et al.* 2007, Klepac *et al.* 2009, McCallum *et al.* 2009), often related to temporal changes in life history stage (Altizer *et al.* 2006). In this simulation, age-specific temporal differences in the prevalence of mange in the Bristol fox population were well described. Some discrepancies between modelled and observed juvenile prevalence could be accounted for by modelling births as a pulse, which is a simplification. Nevertheless, the modelled prevalence reflects the restricted movement of cubs in the months after birth (Robertson *et al.* 2000) and the subsequent increase in opportunities for contacting infectious individuals once cubs start leaving the den. Juveniles thus act as a naïve source of susceptible individuals each year, creating a pulse of infections that also drives the seasonal pattern in adult prevalence.

The predicted difference in the transmission coefficients, β'_{jj} and β'_{aa}, implies that the probability of infection is high for juveniles. The transmission coefficient is a function of contact rates and successful infection given contact with an infected individual

(McCallum *et al.* 2001). The results suggest either that juveniles are more likely compared to adults to become infected once a contact is made, and/or are encountering infected individuals at a higher rate than adults. For example, the probability of a contact resulting in successful infection is expected to be high if juvenile individuals have underdeveloped immune systems; however, understanding heterogeneities in the immune response of mammalian hosts to mange is complex and remains poorly understood (Sarasa *et al.* 2010). Movement patterns at different life stages that alter encounter rates could also give rise to differences in transmission (e.g. Klepac *et al.* 2009), although the high transmission in juveniles does not translate into higher prevalence compared to adults, due to higher observed disease-induced mortality in the younger age class (Newman *et al.* 2002). The shorter disease duration in juveniles means that mange is likely maintained in the population by older individuals; this is plausible, because adults have a lower disease-induced mortality rate and have a longer time to become infected compared to younger individuals. Combining data on all individuals younger than one year is a simplification that hinders insight into the underlying mechanisms behind high mange transmission in juveniles; this age classification encompasses a range of stages, with encounter rates changing during this time (Robertson *et al.* 2000) and immunological development also likely. Prevalence data were not sufficiently detailed, however, to add a pre-emergent age class. Although the age-specific transmission rates in the models in this analysis are simplifications of the true contact patterns in this social species, the addition of more complex contact rates, such as separate rates for between age class transmission, β_{ja}, and $\beta_{aa,}$ and age-specific disease-induced mortality rates, are not supported by the data.

Parameter estimates derived from the analyses were subject to a high degree of uncertainty. This could in part stem from sampling error. Identifying sources of error, such as from undiagnosed or misdiagnosed cases (Lloyd-Smith 2007) remains an issue for detecting mange infections (Pence & Ueckermann 2002). For example, capture rates of individuals with advanced mange infections may be low due to the disease reducing their ability to meet energetic demands. The inference methods used in this study are intended to avoid selecting overly complicated models due to sampling

error. Uncertainty in the transmission coefficients, β, and infectious period, γ, could also be due to unexplained variation in infection such as that which arises from individual variation in parasite load or susceptibility. In the context of mange, although densities of up to 5000 mites cm^{-2} have been reported for foxes (Little *et al.* 1998), inter-individual variation in parasite load is undocumented. So it is unknown if the rate of transmission is dependent on a density threshold of mites or if there is a relationship between the duration and the intensity of infection. In diseases with high individual variation in susceptibility, a higher than average number of secondary infections are caused by individuals known as "superspreaders" (Lloyd-Smith *et al.* 2005b). In such cases, diseases are either subject to infrequent but explosive epidemics or die out rapidly, as observed in SARS (Lloyd-Smith *et al.* 2005b). There is evidently a need for further analysis into individual infectiousness, and insight into parameter estimates could be gained from stochastic simulation models in which such heterogeneities in prevalence can be included.

A further source of variation causing potential uncertainty in parameter estimates of γ, is the potential for individual variation in resistance to mange. For example, longer survival of infected individuals and a degree of recovery among the adult population during the enzootic phase (S Harris *pers. comm.*), suggests some adaptation to the disease in the Bristol population. Since not all class I infections progressed to class II during the enzootic phase (S Harris *pers. comm*.), combining data on these mange classes increases the uncertainty in estimates of the infectious period. Long-term adaptation to mange has been demonstrated by serological studies in a Danish fox population (Davidson *et al.* 2008) and genetic resistance was supported in a simulation of mange in a coyote population, indicating the potential importance of the evolution of resistance for this disease (Leung & Grenfell 2003). Therefore, modelling immunity as a mechanism of long-term persistence of mange in this urban fox population would be worthwhile.

5.4.3 Sociality and disease transmission

The importance of social contacts for mange transmission in the Bristol fox population is supported by the SEI modelling approach in this study, as the findings are most

consistent with frequency-dependence as a transmission mechanism. This is in comparison to the good fit of density-dependent transmission for analogous models of mange in chamois (Lunelli 2010), a less social species than foxes. The fact that density- and frequency-dependent transmission mechanisms are indicated for the same disease in different species probably results from the differing sociality of chamois and this population of red foxes. Differing levels of sociality are implicitly incorporated into compartment models, as contact rates are included in the transmission coefficient β. Indeed, the substantial influence of sociality in disease transmission has been extensively reviewed, highlighting that while group size can be a predictor of infection risk, this relationship is often confounded by social contacts and territoriality (Altizer *et al.* 2003b). Inter-group contact rates were found to determine the spread of canine distemper virus in a multi-host carnivore community; lions *Panthera leo,* a species with low intraspecific inter-group transmission, experienced a greater vulnerability to canine distemper that, in the absence of species with higher inter-group contact rates such as spotted hyaenas *Crocuta crocuta*, may have died out (Craft *et al.* 2008). The role of a stable social structure has been emphasised in inhibiting the spread of bovine Tb in badgers *Meles meles*, where the number of new cases was related to groups undergoing a reduction in size (Vicente *et al.* 2007). The degree of sociality in foxes, compared to other canids, is considered evolutionarily primitive (Baker *et al.* 2004), with low levels of contact even between group members (White & Harris 1994). The non-linear contact rates that lead to heterogeneous transmission risk in social species are a source of variation in prevalence data, and are contrary to the assumption of homogenous mixing in compartment models, thus limiting the ability of these models to incorporate complex social dynamics. Further, spatial behaviour is implicit in the transmission term, β, another limitation of compartment models. The potential importance for mange transmission of changes in territorial (Baker *et al.* 2000) and dispersal (Lindström 1992) behaviours, points towards the application of models with an explicit spatial component. Consequently, individual-based models may offer a more appropriate method to incorporate the social complexities required to describe between and within group mange dynamics in foxes.

5.5 Conclusion

This study provides the first estimates of β and R_0 for mange in a fox population. Despite uncertainty in the empirical data, age-specific heterogeneities in mange transmission were identified, with juveniles having a fourfold higher rate of transmission. Modelling suggests that frequency-dependent transmission is the dominant mechanism in this study population but the contribution of indirect transmission cannot be entirely discounted. The underlying contact rates that led to these results point towards sociality having a significant role in the transmission of mange in foxes. The epidemiological parameter estimates provide an important baseline for the construction of more complex models. Unravelling the mechanisms involved in the transmission of mange in this well-studied fox population highlights the importance of testing long-standing assumptions relating to disease transmission and will be of use for predicting the spread and control of this disease in both this and other susceptible species.

Chapter 6 An individual-based model of the Bristol fox population under high density conditions

6.1 Introduction

One of the challenges of describing disease dynamics in social species is the limited ability of epidemiological compartment models to incorporate processes including group interactions and dispersal (Lloyd-Smith *et al.* 2005a). Traditional epidemiological compartment models are also often unable to capture simultaneously the observed patterns in disease transmission and population density (Leung & Grenfell 2003, Kramer-Schadt *et al.* 2009). Compartment modelling of a sarcoptic mange *Sarcoptes scabiei* outbreak in an urban red fox *Vulpes vulpes* population (chapter 5), illustrated the difficulties in making predictions relating to population density in specific years. Further, the same modelling approach was insufficient to determine the influence of disease-induced behavioural changes in the spatial organisation of the population. Thus, there is a need to develop a model that more realistically captures changes in behaviour and spatial patterns in this fox population such as by simulating the effects of disease at an individual level.

The non-analytical framework of individual-based models (IBMs) (DeAngelis & Mooij 2005, Grimm & Railsback 2005) provides a mechanism to incorporate variables into ecological models that are important for describing sociality, such as dominance, group interactions, territoriality, and dispersal patterns. Specifically, as opposed to structured population-projection or compartment models where average parameter values are ascribed to groups of individuals, the assignment of values and behaviours at an individual level allows the properties of a system to emerge from IBMs (Railsback & Grimm 2011). In doing so, this method promotes model analysis and interpretation through a pattern-orientated approach (Wiegand *et al.* 2003). Here, emergent properties occurring at the individual and population level are compared to multiple observed patterns to ensure structural integrity and measure model performance (Swanack *et al.* 2009, Topping *et al.* 2010, Railsback & Johnson 2011). There is increasing recognition of the value of using IBMs to address issues of applied

importance (McLane *et al.* 2011), such as disease ecology, especially in social species (Haydon *et al.* 2002, Eisinger & Thulke 2008). However, because IBMs are typically more detailed than analytical population models they often require a larger number of parameters, increasing the sources of uncertainty. Thus, validating models with empirical data is vital for ensuring that model parameters remain biologically meaningful (Hilborn & Mangel 1997). In this context, it is useful to determine whether an IBM can better describe population and disease dynamics during the mange outbreak in the Bristol fox population than the compartment models used previously (chapter 5).

The red fox *Vulpes vulpes* is an important predator and disease host (Baker *et al.* 2008) and thus, is often subject to considerable management and research (Baker *et al.* 2001b, Saunders *et al.* 2010). Foxes have an evolutionarily rudimentary social system (Cavallini 1996, Baker *et al.* 1998): "spatial" groups share a territory, benefiting from alloparental care and territorial defence, rather than from cooperative foraging (Macdonald 1983). In Bristol, the fox population has been studied continuously for over 30 years (Whiteside *et al.* 2011), with much insight gained into the costs and benefits of sociality in this carnivore (White & Harris 1994, Baker *et al.* 1998, Baker & Harris 2000, Baker *et al.* 2004, Iossa *et al.* 2008, Soulsbury *et al.* 2008a, Giuggioli *et al.* 2011, Soulsbury *et al.* 2011, Whiteside *et al.* 2011). Prior to a mange outbreak in 1994, the Bristol fox population reached remarkably high densities, resulting in part from an increase in scavenged food and a decrease in territory size (Baker *et al.* 2000). The changes in density affected social processes in this population; for instance, during this high density period, males were most likely to become dominant from dispersal whereas philopatric individuals had a greater chance of attaining dominance during low density conditions (Iossa *et al.* 2009). Previous models of the Bristol fox population were developed prior to the high density period, and were not evaluated using a pattern-orientated approach (Trewhella & Harris 1988, Smith & Harris 1991), which improves structural integrity and measures model performance by comparing emergent properties occurring at the individual and population level to multiple observed patterns (Swanack *et al.* 2009, Topping *et al.* 2010, Railsback & Johnson 2011). Given the influence of density on behavioural strategies and that the high

density conditions prior to the mange epizootic, it is useful to build a model that accurately describes the population dynamics during this high density period.

Here, an IBM was developed using empirical estimates of social and demographic processes in this well-studied urban fox population. A pattern-orientated approach (Wiegand *et al.* 2003) was used to evaluate the ability of the model to replicate empirical demographic patterns in the high density Bristol fox population before the outbreak of mange. Following model validation, the biological processes that caused the greatest variation in the emergent properties of this model were identified. In the following chapter (7), this model will be applied to investigate the outbreak of mange that occurred in the Bristol fox population.

6.2 Methods

6.2.1 Study population and data

Monitoring of the Bristol fox population began in 1977 (Harris 1981) and has been continuous since that time. Demographic parameters were initially estimated for the population over an area of 14 km^2 of Bristol city (Harris & Smith 1987, Harris & Trewhella 1988, Trewhella *et al.* 1988), with particular attention subsequently concentrated on a smaller number of social groups covering 1.5 km^2 within this population (Baker *et al.* 1998, Baker *et al.* 2004). Demographic and social parameters used for the model were compiled from this long-term study, with data collected through methods including mortality, capture-mark-recapture and radio-telemetry (for data collection protocols see Soulsbury *et al.* 2011, Whiteside *et al.* 2011 and references therein). Parameter values used in the model relate to a period of high density prior to a devastating sarcoptic mange epizootic in 1994 (Baker *et al.* 2000).

6.2.2 Model description

6.2.2.1 Overview

The model

The model description follows the ODD protocol (Overview, Design concepts and Details) for IBMs (Grimm *et al.* 2006). The aim of this protocol is to provide a standardised structure for describing IBMs that aids model understanding and replication. The model was implemented in R 2.14.0 (R Development Core Team 2011).

Purpose

The model was designed to determine whether the current knowledge of fox demography and social behaviour is sufficient to replicate empirical demographic patterns of a high density urban fox population.

State variables and scales

The three entities included in the model were individuals, groups and the population. Individuals were characterised by the state variables: sex; age; social status; and group

membership. A group was defined as a reproductive unit that contained a dominant pair, as well as cubs, subadults, and subordinate adults of both sexes. Cubs were defined as individuals less than 6 months old; subadults referred to individuals older than 6 months, but less than 12 months; and adults were individuals older than 12 months (Trewhella & Harris 1988). A further designation used in this chapter is that of juveniles. Juvenile referred to all individuals of less than one year and, thus, includes both cubs and subadults (Trewhella & Harris 1988). Social status was defined as either subordinate or dominant. A subadult individual younger than the age of 12 months could become a dominant individual through the *dispersal* process (see section 6.2.2.3). Time proceeded in discrete steps of one month. Space was included in the model as a grid of territories based on a coordinate system. Territories were defined as the range of a group, following observations that individuals belonging to a group live within a group boundary, whilst having separate home ranges within this area (White *et al.* 1996). The composition of the simulated groups was recorded for each territory. The population in the model was characterised by the size of the total area, and the number of territories. The total area was specified according to the study area of the Bristol population.

Process overview and scheduling

Each individual in the population was followed through its entire lifetime. Within each year and month, the processes below were simulated in a biologically meaningful and computationally practical order for each of the given entities (see Figure 6.1). Individuals and groups were processed in a randomised sequence each month.

6.2.2.2 Design concepts

Emergence

Fox population and group dynamics emerged from the behaviour of individuals, although individual behaviour was entirely imposed by probabilistic empirical rules. Emergent properties included the postbreeding adult population density, adult group size, proportion of juveniles in the population, sex-specific probability of becoming dominant via dispersal or philopatry and the number of years that dominant individuals retained their status (tenure).

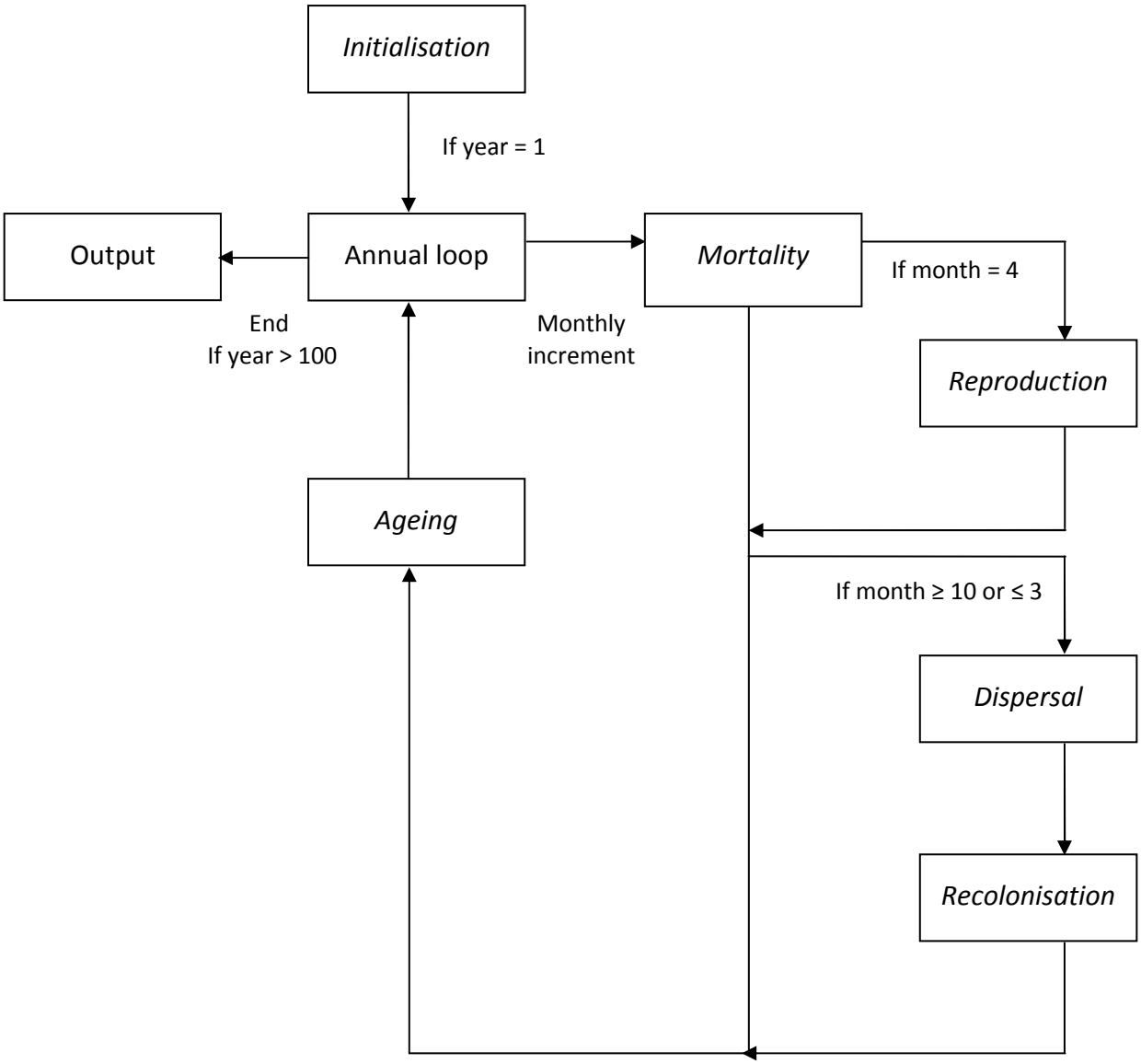

Figure 6.1. Flow chart for scheduling of the processes applied to individuals and groups in the model. The rules defining the processes (in italics) are described in Section 6.2.2.2 and 6.2.2.3.

Interaction

Three types of interaction were modelled implicitly: (i) individuals dispersing and joining a group missing a dominant of the same sex, (ii) resident subordinates or subadults replacing a missing dominant of the same sex, (iii) subordinates replacing a missing dominant of a neighbouring group.

Stochasticity

All demographic and behavioural parameters describing binary processes in the model were interpreted as probabilities using Bernoulli trials to include demographic stochasticity.

Collectives

Individuals were organised into groups that represented independent entities, with some processes explicitly related to these collectives (e.g. *reproduction*).

Observation

For model testing, modelled individual life histories were observed process by process (Grimm & Railsback 2005). To validate the model, characteristic patterns in population and group dynamics were recorded to determine whether the model produced observed patterns at different hierarchical levels of the system, including patterns not explicitly considered in model construction.

Initialisation

Simulations started in April (month 4) with specified numbers of dominant and subordinate individuals per group, and specified numbers of groups in the total population. One male and female per group were randomly selected as dominants. Sex and age in individual groups was randomly assigned: the probability of being male was 0.5 and age was uniformly distributed from 1 to 4 years for dominants, and 1 to 2 years for subordinates.

6.2.2.3 Submodels

Tables 6.1 and 6.2 provide values used in the model for the parameters described in the following processes.

Table 6.1. Monthly mortality and movement probabilities used in the model. Mortality and dispersal values reproduced from Smith and Harris (1991).

	Mortality				***Dispersal***	
	Males		*Females*		*Female*	*Male*
	Adults	*Juveniles*	*Adults*	*Juveniles*	*Juveniles*	*Juveniles*
April	0.035	0.137	0.041	0.129	0.000	0.000
May	0.039	0.045	0.055	0.052	0.000	0.000
June	0.020	0.040	0.035	0.067	0.000	0.000
July	0.028	0.048	0.025	0.037	0.000	0.000
August	0.014	0.036	0.023	0.042	0.000	0.000
September	0.039	0.035	0.034	0.037	0.000	0.000
October	0.036	0.044	0.044	0.044	0.030	0.068
November	0.046	0.044	0.049	0.032	0.030	0.102
December	0.041	0.039	0.035	0.039	0.136	0.182
January	0.121	0.062	0.062	0.025	0.045	0.159
February	0.069	0.032	0.041	0.034	0.045	0.102
March	0.029	0.035	0.036	0.030	0.030	0.057

Table 6.2. Parameter definitions and values used in the model. Parameters were estimated from the literature (Baker *et al.* 2004, Soulsbury *et al.* 2007, S. Harris *unpublished data*).

Parameter definition	***Parameter value***
Total area (km^2)	14.00
Territory size (km^2)	0.18
Initial group size	7.00
Mean litter size	4.00
Annual probability of dominant female breeding	1.00
Annual probability of subordinate female breeding	0.56

Mortality

During each month of the simulation, each individual had an observed monthly sex- and age-specific probability of natural mortality. Since subordinate adult females are known to help rear offspring (Baker *et al.* 2004), only if all the adult females in a group died, did any remaining cubs aged less than two months also die. In cases where a dominant individual died, it was replaced subject to the process of replacement (described in the *dispersal* and *recolonisation* submodels).

Reproduction

Both males and females could reproduce from one year of age. The probability of reproduction within a group in a given year was determined according to the probability of a female breeding. Breeding was not restricted to dominant females; given an opportunity, subordinate females reproduce while remaining in their group (Baker *et al.* 2004). Both dominant and subordinate females mate with extra-territorial males (Baker *et al.* 2004) but, for the purposes of this model, breeding was restricted to pairs on the same territory. Thus, a litter was added annually to each group according to specified probabilities for a dominant female if a dominant male was present and for subordinate females, given the presence of a male of any social ranking. Litter size was randomly selected from a shifted Poisson distribution (Chapter 3). Each cub's gender was allocated randomly, based on an observed probability of 0.5 that the cub was male.

Dispersal

Subadults were assigned a sex-specific monthly probability of dispersing, matching the observed proportion of animals leaving their natal group (Smith & Harris 1991). All potential dispersers had a chance of moving to a new group to attain dominance. All territories missing a dominant individual were identified and a replacement individual of the same sex was matched at random from the disperser pool. Individuals that were in the disperser pool were not allowed to recolonise their natal territory; recolonisation by philopatric individuals occurred during the *recolonisation* submodel. Dispersal distance was not explicitly modelled, as the size of the total area modelled was smaller than the maximum observed dispersal distance (Trewhella *et al.* 1988).

Because dispersal often takes place over a prolonged period (Woollard & Harris 1990), the dispersal process was assumed to take place over an entire time-step. Dispersers that did not attain dominance by replacement were removed from the population. This rule conforms to observed patterns, because dispersers do not remain in an area to form temporary territories or single-sex groups (S. Harris *pers. comm.*).

Recolonisation

If, in a given month, a dominant position remained unoccupied after the dispersal process, an individual of the same sex was randomly selected from the following categories (in order of preference): (i) subadults of the focal group, because individuals typically attain dominance at a young age, with a large proportion consisting of philopatric individuals (Baker *et al.* 1998); (ii) subordinate adults of the focal group; (iii) neighbouring subordinate adults.

Ageing

At each time-step, the age of all individuals increased by one month. Survival was capped at a maximum age. Specifically, to allow for social differences in survival rates (Baker *et al.* 1998), the maximum age of subordinates and dominants was 3 and 5 years, respectively.

6.2.2.4 Model validation and calibration

Model validation involved evaluating the model properties under the initial high population density conditions using a pattern-orientated approach. Population-level emergent properties were recorded from 200 model replicates, each lasting for 100 years, and compared to empirical estimates of these patterns (section 6.2.2.2). In this way, the ability of the initial parameter values to replicate the observed characteristics of the system and the need for calibration of these parameters could be assessed. Calibration is a widely used approach of model parameterisation (van Winkle *et al.* 1998, Beaudouin *et al.* 2008, Stillman & Goss-Custard 2010) to search a range of plausible parameter values to match multiple observed patterns (Railsback & Grimm 2011).

6.2.2.5 Sensitivity analysis

A sensitivity analysis is used to identify parameters that have the most impact on model outcomes and have the most associated uncertainty (van Winkle *et al.* 1998, Wiegand *et al.* 2004). Here, a sensitivity analysis tested the effect on emergent properties of varying nine parameters (age- and sex-specific survival, probability of breeding according to social status, litter size, and sex-specific probability of dispersal). The sensitivity analysis was conducted by independently varying parameter values ± 10% of their mean value, with the exception of the probability with which dominant females breed, the empirical estimate of which was 1.0, such that only a reduction in this parameter could be tested (Table 6.2). Thus, the total number of parameter changes run for each of the eight emergent properties was 17, yielding 136 iterations in total. The mean ratio of change between the emergent properties and empirical estimates was determined for 200 replicates for each of the iterations.

6.3 Results

6.3.1 Model testing

The emergent patterns reproduced by the model (labelled "predicted") during high density conditions are in agreement with empirical estimates from the long-term study of the Bristol fox population (labelled "observed") (Table 6.3). The one exception to this was the mean tenure of dominants which, nevertheless, showed a similar range and order of magnitude to that observed in the field study. Tenure was a mean value across all dominant individuals, because no significant sex differences were observed in the field study (Baker *et al.* 1998). The observed discrepancy could well be attributed to sampling uncertainty, owing to small sample sizes from the field study. Overall, the validation suggests that the model was proficient at describing the dynamics of the Bristol fox population and, therefore, that the initial input parameters did not require calibration.

Table 6.3. Comparison of predicted and observed estimates for variables characterising the fox population under high density conditions. Model values are emergent properties and are not imposed onto the model. The range of observed parameter estimates and mean range of predicted parameter values from 200 model replicates are indicated in parentheses. Empirical estimates are from Harris and Smith, 1987; Baker *et al.* 1998; Baker *et al.* 2000 and Soulsbury *et al.* 2008.

	Parameter value	
	Predicted	***Observed***
Mean adult population density (km^{-2})	36.67 (28.04–41.21)	37.00
Mean adult group size*	6.60 (1.86–14.02)	6.57 (2–10)
Mean proportion of juveniles	0.51 (0.47–0.56)	0.52
Annual probability of attaining dominance through dispersal	0.42 (0.23–0.62) 0.36 (0.23–0.54)	0.00–0.67 (female) 0.17–0.67 (male)
Annual probability of attaining dominance through philopatry	0.14 (0.06–0.25) 0.04 (0.00–0.12)	0.14–0.45 (female) 0.00–0.37 (male)
Mean tenure of dominant individuals (years)	1.80 (0.16–4.56)	2.37 (1– 5)

*Although group size is a "redundant pattern", in that it is not independent of population density, it is included here to illustrate the structural realism of the model at the group level.

6.3.2 Sensitivity analysis

Sensitivity analysis revealed that the model was robust to variation in model parameters and identified the emergent properties most sensitive to parameter variation (Figure 6.2). There was less than a 3% change between the mean model and empirical estimates of emergent properties for 113 of the 136 sensitivity iterations, with survival rates and litter size consistently the least sensitive parameters (Figure 6.2A-H). The model was most sensitive to variation in the dispersal and probability of breeding parameters. The emergent properties that responded most to changes in parameter values were those relating to attaining dominance through philopatry (Figure 6.2F-G). The probability of males attaining dominance through philopatry decreased by 20% and increased by 15% when the male dispersal probability was increased and decreased by 10%, respectively (Figure 6.2G). The equivalent changes in the dispersal probability of females had a much smaller effect, leading to a 1% decrease or a 5% increase in females attaining dominance through philopatry, respectively (Figure 6.2F). Increasing the probability of subordinate breeding lead to a 13% reduction in males attaining dominance through philopatry and a 6% decrease in the same property for females, while a decrease in the breeding probability of subordinate females resulted in a 16% increase in males becoming dominant through philopatry, and an 8% increase for females (Figure 6.2F-G).

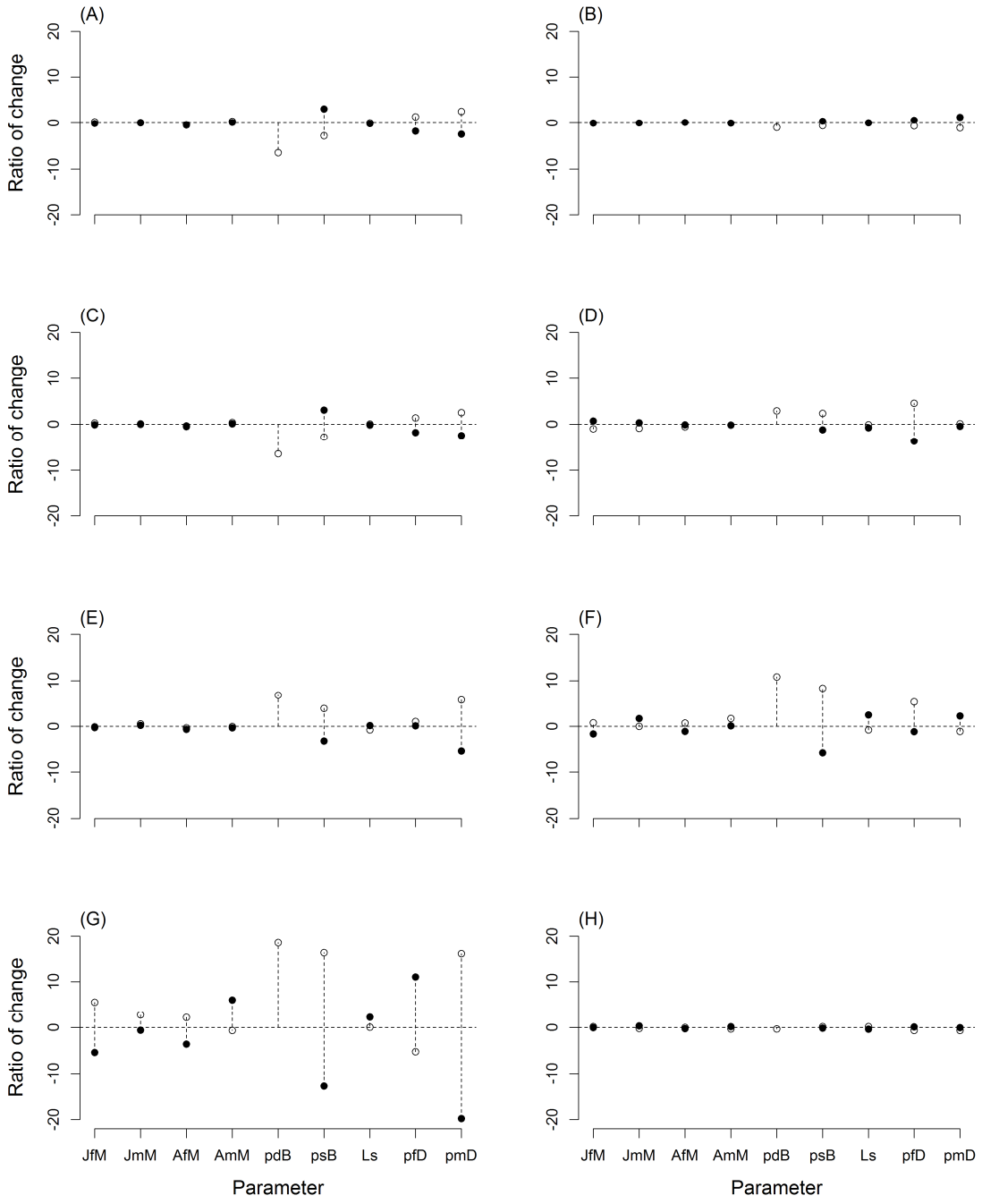

Figure 6.2. Results of the sensitivity analysis, showing the ratio of change between the mean emergent property and observed values (Table 6.3), when varying model parameters by ± 10% of their initial value. Emergent properties are: (A) adult density; (B) proportion of juveniles; (C) adult group size; (D) females attaining dominance through dispersal; (E) males attaining dominance through dispersal; (F) females attaining dominance through philopatry; (G) males attaining dominance through philopatry; (H) tenure of dominants (male and female). Open circles indicate a decrease in the parameter value and closed circles an increase in the parameter value. Parameter codes are: JfM = Juvenile female mortality; JmM = juvenile male mortality; AfM = adult female mortality; AmM = adult male mortality; pdB = probability of dominant breeding (only -10%); psB = probability of subordinate breeding; Ls = litter size; pfD = probability of female dispersal; pmD = probability of male dispersal.

6.4 Discussion

In this chapter, emergent social and demographic properties of a high density urban fox population were successfully described by an IBM parameterised with empirical data. Indeed, the results established that calibration of the initial parameter values was not required. Parameters identified by sensitivity analysis are first discussed in light of their associated uncertainty and importance for shaping population dynamics. The relevance of incorporating social structure using individual-based modelling is then considered in the context of the management concerns facing fox populations.

6.4.1 Model ability to replicate emergent properties of a high density fox population

IBMs can be meaningfully evaluated by comparing multiple observed patterns in a populations' dynamics with properties that emerge from models (Railsback & Grimm 2011). The validation procedure in this study demonstrated the ability of the model to replicate key emergent characteristics of a high density urban fox population, suggesting the detection of dynamics processes that were not imposed on the model. This pattern-orientated approach ensures that IBMs capture characteristics at different hierarchical levels of a system to avoid overfitting of models (Latombe *et al.* 2011). That these fundamental properties were captured at different hierarchical levels, without calibration, provides confidence in the structural realism of the model and in the certainty of key parameter estimates.

The validation of the model was corroborated by a sensitivity analysis, which illustrated that model properties were largely robust to changes in parameter values, especially properties at the population level. Parameters with high sensitivity are of particular interest, both for their importance for shaping model processes and as indicators of highly uncertain empirical estimates (Railsback & Grimm 2011). The sensitivity analysis identified that model outcomes, especially those measured at the level of individual behaviour, were more sensitive to behavioural parameters (e.g. dispersal) than to demographic parameters (e.g. mortality). This suggests that the effects of mortality on population dynamics might be buffered by social processes such

as recolonisation by dispersing individuals. Litter size has previously been shown to have a limited effect on population model outcomes, due in part to the limited variance of this parameter (chapter 3). The sensitivity results pointed towards the importance of breeding females for determining social processes at the individual-level. These breeding individuals are determined largely by reproductive suppression and the costs and benefits of philopatry (Baker *et al.* 2004, Iossa *et al.* 2009). In particular, evidence of reproduction in subordinate female foxes has only recently been verified in the Bristol fox population (Baker *et al.* 2004) and sensitivity analysis highlighted the known uncertainty in empirical estimates of reproduction in these individuals. The sensitivity to dispersal parameters is consistent with other simulations of fox populations (Rushton *et al.* 2006) and underscores the importance of dispersal behaviour for shaping social processes (Soulsbury *et al.* 2008a). The emergent properties most likely to respond to changes in parameter values were those relating to attaining dominance. Uncertainty in these empirical estimates is high, resulting from difficulties in establishing the fate of dispersing and philopatric individuals (Baker *et al.* 1998), and from the variable effects of density (Iossa *et al.* 2009). Further insight could be gained from conducting a sensitivity analysis that simulates the full range of possible conditions by sampling parameters from probability distributions based on known ranges (e.g. Shirley *et al.* 2003) or estimated as in chapter 2.

That the model required relatively few demographic and social parameters to describe accurately the population dynamics of the focal population, supports its use for exploring specific ecological and evolutionary questions, such as those processes relating to sociality or disease dynamics. For example, the model could be modified easily to include inter- and intra-group disease transmission, an important but often uncertain aspect of disease ecology in many species (Altizer *et al.* 2003b). Indeed, this model will be used to explore the dynamics of a mange outbreak that significantly reduced the Bristol fox population in the 1990s (Soulsbury *et al.* 2007). A useful attribute of IBMs is the potential to include decision rules that allow for the adaptive behaviour of individuals and thus the optimisation of fitness (Stephens *et al.* 2002a, McLane *et al.* 2011). Thus, further analyses could incorporate adaptive behaviour to model the fitness components of dispersal and philopatry in fox populations, such as

the relative opportunities for reproduction that are important for determining group formation (Soulsbury *et al.* 2008a). The model in this study therefore provides a useful foundation for future investigations of ecological and evolutionary importance. Future work should aim to validate this model using an independent data set from another population to ensure structural integrity (Railsback & Grimm 2011).

6.4.2 Management implications of modelling social structure in fox populations

Understanding how social structure affects a species' population dynamics is important for determining the success of management actions. Harvests of an alpine marmot *Marmota marmota* population were predicted to be unsustainable when sociality was not accounted for, possibly due to the disruption of dispersal processes (Stephens *et al.* 2002b). Vucetich *et al.* (1997) proposed that management of a population of grey wolves *Canis lupus* should be directed towards packs rather than individuals, since the influence of demographic stochasticity on extinction risk decreases with the number of groups, not population size. An important management issue for many fox populations concerns their status as predators of economic importance (Baker *et al.* 2008). Given the often variable success of controlling fox populations, such as invasive populations in Australia (Saunders *et al.* 2010), insight into fox social structure should be applied to refine management (Newsome 1995). Further analyses are required to determine whether failing to account for the potential importance of dispersal and probability of subordinate breeding for population dynamics, which is especially difficult in analytical population models, could lead to misinformed management predictions.

Diseases such as rabies, sarcoptic mange, and *Echinococcus multilocularis* (Chautan *et al.* 2000, Deplazes *et al.* 2004, Soulsbury *et al.* 2007) are management issues of concern for fox populations worldwide. An understanding of social structure is important for understanding disease transmission, because variation caused by socially-determined contact rates is likely to cause spatiotemporal disease dynamics different to those species exhibiting less sociality. Eisinger and Thulke (2008) demonstrated that the density-invasion threshold required for rabies eradication in foxes was overestimated if models did not account for the spatial structure of groups.

For many species exhibiting social structuring, disease transmission may not be density-dependent (McCallum *et al.* 2009, Johnson *et al.* 2011, Langwig *et al.* 2012), with implications for defining disease invasion thresholds (Lloyd-Smith *et al.* 2005a). Thus, IBMs offer a useful approach to predicting disease spread when group-structuring influences disease transmission. Indeed, by incorporating social structure, IBMs were able to describe the disease dynamics of mange in coyotes *Canis latrans* (Leung & Grenfell 2003) and rabies in foxes (Eisinger & Thulke 2008) better than traditional analytical epidemiological models. The model in this study provides a mechanism to incorporate processes such as recolonisation, extra-territorial reproductive movement and inter-group contact that could provide meaningful information for refining the management of disease in fox populations.

6.5 Conclusion

IBMs are often appropriate for describing social structure, such as territoriality and group formation, which analytical population models are less able to capture. In this study, an individual-based simulation using empirically-derived data was able to reproduce emergent properties of an urban fox population. Using multiple patterns for model validation, the ability of this model to describe demographic and social patterns during a period of high population density was demonstrated. Sensitivity analysis revealed that parameters relating to attaining dominance and probability of subordinate females breeding were associated with the most uncertainty, while pointing towards the potential importance of these parameters for shaping social processes. These results are consistent with empirical observations. Understanding the influence of social structure on population dynamics is important for many management issues.

Chapter 7 Sarcoptic mange *Sarcoptes scabiei* in a red fox *Vulpes vulpes* population: persistence, recovery and sociality

7.1 Introduction

Infectious diseases are recognised as a major driving force in host population dynamics (Tompkins *et al.* 2002) and evolutionary processes (Altizer *et al.* 2003a). Epizootic outbreaks can cause significant population declines and, if neither host nor pathogen is driven to extinction, disease can persist in the population indefinitely at enzootic levels (Keeling & Rohani 2008). Describing these temporal dynamics requires knowledge of the factors driving disease transmission and population recovery. However, understanding these processes is demanding because of host and parasite dynamics at the individual and population levels (Sheldon & Verhulst 1996, Altizer *et al.* 2003b). For example, failing to account for social interactions can result in misguided estimates of the probability of disease invasion and the rate of transmission (Cross *et al.* 2005, Smith *et al.* 2009c). At the population level, rapid evolution of traits that promote host-parasite coexistence such as immunity (Bonneaud *et al.* 2011, Robinson *et al.* 2012), allows populations to recover and disease to persist under enzootic conditions. Thus, a full understanding of disease dynamics requires insight into the ecological interactions and evolutionary changes acting at multiple scales.

Sarcoptic mange, caused by the mite *Sarcoptes scabiei* (Arlian 1989, Bornstein *et al.* 2001), is a highly infectious disease recorded in over 100 domestic and wild mammalian host species, many of which are of management concern (Pence & Ueckermann 2002). Mange outbreaks exhibit cycles; epizootics can occur every 30 to 40 years (Pence & Windberg 1994), drastically reducing some populations (Rossi *et al.* 2007, Soulsbury *et al.* 2007) while having little effect on others (Pence *et al.* 1983, Rossi *et al.* 2007). Often, an enzootic phase follows, with the disease remaining in the population for up to 50 years (Pence *et al.* 1983). The red fox *Vulpes vulpes* is a widespread canid that is an important host of many diseases, including mange (Soulsbury *et al.* 2007), *Echinococcus multilocularis* (Deplazes *et al.* 2004) and rabies (Chautan *et al.* 2000). Foxes have also been implicated as reservoirs of mange for a

number of threatened hosts (Ryser-Degiorgis *et al.* 2002, Oleaga *et al.* 2011). Mange has caused dramatic population declines in several fox populations (Lindström & Morner 1985, Forchhammer & Asferg 2000, Soulsbury *et al.* 2007), with population recovery taking up to 20 years (Lindström *et al.* 1994, Soulsbury *et al.* 2007). Despite being a disease of considerable importance, our understanding of mange dynamics in wild populations remains limited (but see Leung & Grenfell 2003, Lunelli 2010, chapter 5).

An urban fox population in Bristol experienced a mange epizootic from 1994 to 1996 (Baker *et al.* 2000). Prior to the epizootic, this fox population had remarkably high densities, resulting in part from an increase in scavenged food and a decrease in territory size (Baker *et al.* 2000). The initial spread of mange was rapid, causing the population to decline by over 90% in two years (Soulsbury *et al.* 2007). Population recovery was slow and, despite the low population density, mange has persisted at enzootic levels (Soulsbury *et al.* 2007, S. Harris *pers. comm*.). Transmission mechanisms of mange in this population are unclear and a number of behavioural and evolutionary processes may be required to explain the epizootic and enzootic phases. While dispersers may be important for mange transmission (Lindström 1992, Pence & Windberg 1994), the mechanism for infectious contact through dispersal remains undetermined. Unlike in rabies, which has a seasonal peak in infection related to dispersal (Wandeler 1980), mange did not exhibit such temporal patterns in Bristol (S. Harris, *pers.comm.*). Recent work suggests that dispersing foxes avoid the core range of territorial individuals (Soulsbury *et al.* 2011), limiting the opportunity for direct disease transmission.

During the mange epizootic in Bristol, new social groups did not form in territories that became vacant due to disease mortality; rather, neighbouring groups expanded to encompass these spaces (Baker *et al.* 2000). Stable territorial structure is predicted to reduce the spread of disease when levels of inter-group contact are low (Loehle 1995). When host behavioural patterns result in low contact rates, such as the social interactions observed in foxes (White & Harris 1994), direct transmission may not be sufficient to describe patterns of mange spread. The ability of mites to survive off the

host (Arlian *et al.* 1989) increases the potential for indirect mange transmission through contact with fomites, inanimate objects capable of conveying parasites. Indirect transmission occurs in other fox populations (Gerasimov 1958) and, in Bristol, is a potential transmission mechanism (inferred from the incidence of mange in domestic dogs during the epizootic; Soulsbury *et al.* 2007).

The long-term persistence of mange and a possible increase in tolerance to the disease during the enzootic phase indicates a possible role for genetic resistance in promoting population recovery (Soulsbury *et al.* 2007). Although empirical evidence of genetic resistance to mange remains uncertain (Arlian 1989), seroprevalence data suggest long-term adaptation to the disease in a Norwegian fox population (Davidson *et al.* 2008) and selection for resistance was supported by a simulation of a mange epizootic in coyotes *Canis latrans* (2003). In the same model, long-distance recolonisation of territories was required in addition to genetic resistance to allow the coyote population to recover fully (Leung & Grenfell 2003). It is unclear whether a similar recolonisation process occurred in the Bristol foxes during the mange outbreak. That the Bristol fox population has been studied for over 30 years (Whiteside *et al.* 2011) makes it a valuable source of data with which to analyse the dynamics of mange in a group-living species.

In chapter 5, traditional epidemiological compartment models provided meaningful insight into the transmission mechanisms of mange in the Bristol fox population but the limitations of using this approach were also identified. For example, it was difficult to make inferences about both prevalence and population density in specific years, and to account for social interactions. However, compartment models can be useful for initial investigations into disease systems (Smith *et al.* 2009a), as well as providing parameter estimates for, and forming components of, more complex models (Haydon *et al.* 2002, Craft *et al.* 2008). To address some of the limitations of compartment models, in chapter 6, an individual-based model (IBM) that incorporated sociality was developed to describe the Bristol fox population prior to the mange epizootic. Unlike compartment models, IBMs can be fitted not only using disease prevalence data, but also by comparing multiple observed patterns in a population's dynamics with

properties that emerge from the model (Railsback & Grimm 2011). This approach is useful given the considerable uncertainty associated with detecting infection in wild populations (Conner *et al.* 2000, McClintock *et al.* 2010). Here, a pattern-orientated approach was used to evaluate the ability of the IBM, developed in chapter 6, to reproduce the dynamics of mange in the Bristol fox population. Parameters used in this model were estimated both empirically and from compartment modelling (chapter 5). Specifically, this simulation was intended to determine the processes that are important for the spread of mange and the recovery of the population. In particular, the following questions were considered to explain the spread of mange: (1) Is direct transmission alone sufficient to describe mange spread and persistence during the epizootic and enzootic phases? (2) Are dispersing individuals important for the transmission of mange? (3) Does territory collapse increase the spread of mange during the epizootic? The processes important for population recovery were explored by asking: (4) Is there evidence that genetic resistance is required for population recovery? (5) Is the population able to recover without long-distance recolonisation?

7.2 Methods

7.2.1 Study population and data

Mange was introduced into the Bristol fox population in 1994 by a dispersing juvenile male returning to its natal group (Baker *et al.* 2000) and, to date, the disease has persisted in the population. Infected individuals were recorded from capture data or were recovered dead (n = 1662 records), from 1994 to 2010 (Soulsbury *et al.* 2007, S. Harris *pers. comm*.). Mange was classified according to the whether the progression of the disease was pre (Class I) or post (Class II) the development of hyperkeratosis, the crusty skin condition associated with mange (for details see Newman *et al.* 2002). Due to small sample sizes, the observed monthly prevalence was defined as the proportion of the combined class I and II infected individuals in the total sample. For model interpretation, prevalence was also defined as the total mean prevalence (1994 to 2010) and mean epizootic prevalence (1994 to 1996). Demographic rates of infected individuals, including survival and the probability of breeding, were recorded during the epizootic (Baker *et al.* 2000, Newman *et al.* 2002, Soulsbury *et al.* 2007). Annual adult population densities were estimated from capture-mark-recapture data (e.g. Baker *et al.* 2000, Whiteside *et al.* 2011) and four years with missing estimates (1996-97; 2000-01) were determined by linear interpolation. The significant population decline caused by the mange epizootic and the subsequent recovery resulted in conditions of relative low and high density, which were associated with behavioural changes (Iossa *et al.* 2009). Therefore, for model interpretation, it was biologically meaningful to provide density estimates for these periods of high and low density. Mean adult population density was thus estimated for the total period (1994 – 2010), the low density period, defined as 1994 to 2003, and the high density period, from 2004 to 2010, following previous studies (Iossa *et al.* 2009, Whiteside *et al.* 2011).

7.2.2 Model description

7.2.2.1 Overview

Model

The model is based on an IBM developed to describe the Bristol fox population prior to the mange epizootic. For a detailed description of this model, see chapter 6. The following sections describe the modifications to the model that were related to the addition of disease. The model was implemented in R 2.14.0 (R Development Core Team 2011).

Purpose

The model was designed to determine whether the current knowledge of fox demography and behaviour, and of mange epidemiology, is sufficient to replicate empirical patterns of mange spread during epizootic and enzootic phases. The model was also intended to identify mechanisms for population recovery in an urban fox population following this mange outbreak. The theoretical hypotheses proposed to explain these processes were evaluated by defining six model scenarios with specified structural modifications (see section 7.2.2.4).

State variables and scales

In addition to the state variables described in chapter 6, individuals were characterised by their infectious and immune status.

Process overview and scheduling

Within each year and month, processes applied to individuals and groups were simulated in the order depicted in Figure 7.1 for each of the given entities.

7.2.2.2 Design concepts

Emergence

Emergent properties considered here were the mean total prevalence, mean epizootic prevalence, mean adult population density, mean low and high population density (see section 7.2.1), disease persistence, and time of first territory collapse and expansion.

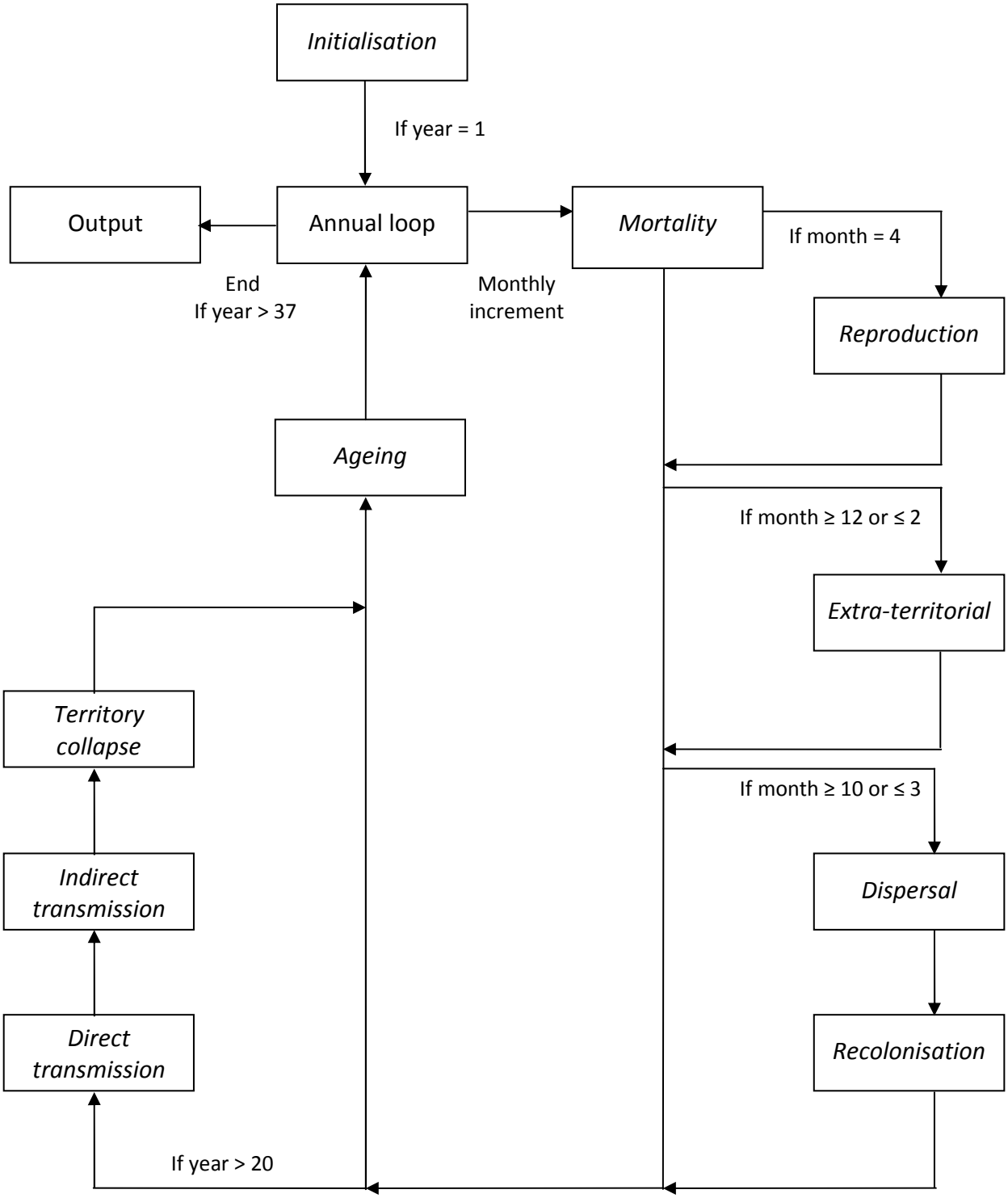

Figure 7.1. Flow chart for scheduling of the processes applied to individuals and groups in the model. The rules defining the processes (in italics) are described in sections 7.2.2.2 and 7.2.2.3 and sections 6.2.2.2 and 6.2.2.3 (chapter 6).

Interaction

Six types of interaction were modelled implicitly: (i) individuals dispersing and joining a group missing a dominant of the same sex; (ii) resident subordinates or subadults replacing a missing dominant of the same sex; (iii) subordinates replacing a missing dominant of a neighbouring group; (iv) inter-group contact; (v) intra-group contact, and (vi) extra-territorial male contact with groups.

Initialisation

Infection was introduced after the model had stabilised over a 20 year period. Following empirical observations (Baker *et al.* 2000), one infected subadult male joined a group at random in the spring (month 5) and was allowed to infect individuals on those territories it crossed entering the system, as described by processes in section 7.2.2.3.

7.2.2.3 Submodels

Table 7.1 provides values for the parameters described in the following processes that relate to disease dynamics and extra-territorial movement.

Mortality

Mortality of infected individuals was higher than that of healthy individuals by a specified, age-specific increment.

Reproduction

Mange infection influenced the probability of breeding, but not litter size (Soulsbury *et al.* 2007); thus, the probability of breeding was reduced to a specified probability for all infected females, regardless of status or age. If either parent was infected, the whole litter was assumed to be exposed to the disease.

Table 7.1. Parameter definitions and values used in the model. Parameters were estimated from the literature, chapter 5 or calibration (see Section 7.2.2.5).

Parameter definition	***Parameter value***	***Source***
Annual probability of infected female breeding	0.34	Soulsbury *et al.* 2007
Proportion of time spent in territory core, T_c	0.59	Soulsbury *et al.* 2011
Proportion of time spent in territory boundary, T_b	0.34	Soulsbury *et al.* 2011
Proportion of time spent in neighbouring territory core, T_{nc}	0.01	Soulsbury *et al.* 2011
Proportion of time spent in neighbouring territory boundary, T_{nb}	0.05	Soulsbury *et al.* 2011
Proportion of time disperser spent in territory core, T_{dc}	0.16	Soulsbury *et al.* 2011
Proportion of time disperser spent in territory boundary, T_{db}	0.84	Soulsbury *et al.* 2011
Proportion of time extra-territorial spent in territory core, T_{xc}	0.56	Soulsbury *et al.* 2011
Proportion of time extra-territorial spent in territory boundary, T_{xb}	0.44	Soulsbury *et al.* 2011
Mean distance for extra-territorial movement, *d* (km)	4.28	Soulsbury *et al.* 2011
Monthly extra-territorial adult male movement probabilities	0.05 (December) 0.80 (January) 0.15 (February)	Soulsbury *et al.* 2011
Annual disease-induced mortality, cubs	0.95	Soulsbury *et al.* 2007
Annual disease-induced mortality, subadults	0.90	Soulsbury *et al.* 2007
Annual disease-induced mortality, adults	0.75	Soulsbury *et al.* 2007
Infection constant, α	-	Calibration
Monthly rate of intra-group juvenile disease transmission, β_j	6.80 (2.61-11.34)	Chapter 5/ Calibration
Monthly rate of intra-group adult disease transmission, β_a	1.70 (0.96 - 4.80)	Chapter 5/ Calibration
Monthly rate of indirect transmission via fomite load, ε	-	Calibration
Initial proportion of population with resistance allele, v	-	Calibration
Infectious period (months) of individual with resistance allele, τ	-	Calibration

Dispersal

The number of territories crossed by a dispersing individual to reach its destination was estimated by assuming linear dispersal through the landscape. This estimate was then used to determine contact probabilities for disease transmission. Dispersal of infected individuals was observed to be negligible once symptoms became apparent (S. Harris *pers. comm.*); therefore, only uninfected or exposed individuals were allowed to disperse. However, dispersing individuals could become exposed through contact with infected groups.

Recolonisation

Only uninfected or exposed individuals were allowed to become dominant. Following observations that new groups were not formed during the epizootic (Baker *et al.* 2000), groups that died out completely during this period were not recolonised. After the epizootic, new groups could form once all vacant dominant positions in existing groups were filled, through recolonisation of empty territories via *dispersal* and the previously described recolonisation processes (i to iii, described in section 6.2.2.3): (i) subadults of the focal group; (ii) subordinate adults of the focal group; (iii) neighbouring subordinate adults. In addition to the recolonisation processes (i) to (iii), an additional process (iv) included in *Scenario 6* (section 7.2.2.4) allowed any subordinate adult to become dominant from any territory in the landscape.

Extra-territorial movement

During the breeding season, adult males search other territories for extrapair mating opportunities, before returning to their own group (Soulsbury *et al.* 2011). All adult males were allowed to make extra-territorial movements with a set monthly probability and a randomly assigned distance and direction. Distances moved, D, were randomly generated from a negative exponential probability density function, re^{-rD}, where the dispersal constant, r, was the reciprocal of d, the mean distance travelled (i.e. $r = 1/d$). The number of territories crossed was determined for estimating rates of disease transmission (see below), and the individual was assumed to return along the same route. Only uninfected males made extra-territorial movements, but these individuals could become exposed through contact with infected groups.

Direct transmission

Direct disease transmission was based on interactions as described in section 7.2.2.2. Contacts between group members and neighbouring groups, dispersers and extra-territorial males were determined according to the proportion of time that infected individuals spent in the core and boundary of a territory. The probability of encounters was assumed to increase linearly with the proportion of time foxes spent in the same area. The proportion of time that any two individuals came into contact was calculated according to the following equations (*sensu* Leung & Grenfell 2003). Contact between individuals of neighbouring groups, P_t, was defined as:

$$P_t = \frac{2(T_c T_{nc} + T_b T_{nb})}{N_n}, \quad (1)$$

where T_c is the time an individual spends in the core of their territory, T_b is the time an individual spends in the boundary of their territory, T_{nc} is the time an individual spends in the core of a neighbouring territory, T_{nb} is the time an individual spends in the boundary of a neighbouring territory. N_n is the number of adjacent neighbouring territories, here defined as a maximum of eight. Interactions between individuals crossing territories during *dispersal*, P_d were calculated as:

$$P_d = \frac{(T_c T_{dc} + T_b T_{db})^{\frac{t}{N_c}}}{N_c}, \quad (2)$$

where, T_{dc} is the time a disperser spends in the core of a territory it crosses and T_{db} is the time a disperse spends in the boundary of a territory. N_c was defined as the number of territories traversed in a monthly dispersal movement. An interaction between individuals crossing territories during *extra-territorial movement*, P_x, was defined as:

$$P_x = \frac{(T_c T_{xc} + T_b T_{xb})^{\frac{t}{2N_c}}}{N_c}, \quad (3)$$

where, T_{xc} is the time a male spends in the core of a territory it crosses and T_{xb} is the time a male spends in the boundary of a territory. N_c was defined as the number of territories crossed in a monthly extra-territorial movement, assuming that individuals returned along the same route. The proportion of time spent in contact between groups was then multiplied by a constant, α, to determine the probability of successful inter-group disease transmission:

$$P_{(inter)} = \alpha\left(1-\left[\prod_{n=1}^{N_I} 1-P_i\right]\right), \quad (4)$$

where N_I is the number of adjacent groups or groups crossed with infected individuals present, and P_i is the proportion of inter-group contact for an individual, corresponding to equations (1-3).

Intra-group disease transmission was modelled according to frequency-dependent SEI (Susceptible-Exposed-Infected) dynamics (Chapter 5). Susceptible individuals, $S_{x,i}$, of age class x, became exposed, $E_{x,i}$, according to the proportion of infected individuals, $I_{x,i}$, in a given group, i, of size N_i ($N_i = S_{x,i}+E_{x,i}+I_{x,i}$). Susceptible juveniles, $S_{j,i}$, became exposed at a rate:

$$C_j = \frac{(\beta_j I_{j,i} + \beta_j I_{a,i})S_{j,i}}{N_i}, \quad (5)$$

and susceptible adults, $S_{a,i}$ at a rate:

$$C_a = \frac{(\beta_a I_{a,i} + \beta_a I_{j,i})S_{a,i}}{N_i}, \quad (6)$$

where β_x is the age specific transmission coefficient for juveniles, j, or adults, a. The probability of infection for a given age class was then:

$$P_{(intra)} = 1-\exp(-C_x). \quad (7)$$

Once a successful disease contact was made, an individual was assigned an exposed status for one month before becoming infectious, to simulate the delay in the manifestation of mange symptoms (Bornstein *et al.* 2001). Disease could also move through the landscape through the *dispersal* of, or *recolonisation* by those exposed individuals that subsequently became infectious after attaining dominance on another territory. Due to their restricted movement from the natal den after birth (Robertson *et al.* 2000), cubs of less than two months became infected only through intra-group transmission.

Indirect transmission

This process was included in model scenarios, as defined in section 7.2.2.4. Indirect disease transmission was incorporated into the system through assigning each territory a "fomite load". This parameter was determined each month according to the number of infected individuals on a territory, and the proportion of time that neighbouring infected individuals spent in the territory boundary. This provided a mechanism to incorporate the time that infected individuals excreted mites into the environment. For simplicity, only time spent in the boundary of neighbouring territories was considered; the mechanism by which mites are transferred into the environment is unknown, but possible processes such as the inter-group sharing of dens are less likely to occur in territory cores. Thus, for each territory the fomite load was defined as:

$$F_t = I_r(T_c + T_b) + \sum_{i=1}^{N_n} I_i(T_b T_{nb}), \qquad (8)$$

where I_r is the number of infected members in the focal group and I_i is the number of infected individuals on a neighbouring territory. Each route travelled by an individual moving across the landscape was assigned a fomite load, determined according to the proportion of time an individual spent crossing the boundary of those territories with infected individuals. For the route travelled by dispersing individuals, the fomite load was determined by:

$$F_d = \sum_{i=1}^{N_c} I_i (T_b T_{db}), \quad (9)$$

were I_i was the number of infected individuals on the territories crossed by a dispersing individual. The fomite load for the route travelled by an extra-territorial male was then:

$$F_x = \sum_{i=1}^{N_c} I_i (T_b T_{xb}), \quad (10)$$

were I_i was the number of infected individuals on the territories crossed by a extra-territorial male. Individuals then became infected given the probability of contact with the fomites:

$$P_{(fom)} = 1 - \exp(-\varepsilon F_i), \quad (11)$$

where ε is the rate of successful infection through indirect transmission for a given fomite load, F_i.

Territory Collapse

Empirical data suggest that groups that died out during the epizootic were not recolonised, but neighbouring groups were observed to expand their territory to encompass the space created by the missing group (Baker *et al.* 2000). If a territory became vacant during the epizootic, a neighbouring group with uninfected or exposed individuals was randomly selected to expand into the empty territory space. During the time step following a territory collapse, a fomite load remained on the empty territory. If the group that expanded into the empty territory subsequently disappeared through mortality, the "expanded" territory space was restored to the original territories and neighbouring groups were given the opportunity at random to expand as described above. No limit was placed on the number of empty adjacent territories into which a group could expand.

Genetic resistance

This process was included in model scenarios, as defined in section 7.2.2.4 and applied to individuals during *mortality* and *reproduction*. Resistance was modelled as a dominant allele (Leung & Grenfell 2003), which a specified proportion, v, of the population were initially assumed to carry. The allele was passed onto offspring by either parent. Here, resistance influenced recovery rather than susceptibility (Gandon & Michalakis 2000). Infected individuals with the resistance allele recovered after a specified period, τ, while those without the allele died of the disease as specified above.

7.2.2.4 Model Scenarios

To investigate the mechanisms driving mange spread, persistence and population recovery, the following model scenarios were compared. For each scenario, specified submodels relating to disease transmission (section 7.2.2.3) were incorporated and the relevant set of parameters was calibrated (see section 7.2.2.5).

Scenario 1: *direct transmission.* The first disease model included only *direct transmission*. The parameter set that required calibration for this scenario was $\{\alpha, \beta_j, \beta_a\}$.

Scenario 2: indirect and direct transmission. In this model, *indirect transmission* was added to *Scenario 1*. The parameter set calibrated in this scenario was $\{\alpha, \beta_j, \beta_a, \varepsilon\}$.

Scenario 3: direct transmission and genetic resistance. In this model, *Scenario 1* was run with the inclusion of *genetic resistance*. The parameter set calibrated in this scenario was $\{\alpha, \beta_j, \beta_a, v, \tau\}$.

Scenario 4: direct and indirect transmission with genetic resistance. In this model, both the *indirect transmission* and *genetic resistance* submodels were incorporated into *Scenario 1*. Here, the parameter set $\{\alpha, \beta_j, \beta_a, \varepsilon, v, \tau\}$ was calibrated.

The following structural changes were then made to the best-fitting model scenario:

Scenario 5: Removal of territory collapse. To determine the effects of territorial changes on mange transmission, the model was run without the *territory collapse* process. In this model, empty groups could be filled during the epizootic by the *recolonisation* processes (i) to (iii).

Scenario 6: Inclusion of long-distance recolonisation. To consider whether recolonisation by non-neighbouring subordinates was required for population recovery, the model was run with the addition of long-distance *recolonisation* (see section 7.2.2.3).

7.2.2.5 Model validation and calibration

Model validation was conducted to evaluate the ability of the model to reproduce observed patterns of mange spread. In order to find plausible values for those parameters relating to disease transmission that could not be estimated directly, it was necessary to conduct calibration. Here, parameters associated with high uncertainty are systematically varied to determine the values generating patterns that best fit the predetermined criteria (Railsback & Grimm 2011). Empirical observations of the mean disease prevalence and adult population density were used as criteria for calibration. Parameter sets requiring calibration varied among model scenarios (see section 7.2.2.4). Models were evaluated for a range of values within the parameter set using Latin Hypercube Sampling (Vose 2008, pp. 59-62), which is an efficient way to sample equally within the parameter space. For each scenario, ten model replicates were run for each of the 200 parameter combinations that were tested for a given parameter set. Parameter values were selected according to the parameter combinations that reduced the distance between model output and empirical estimates of the chosen criteria. The data to which the model was fitted were characterised by the mean and variance of observed values, $\overline{X}$ ($\pm \sigma^2_X$) and $\overline{Y}$ ($\pm\sigma^2_Y$), where *X* denotes disease prevalence and *Y* refers to adult density. Model estimates of each value, $\overline{x}_{j,i}$ and $\overline{y}_{j,i}$, were determined for each replicate, *j*, of each parameter combination, *i*. Thus, the model was evaluated with respect to the parameters in relation to $\overline{x}_{j,i}$ and $\overline{y}_{j,i}$, assuming that the combination of parameter values, *i*, that yielded the lowest

standardised error, Δ_i, was the best fit. For each parameter value combination, the standardised error of the model was calculated as:

$$\Delta_i = \sum_{j=1}^{m} \left[\frac{(\overline{x}_{j,i} - \overline{X})}{\sigma_X} \right]^2 + \left[\frac{(\overline{y}_{j,i} - \overline{Y})}{\sigma_Y} \right]^2, \quad (12)$$

where *m* is the total number of replicates for each parameter combination. Following parameterisation, the performance of each calibrated scenario was evaluated by estimating specified emergent properties from 200 replicates, each run for 17 years following the introduction of mange to the system.

7.3 Results

7.3.1 Calibration of disease parameters

Calibrated parameter values for *Scenarios 1* to *6* are presented in Table 7.2. The region of parameter combinations that provide the best fit to the data corresponds to the minimum standardised error, Δ_i, as illustrated for calibration of parameters for *Scenario 1* in Figure 7.2(A-C). Estimates of the monthly age-specific transmission coefficients, β_j and β_a (Table 7.2), were consistent with the same parameters estimated from SEI modelling (Table 7.1). Variation in the parameter values between models is likely to stem partly from interactions between the components describing the disease dynamics. For example, to compensate for the recovery of infected individuals, ε is higher in the models with genetic resistance (*Scenarios 4-6*), compared to the model without (*Scenario 2*) (Table 7.2).

Table 7.2. Best parameter estimates for model *Scenarios 1* to *6*, fitted by calibration. Showing the standardised error between the empirical and model estimates of prevalence, Δ_X, and density, Δ_Y, and the total associated standardised error, Δ_i, for the given parameter estimates.

	Scenario 1	***Scenario 2***	***Scenario 3***	***Scenario 4***	***Scenario 5***	***Scenario 6***
Juvenile disease transmission coefficient, β_j	5.08	5.84	6.85	5.08	5.77	5.13
Adult disease transmission coefficient, β_a	1.64	1.67	1.84	1.99	1.75	2.02
Infection constant, α	0.75	0.70	0.69	0.68	0.64	0.64
Rate of indirect transmission, ε	-	0.67	-	1.50	0.94	1.54
Initial proportion with resistance allele, ν	-	-	0.01	0.02	0.04	0.02
Recovery time (months) with resistance allele, τ	-	-	2	2	2	2
Δ_X	0.75	28.67	1.08	5.68	3.69	4.73
Δ_Y	1.17	0.22	8.96	2.86	7.73	1.28
Δ_i	1.92	28.89	10.03	8.54	11.42	6.01

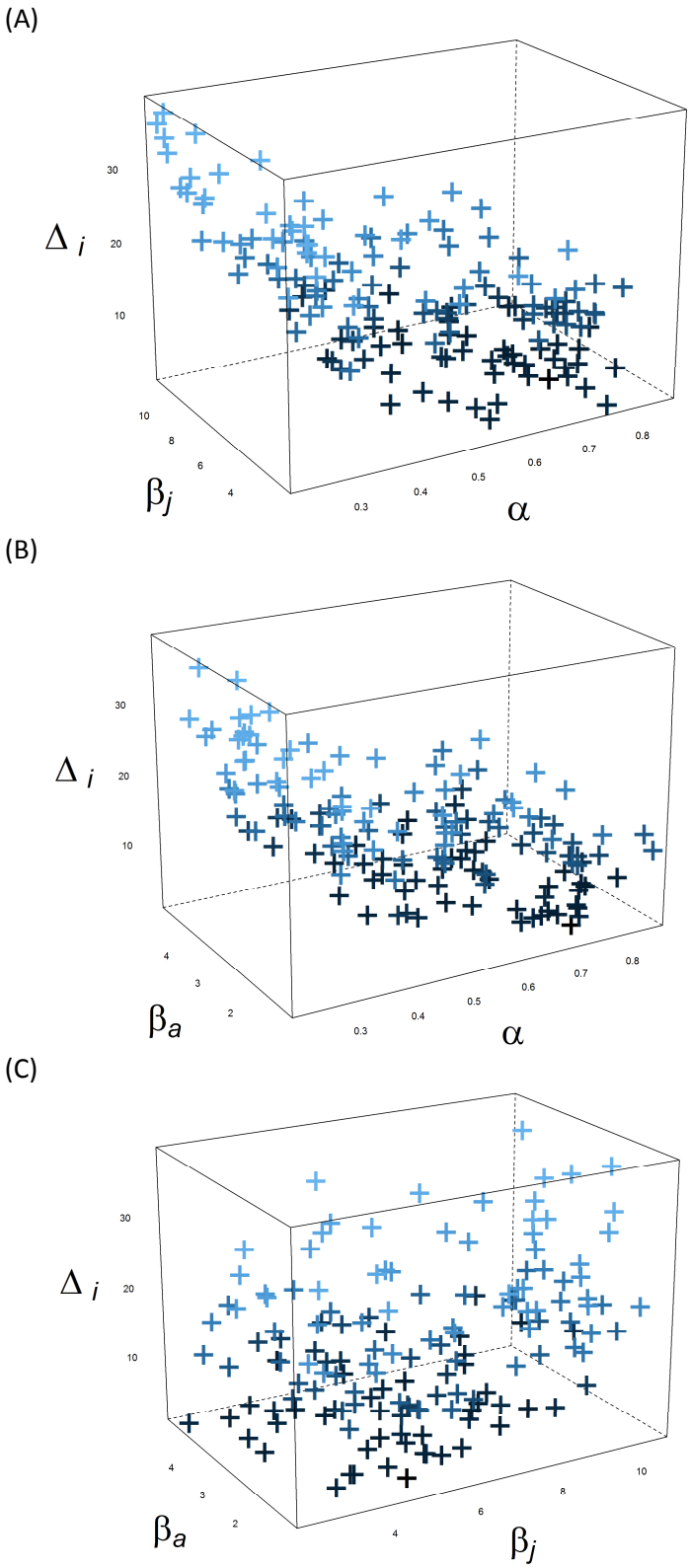

Figure 7.2. Calibration results for *Scenario 1*, showing the standardised error Δ_i determined for the parameter spaces of: (A) β_a and α; (B) β_j and α; and (C) β_a and β_j. The gradient of light to dark blue indicates the increasingly better fit of parameter combinations.

7.3.2 Model application: mange spread

The probability that mange failed to establish in the population (mean = 0.09, SD ± 0.02), because no infectious contacts arose from the initial infected individual, was not significantly different between all the model scenarios (χ^2 = 5.04, df = 3, p = 0.41). Fitting the models to the mean prevalence (Figure 7.3A) and mean adult density (Figure 7.3D), enabled evaluation of the models using the finer scale properties that emerged, such as the mean epizootic prevalence, as described in the following sections.

7.3.2.1 Transmission during the epizootic and enzootic mange phases

Direct transmission alone was insufficient to reproduce the spread of mange, and was particularly poor at explaining mean epizootic prevalence and the periods of high and low density (Figure 7.3B&E-F, Figure 7.4A, Figure 7.5A). Indirect transmission (*Scenarios 2, 4-6*) acted to increase mange transmission (Figure 7.4B-F), resulting in an improved ability of these scenarios to reproduce the mean epizootic prevalence (Figure 7.3B) and observed population decline (Figure 7.5B-F). However, the asymptote of the predicted cumulative proportion of infected individuals in *Scenario 2* indicated that no new infections occurred after the epizootic (Figure 7.6) and thus, is reflected in the short disease persistence and likelihood of population extinction. During the enzootic phase, the persistence of mange increased with the addition of genetic resistance in *Scenarios 3 to 6*, to capture the observed duration of the disease in the Bristol fox population (Figure 7.3C). Saturation of new infectious contacts was not reached in *Scenarios 3* to *6*, as indicated by the asymptotes in Figure 7.6*,* implying the continued persistence of the disease in the simulation. Thus, based on these measures of disease spread, *Scenarios 4* to *6* were able to adequately reproduce empirical patterns. Although the mean predicted density suggests that *Scenario* 6 was best able to reproduce empirical observations, the wide confidence intervals around the predicted density estimates of *Scenarios 4* and *5* indicate that these scenarios merit further consideration.

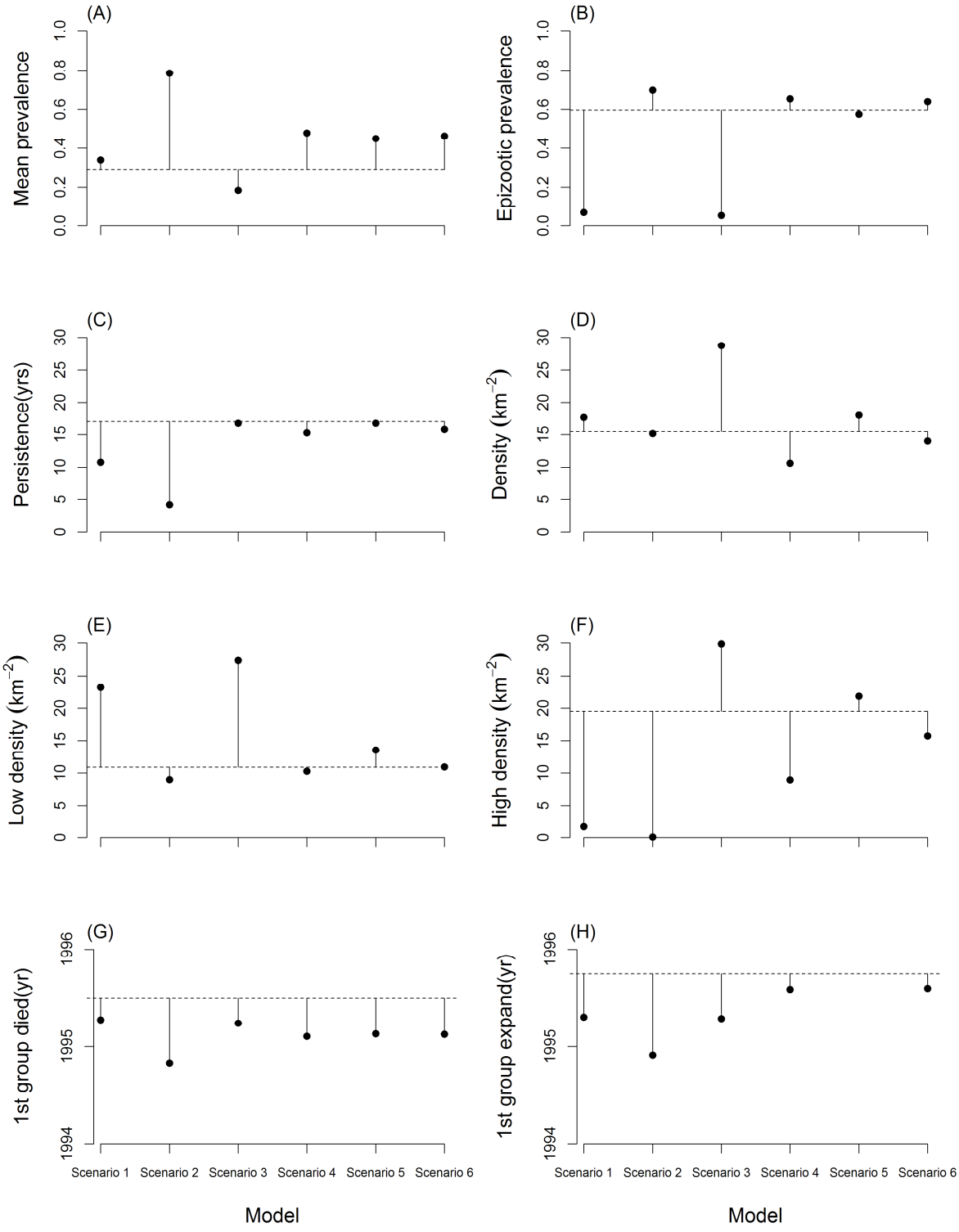

Figure 7.3. Summary of the mean estimates of emergent properties relating to disease transmission in the Bristol fox population. Estimates are presented for model replicates where the disease became established, for *Scenarios 1* to *6.* (A) Mean prevalence; (B) mean epizootic prevalence (1994-1996); (C) mean persistence (years); (D) mean adult density; (E) mean adult low density (1994-2003); (F) mean adult high density (2004-2010); (G) time of first group collapse (year); (H) time of first group expansion (year). Dashed lines are the mean empirical estimates for the specified emergent property.

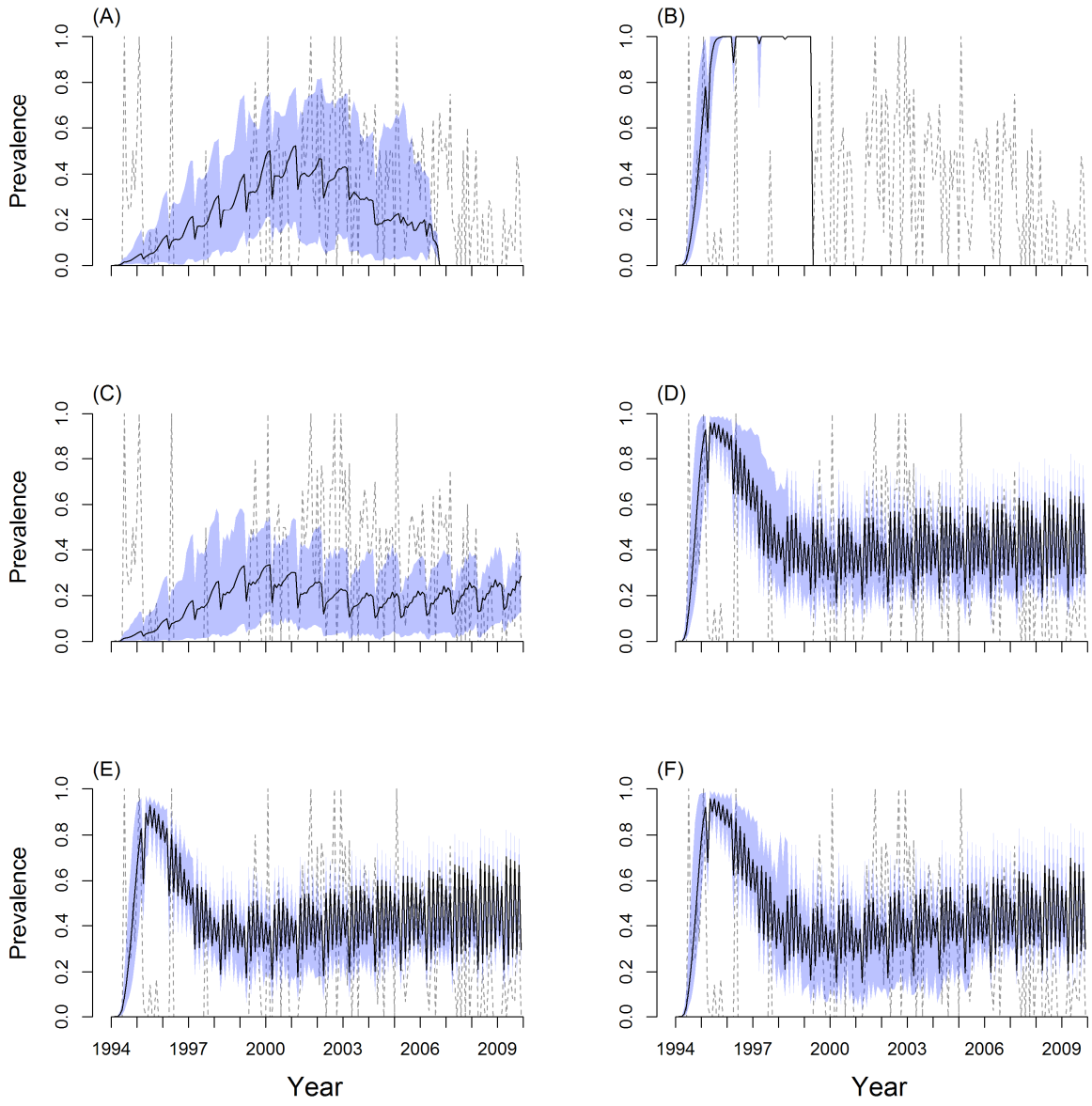

Figure 7.4. Temporal dynamics of a simulated mange outbreak. Observed monthly mange prevalence (dashed line, see section 7.2.1) and predicted estimates (black line) of the monthly mange prevalence for runs where infections were still present. Model estimates are the mean values from 200 replicates, with 95% confidence intervals indicated by the shaded areas. (A) *Scenario 1*, direct transmission only; (B) *Scenario 2*, with indirect transmission; (C) *Scenario 3*, with genetic resistance; (D) *Scenario 4*, with indirect transmission and genetic resistance; (E) *Scenario 5*, with indirect transmission and genetic resistance and without territory expansion and (F) *Scenario 6*, with indirect transmission, genetic resistance and long-distance recolonisation.

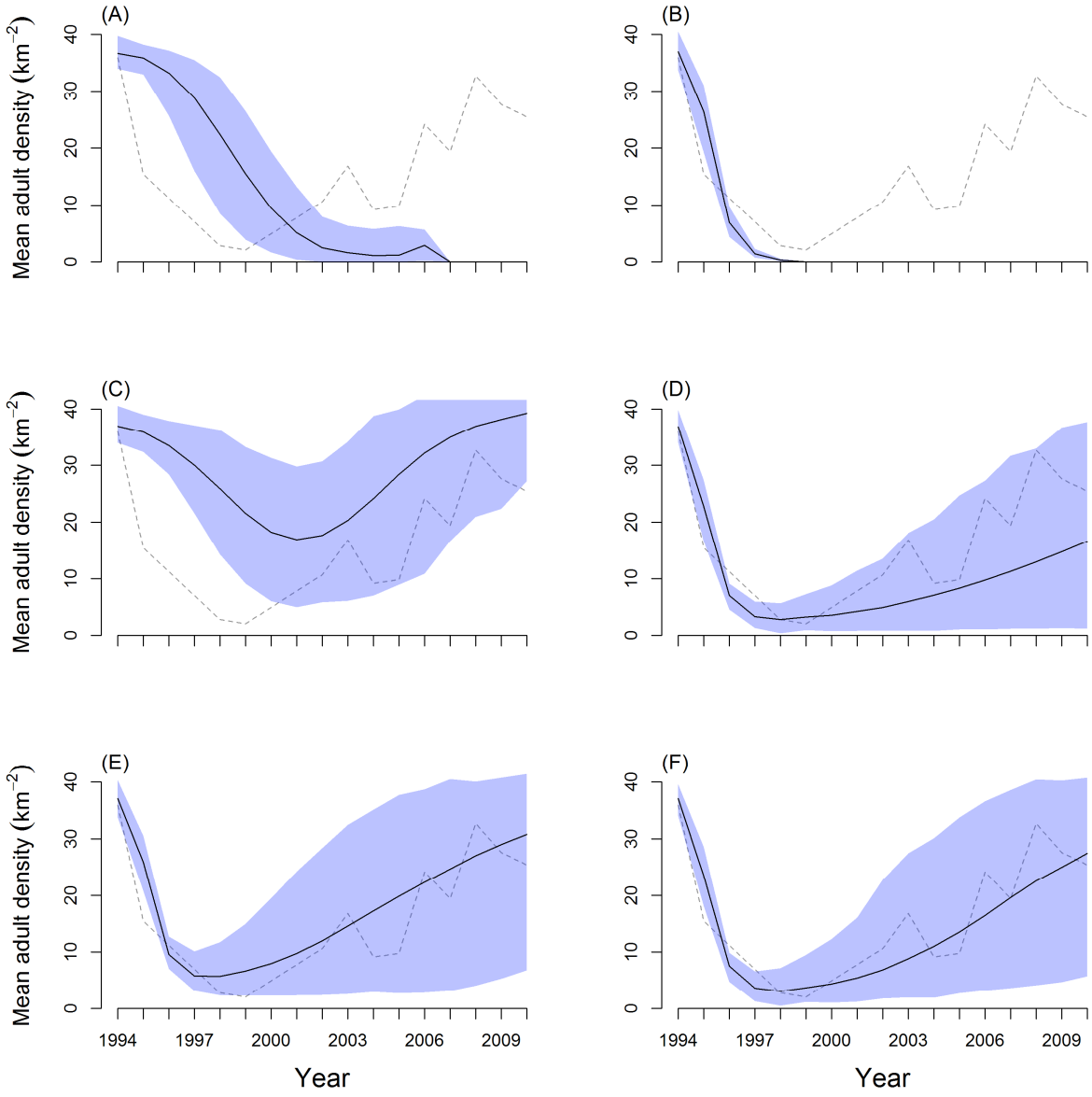

Figure 7.5. Temporal dynamics of population density in response to a simulated mange outbreak. Observed (dashed line) and model estimates (black line) of adult population density (km^{-2}). Model estimates are the mean values from 200 replicates for runs where infections were still present, with 95% confidence intervals indicated by the shaded areas. (A) *Scenario 1*, direct transmission only; (B) *Scenario 2*, with indirect transmission; (C) *Scenario 3*, with genetic resistance; (D) *Scenario 4*, with indirect transmission and genetic resistance; (E) *Scenario 5*, with indirect transmission and genetic resistance and without territory expansion and (F) *Scenario 6*, with indirect transmission, genetic resistance and long-distance recolonisation.

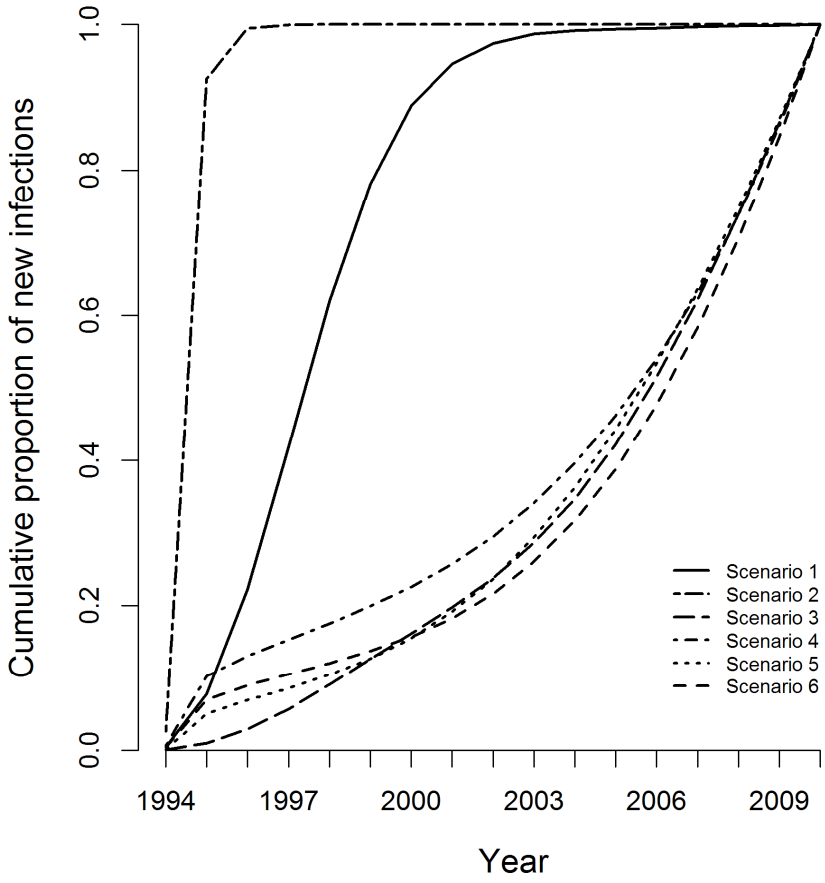

Figure 7.6. Mean cumulative proportion of new infections per year, indicating the time taken to reach saturation of new infections in a given system. Estimates are the mean from 200 model replicates for *Scenarios 1* to *6*.

7.3.2.2 The relative importance of social contacts for mange transmission

Exposure to mange arising from both intra-group contact and parental contact at birth was greater than inter-group contact for all model scenarios (Figure 7.7), reflecting empirical encounter rates. The proportion of infectious contacts resulting from fomites was higher than intra-group contact for models with indirect transmission (*Scenarios 2* and *4-6*). The importance of inter-group contact for mange transmission was reduced with the inclusion of indirect transmission. For all scenarios, infection during dispersal or extra-territorial movement contributed to a small proportion of all infections. However, compared to dispersers, extra-territorial males consistently had more contacts with infectious fomites.

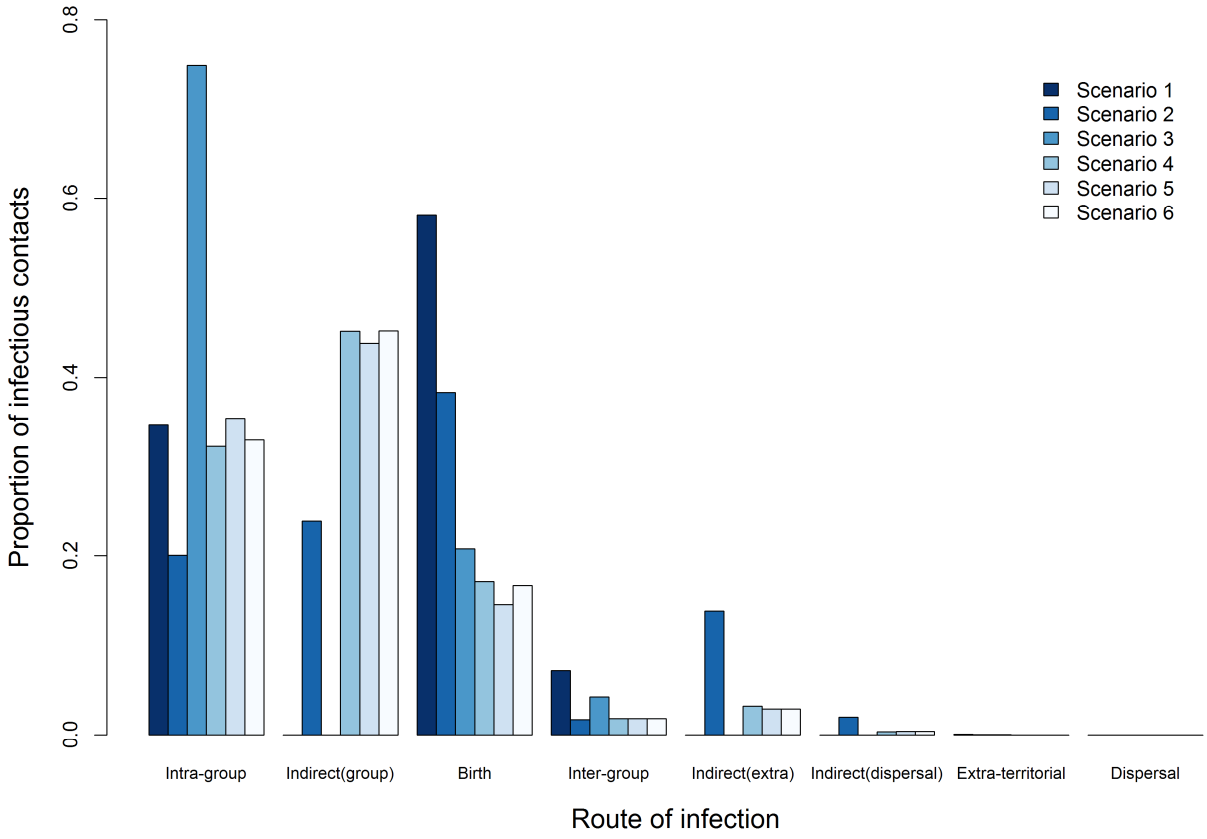

Figure 7.7. Model output for the mean proportion of contacts resulting in infections from different routes. Estimates are from 200 model replicates for *Scenarios 1* to *6*. Infectious contacts are from individuals becoming exposed due to intra-group contact, transmission via fomites for territorial individuals, infection at birth, inter-group contact, transmission via fomites for extra-territorial males and dispersers, and direct contact during extra-territorial movement and dispersal.

7.3.2.3 The effects of territory collapse for mange spread

Scenarios 4 and *6* were best able to reproduce the timing of territory collapse and expansion (Figure 7.3I&J). The size of territory expansion was underestimated by all models. In autumn 1995, the observed mean territory size had increased by a factor of 3.1 and, by winter 1995, territories had increased by a total of 7.8 times the original size (Baker *et al.* 2000). In the best-fitting model (*Scenario 6*), the predicted mean increase of the original simulated territory size was 2.10 (SD ± 0.21) in autumn 1995 and by a total of 2.32 (SD ± 0.25) in winter 1995. The mismatch between the model and empirical estimates of the territory expansions might be due to model simplifications or empirical uncertainties, given the small sample sizes. Observations were also derived from a smaller area (Baker *et al.* 2000) than was modelled, with territorial processes restricted by the physical boundaries of the study area including the river and uncolonised open areas (S. Harris *pers. comm.*).

The sources of infectious contacts between *Scenarios 4* and *5* (with and without *territory collapse*, respectively) were compared to determine the effects of territory formation on disease spread. Infections from fomites are predicted to increase due to contact by expanding groups with infected substances remaining on empty territories. There was a significant difference in the mean proportion of fomite infections of territorial individuals during the epizootic between *Scenario 4* (mean = 0.42, SD ± 0.03) and *Scenario 5* (mean = 0.37, SD ± 0.03) (Mann-Whitney U-test: $U = 1334$, $p = 0.002$). This difference was reflected in the faster cumulative rate of infections in *Scenario 4* compared to *Scenario 5* (Figure 7.6). Perturbation of social organisation might be expected to alter inter-group encounters, but the difference in inter-group infections between *Scenario 4* (mean = 0.02, SD ± 0.004) and *Scenario 5* (mean = 0.02, SD ± 0.004) was marginally non-significant (Mann-Whitney U-test: $U = 2204$, $p = 0.08$).

Territory collapse may be expected to reduce breeding opportunities, due to fewer vacancies for dominant individuals if new groups are not formed. Thus, the probability of breeding during the epizootic was compared between *Scenarios 4* and *5*. During the epizootic, the mean proportion of breeding dominant females was significantly lower (0.37, SD ± 0.18) in *Scenario 4* than in *Scenario 5* (0.48, SD ± 0.17) (Mann-Whitney U-

test: $U = 3921$, $p < 0.001$). There was no significant difference in the mean proportion of breeding subordinate females in *Scenario 4* (0.14, SD ± 0.06) and in *Scenario 5* (0.16, SD ± 0.04) (Mann-Whitney U-test: $U = 10328$, $p = 0.109$).

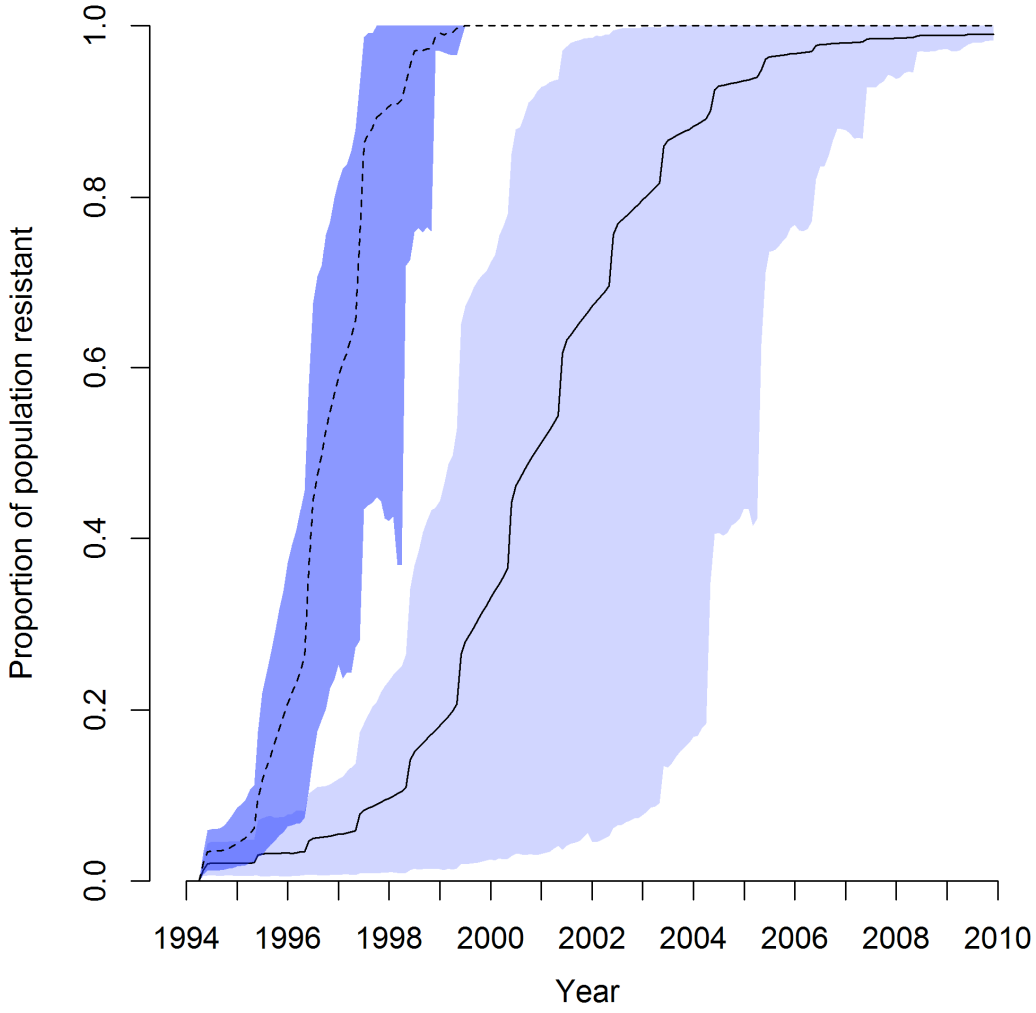

Figure 7.8. Mean proportion of the population with the resistance allele for *Scenario 3* (solid line, light blue shading indicates 95% confidence intervals) and *Scenario 4* (dashed line, dark blue shading indicates 95% confidence intervals). Model estimates are from 200 model replicates for runs where infections were still present.

7.3.3 Model application: population recovery

7.3.3.1 The importance of genetic resistance for population recovery

The proportion of model replicates when the population did not go extinct by the end of the simulated period, was significantly higher when genetic resistance was added to the model [mean (*Scenarios 1-2*) = 0.32, SD ± 0.34, mean (*Scenarios 3- 6*) = 0.87, SD ± 0.11, χ^2 = 525.84, df = 3, $p < 0.001$], reflecting the higher likelihood of population recovery in these models. The low density period, encompassing the population decline caused by the epizootic, was overestimated without the inclusion of genetic resistance (Figure 7.3E). Thus, the longer disease persistence in the model scenarios with genetic resistance reflected the continuation of mange infections, sustained by individuals surviving to produce new susceptible offspring. In *Scenario 4*, the allele spread rapidly and, once the population started to recover (year 1999), the allele was present in all individuals. This is in comparison to *Scenario 3,* where the increase in the mean proportion of resistant individuals was gradual and only 20% of the population were resistant by 1999 (Figure 7.8).

7.3.3.2 Long-distance recolonisation and population recovery

Estimates of the high density period, indicative of the population recovery, were better reproduced by models with long-distance recolonisation (*Scenario 6*) (Figure 7.3F), suggesting that genetic resistance alone was inadequate as a mechanism for population recovery. Allowing subordinate individuals to attain dominance in non-neighbouring groups improved estimates of population recovery (Figure 7.5F). Figure 7.9 illustrates the importance of long-distance recolonisation as a source of dominant individuals, particularly for the years immediately following the epizootic. During the high density period, dominant females were most likely to originate through philopatry (Figure 7.9A) and dominant males were predominantly replaced by dispersing individuals (Figure 7.9B), which is consistent with empirical observations (Iossa *et al.* 2009).

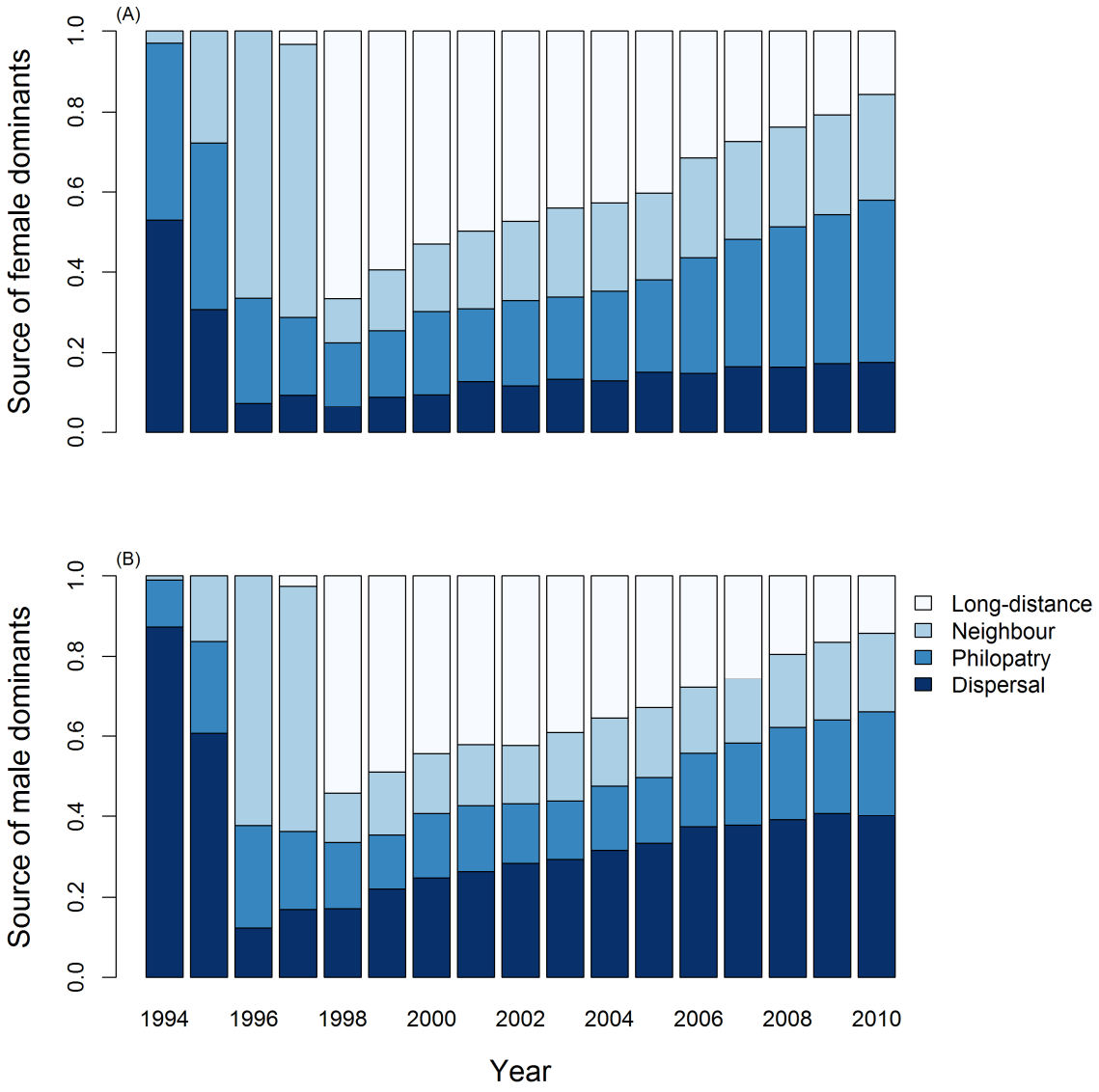

Figure 7.9. Mean source of dominants in *Scenario 6*, showing the proportion of dominants that were replaced by dispersing, philopatric, neighbouring and long-distance females (A) and males (B). Mean model estimates from 200 model replicates for runs where infections were still present.

7.4 Discussion

In this study I demonstrated the ability of an IBM to reproduce the dynamics of mange in an urban fox population, using a combination of empirical and calibrated parameters. The results suggest that direct transmission alone is not sufficient to describe the spread of this disease. The simulation outcomes emphasised that additional processes, namely indirect transmission, genetic resistance and long-distance recolonisation, are needed to capture the temporal dynamics of mange in the Bristol fox population. Contrary to predictions, dispersers did not contribute significantly to mange spread. The model suggested that the collapse of territories affected the spread of mange by promoting contact with fomites. Here, the relative importance of indirect and direct transmission mechanism is discussed, particularly in light of data uncertainty. The influence of social processes, namely, extra-territorial movement, recolonisation, and stable territorial structure, on mange spread and population recovery is examined. Consideration is given to the evolutionary adaptation of genetic resistance to mange. The results of this simulation are also discussed in the context of the contrasting intra-and inter-specific impacts of mange on host populations.

7.4.1 The role of direct and indirect transmission in mange dynamics

The model scenarios in this study were used to explore the role of direct and indirect transmission mechanisms for the spread of mange in the Bristol fox population. Direct transmission alone, using empirically estimated contact rates, was unable to reproduce the observed mange dynamics. Fox social organisation appears to yield encounter rates that are insufficient to promote mange prevalence, lending support to the hypothesis that an additional transmission pathway, such as through fomites (Soulsbury *et al.* 2007), is required to explain the rapid spread of this disease. This contrasts with the dynamics of mange in a coyote population, which were adequately described by direct transmission alone (Leung & Grenfell 2003). Direct encounter rates among coyotes might be sufficient to describe the outbreak; unlike foxes, groups of transient juveniles overlap with resident groups (Leung & Grenfell 2003), possibly

increasing the opportunity for infection and potentially masking infections from indirect transmission. Interspecific variation in the relative importance of transmission pathways is therefore likely to be influenced in part by host behaviour. This simulation suggests that indirect transmission is important for describing mange dynamics in foxes.

Indirect transmission acts by increasing opportunities for infection through contact with fomites, augmenting the number of infected individuals in the population and thereby, increasing the speed of disease spread. Indirect transmission has been implicated in the long-term persistence of several diseases, including the transmission of avian influenza through contaminated drinking water (Rohani *et al.* 2009), chronic wasting disease through infected faeces or carcasses (Miller *et al.* 2006), and hantavirus excreted in scent marks (Sauvage *et al.* 2003). The source of environmental contamination for mange remains unclear. The incidence of inter-group den sharing, a known route of indirect mange transmission in Russian fox populations (Gerasimov 1958), is undetermined, but likely to be low in the Bristol fox population. Snoop zones, territory boundaries where scent-marking reduces direct contact during territorial defence (Giuggioli *et al.* 2011), could also act as a conduit for mange transfer through mutual contact with infected substances in these zones. In particular, foxes typically favour certain routes (S. Harris *pers. comm.*) into gardens (a preferred habitat of urban foxes; Harris & Rayner 1986b); thus, the likelihood of indirect transmission between groups in these overlapping zones is increased by multiple individuals using the same preferred entry points, such as when scraping under fences. A further potential route of indirect infection identified in this study is the expansion of territories, leading to contact with fomites that remain on empty territories, which could increase opportunities for indirect transmission. The increased rate of indirect transmission when allowing territories to collapse provided some support for this mechanism in the Bristol population. Another proposed route, environmental contamination by domestic dogs (Soulsbury *et al.* 2007), was not considered here, but deserves further attention. It is also possible that support for indirect transmission is indicative of other processes involved in increasing transmission, including individual heterogeneity in infectivity. Such heterogeneity can occur with the existence of superspreaders (see below and

chapter 5). It is also possible that there were a greater number of infected individuals involved in the initial introduction of mange to the Bristol population. A simplification of modelling transmission in this simulation was the restriction of breeding pairs to individuals on the same territory, which may overlook an additional route of direct mange transmission. Inter-group mating occurs in Bristol (Baker *et al.* 2004); however, allowing extrapair mating is unlikely to increase direct transmission to the levels required to describe the spread of this disease. Hence, while the model indicated the importance of an additional transmission pathway, identifying the mechanism of this increased transmission requires further research.

The simulation in this study used proxies for explicitly modelling direct and indirect inter-group disease transmission. Here, the probability of becoming infected through direct inter-group encounters was independent of the number of infected individuals. Modifying this transmission mechanism to identify the individuals responsible for infectious contacts would enable the detection of finer scale processes, such as the group-based measure of the basic reproductive number, R_0, which takes into account inter-group differences in infection rates (Cross *et al.* 2005). Similarly, the approach used to model indirect transmission requires further refinement, given the unknown rates of shedding from infected individuals into the environment, mortality of mites in the environment, and rates of fox contact with fomites, which were implicit within the fomite load and the parameter ε. Like direct transmission, the source of the infection could not be determined for a given infectious contact. This is the first time indirect transmission has been simulated for mange transmission in foxes and there appear to be few instances of IBMs incorporating indirect disease transmission in a wild mammalian population; therefore, this study provides a foundation for future work.

The effects of variation in infectious status were not accounted for in this study, due to data limitations and model simplifications. Combining class I and class II mange infections might have led to overestimating processes such as reproduction; infected males were allowed to reproduce, whereas evidence suggests that individuals with Class II mange were not capable of reproducing (Soulsbury *et al.* 2007). Similarly, the influence of social ranking on infection was not included. Although there is evidence in

other species that dominance behaviour can either increase or decrease disease risk (Møller *et al.* 1993), the effects of social status for mange infection are undocumented in foxes. Individual variation in susceptibility is an important influence in disease dynamics (Woolhouse *et al.* 1997, Kramer-Schadt *et al.* 2009). For instance, disease invasion can be highly dependent on individuals that have a greater than average risk of causing infections, so-called superspreaders (Lloyd-Smith *et al.* 2005b). Given that little is known about variation in parasite loads in mangy foxes, further data are necessary to determine whether mange infestations are disproportionate in some individuals and whether this could influence the relative importance of direct and indirect transmission mechanisms.

7.4.2 The relative importance of dispersal for mange transmission

Disease transmission in social species is influenced by interactions between and within groups (Altizer *et al.* 2003b) and, the potential importance of contact with dispersing individuals has been emphasized for the spread of mange in foxes (Lindström 1992). Whilst these results indicated that dispersing individuals contributed little to the local spread of mange in Bristol, this should be viewed in the light of model simplifications; data limitations prevented the incorporation of explicit encounter rates between territory holders and dispersers. While dispersing individuals spend little time in territory cores (Soulsbury *et al.* 2011), the high proportion of bite wounds in dispersers indicates that there may be substantial contact with territory holders (Soulsbury *et al.* 2008a). The role of dispersing foxes for moving mange over longer distances than that modelled here requires further work, especially given the support for infrequent long-distance dispersal in simulating the wave-like spread of a rabies outbreak (Trewhella & Harris 1988, Jeltsch *et al.* 1997).

Individuals moving through the landscape were more important for indirect than for direct mange transmission. That extra-territorial males were more important for indirect transmission than dispersers could be attributed to the fact that that all adult males were given the opportunity for reproductive movement and, by returning to their territory along the same route, passed through infected territories twice. However, the role of movement by individuals other than dispersers for disease

transmission has been demonstrated in lions *Panthera leo* through network modelling. Specifically, infrequent long-distance contact between non-neighbouring prides, driven by food availability, reduced the effective distance between prides and increased disease transmission more than through the movement of dispersers (Craft *et al.* 2011). Thus, the effect on disease transmission of the movement of individuals other than dispersing juveniles requires further consideration.

7.4.3 Territory collapse as a mechanism for promoting disease spread

The increase in the number of mange infections in those scenarios that incorporated the collapse of territories is consistent with theory suggesting that group-structuring reduces the spread of disease (Loehle 1995, Cross *et al.* 2005, Vicente *et al.* 2007). The mechanisms underlying this process are complex and system-specific. Disruption to territorial behaviour in a completely susceptible badger *Meles meles* population increased the likelihood of colonisation by diseased individuals (Carter *et al.* 2007). The outbreak of mange in the Bristol fox population provided the opportunity to gain insight into territory collapse during an epizootic. That the simulated inter-group contact rate remained unchanged following the collapse of territories, is consistent with observations that scent-marking behaviour and underlying social organisation were unaffected during the epizootic (Baker *et al.* 2000). However, the simulated spread of mange increased with the collapse of territories. The increased contact with fomites, arising from groups expanding into infected territory spaces, appears to have been sufficient to cause this outcome.

The low population recovery in *Scenario 4* (with territory collapse) implies the existence of a threshold below which the population could not recover. Leung and Grenfell (2003) found that, without long-distance recolonisation, the coyote population exhibited Allee effects and declined to extinction because territories remained empty. That the simulation predicted a faster recovery of the Bristol fox population without territory collapse is plausible, since recolonisation of empty groups during the epizootic promoted reproduction, thus preventing the population from declining below a critical threshold. These findings are indicative of the long-term impacts of territorial perturbations on population dynamics. Similar long-term effects

were observed in a badger population, where recovery to pre-perturbation densities after territory expansion induced by culling was slow, despite initial rapid immigration (Carter *et al.* 2007). This simulation of mange in an urban fox population therefore points towards the importance of a stable territory structure for epidemiological and population dynamics.

7.4.4 The influence of resistance for mange persistence

In these analyses, the recovery of the Bristol fox population was driven partly by the evolution of heritable resistance, as was found for a simulation of mange in a coyote population (Leung & Grenfell 2003). Increasingly, evolution in host-parasite systems is demonstrated to occur over much shorter timescales than previously thought, such as over decades (Altizer *et al.* 2003a). For instance, simulations predicted the evolution, over 50 years, of resistance alleles to phocine distemper virus in harbor seals *Phoca vitulina* (Harding *et al.* 2005). However, empirical evidence for resistance alleles in wild mammalian populations is still limited. In Australia, resistance to the myxoma virus developed rapidly after the introduction of a highly virulent strain in European rabbits, *Oryctolagus cuniculus*; virulence of the virus subsequently decreased to intermediate levels, promoting the persistence of the disease (Dwyer *et al.* 1990). Recently, a gene increasing the resistance to prion disease was identified in a white-tailed deer *Odocoileus virginianus* population subject to high infection rates (Robinson *et al.* 2012). Thus, the high mortality rate, possibly in combination with a high rate of mange infection arising from indirect transmission in the scenarios examined in this study was likely to act as a selection pressure for the inheritance of resistance over the duration of the mange epizootic.

The rapid evolution of resistance to mange simulated here is consistent with the cycles of mange epizootics that are followed by a long endemic phase. This evolutionary process is compatible with theory suggesting that new outbreaks are caused by mutations of mites (Pence & Ueckermann 2002), because resistance acts directly on the evolution of parasite virulence (Gandon & Michalakis 2000). However, the model in this study did not incorporate selection pressures on the parasite. The simplest system of genetic inheritance was modelled following Leung and Grenfell (2003),

allowing for the selection but not the mutation of alleles. Here, the mutation giving rise to the allele was assumed to have occurred prior to the outbreak of mange; rapid evolution in allelic frequency following disease introduction suggests prior variability in genetic resistance (Bonneaud *et al.* 2011). In light of the cyclical outbreaks of mange and the importance of resistance identified here, further research is needed on the evolution of mange virulence.

Differences in the degree of prior genetic resistance or exposure to mange could explain the varying impact of this disease on host populations. The dramatic population decline observed in foxes following a mange epizootic in Bristol did not occur in a population of coyotes (Pence & Windberg 1994). Indeed, the initial proportion of resistant individuals was estimated to be over five times higher in this coyote population (Leung & Grenfell 2003) than the Bristol fox population. Further, the slower allele spread through the coyote population (Leung & Grenfell 2003) than in this study could reflect the potential difference in infection rates between these two canid species. Empirical data are required to determine whether these differences are a reflection of genuine patterns in the processes driving resistance to mange or are an artefact of interspecific variation.

7.4.5 Long-distance recolonisation as a mechanism for population recovery

The protracted recovery of the Bristol fox population is consistent with observations of mange epizootics in Scandinavian fox populations (Lindström *et al.* 1994, Forchhammer & Asferg 2000). The best-fitting simulation in this study was able to reproduce population recovery with the addition of long-distance recolonisation. Leung and Grenfell (2003) hypothesised that long-distance recolonisation by dispersers and subordinate adults was necessary for the coyote population to recover from a mange epizootic. In fox populations, dispersal by adults is uncommon (Harris & Trewhella 1988). However, given the extremely low population density by the end of the epizootic in Bristol, the opportunity to attain dominance could have promoted the dispersal of juvenile and adult subordinate individuals into empty territories across the landscape. Indeed, rapid recolonisation has been observed in some fox populations after control efforts or disease due to their dispersal abilities (Bögel *et al.* 1974, Gentle

et al. 2007). The results of this simulation suggested that long-distance recolonisation was particularly important early in the recovery phase; the need for this process implies that a mechanism existed to promote group formation immediately after the epizootic. In badgers, culling-induced perturbation of territories triggered an increase in immigrant badgers (Carter *et al.* 2007). Immigration rates in the Bristol fox population were low prior to the epizootic (Harris & Smith 1987), and so, it was considered suitable to model a closed population. However, the collapse of territories could have led to an increase in immigration, stimulating the observed recovery. This model therefore exposes the need for a better mechanistic understanding of the recovery of the Bristol population.

7.5 Conclusion

An individual-based simulation using empirically-derived data was able to reproduce emergent properties of an urban fox population during a mange outbreak. The ability of this model to describe epidemiological and demographic patterns during both the epizootic and enzootic phases was demonstrated using multiple patterns for model validation. This study provided compelling support for several theoretical hypotheses proposed to explain epidemiological and population dynamics during outbreaks of mange. Empirically estimated direct encounter rates alone were insufficient to describe the dynamics of this disease in a high density fox population. The importance of an additional process leading to increased mange transmission, such as indirect transmission, was inferred from the simulation. Contrary to predictions, dispersing individuals contributed to a relatively small proportion of infectious contacts in the model. Results were suggestive of the influence of territory collapse on disease spread. The influence of rapid evolutionary dynamics on the selection of resistance alleles was illustrated. However, genetic resistance alone was not sufficient to reproduce population recovery and the need for a mechanism to promote recolonisation after the epizootic was supported. The support for genetic resistance and long-distance recolonisation, as also found for coyotes, implies some consistency among the processes shaping this disease in canids. However, the processes identified in this study require empirical validation; the evolution of immunity and the underlying

mechanisms of fomite transmission demand particular attention. While the model in this study was able to describe the mange dynamics, finer scale processes at the individual level remain to be determined. Further analyses, such as robustness analysis (Railsback & Grimm 2011) or information-theoretic approaches (Burnham & Anderson 2002) will then be required to confirm the structural integrity of the model. Understanding the processes driving mange outbreaks is of wider relevance for the management of this disease, given that mange affects a wide range of mammalian species.

Chapter 8 General discussion

In this thesis I investigated the demography of red foxes *Vulpes vulpes* and the dynamics of sarcoptic mange *Sarcoptes scabiei*, a major disease of the fox. I found that despite substantial sampling effort worldwide, fox demography is surprisingly poorly known due to weaknesses in the data. However, analyses of the better studied populations provided evidence of intraspecific demographic variation, which I showed has implications for data substitution in population models. Prompted by the high uncertainty in fox demographic data, I developed a method to account for uncertainty in vital rates estimated from mortality data, and also determined that litter size can be included in demographic models with a number of suitable probability distributions. By modelling mange for the first time in a fox population, I established that transmission is frequency-dependent, but that direct transmission alone was insufficient to reproduce the observed spread of this disease. I also identified the importance of indirect transmission, genetic resistance and long-distance recolonisation for describing mange dynamics in this fox population. Here, I will begin by discussing the major findings of chapters 2 to 7 in the context of the broad themes of this thesis. I will then go on to consider how the results of this thesis fit into the wider implications of the areas of this study, and suggest directions for future work.

8.1 Synthesis

Vertebrate species are increasingly at risk from a suite of threats including disease, hunting, climate change and human-wildlife conflict (Hulme 2005, Milner-Gulland & Rowcliffe 2007, Smith *et al.* 2009b). Accurate management predictions are dependent on resolving the challenges facing population ecologists such as demographic uncertainty, intraspecific variation, environmental variation and undefined interspecific interactions. Given that red foxes are abundant, widespread and the subject of much management and sampling effort (Baker *et al.* 2008, Saunders *et al.* 2010), this species was used as an archetype to explore questions relating to data uncertainty, demographic variation and disease dynamics.

8.1.1 Modelling demography with uncertainty

Disregarding uncertainty in demographic parameters and resultant model predictions of population dynamics is likely to lead to less effective management (Beissinger & Westphal 1998), but it is nethertheless a widespread practice in the published literature (chapter 2). This has implications for demographic models, which often demand the use of published data (chapters 2, 3 and 4). As chapters 2 and 4 illustrated, published demographic data from fox populations are typically derived from hunting returns and presented as point estimates. Using published data on this widespread carnivore, the potentially substantial oversights from basing management on a point estimate of the population growth rate (λ) were illustrated (chapter 2). This type of oversight could be of particular consequence for the management of populations close to extinction, or species that pose a risk to other wildlife or human populations. Indeed, a previously published point estimate of λ for a wolf *Canis lycaon* population was shown to be overly pessimistic, because uncertainty in the vital rates determined from detailed capture-mark-recapture data was not incorporated (Patterson & Murray 2008). However, as demonstrated in this thesis, confidence intervals are also easily determined from parameters based on point estimates of vital rates, using a novel combination of widely applied techniques, for both λ (chapter 2) and life history speed metrics (chapter 4). This study therefore illustrated that, if uncertainty is accounted for, meaningful information for management can be gained from using published or minimal data.

A particular source of uncertainty in demographic models is the parametric distribution chosen to simulate demographic stochasticity in life history parameters, such as offspring number (Fieberg & Ellner 2001, Kendall & Wittmann 2010). In chapter 3, it was established that several distributions were suitable for describing litter size in carnivores and that distribution choice had a negligible effect on model predictions of quasi-extinction risk and disease control in canids. Given the often deficient life history data for many carnivores (Gese 2001), this finding is reassuring for those population ecologists and conservation biologists faced with minimal data and increases the confidence with which demographic stochasticity in this life history trait is modelled.

This study represents the first published evaluation for a multiparous species of the robustness of population models to the choice of litter size probability distributions. Yet, the importance of phylogeny as an intrinsic constraint on offspring number (Shine & Greer 1991, Jetz *et al.* 2008) suggests that caution should be applied when extending the results to other mammalian orders or multiparous taxa such as birds or reptiles.

One of the shortcomings with addressing uncertainty in population modelling is data availability, limited largely by sampling constraints (Morris & Doak 2002, Mills 2007). Sampling efficiency can be improved by testing the effects of sampling constraints on model output, such as quantifying the magnitude of variance (Doak *et al.* 2005) or bias (Fiske *et al.* 2008) in λ due to study duration or sample size, respectively. The findings in this thesis build upon and complement existing recommendations, such as increasing sample sizes when survival is low (Fiske *et al.* 2008) and extending study duration (Doak *et al.* 2005) as sample sizes increase. In chapter 2, small sample sizes conveyed more uncertainty in vital rate estimates, especially when mean values were at the extremes of the probability distribution. In chapter 3, the number of parsimonious probability distributions supported for empirical litter size frequencies decreased with increasing sample size, indicating the increasing certainty in the distribution of this parameter. The effects of sample size on λ estimates were clearly illustrated in chapter 2, with direct applicability to data collection: increasing sample size fourfold decreases confidence intervals of λ by half. Further, the approach taken in this thesis (chapter 2) could be incorporated into existing methods that aim to improve sampling design, such as combined power and population viability analyses to determine the optimal duration of data collection required to minimise uncertainty in model outcomes (Thompson *et al.* 2000).

8.1.2 Understanding red fox demography

In chapter 4, a broad-scale review of fox demography brought together over 70 years of sampling effort for the first time, underlining that, despite this extensive sampling effort and the wide geographic range for which data are available, many fox populations lacked the data required for comprehensive demographic analyses. That there are substantial gaps in the demographic data of a widespread species is a

cautionary note for our understanding of the ecology of many common species. Indeed, this finding mirrors other common species noted for their role as predators, disease hosts or economic importance, where demographic data are lacking despite a large body of published literature, such as the big brown bat *Eptesicus fuscus* (Agosta 2002) and yellow-legged gull *Larus cachinnans* (Vidal *et al.* 1998). In contrast to the overarching finding of data deficiency, the individual-based model (IBM) developed in chapter 6 illustrated that given the appropriate data on a well-studied population, patterns in population dynamics could be reproduced accurately. Ultimately, such insight into the population dynamics of widespread species is fundamental for our understanding of ecological interactions, especially since common species are of importance for ecosystem functioning (Gaston & Fuller 2008) and can also face extinction themselves (Lindenmayer *et al.* 2011).

The significance of intraspecific population dynamics continues to gain recognition for management and ecology (Nilsen *et al.* 2009, Johnson *et al.* 2010, Servanty *et al.* 2011). Previous comparisons of life history tactics have been conducted predominantly at interspecific levels (e.g. Promislow & Harvey 1990, Ferguson & Larivière 2002) and, significantly, this thesis represents the first analysis of inter-population differences in a carnivore species (chapter 4). Comparing eight fox populations with sufficient data for demographic modelling (chapter 4) revealed overarching demographic themes: juvenile survivorship and fecundity consistently made the greatest contributions to λ and life history speed fell at the medium-fast end of the fast-slow continuum. However, within these broad themes, there was intraspecific variation in the contributions of vital rates to λ and in life history speed that may reflect environmental productivity rather than a trade-off between survival and fecundity. Given the importance of local conditions it is useful to consider how climatic changes shape future population growth, knowledge of which is currently lacking. Studies of the effects of climate on demography have focused on single, often threatened, populations; thus, there is a need to examine whether intraspecific populations of common and widespread species respond differently to climate change (Gaillard *et al.* 2013). For example, although recruitment drives a decline in λ for conspecific roe deer *Capreolus capreolus* populations during earlier springs, populations in less productive

habitats may be forced to undergo seasonal migration because they cannot increase reproduction in response to a climate-induced decline in resource availability (Gaillard *et al.* 2013). Recent advances have coupled climate models with demographic models (Jenouvrier *et al.* 2012), where scope exists to improve the resultant model predictions by incorporating intraspecific dynamics, such as those demonstrated in this thesis.

Chapter 4 culminated with an example of data substitution between fox populations, thereby assimilating aspects of parameter uncertainty, life history variation and the use of published data. Surrogate data are needed when data for a focal species or populations are unavailable (Caro *et al.* 2005); however, the effects of such data on demographic model estimates remain poorly understood. Given the contrasting fox life histories described above, inter-population data substitution illustrated that comparable levels of anthropogenic pressure or close geographic proximity did not predict demographic similarity (chapter 4). Replacing values for the most variable parameter, fecundity, typically had the greatest impact on the accuracy of λ estimates. Further, these results indicated that demographic models might be particularly sensitive to the substitution of certain components of fecundity; replacing breeding probabilities generally caused a greater change in λ than did substituting litter size. Variation in the response of fox population models to components of fecundity was also apparent in other chapters. In chapter 3, low variation in litter size meant that the choice of parametric distributions had a limited impact on predictions of quasi-extinction risk and disease control in three canids, including foxes, and further, no evidence of inter-population variation in the underlying distributions describing litter size was found for foxes. Interestingly, whereas the IBM of the Bristol fox population was sensitive to changes in breeding probabilities, the model was robust to varying litter size (chapter 6). These findings point to commonalities for modelling variation in these parameters for foxes. While data substitution will continue out of necessity, continued work is needed to define guiding principles for this practice and determine whether similar inferences can be made for data substitution in other species. This study contributes to our understanding of how intraspecific differences and parameter variation affects model estimates based on data substitution and, thus, how the poor use of surrogate data can yield flawed management decisions.

8.1.3 Sarcoptic mange dynamics

It is useful to consider the potential role of host-parasite interactions as one of several important interspecific processes that shape a population's dynamics (Dobson & Hudson 1986). These systems are complex due to the dynamics of the host and parasite populations and their interaction, as well as within-host dynamics and potential intermediate or free-living stages. Deterministic epidemiological models are useful for gaining initial understanding of disease systems, especially within the host population, while individual-based stochastic models are important for describing emergent properties, particularly if heterogeneities in susceptibility or social status are important for disease transmission (Smith *et al.* 2009a). Noisy prevalence data for mange in the Bristol fox population resulted in uncertainty in epidemiological parameter estimates as well as difficulties in making predictions about specific years from the SEI models (chapter 5); however, this modelling approach was important for elucidating mange transmission mechanisms thereby providing support for frequency-dependent transmission. In chapter 7, through a pattern-orientated approach (Wiegand *et al.* 2003), an IBM using the same data was able to reproduce temporal population density and prevalence patterns, which compartment models are often unsuccessful at predicting simultaneously (Leung & Grenfell 2003, Kramer-Schadt *et al.* 2009). The results from chapters 5 and 7 demonstrate the insight into disease dynamics that can be gained from two contrasting modelling approaches, illustrating the value of testing theoretical models with empirical data.

Identifying the mechanism of disease transmission is important for predicting disease spread in host populations (Begon *et al.* 2003, Wasserberg *et al.* 2009). The recognition that frequency-dependent transmission is not restricted to sexually-transmitted or vector-borne diseases is growing, as determined recently for facial tumour disease in the Tasmanian devil *Sarcophilus harrisii* (McCallum *et al.* 2009) and *Gyrodactylus turnbulli* in the guppy *Poecilia reticulata* (Johnson *et al.* 2011). That frequency-dependent transmission was supported for mange in the Bristol fox population (chapter 5), reflects transmission driven by socially determined contact rates. This finding is important for the future control of mange, and potentially other diseases, in

fox populations and other social species. However, control of diseases with frequency-dependent transmission is challenging given the absence of a critical host density threshold for disease invasion, since transmission is independent of population density (Lloyd-Smith *et al.* 2005a). Unsuccessful culling to control rabies in canids (Morters *et al.* 2013) and vampire bats *Desmodus rotundus* (Streicker *et al.* 2012) has been attributed to a complex relationship between disease transmission and density that is influenced by demographic heterogeneity, compensatory mechanisms and sociality. Thus, further data and modelling are required to establish the most effective method of control for diseases with transmission that is predominantly frequency-dependent.

Understanding the impact of age or social structure on transmission is of applied importance, including for designing targeted disease control (Bolzoni *et al.* 2007, Carter *et al.* 2009). Age-structured modelling has advanced particularly for notifiable childhood diseases such as measles (Keeling & Grenfell 1997) and whooping cough (Rohani *et al.* 2010). Such models point to the importance of accounting for complex social factors; for example, seasonal forcing of contact rates captured the dynamics of measles during school terms (Keeling & Grenfell 1997). In the SEI model (chapter 5), likelihood-based estimates of age-specific transmission coefficients, β, were fourfold higher for juveniles than adults, reflecting possible differences in immune response or movement patterns determined by life history stage. Calibration of age-specific β, used in the IBM (chapter 7) to model intra-group mange transmission, produced estimates that were consistent with the results of the SEI model (chapter 5). Even though the parameter estimates of β were consistent in both models, the dynamics differed between the two models (see below), partly because of the addition of stochasticity and social structure but also because age-specific transmission rates were limited to intra-group transmission in the IBM (chapter 7). Apart from some notable examples, e.g. cowpox in voles (Smith *et al.* 2009c), our understanding of wildlife disease dynamics lags behind that of human infectious diseases due to a paucity of data. This thesis (chapters 5 and 7) illustrates the importance of age for mange transmission, but also highlights how data limitations preclude the elucidation of seasonality and other complex factors influencing age-specific transmission.

Increasingly, models are revealing the importance of indirect transmission for describing disease dynamics (Barlow *et al.* 2002, Miller *et al.* 2006, Roche *et al.* 2009). While SEI models (chapter 5) did not support the inclusion of an indirect transmission pathway, possibly in part because of a lack of data, direct transmission alone did not adequately describe the outbreak of mange in the IBM (chapter 7). Since social groups were not incorporated into the SEI model due to the difficulties of incorporating group interactions in compartment models (Lloyd-Smith *et al.* 2005a), the varying transmission rates stemming from these social interactions, particularly the low inter-group contact rates, could have led to an overestimation of direct transmission in the compartment model. While indirect transmission was supported in the IBM (chapter 7), in many disease systems, the relative contribution of environmental and direct transmission is thought to vary temporally. Processes leading to such variation in transmission mechanisms include interactions between resident and migratory shorebirds in avian influenza (Brown *et al.* 2012), varying host density in human influenza (Spicknell *et al.* 2010) and environmental contamination increasing with the duration of chronic wasting disease outbreaks (Almberg *et al.* 2011). In light of the uncertainty associated with the mechanism of fomite contact during the mange outbreak in the Bristol fox population, the temporal contribution of direct and indirect transmission modes remains undetermined. However, it is also possible that the inferred support for indirect transmission reflects unknown processes that increase the rate of disease transmission, such as individuals acting as reservoirs or superspreaders.

Social interactions can play a major role in determining patterns of disease transmission (Altizer *et al.* 2003b, chapter 5). Contrary to some suggestions (Lindström 1992), dispersing foxes did not play a large role in local disease transmission in simulations of the mange outbreak (chapter 7), although there were limitations to modelling the dispersal process. These individual-based simulations also suggested that the collapse of territory formation observed during the epizootic (Baker *et al.* 2000) increased the spread of mange and decreased the chance of population recovery, due to an increase in transmission via fomites and a reduction in reproduction, respectively (chapter 7). Understanding how social perturbation affects disease spread and subsequent recovery is important for management. For instance, in

European badgers *Meles meles*, social perturbation through culling promoted the movement of individuals infected with Tb (Carter *et al.* 2007, Pope *et al.* 2007). Pack formation collapsed during a rabies outbreak in Ethiopian wolves *Canis simensis*, prompting a recommendation to vaccinate entire packs in order to maintain behavioural functionality within the population (Randall *et al.* 2004). In foxes, simulations indicated that immigration increased following culling (Rushton *et al.* 2006), and indeed in the IBM (chapter 7), long-distance recolonisation, possibly by immigrating individuals, was necessary for population recovery after the mange epizootic in Bristol. This study contributes to our understanding of the social processes involved in the transmission of mange and underlines the complex ecological processes involved in the control of disease in wildlife populations.

The role of genetic resistance for mange dynamics has been indicated in both empirical and theoretical work in canids (Leung & Grenfell 2003, Davidson *et al.* 2008). Consistent with simulations of mange in a coyote *Canis latrans* population (Leung & Grenfell 2003), resistance was required in the IBM to reproduce both population density and prevalence patterns (chapter 7). These results are supportive of evidence for rapid evolution occurring over shorter evolutionary timeframes than previously thought (Altizer *et al.* 2003a). That the proportion of resistant individuals was higher in the IBM incorporating indirect transmission than with direct transmission alone (chapter 7), is consistent with the suggestion that infection rates influence the selection of alleles (Robinson *et al.* 2012). Indeed, the faster evolution of immunity to myxomatosis in Australian rabbits *Oryctolagus cuniculus* compared to UK populations was attributed partly to the relatively higher virulence in the former population (Kerr *et al.* 2012). Although the actual physiological mechanism for immunity to mange remains unclear in all susceptible species, including humans (Walton 2010), the recent development of an experimental animal model for porcine scabies aims to determine the evolution and adaptation of the immune response to this disease (Mounsey *et al.* 2010). Importantly, the modelling approach in this study (chapter 7) provides further support that immunity is important for mange dynamics in canids.

8.2 Further implications

8.2.1 Demographic models: only as good as uncertainty allows

Data limitations frequently prevent the quantification of sources of demographic uncertainty (Wisdom *et al.* 2000). This was illustrated in chapter 4, where the study duration of focal populations was too short to enable separation of process and sampling error for all but one fox population. Future data collection should strive to maximise the efficiency of the sampling effort invested in fox populations. Often, fox population samples are biased due to seasonal variations in the catchability of different age classes (Kolb & Hewson 1980, Tryjanowski *et al.* 2009). Demographic modelling could be used to determine the representativeness of population structure from different sampling techniques and schedules, using existing data such as the long-term study of the Bristol population. Such approaches include information theoretic approaches and multi-state models, as applied to capture-mark-recapture studies of snakes and seabirds to incorporate behavioural responses to trapping (Willson *et al.* 2011) and improve accounting for biases in survey design (Kendall *et al.* 2009), respectively. By establishing synchrony of data collection and following guidelines (chapter 4), including those for rigorous parameter definitions, future studies of fox demography will be better able to determine inter-annual variation, sources of uncertainty, potential correlations among vital rates, and biases in existing samples, as well as serving to address many of the questions posed in the following sections.

Integrated population models (IPMs) are an emerging method that provide a useful means for combining multiple data sources and for estimating sampling and process error (Abadi *et al.* 2010). The methods in this thesis for addressing uncertainty (chapters 2, 3 and 4) could be incorporated into either Bayesian or frequentist IPMs. Of particular interest is the promise of using IPMs to estimate unknown demographic rates with existing data, thus eliminating the need for surrogate parameters when faced with sparse data. Using a Bayesian framework, Abadi *et al.* (2010) accurately estimated fecundity parameters by fitting likelihoods determined from simulated capture-mark-recapture (CMR) and population size data. Observed cycles in cub production were predicted by a frequentist IPM of black bear *Ursus americanus* that

used only age-at-harvest and CMR data (Fieberg *et al.* 2010). This use of age-at-death data is of direct application to foxes, given that mortality data are readily available for many populations. IPMs also reduce uncertainty in λ: the width of the λ confidence intervals was reduced by a third with a Bayesian IPM using radio-tracking data and population density estimates of koala *Phascolarctos cinereus*, compared to a model using only the radio-tracking data (Rhodes *et al.* 2011). IPMs therefore have the potential to be applied to those fox populations in chapter 4 that lack fecundity data, but which have an independent estimate of population size.

8.2.2 Same species, different demographics

Recognition of population-level variation is increasing in many areas of ecology and evolution, including reproductive effort (Mason *et al.* 2011), evolutionary-stable strategies (Hesse *et al.* 2008) and bet-hedging (Nevoux *et al.* 2010). The contrasting demography of fox populations (chapter 4) is of direct relevance to management. The intimation that UK rural fox populations respond differently to hunting pressure (Heydon & Reynolds 2000) and the varying success of baiting in Australian fox populations (Saunders *et al.* 2010), present opportunities for examining relationships between intraspecific population dynamics and local conditions. Additional data are required to obtain a more comprehensive picture of the mechanisms driving these demographic differences. Particularly valuable are long-term data on population-specific climate and hunting effort, given the importance of local conditions inferred from this study (chapter 4). The insight that such data provide is illustrated for leatherback turtles *Dermochelys coriacea*; lower reproductive effort in Pacific populations, driven by high temporal variability in resources, has resulted in decreased resilience to anthropogenic mortality compared to Atlantic populations and led to population-specific management recommendations (Wallace & Saba 2009). While it was not possible to substantiate widely the presence of inter-annual demographic variation in this study (chapters 3 and 4), future research should examine whether contrasting demographic tactics are supported in light of pervasive environmental stochasticity.

It is important to distinguish whether the inter-population differences seen in this study are conditional not only on possible inter-annual variation but also on transient dynamics. These short-term dynamics occur before or if a population converges on a stable stage distribution and asymptotic growth, for example as a result of a perturbation such as harvesting (Ezard *et al.* 2010). A study of metapopulation dynamics in yellow-bellied marmots *Marmota flaviventris* found that the relative contribution of patches to the total λ differed between transient and asymptotic dynamics, and that transient, but not asymptotic dynamics were driven in part by patch-specific population size and structure (Ozgul *et al.* 2009). Transient dynamics in fast-living bird and mammal species were found to be less variable and deviated less from asymptotic dynamics than in slow species, possibly because the longer generation times of the latter result in a higher chance for demographic variability (Koons *et al.* 2005). Future analyses should determine the relationship of transient and asymptotic dynamics with local conditions, life history speed and perturbations. Given the relevant data, the methods in this thesis (chapters 2 and 4) could easily be applied to exploring intraspecific transient and inter-annual dynamics. For example, do fast fox populations converge more rapidly on "stable" dynamics following a perturbation, and does life history change in response to environmental conditions?

Current understanding of intraspecific responses to disease is limited in wild populations, but worthy of further consideration. Cahn *et al.* (2011) simulated disease in Sierra Nevada bighorn sheep *Ovis canadensis sierrae* populations, finding that a stable population (λ = 1) was unable to recover from mild or severe disease outbreak without management intervention, a slowly growing population (λ = 1.07) could only withstand mild disease outbreaks, whereas a faster growing population (λ = 1.1) was sufficiently robust to the impacts of severe disease to recover without management. Further work is required to determine whether these types of analyses are useful in light of the underlying causes of variation in λ and density-dependent processes, although in this example, both density-dependent and independent simulations produced equivalent results. Contrasting prevalence and recovery among local fox populations exposed to rabies was thought to arise from intraspecific responses to hunting pressure (Zimen 1982). Given the possible intraspecific demographic variation

(chapter 4), population-specific responses to mange could be explored through simulations, as demonstrated in this study (chapter 7).

8.2.3 The adaptive modelling loop

An important role of models is to guide future multidisciplinary research, which is especially pertinent in wildlife disease ecology given the multi-species interactions of population dynamics, behaviour and epidemiology. For example, modelling of the poorly understood Lagos bat virus in straw-coloured fruit bats *Eidolon helvum* directed work to determine age-specific demographic rates and the existence of protective acquired immunity (Restif *et al.* 2012). Field and laboratory studies on plague *Yersinia pestis* in black-tailed prairie dogs *Cynomys ludovicianus*, guided by modelling, revealed that previous hypotheses underestimated the importance of early-phase and environmental transmission for disease spread (Restif *et al.* 2012). Indeed, frequent updating of models with revised data can then be used to refine management actions. In this context, modelling was recommended to guide and evaluate management efforts to control facial tumour disease in Tasmanian devils, given the continued failure of culling and the uncertainty over transmission mechanisms (Lachish *et al.* 2010). The following sections discuss some of the demographic, social and epidemiological uncertainties highlighted by modelling mange (chapters 5 and 7), that require further theoretical, field, experimental and captive studies to increase our understanding of mange dynamics and to refine management actions.

8.2.4 Faster life history, higher infection

The relevance of life history speed for explaining variation in aspects of ecology is gaining recognition (e.g. for senescence see Jones *et al.* 2008c, and for personality see Careau *et al.* 2009) and recent work suggests that the fast-slow continuum can be used to predict species-specific susceptibility to infection (Lee 2006, Martin *et al.* 2006, Johnson *et al.* 2012). Such insight improves our understanding of the relationship between life history strategy and disease dynamics, both within and between species. Relative to fast species, slower amphibian species were found to invest more in immunity, resulting in lower parasite loads (Johnson *et al.* 2012). Lee *et al.* (2008)

proposed that fast-living bird species invest less in immunity compared to slow species in order to minimise the resources devoted by juveniles to the immune response. Notably, the innate traits of fast species, e.g. high productivity and low investment in immunity, increase not only their susceptibility to infection but also their resilience to biodiversity loss, which is likely to be of consequence for future disease spread in light of the increasing perturbation of natural ecosystems (Keesing *et al.* 2010). Given the inferred importance of resistance to mange (chapter 7), and that foxes exhibit a range of speeds within the medium-fast classification (chapter 4), the investment that this widespread canid makes in immunity could be considered in relation to its life history. Specifically, such insight could be gained through a comparison among conspecific fox populations and to other carnivores along the fast-slow continuum.

8.2.5 Socialising with infection

Sociality can influence disease transmission by changing the rate of contact with infectious individuals or substances. Social-ranking can cause dominant individuals to experience an above average number of encounters; however, there is evidence that the directionality of these contacts is important in determining successful disease transmission. For example, meerkats *Suricata suricatta* receiving aggression are more at risk of *Tb* infection than those individuals directing the aggression (Drewe 2010). Disease-induced changes in social behaviour, such as the restricted movement observed in mangy foxes (Overskaug 1994) and increased diurnal movement in mangy bare-nosed wombats *Vombatus ursinus* (Borchard *et al.* 2012), are hard to document and often unpredictable, but may result in reduced encounter rates; simulations show that disease transmission can decrease in a population if infected individuals become isolated from other group members (Gudelj *et al.* 2004). Social perturbation potentially influences disease transmission by increasing contact rates (Carter *et al.* 2007), although the processes relating to social disruption are still poorly understood. In the IBM (chapter 7), although inter-group contact rates did not change, territory collapse resulted in increased contact with fomites, speeding up the transmission of mange. Insight into social contacts in fox populations could be gained through proximity data loggers (Böhm *et al.* 2009).

The models in this thesis provide a foundation for future work. Specifically, the IBM developed in chapter 6 could be modified to include decision rules that allow for the adaptive behaviour of individuals (e.g. Stephens *et al.* 2002a). In addition, network modelling is increasingly being applied to determine the role of social processes for disease transmission in wild populations (Cross *et al.* 2004, Drewe 2010, Craft *et al.* 2011), where the relationship between individuals can be simulated to determine the direction and intensity of interactions. Network modelling is often used to simulate "small-worlds", where most individuals are not neighbours but any two individuals are connected by a short number of "steps". For instance, occasional pride-to-pride contact was sufficient to promote the persistence of canine distemper in a small-world network of lions *Panthera leo* (Craft *et al.* 2011) and in schools with small-world networks, vaccination based on contact structure was recommended for influenza (Salathe *et al.* 2010). Disease control strategies in social species should therefore include measures to account for the nature of such contacts.

Not only does sociality influence disease transmission through the behavioural changes described above, but social status can alter an individual's physiological likelihood of infection. Higher-ranking individuals had better immunity than lower-ranking conspecifics due to greater access to resources in spotted hyena *Crocuta crocuta* (Höner *et al.* 2012) or high levels of testosterone in baboons *Papio cynocephalus* (Archie *et al.* 2012). However, endoparasite loads in fur seals *Arctocephalus forsteri* were found to be higher in dominant individuals that had high levels of testosterone (Negro *et al.* 2010). The role of social status in the immune response to mange remains to be determined. Important progress towards understanding the impact of sociality for immunity to mange could be made by combining field-based immunological tests with a long-term history of individual infections (Pedersen & Babayan 2011), such as that of the Bristol fox population. The influence of sociality on immunological function has long been recognised in humans (Berkman & Syme 1979, Uchino 2006). In this context, learning about immune processes in closely-related human diseases and using techniques developed for their study can provide insight into immune responses in wildlife diseases (O'Brien *et al.* 2006). Understanding such physiological processes is

especially important given that selection pressures can act on immunity over evolutionary short time frames (Altizer *et al.* 2003a, chapter 7).

The interface of disease ecology and ecological immunology is an emerging field that bridges within- and between-host processes, thus linking transmission dynamics and variation in immune responses (Hawley & Altizer 2011). Underlying immunological processes can be better understood through insight into the influence of sociality and life history on disease transmission (see above and previous section). Recent work suggests that negative and positive covariation between behavioural and physiological processes is important for determining R_0, and hence, the likelihood of a disease invading (Hawley *et al.* 2012). Changes in contact rates or immunity can give rise to superspreaders, individuals that cause significantly higher than average infections (Lloyd-Smith *et al.* 2005b). In such diseases infrequent but explosive epidemics can occur after the introduction of a single case, as illustrated in the recent SARS outbreak (Lloyd-Smith *et al.* 2005b). Evidence from porcine mange (*S*. *scabiei* var. *suis*) in captive pigs suggests that a small number of individuals have significantly higher than average mite loads and intensity of infection (Davies 1995), but the relationship with transmission is unknown. The fact that mange remains endemic in the Bristol fox population provides a useful opportunity to explore disproportionate infection risk, especially since asymmetrical infection rates are inferred from the age-specific variation in mange transmission (chapter 5). Further data are required to determine the relative influence of social status or life history stage for individual infection risk in foxes and whether resistance allowed certain individuals to live with the disease, or recover and become re-infected. Ultimately, greater insight into the consequences of individual immunity for fitness and the adaptive significance of life history for host-pathogen dynamics can be gained by taking a multidisciplinary approach, combining immunological and ecological data, to studying disease in wild populations.

With the exception of a few relatively well-studied wildlife diseases such as avian influenza (Lebarbenchon *et al.* 2009), epidemiological parameters of indirect transmission are typically estimated through modelling. In compartment models, the density of fomites in the environment is often simulated by fitting the parameters

related to fomite transmission to data (chapter 5, Barlow *et al.* 2002, Miller *et al.* 2006, Roche *et al.* 2009). However, there are few published studies of indirect transmission in IBMs and within these examples there is no consistent method of modelling the pathogen load in the environment. The approaches taken thus far include incorporating the viral load of avian influenza in lakes as a deterministic "compartment" (Roche *et al.* 2011), ascribing a proportion of individuals as "superexcretors" of Tb in badgers (Shirley *et al.* 2003) and a climate-dependent probability of infection given a fixed number of water sources contaminated with brown rot fungus (Breukers *et al.* 2006). In chapter 7, a novel approach was taken, determining a "fomite load" based on the number of infected individuals and the proportion of contact between territories, with a fitted rate of successful infection from contact with the infested substance. Given the difficulties of observing contact rates with fomites in wild populations, a benefit of the fomite load approach taken in chapter 7 was that it constrains the number of parameters requiring model fitting by implicitly incorporating the many unknown processes. Thus, more realistic incorporation of indirect transmission into IBMs, not only in mange systems, could be improved by simulating finer-scale behavioural and epidemiological data to provide further insight into the relative importance of direct and indirect pathways. Research is now required to identify possible mechanisms of indirect mange transmission, such as the limited entry points into gardens that could be a bottleneck for transmission, as well as rates of mite uptake and shedding by foxes. Such information is also useful since diseases with indirect transmission can be harder to control due to the additional transmission component. For example, low culling success was predicted for white-nose syndrome in bats due to the persistence of the fungus in the environment promoting indirect transmission (Hallam & McCracken 2010). In this context, the identification of the most influential parameters for indirect transmission, such as the viral inactivation rate in water for avian Influenza (Lebarbenchon *et al.* 2009), will be of consequence for designing optimal control programmes.

Uncertainty exists over whether conventional epidemiological parameters realistically describe disease transmission in social species. The basic reproductive number, R_0, is a population measure and thus, group structure and individual differences are not

explicitly accounted for (Cross *et al.* 2007). In reality, low contact rates and high spatial clustering, as seen in social species, can result in underestimating R_0 (Keeling 2005). R_0 can be adapted to account for variation in infection rates by determining the number of groups, R_*, infected by an initial diseased group in an otherwise susceptible population (Ball *et al.* 1997). Estimating R_* requires following those individuals that infect new groups (Cross *et al.* 2005). In this thesis, for reasons of computational simplicity, inter- and intra-group disease transmission was modelled without assigning a specific individual as the source of infection. Therefore, the transmission mechanism in chapter 7 requires modification in order to determine R_* for the simulated fox-mange system.

8.2.6 Foxes and mange: a community affair?

Disease systems do not exist in isolation. Interactions between and within multi host-pathogen systems can be a selective pressure on immunity (Bordes & Morand 2009) and trade-offs in the effects of parasites on host populations are thought to contribute to the evolution of optimal group size (Møller *et al.* 1993). In wild field vole *Microtus agrestic* populations, relative infection with microparasites explained more variation in infection risk than factors such as age and season (Telfer *et al.* 2010). Foxes are susceptible to multiple, often concurrent pathogens, including rabies*, E. multilocularis*, *Toxocara canis*, and many ecto-and endo-parasites (Deplazes *et al.* 2004, Vitasek 2004, Barbosa *et al.* 2005, Kočišová *et al.* 2006). Inter-population variation in parasite distributions was found in Spain, with rural fox populations having a higher diversity of parasites than urban populations (Barbosa *et al.* 2005). Within-host parasite interactions were inferred by the high prevalence of intestinal worms found in mangy foxes in Italy, where the relationship between mange mites and helminths was suggested to result in part from a trade-off in immunological response (Balestrieri *et al.* 2006). Within-host parasite interactions are not only important to consider for understanding disease dynamics but will also ultimately influence the success of control programmes.

Multi-host systems for mange are widespread: for example, foxes are potential vectors for domestic dogs in the UK (Soulsbury *et al.* 2007) and for the Iberian wolf *Canis lupus*

in Spain (Oleaga *et al.* 2011). Models are increasingly providing insight into multi-species infections. For instance, modelling suggested that rabies was more likely to die out when a multi-host outbreak started in foxes than in badgers, but that a single cross-species transmission to badgers was sufficient to promote disease persistence (Singer & Smith 2012). A lag in the observed and predicted incidence of poxvirus in red squirrels *Sciurus vulgaris* following the invasion of infected grey squirrels *Sciurus carolinensis* indicated low rates of direct contact between the two species (Rushton *et al.* 2000). Modelling disease control can also reveal unintended consequences in multi-species population dynamics, such as decreased cub survival in cheetahs *Acinonyx jubatus* due to increased predation by lions *Panthera leo* following their vaccination against canine distemper (Chauvenet *et al.* 2011). Community disease ecology remains an under-studied issue in fox-parasite systems. In particular, the potential effects for fox-mange dynamics warrant further research, especially given the inferred importance of indirect transmission and the evolution of immunity (chapter 7).

8.2.7 Conservation implications of mange: the bigger picture

The relevance of disease ecology to conservation is now widely recognised (Dobson & Hudson 1986, Daszak *et al.* 2000, Altizer *et al.* 2003a, Pedersen *et al.* 2007, Jones *et al.* 2008a), although integration of disease control into conservation management is constrained either by a lack of information or understanding (Woodroffe 1999, Lafferty & Gerber 2002). One reason epidemiology is overlooked by conservationists is the conjecture that disease is unlikely to cause extinction in small populations because transmission is density-dependent and, as a result, there is a host density threshold below which a pathogen cannot invade (Lafferty & Gerber 2002). However, as demonstrated in this thesis (chapter 5), traditional transmission assumptions are increasingly being challenged through modelling. Hence, frequency-transmitted pathogens, especially in social species, could pose a significant risk to already compromised populations. For example, modelling of white-nose syndrome and facial tumour disease demonstrates that extinction is a real possibility in US bat (Langwig *et al.* 2012) and Tasmanian devil populations (McCallum *et al.* 2009), respectively. In the context of mange, this disease has caused significant declines in isolated southern

hairy-nosed wombat *Lasiorhinus latifrons* populations (Ruykys *et al.* 2009) and is a major cause of mortality in the threatened Masai Mara cheetah population (Gakuya *et al.* 2012). Given the complex nature of disease transmission (chapters 5 and 7), understanding population dynamics and epidemiology is essential for successful wildlife disease management (Woodroffe 1999, Breed *et al.* 2009).

A pressing issue for conservation is the transmission of disease between domesticated and wild species. Over 80% of domesticated animal pathogens have the potential to infect wildlife species (Cleaveland *et al.* 2001). Mammalian orders with the highest number of domesticated or human-associated species (e.g. carnivores, ungulates and rodents) face a disproportionate risk of infectious disease outbreak (Pedersen *et al.* 2007). Indeed, domestic-wildlife mange transmission is a current and potential threat to many species (Daszak *et al.* 2000, Gortazar *et al.* 2007); domestic dogs were the source of rabies epizootics in Ethiopian wolves (Randall *et al.* 2004) and the extinction of a Spanish ibex *Capra pyrenaica hispanica* population was caused by a mange outbreak stemming from domestic goats (Leon-Vizcaino *et al.* 1999). Insight into mange dynamics, such as that provided by chapters 5 and 7, as well as understanding how age classes that are important for disease transmission contribute to population growth (chapter 4), can contribute to refining the management of this disease. The potential for inter-species transmission could be reduced trough the targeted control or treatment of specific sexes or age classes of mangy individuals (Gressmann & Deutz 2001) in wild and/or domestic species, identifying direct or indirect routes of transmission between domestic and wild populations, or by acting to increase the proportion of the disease-resistant population, such as by translocation of individuals with resistance alleles (Hamede *et al.* 2012).

Global change, including climate change and biodiversity loss will inevitably alter the persistence and range of parasites. The loss of species that are more resilient to infection can alter disease dynamics due to differences in life history (see section 8.2.4) or encounter dilution effects. For example, hantavirus prevalence increased when the experimental reduction of small mammal species richness resulted in higher densities of the generalist reservoir host *Zygodontomys brevicauda* (Suzán *et al.* 2009). In this

context, changes in multi-host community composition could be of importance for mange dynamics in susceptible threatened species. In light of climatic changes, increasingly favourable temperatures have resulted in the expansion of Bluetongue into Northern Europe in recent years due to the increased survival of the disease's main vector (Purse *et al.* 2005) and several vector-bourne diseases, including malaria, have expanded into previously disease-free latitudes and altitudes (Kovats *et al.* 2001). A slower spread of mange was observed in Spanish ibex *Capra pyrenaica* during dry years due to the inhospitable climate for the mites (Perez *et al.* 1997). Given the potential for indirect transmission (chapter 7) and that the persistence of mange mites in the environment is driven by temperature and moisture (Arlian *et al.* 1989), changes in climate may alter the prevalence or intensity of this disease.

8.3 Conclusion

In this study, I established that demographic analyses of a common species could provide insight into methodologies to account for data uncertainty, identify intraspecific demographic variation, and provide meaningful information on the dynamics of an important disease. Importantly, I found support for inter-population differences in the contributions of vital rates to population growth in the red fox. However, I highlighted the significant gaps in our understanding of global fox demography through reviewing the quality and quantity of demographic data and illustrated the management implications of ignoring uncertainty in demographic modelling. Using a long-term data set on an urban fox population, I made considerable progress towards elucidating the processes driving epizootic and enzootic phases of sarcoptic mange outbreaks and determining the impacts of sociality for disease transmission. Ultimately, I demonstrated that increasing our knowledge of a species' demography, and the pressures upon it, will enable the refinement of management decisions.

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Appendix 1. Summary of a review of global fox population dynamics

Underlined populations were selected for demographic analysis in chapter 4. ± standard deviations, where provided. [1]Data type: MD: Mortality data; CMR: Capture-mark-recapture; RT: Radiotelemetry; SS: Sign surveys; BE: Behavioural observations; G: Genetic. – Data not provided; [2]Habitat: 1 – Rural agricultural; 2 – Rural non-agricultural 3: Low population density; 4 – High population density.

Study #	*Study population*	*References*	*Data type*[1]	*Total study duration (years)*	*Max study area (km²)*	*Max sample size (from one study)*	*Habitat*[2]	*Sex ratio: all ages*; adults**; juveniles^; embryos^^*	*Density km⁻² (individual, litter* or group**)*	*Home range (km⁻²)*
1	**UK: London**	1, 2, 3	MD	6	1618	1141	4	1 : 0.96*	-	-
2	**UK: London**	4	CMR, SS	6	7.6	209	4		2.33 ± 0.39 1.03*	1.65
3	**UK: Bristol**	5, 3, 6, 7, 8, 9, 10, 11, 12	MD, RT, BE, SS, CMR, G	30+	116	1701	4	1 : 0.81* 1.2:1.0**	14.00± 8.34 1.82*	0.51 ± 0.48
4	**UK: Oxford**	13, 14, 15, 16	RT	10	9.17	>120	3,4	-	2.15 2.5**	0.92 ± 0.66
5	**UK: Wales**	17, 18	CMR	6	580	476	1,2	1:82**	1.85 ± 1.27 0.90 ± 0.57*	2.35 ± 2.33
6	**UK: Hampshire**	19	BE	1	53	124	2	-	0.57*	-
7	**UK: Dorset**	20	RT, SS	2	11	14	2	-	-	2.43 ± 0.97
8	**UK**	21, 22	MD	3	2322	656	1,2	1 : 1**	0.94 ± 0.85	-
9	**UK: Scotland**	23, 24	MD	23	48760	4765	1,2	-	1.09 ± 0.67	-
10	**Ireland**	25, 26	CMR	2	-	292	-	-	-	-
11	**Belarus**	27	SS	3	300	-	2	-	0.92 ± 0.93	-
12	**Belgium**	28	MD	2	589	314	3,4	0.95:1*	-	-
13	**France: north-east**	29, 30, 31, 32	RT, SS, MD, G	7	250	1259	1,3	-	-	1.18 ± 0.75

Study #	*Study population*	*References*	*Data type*[1]	*Total study duration (years)*	*Max study area (km²)*	*Max sample size (from one study)*	*Habitat*[2]	*Sex ratio: all ages*; adults**; juveniles^; embryos^^*	*Density km⁻² (individual, litter* or group**)*	*Home range (km⁻²)*
14	**France**	33	-	-	-	-	-	-	-	-
15	**Germany**	34	MD, BE	15	130	955	2	1.5: 1**	0.73 ± 0.25 0.55 ± 0.17*	7.00
16	**Germany**	35, 36	MD, CMR	5	1012	1371	1,2	-	0.74 0.31*	-
17	**Italy**	37, 38	RT, MD	2	2448	317	1,2,4	1 : 0.96^^	-	1.98 ± 1.28
18	**Netherlands**	39	RT	5	-	150	2	-	0.55*	3.48 ± 3.77
19	**Netherlands**	40, 41	RT	6	300	311	2	-	-	-
20	**Norway**	42	SS	3	18	2	2	-	-	5.47 ± 0.46
21	**Poland**	43, 44	SS, MD, BE	9	89	113	1,2	1.17 : 1**	0.71 ± 0.18 0.0.94-0.171*	-
22	**Poland**	45	SS	3	66	-	1,2	-	1.30 ± 0.31 0.31 ± 0.02*	-
23	**Russia**	46	MD	5	-	759	-	-	-	-
24	**Spain: Doñana**	47, 48	MD, SS	4	500	116	-	0.9:1^^	1.70	-
25	**Spain: Ebro**	49	MD	7	-	413	1,2	1:0.76*	-	-
26	**Sweden: South**	50, 51	MD, CMR	6	-	799	1,2	-	-	-
27	**Sweden: North**	50, 51	MD, CMR	4	-	870	1,2	-	-	-
28	**Sweden**	52	BE	6	3	13	1,2	-	-	4.00 ± 1.84
29	**Sweden**	53, 54, 55, 56, 57	MD, RT, SS	17	130	874	2	-	-	-

Study #	*Study population*	*References*	*Data type*[1]	*Total study duration (years)*	*Max study area (km²)*	*Max sample size (from one study)*	*Habitat*[2]	*Sex ratio: all ages*; adults**; juveniles^; embryos^^*	*Density km⁻² (individual, litter* or group**)*	*Home range (km⁻²)*
30	**Switzerland**	58, 59, 60	MD, SS	8	30	88	1,2	-	0.4 - 3.2 0.37 ± 0.04*	5.66 ± 11.68
31	**Japan**	61	MD	4	6800	690	1,2	-	-	-
32	**Japan**	62	RT	1	24	4	-	1 : 0.65** 1 : 0.74^	-	3.95 ± 1.98
33	**Japan**	63	-	1	-	6	-		-	4.94 (3.57-6.31)
34	**USA: New York State**	64	-	2	-	175	-	0.95 : 1^^	-	-
35	**USA: Indiana**		MD	1	-	104	-	-	-	-
36	**USA: Midwest**	65, 66	MD, SS, CMR, RT	9	84	2049	1,2	1 : 0.79** 1 : 0.82^ 1 : 0.96^^	-	9.71
37	**USA: Minnesota**	67	SS, RT	2	41.44	32	-	-	-	6.993 ± 1.372
38	**USA (Midwest): Wisconsin**	68, 69	-	4	83.73	-	-	1 : 1.04^	0.09 ± 0.03**	-
39	**USA: Illinois**	70	RT, MD	5	3000	611	1,4		-	-
40	**USA: New York State**	71, 72, 73	CMR, MD	5	26	2848	1,2	1.06:1** 1.35:1^	0.74 0.97 ± 0.09**	-
41	**USA (East): Maryland**	74	MD	3	-	210	1,2	1:1*	-	-
42	**USA: North Dakota**	75, 76	MD, RT	5	-	363	1,2	1.33:1** 1: 0.93^^	0.10 ± 0.04**	-
43	**USA: Alaska**	77	CMR, BE	4	3	30	2	-	9.53 ± 0.45	-

Study #	*Study population*	*References*	*Data type*[1]	*Total study duration (years)*	*Max study area (km²)*	*Max sample size (from one study)*	*Habitat*[2]	*Sex ratio: all ages*; adults**; juveniles^; embryos^^*	*Density km⁻² (individual, litter* or group**)*	*Home range (km⁻²)*
44	**Canada: Alberta**	78	SS, BE	9	21	-	1,2	-	-	-
45	**Canada: Ontario**	15, 79	RT	8	-	120	1	-	0.54 ± 0.65	9 (5.00-20.00)
46	**Canada: Ontario**	80	RT	1	4	7	3	-	0.57**	0.77 ± 0.39
47	**Australia: Canberra**	81	-	2	-	437	-	1:0.87*	-	-
48	**Australia: NSW**	82	-	5	-	838	-	-	-	-
49	**Australia: Victoria**	83, 84	MD	4	24	317	-	1: 0.79**	2.7 ± 1.38	2.56 ± 2.30
50	**Australia: Melbourne**	85, 86, 87	RT, MD, SS	5	21	50	4	-	5.99 ± 4.93 1.18 ±0.96*	0.28 ± 0.12
51	**Australia (Hunted): NSW**	88, 89	RT, MD, SS	3	-	534	1,2	1 : 0.72* 1:0.72^	-	-
52	**Australia: NSW**	90	-	2	77	21	2,4	-	-	1.35 ± 0.042
53	**Australia: NSW**	91	SS,MD	2	108	276	1	-	-	-
54	**Australia (Non-hunted): Western**	92	MD, SS,	1	200	204	1	1:1*	0.46–0.52	-
55	**Australia: south**	93	SS	10	20 km transect	-	2,4	-	0.60	-
56	**Australia: Melbourne**	94	RT	2	26	9	2,3	-	-	0.45 ± 0.13

Appendix 2. Demographic parameters from a review of global fox populations

Study numbers refer to Appendix 1, ± standard deviations, where provided. Studies from Appendix 1 that do not report relevant information are omitted. Underlined populations were selected for demographic analysis.

Study #	Study population	Age definition[1]	Juvenile: adult ratio	Survival (age-specific)	Litter size definition[2]	Breeding probability definition[3]	Litter size[4] (mean - all ages)	Litter size (age-specific)	Percent non-breeding (mean)	Percent non-breeding (age-specific	Percent dispersing - juvenile males (mean)	Percent dispersing –juvenile females (mean)
1	**UK: London**	1	0.53:0.47	0+0.38 1+0.43 2+0.49 3+0.44	1	1	-	0+4.6 1+5.0 2+4.9 3+4.9	-	0+ 24.6 1+8.1 2+4.9 3+3.5	-	-
2	**UK: London**	3	-	-	2	NA	-	-	-	-	-	-
3	**UK: Bristol**	1	0.50:0.50	0+ 0.44 1+0.53 2+ 0.52 3+0.51	1	1	-	0+4.5 1+4.9 2+4.8 3+4.7	-	0+24.4 1+17.1 2+19.1 3+2.9	44.0 ± 25.9	22.7 ± 12.6
4	**UK: Oxford**	NA	-	-	1	2	-	-	40.6± 25.5	-	-	-
5	**UK: Wales**	1	-	0.75-1: 0.45 1.75-2: 0.43 2.75-3: 0.44 3.75-4: 0.43 4.75-5: 0.50	1	1	4.6**	-	20.5	-	25.0 ± 16.2	32.5 ± 1.7
7	**UK: Dorset**	NA	-	-	1	NA	5.8 ± 1.9^	-	-	-	-	-
8	**UK**	1	-	0+ 0.45 1+ 0.45 2+ 0.30 3+ 0.45	1	1	5.55 ± 0.9	-	9.7 ± 13.72	-	-	-
9	**UK: Scotland**	1	0.67:0.33	0+ 0.34 1+ 0.45 2+ 0.43 3+ 0.13	1	NA	5.0**	-	-	-	-	-

Study #	Study population	Age definition[1]	Juvenile: adult ratio	Survival (age-specific)	Litter size definition [2]	Breeding probability definition[3]	Litter size[4] (mean - all ages)	Litter size (age-specific)	Percent non-breeding (mean)	Percent non-breeding (age-specific	Percent dispersing - juvenile males (mean)	Percent dispersing –juvenile females (mean)
10	**Ireland**	3	0.64:0.36	-	1	3	-	-	9.8 ± 2.8	-	30.0	20.0
12	**Belgium**	1	0.51:0.49	0+ 0.42 1+ 0.51 2+ 0.63 3+ 0.92 4+0.36	NA	NA	-	-	-	-	-	-
14	**France**	1	0.54:0.46	-	NA	NA	-	-	-	-	-	-
15	**Germany**	1	0.66:0.34	0+ 0.35 1+ 0.34 2+ 0.35 3+ 0.32 4+ 0.23	2	NA	4.8 ± 1.1* 6.8 ± 0.9**'	-	-	-	-	-
16	**Germany**	1	0.56:0.44	-	1	1	4.6*	0+ 4.5^ 1+ 5.3 2+ 4.7 3+ 4.9	-	0+ 24 1+ 17.9 2+ 0.0 3+ 6.8	-	-
17	**Italy**	1	0.52:0.48	-	1	2	4.0 ± 1.3^ 3.9 ± 1.6**	-	20	-	-	-
21	**Poland**	1	0.54:0.46	0-0.167: 0.69 0.167-0.5: 0.76 0.5-1: 0.45 1+ 0.56 2+ 0.428 3+ 0.38 4+ 0.32	1	NA	3.8 (2.7 - 4.5)* 5.5^	-	-	-	-	-

Study #	Study population	Age definition[1]	Juvenile: adult ratio	Survival (age-specific)	Litter size definition[2]	Breeding probability definition[3]	Litter size[4] (mean - all ages)	Litter size (age-specific)	Percent non-breeding (mean)	Percent non-breeding (age-specific	Percent dispersing - juvenile males (mean)	Percent dispersing –juvenile females (mean)
23	**Russia**	1	0.62:0.38	0+ 0.34 1+ 0.49 2+ 0.52 3+ 0.50 4+ 0.60	2	NA	-	-	-	-	-	-
24	**Spain: Donana**	1	-	-	1	1	3.1 (2.5-3.6)* 3.3 ±0.7 **	-	-13.2	-	-	-
25	**Spain:Ebro**	2	0.58:0.42	1+ 0.56 2+ 0.52 3+ 0.55 4+ 0.64	1	1	3.6 ± 0.4^	-	10.5 ±12.5	-	-	-
26	**Sweden (South)**	1	0.60:0.40	0+ 0.43 1+ 0.53 2+ 0.75 3+ 0.55	1	1	-	0+ 3.93^ 1+ 4.77 2+ 4.53 3+ 4.20	-	0+ 46 1+ 62 2+ 81	-	-
27	**Sweden (North)**	1	0.54:0.46	0+ 0.33 1+ 0.71 2+ 0.50 3+ 0.59	1	1	-	0+ 4.17^ 1+ 4.30 2+ 4.77 3+ 4.20	-	0+ 59 1+ 48 2+ 33	-	-
28	**Sweden**	NA	-	-	1	2	4.8 ± 0.7*	-	50	-	-	-
29	**Sweden**	1	-	0+ 0.53 1+ 0.67 2+ 0.66 3+ 0.61 4+ 0.66	1	NA	4.1 ± 0.5^	-	-	-	-	-
30	**Switzer-land**	NA	-	-	2	NA	3.9 ± 0.4*	-	-	-	-	-

Study #	Study population	Age definition[1]	Juvenile: adult ratio	Survival (age-specific)	Litter size definition[2]	Breeding probability definition[3]	Litter size[4] (mean - all ages)	Litter size (age-specific)	Percent non-breeding (mean)	Percent non-breeding (age-specific	Percent dispersing - juvenile males (mean)	Percent dispersing –juvenile females (mean)
31	**Japan**	2	0.70:0.30	0+ 0.19 1+ 0.51 2+ 0.53 3+ 0.40 4+ 0.75	NA	NA	-	-	-	-	-	-
32	**Japan**	1	0.62:0.38	0+ 0.20 1+ 0.88 2+0.43 3+ 0.70	NA	NA	-	-	-	-	-	-
34	**USA: New York State**	NA	-	-	1	2	5.4 (1-9) **	-	4.7	-	-	-
35	**USA: Indiana**	NA	-	-	2	2	6.8 ± 0.3	-	40	-	-	-
36	**USA: Midwest**	1	0.64:0.36	0+ 0.35 1+ 0.53 2+ 0.80 3+ 0.80 4+ 0.86	1	3	4.2 ± 0.1* 7.1 ± 1.9^ 6.8 ± 0.1**	-	-	-	87.4 ± 9.2	44.6 ± 11.5
38	**<u>USA (Midwest): Wisconsin</u>**	1	0.59:0.41	1+ 0.33 2+0.40 3+0.95 4+0.43	1	2	-	0+ 5.9** 1+ 5.4 2+ 6.8 3+ 5.3 4+ 8.0	-	0+ 41 1+ 10 2+11 3+ 25 4+ 0	-	-
39	**USA: Illinois**	3	-	0+ 0.27 1+ 0.35 0+ 0.63	NA	NA	-	-	-	-	-	-
40	**USA: New York State**	1	0.69:0.31	1+ 0.33 2+ 0.57 3+ 0.25 4+ 0.58	NA	NA	-	-	-	-	58.3 ± 14.0	47.5 ± 26.7

Study #	Study population	Age definition[1]	Juvenile: adult ratio	Survival (age-specific)	Litter size definition[2]	Breeding probability definition[3]	Litter size[4] (mean - all ages)	Litter size (age-specific)	Percent non-breeding (mean)	Percent non-breeding (age-specific	Percent dispersing - juvenile males (mean)	Percent dispersing –juvenile females (mean)
41	**USA (East): Maryland**	2	0.55:0.45	0+ 0.34 1+ 0.87 2+ 0.56 3+ 0.63 4+ 0.58	2	2	-	0+ 5.32^ 1+ 6.68 2+ 6.26 3+ 6.10	-	0+ 83 1+ 17	-	-
42	**USA: North Dakota**	2	0.44:0.56	-	1	1	-	0+ 3.1±2.3 1+ 4.7±2.2 2+ 4.9±2.2 3+ 5.6±1.9 4+ 4.8±1.3	-	0+ 28.3 1+ 7.7 2+ 7.7 3+ 5.3 4+ 0.0	62.0± 10.1	31.0 ± 34.7
43	**USA: Alaska**	3	-	-	2	2	4.2 ± 0.2*	-	78.8 ± 14.1	-	-	-
44	**Canada: Alberta**	3	-	-	NA	NA	5.0*	-	-	-	-	-
45	**Canada: Ontario**	3	0.79:0.21	Juv+ 0.20 1.5+ 0.40 2.5+ 0.83	2	3	8.0^	-	-	-	90.5	77.0
47	**Australia: Canberra**	3	-	-	2	3	3.8 (1-8)* 4.3 (1.8)^ 3.8 (1-6) **	-	2.6	3	-	-
48	**Australia: NSW**	2	-	-	2	3	3.7 ± 1.5^ 4.0 ± 1.6**	-	30	-	-	-

Study #	Study population	Age definition[1]	Juvenile: adult ratio	Survival (age-specific)	Litter size definition[2]	Breeding probability definition[3]	Litter size[4] (mean - all ages)	Litter size (age-specific)	Percent non-breeding (mean)	Percent non-breeding (age-specific	Percent dispersing - juvenile males (mean)	Percent dispersing –juvenile females (mean)
49	**Australia: Victoria**	1	0.55:0.45	-	1	NA	3.3*	-	-	-	31.0	23.5
50	**Australia: Melbourne**	1	-	-	1	NA	4.4 ± 0.2* 4.6^	-	-	-	-	-
51	**Australia (Hunted): NSW**	1	0.61:0.39	0+ 0.29 1+ 0.38 2+ 0.55 3+ 0.64 4+ 0.70	1	3	-	0+ 3.0 ± 1.8 1+ 3.9± 1.5 2+ 4.8± 1.3 3+ 4.1± 2.0 4+5.2± 1.8	-	0+30.6 1+14.8 2+13.3 3+8.3 4+8.3	-	-
53	**Australia: NSW**	1	-	-	NA	NA	-	-	-	-	-	-
54	**Australia (Non-hunted): Western**	1	0.54:0.46	0+ 0.39 1+ 0.65 2+ 0.92 3+ 0.17 4+ 0.5	1	2	-	0+ 3.5^ 1+ 3.9 2+ 3.1 3+ 4.5 4+3.0	-	0+ 0 1+ 0 2+ 0 3+ 0 4+ 0	-	-

[1]Age definition: 1 – Well defined: Clear description of technique, with juveniles clearly defined; 2 – Adequately defined: Technique stated, but juveniles poorly defined; 3 – Poorly defined: No definition provided.
[2]Litter size definition: 1 – Well defined: Clear description of technique, e.g. defining grades of placental scars, or live embryos; 2 – Adequately defined: Technique stated but lack of detail; 3 – Poorly defined: No definition provided. NA – not applicable for study purpose.
[3]Breeding probability: 1 – Well defined: Clear description of technique, e.g. stating inclusion of post-implantation loss/reabsorptions; 2 – Adequately defined: Technique stated but lack of detail; 3 – Poorly defined: No definition provided. Litter size: ^Placental scars; *direct counts; ** embry

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Appendix 3. Functional forms for litter size probability distributions

The 12 probability distributions, $f(x)$, fitted to the empirical litter size frequencies are described below. Here, x is the litter size, y is (x-1) and x_{max} is the maximum litter size for a given population. Γ is the complete gamma function and λ, s and f_{max} are the parameters of the distributions fitted by maximum likelihood. Continuous distributions* were converted into discrete forms by calculating values for x = 1,2,…x_{max} and rescaling the probabilities sum to unity.

Distribution	***Functional form***	***Estimated parameters and possible range***	***Possible range of x***
Shifted Poisson	$f(x \mid \lambda) = \frac{\lambda^x e^{-\lambda}}{x!}$	λ > 0	$x \geq 0$
Zero-truncated Poisson	$f(y \mid \lambda) = \frac{\lambda^y e^{-\lambda}}{(1-e^{-\lambda})y!}$	λ > 0	$y \geq 1$
Shifted Generalised Poisson	$f(x \mid \lambda, s) = \frac{\lambda(\lambda + xs)^{(x-1)} e^{-(\lambda+xs)}}{x!}$	λ > 0	$x \geq 0$
Zero-truncated Generalised Poisson	$f(y \mid \lambda, s) = \frac{\lambda(\lambda + ys)^{(y-1)} e^{-(\lambda+ys)}}{(1-e^{-\lambda}+ys)y!}$	λ > 0	$y \geq 1$
Shifted Binomial	$f(x \mid \lambda, s) = \frac{\lambda!}{(\lambda-x)!x!} s^x (1-s)^{(\lambda-x)}$	λ is a positive integer, $0 \leq s \leq 1$	$0 \leq x \leq \lambda$
Zero-truncated Binomial	$f(y \mid \lambda, s) = \frac{\lambda!}{(1-s)^{\lambda}(\lambda-y)!y!} s^y (1-s)^{(\lambda-y)}$	λ is a positive integer, $0 \leq s \leq 1$	$1 \leq y \leq \lambda$
Shifted Negative Binomial	$f(x \mid \lambda, s) = \frac{\Gamma(x+\lambda)}{\Gamma(\lambda)x!} s^{\lambda} (1-s)^x$	λ > 0, s > 0	$0 \leq x \leq \infty$
Zero-truncated Negative Binomial	$f(y \mid \lambda, s) = \frac{\Gamma(y+\lambda)}{(1-s)^{\lambda}\Gamma(\lambda)y!} s^{\lambda} (1-s)^y$	λ > 0, s > 0	$1 \leq y \leq \infty$
Discretised normal*	$f(x \mid \lambda, s) = \frac{1}{\sqrt{(2\pi)s^2}} e^{\frac{(x-\lambda)^2}{2s^2}}$	s > 0	$1 \leq x \leq x_{max}$

Distribution	*Functional form*	*Estimated parameters and possible range*	*Possible range of x*
Discretised lognormal*	$f(x \mid \lambda, s) = \frac{1}{\sqrt{(2\pi)}sx} e^{-\frac{(\ln x - \lambda)^2}{2s^2}}$	λ > 0, s > 0	$1 \le x \le x_{max}$
Discretised stretched beta, 2 parameter form*	$f(z \mid \lambda, s) = \frac{\Gamma(a+b)}{\Gamma(a)\Gamma(b)} z^{(a-1)}(1-z)^{(b-1)}$, where: $z = \frac{x - f_{min}}{f_{max} - f_{min}}$, $a = m\left[\frac{m(1-m)}{v} - 1\right]$, $b = (1-m)\left[\frac{m(1-m)}{v} - 1\right]$, $m = \frac{\lambda - f_{min}}{f_{max} - f_{min}}$, $v = s\left[\frac{1}{(f_{max} - f_{min})}\right]^2$, $f_{min} = 1$, $f_{max} = x_{max}$	λ > 0, s > 0	$1 \le x \le x_{max}$
Discretised stretched beta, 3 parameter form*	$f(z \mid \lambda, s) = \frac{\Gamma(a+b)}{\Gamma(a)\Gamma(b)} z^{(a-1)}(1-z)^{(b-1)}$, where: $z = \frac{x - f_{min}}{f_{max} - f_{min}}$, $a = m\left[\frac{m(1-m)}{v} - 1\right]$, $b = (1-m)\left[\frac{m(1-m)}{v} - 1\right]$, $m = \frac{\lambda - f_{min}}{f_{max} - f_{min}}$, $v = s\left[\frac{1}{(f_{max} - f_{min})}\right]^2$, $f_{min} = 1$	λ > 0, s > 0, f_{max} > 0	$1 \le x \le f_{max}$

Appendix 4. Summary of terrestrial carnivore litter size data from published studies

The study duration in years and the number of populations that the data refer to are indicated ('M' indicates multiple years or populations). The method of litter size determination refers to placental scars (ps), embryo counts (ec) or direct counts (dc). Sample size refers to the number of litters.

Species[Reference]	*Duration [Population]*	*Method*	*Sample size*	*Mean litter size*	*Variance*	*Variance/ mean*	*Skewness*
Vulpes velox [1]	2 [1]	dc	9	4.78	0.840	0.176	-0.126
Vulpes macrotis [2]	15 [1]	dc	101	3.75	1.632	0.435	-0.078
Vulpes macrotis [3]	4 [1]	dc	50	4.55	0.248	0.055	-0.024
Vulpes vulpes [4]	4 [1]	ps	112	4.77	1.660	0.348	-0.072
Vulpes vulpes [5]	6 [1]	ec	114	4.46	2.774	0.623	0.285
Vulpes vulpes [6]	14 [1]	dc	106	4.85	3.713	0.766	0.220
Vulpes vulpes [7]	6 [1]	ec	114	5.05	2.576	0.510	-0.125
Vulpes vulpes [8]	3 [1]	ps	113	4.40	4.877	1.109	0.144
Vulpes vulpes [8]	4 [1]	ps	58	4.29	2.104	0.490	0.106
Vulpes vulpes [8]	4 [1]	ps	109	4.79	2.754	0.575	0.059
Vulpes vulpes [9]	6 [1]	ps	158	4.62	2.375	0.514	0.243
Vulpes vulpes [10]	6 [1]	ec	42	5.05	1.807	0.358	0.015
Vulpes vulpes [11]	13 [1]	ps	340	4.69	2.351	0.502	0.066
Vulpes vulpes [12]	13 [1]	ec	60	4.17	3.206	0.769	0.046
Vulpes vulpes [13]	17 [1]	dc	191	5.35	2.332	2.332	0.667
Urocyon littoralis [14]	2 [1]	dc	20	2.50	0.550	0.220	-0.089
Urocyon littoralis [15]	5 [1]	ec	34	2.06	0.938	0.455	0.223
Urocyon cinereoargenteus [16]	2 [1]	ec	7	3.14	0.980	0.312	0.185
Urocyon cinereoargenteus [17]	5 [1]	ps	98	4.56	1.185	0.260	0.082
Alopex lagopus [18]	19 [1]	dc	167	6.31	11.003	1.745	1.233
Alopex lagopus [19]	3 [1]	dc	17	6.41	1.772	0.276	0.219
Canis lupus [20]	4 [1]	ps	12	5.42	3.076	0.568	0.552
Canis lupus [21]	12 [1]	dc	26	5.27	4.658	0.884	0.080
Lycaon pictus [22]	M [M]	dc	246	10.76	11.156	1.044	-0.539
Lycaon pictus [23]	15 [1]	dc	39	10.31	26.162	2.538	-0.565
Lycaon pictus [23]	15 [1]	dc	36	10.39	9.293	0.895	1.654
Lycaon pictus [23]	6 [1]	dc	25	8.88	16.746	1.886	-0.377
Nyctereutes procyonoides [24]	4 [1]	ps	15	8.13	2.916	0.358	0.077
Procyon lotor [25]	3 [1]	dc	15	8.13	2.916	0.358	-0.864
Crocuta crocuta [26]	3 [1]	dc	53	1.68	0.369	0.220	0.043
Crocuta crocuta [26]	3 [1]	dc	55	1.56	0.246	0.157	-0.027
Crocuta crocuta [27]	8 [1]	dc	106	1.44	0.266	0.184	0.047
Acinonyx jubatus [28]	3 [1]	dc	21	3.76	0.753	0.200	0.594
Felis concolor [29]	9 [1]	dc	26	2.38	0.621	0.261	0.013
Felis concolor [30]	9[1]	dc	27	2.22	0.469	0.211	-0.079
Felis concolor [31]	18 [M]	dc	258	2.87	0.825	0.287	-0.058
Felis iriomotensis [32]	13 [1]	dc	41	1.10	0.088	0.080	0.058

Species[Reference]	*Duration [Population]*	*Method*	*Sample size*	*Mean litter size*	*Variance*	*Variance/ mean*	*Skewness*
Lynx pardinus [33]	9 [1]	dc	15	3.13	0.516	0.165	0.179
Panthera tigris altaica [34]	8 [1]	dc	16	2.38	1.234	0.520	0.018
Panthera onca [35]	2 [1]	dc	23	1.61	0.499	0.310	0.136
Panthera leo [36]	2 [1]	dc	34	2.32	0.807	0.347	0.203
Panthera leo [37]	4 [1]	dc	28	2.68	0.504	0.188	-0.010
Panthera leo [37]	4 [1]	dc	38	2.82	0.677	0.240	0.017
Panthera leo [38]	24 [1]	dc	110	2.54	1.049	0.413	0.044
Panthera leo [38]	24 [1]	dc	200	2.46	1.028	0.418	0.151
Panthera leo [38]	24 [1]	dc	159	2.48	1.130	0.455	0.144
Panthera pardus [39]	5 [1]	dc	11	1.73	0.198	0.115	0.130
Leopardus pardalis [40]	13 [1]	dc	13	1.23	0.178	0.144	0.141
Ursus maritimus [41]	26 [1]	dc	261	1.89	0.336	0.178	-0.101
Ursus maritimus [42]	3 [1]	dc	61	1.74	0.193	0.111	0.001
Ursus maritimus [42]	2 [1]	dc	44	1.86	0.163	0.088	-0.081
Ursus maritimus [42]	1 [1]	dc	15	2.27	0.329	0.145	0.299
Ursus arctos [43]	17 [1]	dc	46	2.56	0.507	0.197	-0.095
Ursus arctos [43]	17 [1]	dc	51	2.06	0.487	0.236	-0.108
Ursus arctos [43]	16 [1]	dc	91	2.09	0.476	0.228	-0.014
Ursus arctos [44]	43 [1]	dc	56	2.39	0.524	0.219	0.014
Ursus americanus [45]	4 [1]	dc	15	2.53	0.516	0.204	0.073
Ursus americanus [46]	16 [1]	dc	86	2.35	0.599	0.255	0.184
Ursus americanus [47]	4 [1]	dc	12	2.75	0.688	0.250	-0.047
Ursus americanus [48]	4 [1]	dc	23	1.65	0.401	0.243	-0.111
Ursus americanus [49]	4 [1]	dc	50	2.41	0.538	0.223	0.084
Ursus americanus [50]	12 [1]	dc	105	2.49	0.593	0.238	0.065
Ursus americanus [51]	3 [1]	dc	35	2.74	0.477	0.174	0.009
Lutra lutra [52]	M [M]	dc	160	2.45	0.346	0.847	0.124
Lutra lutra [53]	11 [M]	ec	17	2.06	0.526	0.255	-0.021
Lutra lutra [54]	5 [1]	dc	28	1.64	0.515	0.314	0.260
Lutra lutra [55]	50 [7]	ps	30	2.27	0.662	0.292	0.057
Lutra lutra [55]	50 [7]	dc	121	2.39	0.833	0.349	0.082
Lutra lutra [55]	50 [7]	ec	46	2.02	0.673	0.333	0.166
Lontra canadensis [56]	M[M]	ec	22	2.41	0.543	0.257	0.148
Mustela erminea [57]	4 [M]	ec	12	8.58	2.910	0.339	-0.030
Mustela nigripes [58]	4 [1]	dc	68	3.29	0.796	0.242	0.074
Martes pennanti [59]	6 [1]	ec	9	3.33	0.222	0.067	0.053
Martes americana [60]	5 [1]	dc	10	1.40	0.240	0.171	-0.210
Spilogale putorius [61]	1 [1]	dc	12	3.58	1.576	0.440	-0.024
Gulo gulo [62]	19 [M]	dc	28	2.46	0.606	0.246	-0.025
Meles meles [63]	M[M]	ps	37	2.95	0.869	0.295	0.142
Meles meles [63]	M[M]	obs	23	2.36	0.686	0.290	-0.126
Mean ± SD			**67.89 ± 67.953**	**3.52 ± 2.134**	**1.99 ± 3.872**	**0.40 ± 0.394**	**0.074 ± 0.310**

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Appendix 5. Model selection for 12 probability distributions fitted to carnivore litter size frequencies, showing ΔAIC values

Bold indicates the distributions for which ΔAIC ≤ 6. References refer to those in Appendix 4. Distribution abbreviations: SP: Shifted Poisson; ZTP: Zero-truncated Poisson; SB: Shifted binomial; ZTB: Zero-truncated binomial; SNB: Shifted negative binomial; ZTNB: Zero-truncated negative binomial; SGP: Shifted generalised Poisson; ZTGP: Zero-truncated generalised Poisson; DN: Discretised normal; DLN: Discretised lognormal; DSB3; Discretised stretched-beta (3 parameter form); DSB2; Discretised stretched-beta (2 parameter form).

	Distribution											
Species [(reference)]	***SP***	***ZTP***	***SB***	***ZTB***	***SNB***	***ZTNB***	***SGP***	***ZTGP***	***DN***	***DLN***	***DSB3***	***DSB2***
Vulpes velox[1]	**4.79**	6.29	**1.89**	**2.03**	6.40	7.81	7.00	8.57	**0.00**	**0.71**	**1.83**	**0.12**
Vulpes macrotis[2]	13.56	23.17	**1.87**	**0.00**	12.20	21.33	16.51	26.63	**0.10**	17.01	**0.09**	7.64
Vulpes macrotis[3]	18.17	20.42	12.41	12.79	19.62	21.83	20.53	22.86	**0.00**	**0.00**	**2.00**	**0.00**
Vulpes vulpes[4]	29.70	43.24	**0.46**	**0.22**	28.35	41.20	33.17	47.25	**0.00**	15.00	**2.79**	6.71
Vulpes vulpes[5]	**0.51**	6.78	**0.00**	**0.10**	**1.39**	6.17	**2.91**	9.61	**0.68**	8.45	**5.73**	**4.07**
Vulpes vulpes[6]	**0.00**	**2.63**	**1.93**	**1.95**	**1.95**	**3.50**	**2.09**	**5.14**	**4.46**	7.71	**2.16**	**2.00**
Vulpes vulpes[7]	12.86	20.61	**3.39**	**1.12**	12.73	18.70	15.91	24.25	**0.00**	23.27	**1.17**	10.53
Vulpes vulpes[8]	22.19	9.02	14.21	9.14	25.19	11.99	23.39	10.63	**0.00**	25.08	**5.57**	12.61
Vulpes vulpes[8]	**3.69**	8.80	**0.43**	**0.25**	**4.52**	8.97	6.12	11.50	**0.00**	**5.45**	**3.48**	**2.63**
Vulpes vulpes[8]	**5.74**	11.97	**2.41**	**1.05**	6.13	11.51	8.31	14.97	**0.00**	16.85	7.98	8.85
Vulpes vulpes[9]	9.77	23.18	**0.00**	**0.96**	8.65	17.44	12.77	26.78	**0.21**	9.57	6.85	**5.15**
Vulpes vulpes[10]	9.11	14.21	**0.00**	**0.17**	9.81	14.96	11.70	17.01	**0.26**	**2.43**	**1.46**	**0.39**
Vulpes vulpes[11]	31.15	59.49	**2.21**	**1.13**	25.75	43.60	35.43	65.10	**0.00**	35.10	12.17	17.23
Vulpes vulpes[12]	**2.24**	**1.38**	**4.24**	**2.19**	**4.38**	**2.91**	**4.24**	**3.64**	**0.00**	11.84	**3.37**	**5.59**
Vulpes vulpes[13]	51.26	69.99	46.43	71.87	57.63	72.36	52.29	72.19	157.74	**0.00**	8.81	16.22
Urocyon littoralis[14]	6.43	9.66	**2.02**	**3.55**	7.85	11.01	8.67	11.97	**0.00**	**2.21**	**4.01**	**1.22**
Urocyon littoralis[15]	**0.00**	**1.18**	**1.73**	**1.63**	**1.83**	**2.85**	**2.05**	**3.32**	**1.97**	**2.24**	**3.91**	**1.44**
Urocyon cinereoargenteus[16]	**0.18**	**1.35**	**0.61**	**0.95**	**1.98**	**3.15**	**2.26**	**3.46**	**0.67**	**0.00**	**1.99**	**0.07**
Urocyon cinereoargenteus[17]	42.28	57.57	6.47	8.49	36.73	54.91	45.94	61.75	**1.06**	**1.30**	**2.19**	**0.00**
Alopex lagopus[18]	56.96	35.90	**3.31**	**5.81**	59.62	38.24	56.07	35.50	34.28	**3.60**	**1.85**	**0.00**
Alopex lagopus[19]	6.59	8.62	**1.76**	**1.95**	7.98	9.65	8.90	11.01	**1.13**	**0.00**	**2.29**	**0.53**
Canis lupus[20]	**0.00**	**0.91**	**1.25**	**1.32**	**1.79**	**2.64**	**2.10**	**3.08**	**1.54**	**1.50**	**2.59**	**0.88**
Canis lupus[21]	**2.94**	**1.37**	**4.80**	**3.22**	**5.08**	**3.27**	**4.89**	**3.44**	**0.99**	9.90	**0.00**	**4.49**
Lycaon pictus[22]	26.15	17.42	26.83	19.42	29.46	19.66	27.52	19.55	**0.00**	86.84	11.70	36.18
Lycaon pictus[23]	31.52	23.62	**0.35**	**0.00**	16.94	25.82	32.00	24.20	**3.54**	**0.63**	404.40	52.42
Lycaon pictus[23]	**0.00**	**0.00**	**2.00**	**1.77**	**2.03**	**1.82**	**2.02**	**2.14**	**1.20**	**5.11**	**0.27**	**1.52**
Lycaon pictus[23]	13.35	8.03	**4.31**	**2.76**	15.43	10.12	14.72	9.48	**0.00**	7.12	36.79	38.13
Nyctereutes procyonoides[24]	6.83	7.91	**3.30**	**3.09**	8.29	9.08	9.15	10.30	**3.65**	7.65	**0.00**	**4.67**
Procyon lotor[25]	8.02	12.44	**0.90**	**3.28**	8.84	13.44	10.29	14.80	**0.00**	**0.33**	**2.28**	**0.06**
Crocuta crocuta[26]	20.19	25.14	6.93	12.12	20.38	25.99	22.63	27.67	**0.00**	**0.00**	**2.00**	**0.00**
Crocuta crocuta[26]	15.11	20.62	**1.80**	**5.03**	14.55	20.71	17.42	23.02	**0.00**	**0.01**	**2.01**	**0.00**

	Distribution											
Species[(reference)]	**SP**	**ZTP**	**SB**	**ZTB**	**SNB**	**ZTNB**	**SGP**	**ZTGP**	**DN**	**DLN**	**DSB3**	**DSB2**
Crocuta crocuta[27]	**2.34**	**3.96**	**2.51**	**3.01**	**4.07**	**5.66**	**4.46**	6.12	**2.62**	**0.00**	**2.51**	**1.12**
Acinonyx jubatus[28]	10.76	14.70	**3.63**	**4.71**	11.97	15.80	13.18	17.25	**0.26**	**0.59**	**2.29**	**0.00**
Felis concolor[29]	**5.80**	9.26	**1.10**	**2.44**	7.01	10.51	8.05	11.60	**0.00**	**3.16**	**4.87**	**1.77**
Felis concolor[30]	8.71	12.77	**2.50**	**4.82**	9.81	13.72	11.04	15.21	**0.00**	**2.29**	**4.11**	**0.67**
Felis concolor[31]	68.08	108.76	7.17	19.71	61.30	100.73	72.28	113.89	**0.50**	**3.78**	**3.87**	**0.00**
Felis iriomotensis[32]	9.54	12.73	**5.21**	6.63	10.92	14.13	11.78	15.05	**1.33**	**0.00**	**2.22**	**0.59**
Lynx pardinus[33]	**0.00**	**0.10**	**1.87**	**1.17**	**1.94**	**1.94**	**2.03**	**2.19**	**0.36**	**3.24**	**4.35**	**1.63**
Panthera tigris altaica[34]	**0.00**	**0.66**	**1.43**	**1.22**	**1.78**	**2.44**	**2.04**	**2.73**	**1.24**	**1.94**	**3.85**	**1.41**
Panthera onca[35]	**3.00**	6.92	17.26	6.52	7.71	13.47	**5.00**	8.92	**2.34**	**0.00**	**2.54**	**0.44**
Panthera leo[36]	13.25	18.61	**3.49**	18.69	19.19	28.67	15.25	20.61	**0.00**	**1.27**	**2.46**	**0.46**
Panthera leo[37]	12.79	19.25	**4.45**	15.70	18.24	30.54	14.79	21.25	**0.00**	**2.09**	**2.71**	**0.71**
Panthera leo[37]	7.44	16.28	21.04	13.55	15.09	33.57	9.44	18.28	**0.00**	8.59	**5.55**	**4.40**
Panthera leo[38]	9.91	27.17	333.42	12.03	18.61	51.26	11.91	29.17	**3.29**	**2.72**	**2.00**	**0.00**
Panthera leo[38]	**3.14**	14.03	168.02	38.55	10.20	32.22	**5.14**	16.04	**0.00**	**4.15**	**2.07**	**0.07**
Panthera leo[38]	**0.00**	**0.21**	**1.27**	**4.61**	**1.97**	**2.12**	**2.02**	**2.24**	**1.25**	**1.25**	**3.25**	**1.25**
Panthera pardus[39]	**0.00**	**0.13**	**1.60**	**2.21**	**1.89**	**2.03**	**2.01**	**2.14**	**1.61**	**1.61**	**3.60**	**1.60**
Leopardus pardalis[40]	6.20	7.79	**3.93**	**5.58**	7.81	9.46	8.35	9.96	**0.00**	**0.00**	**2.00**	**0.00**
Ursus maritimus[41]	104.86	140.98	30.90	57.46	98.59	134.43	109.25	145.54	**0.00**	**2.22**	**3.93**	**0.43**
Ursus maritimus[42]	38.78	46.84	21.48	29.32	38.85	47.12	41.29	49.45	**0.00**	**0.00**	**2.00**	**0.00**
Ursus maritimus[42]	10.08	13.35	6.87	9.07	11.53	14.81	12.26	15.57	**2.88**	**0.00**	**2.13**	**0.94**
Ursus maritimus[42]	45.17	54.17	23.05	32.42	44.49	54.24	48.00	57.16	**0.00**	**0.00**	**2.00**	**0.00**
Ursus arctos[43]	21.56	29.47	7.02	10.95	21.81	29.64	24.16	32.26	**0.00**	6.37	7.93	**3.69**
Ursus arctos[43]	12.29	18.64	**1.91**	**5.28**	12.84	19.36	14.77	21.29	**0.00**	**2.92**	**4.66**	**0.80**
Ursus arctos[43]	25.46	37.80	**4.78**	11.51	24.78	37.01	28.15	40.73	**0.00**	**2.59**	**4.28**	**1.16**
Ursus arctos[44]	20.42	30.07	6.18	11.24	20.67	30.38	23.02	32.87	**1.37**	**0.26**	**2.09**	**0.00**
Ursus americanus[45]	6.36	9.36	**3.65**	**5.29**	7.76	10.86	8.55	11.61	**1.92**	**0.00**	**2.01**	**0.46**
Ursus americanus[46]	23.92	35.60	**3.74**	8.60	24.16	34.60	26.73	38.70	**0.00**	8.70	9.92	**4.76**
Ursus americanus[47]	**2.50**	**4.36**	**0.83**	**1.48**	**4.07**	**5.91**	**4.66**	6.57	**0.00**	**1.63**	**3.43**	**0.77**
Ursus americanus[48]	**0.86**	**2.41**	**0.01**	**0.55**	**2.36**	**4.03**	**2.95**	**4.54**	**0.00**	**0.23**	**2.20**	**0.05**
Ursus americanus[49]	18.86	28.04	**5.34**	10.04	17.18	27.85	21.44	30.82	**1.03**	**0.45**	**2.25**	**0.00**
Ursus americanus[50]	35.18	51.60	8.25	15.24	34.02	49.15	38.11	54.87	**0.00**	6.64	8.35	**4.89**
Ursus americanus[51]	20.03	26.62	7.87	11.09	20.42	26.98	22.58	29.33	**0.00**	**5.01**	6.64	**2.85**

	Distribution											
Species[(reference)]	**SP**	**ZTP**	**SB**	**ZTB**	**SNB**	**ZTNB**	**SGP**	**ZTGP**	**DN**	**DLN**	**DSB3**	**DSB2**
Lutra lutra[52]	20.88	40.02	**3.58**	7.70	15.13	38.62	23.85	43.47	**5.38**	**0.88**	**2.23**	**0.00**
Lutra lutra[53]	**2.04**	**3.97**	**0.32**	**1.21**	**3.56**	**5.53**	**4.19**	6.17	**0.00**	**1.19**	**3.08**	**0.33**
Lutra lutra[54]	**0.00**	**1.25**	**1.44**	**1.87**	**1.71**	**2.92**	**2.05**	**3.33**	**2.05**	**0.58**	**2.66**	**1.04**
Lutra lutra[55]	**3.66**	7.32	**0.19**	**1.38**	**4.84**	8.67	**5.89**	9.64	**0.00**	**0.80**	**2.61**	**0.14**
Lutra lutra[55]	12.39	25.20	**0.02**	**1.75**	11.83	24.68	15.07	28.22	**0.00**	**3.35**	**4.42**	**1.12**
Lutra lutra[55]	**2.43**	6.44	**0.87**	**1.65**	**3.61**	7.62	**4.63**	8.75	**1.47**	**0.00**	**1.90**	**0.05**
Lontra canadensis[56]	**0.25**	**1.29**	**0.20**	**0.67**	**2.01**	**3.08**	**2.33**	**3.40**	**0.00**	**0.77**	**2.71**	**0.23**
Mustela erminea[57]	**2.28**	**3.24**	**0.16**	**0.20**	**3.90**	**4.81**	**4.51**	**5.52**	18.60	**0.18**	**2.01**	**0.00**
Mustela nigripes[58]	27.40	39.28	**5.59**	9.16	26.56	37.76	30.42	42.64	**0.00**	**4.72**	**5.31**	**1.48**
Martes pennanti[59]	12.02	14.17	8.77	9.76	13.59	15.75	14.26	16.47	**0.01**	**0.00**	**2.00**	**0.00**
Martes americana[60]	**0.00**	**0.47**	**0.34**	**0.74**	**1.78**	**2.33**	**2.04**	**2.52**	**0.13**	**0.13**	**2.13**	**0.13**
Spilogale putorius[61]	**0.08**	**1.04**	**0.45**	**0.02**	**1.84**	**2.69**	**2.20**	**3.23**	**0.00**	**2.90**	**2.98**	**1.19**
Gulo gulo[62]	7.35	11.60	**1.60**	**3.44**	8.41	12.27	9.65	14.01	**0.00**	**2.06**	**3.82**	**0.96**
Meles meles[63]	9.26	14.84	**1.36**	**2.62**	10.05	15.64	11.68	17.43	**0.00**	**4.38**	**3.31**	**2.61**
Meles meles[63]	24.48	40.10	7.65	13.23	23.92	39.50	27.23	43.15	6.56	**0.00**	**2.12**	**0.89**
Frequency of ΔAIC = 0	0.13	0.00	0.04	0.03	0.00	0.00	0.00	0.00	0.47	0.15	0.03	0.15
Frequency of ΔAIC ≤ 6	0.37	0.26	0.73	0.63	0.31	0.26	0.32	0.22	0.95	0.77	0.86	0.87

Appendix 6. Results of the Fisher Exact test goodness-of-fit of probability distributions to empirical carnivore litter size frequencies

Distributions with $p < 0.05$ were classified as not fitting. Bold indicates the distributions for which model selection determined ΔAIC ≤ 6 (Appendix 5). References refer to those in Appendix 4. Distribution abbreviations: SP: Shifted Poisson; ZTP: Zero-truncated Poisson; SB: Shifted binomial; ZTB: Zero-truncated binomial; SNB: Shifted negative binomial; ZTNB: Zero-truncated negative binomial; SGP: Shifted generalised Poisson; ZTGP: Zero-truncated generalised Poisson; DN: Discretised normal; DLN: Discretised lognormal; DSB3; Discretised stretched-beta (3 parameter form); DSB2; Discretised stretched-beta (2 parameter form).

	Distribution											
Species *(reference)*	***SP***	***ZTP***	***SB***	***ZTB***	***SNB***	***ZTNB***	***SGP***	***ZTGP***	***DN***	***DLN***	***DSB3***	***DSB2***
Vulpes velox[1]	**0.846**	0.837	**0.991**	**0.990**	0.875	0.859	0.853	0.824	**0.811**	**0.764**	**0.713**	**0.795**
Vulpes macrotis[2]	0.205	0.025	**0.693**	**0.885**	0.268	0.059	0.162	0.019	**0.914**	0.148	**0.841**	0.409
Vulpes macrotis[3]	0.132	0.093	0.361	0.354	0.145	0.086	0.149	0.100	**1.000**	**1.000**	**1.000**	**1.000**
Vulpes vulpes[4]	0.018	0.001	**0.982**	**0.994**	0.038	0.003	0.009	0.002	**0.996**	0.430	**0.955**	0.729
Vulpes vulpes[5]	**0.238**	0.084	**0.274**	**0.359**	**0.271**	0.123	**0.199**	0.073	**0.253**	0.119	**0.147**	**0.094**
Vulpes vulpes[6]	**0.652**	**0.662**	**0.644**	**0.611**	**0.623**	**0.699**	**0.748**	**0.699**	**0.353**	0.538	**0.637**	**0.634**
Vulpes vulpes[7]	0.246	0.058	**0.704**	**0.819**	0.299	0.146	0.242	0.054	**0.844**	0.104	**0.633**	0.321
Vulpes vulpes[8]	0.004	0.154	0.182	0.344	0.004	0.098	0.007	0.160	**0.777**	0.086	**0.419**	0.260
Vulpes vulpes[8]	**0.538**	0.320	**0.580**	**0.662**	**0.630**	0.431	0.565	0.300	**0.705**	**0.555**	**0.394**	**0.522**
Vulpes vulpes[8]	**0.504**	0.282	**0.712**	**0.810**	0.598	0.391	0.542	0.253	**0.923**	0.164	0.478	0.386
Vulpes vulpes[9]	0.031	0.003	**0.184**	**0.176**	0.066	0.013	0.030	0.005	**0.283**	0.194	0.157	**0.096**
Vulpes vulpes[10]	0.818	0.515	**0.992**	**0.993**	0.838	0.595	0.787	0.527	**0.986**	**0.904**	**0.994**	**0.968**
Vulpes vulpes[11]	0.006	0.000	**0.604**	**0.673**	0.033	0.000	0.005	0.000	**0.704**	0.072	0.371	0.162
Vulpes vulpes[12]	**0.209**	**0.392**	**0.190**	**0.282**	**0.147**	**0.384**	**0.207**	**0.403**	**0.379**	0.124	**0.211**	**0.215**
Vulpes vulpes[13]	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	**0.050**	0.000	0.000
Urocyon littoralis[14]	0.301	0.179	**0.717**	**0.599**	0.352	0.167	0.299	0.157	**0.849**	**0.517**	**0.528**	**0.577**
Urocyon littoralis[15]	**0.742**	**0.739**	**0.733**	**0.672**	**0.766**	**0.752**	**0.727**	**0.736**	**0.600**	**0.729**	**0.758**	**0.749**
Urocyon cinereoargenteus[16]	**0.939**	**0.854**	**0.848**	**0.925**	**0.942**	**0.872**	**0.940**	**0.852**	**0.728**	**0.773**	**0.825**	**0.776**
Urocyon cinereoargenteus[17]	0.001	0.000	0.543	0.448	0.000	0.000	0.000	0.000	**0.713**	**0.802**	**0.792**	**0.719**
Alopex lagopus[18]	0.000	0.001	**0.698**	**0.505**	0.000	0.003	0.000	0.001	0.001	**0.778**	**0.799**	**0.601**
Alopex lagopus[19]	0.721	0.669	**0.630**	**0.558**	0.739	0.694	0.715	0.662	**0.585**	**0.588**	**0.633**	**0.575**
Canis lupus[20]	**0.920**	**0.949**	**0.742**	**0.772**	**0.915**	**0.952**	**0.936**	**0.946**	**0.748**	**0.742**	**0.843**	**0.789**
Canis lupus[21]	**0.059**	0.146	**0.081**	**0.092**	**0.044**	**0.104**	**0.060**	**0.153**	**0.183**	0.086	**0.188**	**0.117**
Lycaon pictus[22]	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.001	**0.001**	0.000	0.000	0.000
Lycaon pictus[23]	0.001	0.002	**0.926**	**0.945**	0.449	0.001	0.001	0.002	**0.521**	**0.958**	0.000	0.000
Lycaon pictus[23]	**0.893**	0.937	**0.868**	**0.895**	**0.847**	**0.904**	**0.882**	**0.960**	**0.936**	**0.836**	**0.977**	**0.899**
Lycaon pictus[23]	0.003	0.007	**0.224**	**0.255**	0.002	0.006	0.002	0.004	**0.201**	0.248	0.000	0.000
Nyctereutes procyonoides[24]	0.725	0.733	**0.363**	**0.398**	0.707	0.735	0.689	0.737	**0.452**	0.314	**0.921**	**0.391**
Procyon lotor[25]	0.137	0.036	**0.836**	**0.429**	0.177	0.048	0.127	0.053	**1.000**	**0.955**	**0.957**	**1.000**
Crocuta crocuta[26]	0.004	0.000	0.465	0.025	0.001	0.002	0.002	0.001	**1.000**	**1.000**	**1.000**	**1.000**
Crocuta crocuta[26]	0.007	0.003	**0.906**	**0.301**	0.015	0.002	0.007	0.000	**1.000**	**1.000**	**1.000**	**1.000**
Crocuta crocuta[27]	**0.577**	0.513	**0.413**	**0.375**	**0.594**	**0.478**	**0.568**	0.518	**0.296**	**0.473**	**0.437**	**0.401**

	Distribution											
Species[(reference)]	**SP**	**ZTP**	**SB**	**ZTB**	**SNB**	**ZTNB**	**SGP**	**ZTGP**	**DN**	**DLN**	**DSB3**	**DSB2**
Acinonyx jubatus[28]	0.387	0.175	**0.809**	**0.759**	0.352	0.199	0.369	0.171	**0.762**	**0.767**	**0.788**	**0.805**
Felis concolor[29]	0.300	0.140	**0.672**	**0.600**	0.367	0.155	0.279	0.128	**0.739**	**0.433**	**0.395**	**0.492**
Felis concolor[30]	0.124	0.061	**0.567**	**0.403**	0.167	0.068	0.151	0.053	**0.828**	**0.528**	**0.517**	**0.668**
Felis concolor[31]	0.000	0.000	0.358	0.035	0.000	0.000	0.000	0.000	**0.843**	**0.722**	**0.868**	**0.935**
Felis iriomotensis[32]	0.210	0.104	**0.384**	0.335	0.205	0.124	0.192	0.097	**0.366**	**0.542**	**0.473**	**0.408**
Lynx pardinus[33]	0.610	0.739	**0.532**	**0.608**	**0.619**	**0.711**	**0.669**	**0.725**	**0.799**	**0.584**	**0.671**	**0.628**
Panthera tigris altaica[34]	0.920	0.885	**0.763**	**0.820**	**0.903**	**0.880**	**0.887**	**0.907**	**0.919**	**0.795**	**0.839**	**0.874**
Panthera onca[35]	0.519	0.255	0.605	0.446	0.508	0.269	**0.535**	0.230	**0.432**	**0.927**	**0.923**	**0.822**
Panthera leo[36]	0.080	0.019	**0.614**	0.392	0.109	0.035	0.110	0.023	**1.000**	**0.792**	**0.785**	**0.866**
Panthera leo[37]	0.091	0.013	**0.615**	0.440	0.092	0.026	0.089	0.013	**0.788**	**0.542**	**0.530**	**0.616**
Panthera leo[37]	0.407	0.074	0.852	0.950	0.508	0.089	0.373	0.056	**0.957**	0.432	**0.505**	**0.690**
Panthera leo[38]	0.177	0.003	0.794	0.641	0.282	0.017	0.156	0.001	**0.520**	**0.750**	**0.713**	**0.841**
Panthera leo[38]	0.371	0.044	0.761	0.634	0.532	0.076	**0.363**	0.041	**0.769**	**0.518**	**0.610**	**0.490**
Panthera leo[38]	0.794	0.799	**1.000**	**0.726**	**1.000**	**0.803**	**0.796**	**0.808**	**1.000**	**1.000**	**1.000**	**1.000**
Panthera pardus[39]	0.814	0.808	**1.000**	**0.821**	**1.000**	**1.000**	**0.800**	**0.816**	**1.000**	**1.000**	**1.000**	**1.000**
Leopardus pardalis[40]	0.169	0.153	**0.351**	**0.152**	0.162	0.123	0.169	0.141	**1.000**	**1.000**	**1.000**	**1.000**
Ursus maritimus[41]	0.000	0.000	0.001	0.000	0.000	0.000	0.000	0.000	**1.000**	**0.948**	**0.948**	**0.988**
Ursus maritimus[42]	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	**1.000**	**1.000**	**1.000**	**1.000**
Ursus maritimus[42]	0.035	0.008	0.034	0.037	0.026	0.011	0.031	0.010	**0.069**	**0.225**	**0.215**	**0.139**
Ursus maritimus[42]	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	**1.000**	**1.000**	**1.000**	**1.000**
Ursus arctos[43]	0.002	0.001	0.137	0.071	0.002	0.001	0.002	0.000	**0.418**	0.061	0.068	**0.134**
Ursus arctos[43]	0.049	0.013	**0.643**	**0.247**	0.058	0.010	0.042	0.006	**0.853**	**0.511**	**0.543**	**0.710**
Ursus arctos[43]	0.001	0.000	**0.411**	0.046	0.002	0.000	0.001	0.000	**0.997**	**0.748**	**0.765**	**0.876**
Ursus arctos[44]	0.009	0.000	0.284	0.103	0.014	0.000	0.006	0.001	**0.619**	**0.975**	**0.983**	**0.952**
Ursus americanus[45]	0.220	0.117	**0.366**	**0.277**	0.230	0.118	0.218	0.105	**0.250**	**0.541**	**0.551**	**0.405**
Ursus americanus[46]	0.007	0.003	**0.906**	**0.301**	0.015	0.002	0.007	0.000	**1.000**	**1.000**	**1.000**	**1.000**
Ursus americanus[47]	0.002	0.000	**0.270**	0.130	0.003	0.000	0.002	0.000	**0.570**	0.066	0.081	**0.157**
Ursus americanus[48]	0.727	**0.568**	**0.946**	**0.908**	0.737	**0.578**	**0.702**	0.550	**0.840**	**0.611**	**0.676**	**0.677**
Ursus americanus[49]	0.645	**0.501**	**1.000**	**0.910**	0.685	**0.493**	**0.639**	**0.506**	**1.000**	**0.912**	**0.907**	**1.000**
Ursus americanus[50]	0.013	0.001	**0.382**	0.146	0.024	0.002	0.011	0.001	**0.717**	**0.961**	**0.981**	**0.965**
Ursus americanus[51]	0.000	0.000	0.001	0.000	0.000	0.000	0.000	0.000	**0.016**	0.000	0.001	**0.001**
Lutra lutra[52]	0.627	**0.456**	**0.905**	**0.843**	0.654	**0.446**	**0.612**	0.444	**0.857**	**0.752**	**0.770**	**0.822**
Lutra lutra[53]	0.425	**0.346**	**0.312**	**0.311**	**0.438**	**0.357**	**0.429**	**0.382**	**0.272**	**0.485**	**0.481**	**0.424**
Lutra lutra[54]	0.609	0.317	**0.993**	**0.887**	**0.653**	0.363	**0.620**	0.285	**0.982**	**0.919**	**0.919**	**0.987**
Lutra lutra[55]	0.120	0.007	**0.962**	**0.771**	0.210	0.008	0.090	0.004	**0.936**	**0.775**	**0.834**	**0.848**
Lutra lutra[55]	0.445	0.213	**0.569**	**0.499**	**0.544**	0.233	**0.445**	0.223	**0.389**	**0.791**	**0.827**	**0.711**

Species[(reference)]	Distribution											
	SP	**ZTP**	**SB**	**ZTB**	**SNB**	**ZTNB**	**SGP**	**ZTGP**	**DN**	**DLN**	**DSB3**	**DSB2**
Lutra lutra[55]	0.748	**0.686**	**0.933**	**0.896**	**0.753**	**0.677**	**0.730**	**0.679**	**0.862**	**0.764**	**0.756**	**0.795**
Lontra canadensis[56]	0.005	0.000	**0.699**	0.411	0.009	0.000	0.005	0.000	**0.969**	**0.542**	**0.692**	**0.800**
Mustela nigripes[58]	0.727	**0.647**	**1.000**	**0.759**	**0.699**	**0.659**	**0.712**	**0.672**	**1.000**	**1.000**	**1.000**	**1.000**
Martes pennanti[59]	**0.674**	**0.678**	**0.473**	**0.521**	**0.660**	**0.713**	**0.652**	**0.690**	**0.601**	**0.460**	**0.567**	**0.468**
Martes americana[60]	0.344	0.145	**0.871**	**0.713**	0.345	0.153	0.311	0.143	**0.966**	**0.752**	**0.734**	**0.820**
Spilogale putorius[61]	0.175	0.071	**0.564**	**0.579**	0.225	0.060	0.163	0.052	**0.665**	**0.322**	**0.403**	**0.391**
Gulo gulo[62]	0.002	0.000	0.142	0.028	0.004	0.000	0.005	0.000	0.207	**0.983**	**0.934**	**0.892**
Meles meles[63]	**0.846**	0.837	**0.991**	**0.990**	0.875	0.859	0.853	0.824	**0.811**	**0.764**	**0.713**	**0.795**
Meles meles[63]	0.205	0.025	**0.693**	**0.885**	0.268	0.059	0.162	0.019	**0.914**	0.148	**0.841**	0.409

Appendix 7. Publications resulting from this study

Chapter 2. Published in a modified format, as: Devenish-Nelson, E. S., Harris, S., Soulsbury, C. D., Richards, S. A., & Stephens, P. A. (2010) Uncertainty in population growth rates: determining confidence intervals from point estimates of parameters. *PLoS ONE,* 5(10), e13628.

Chapter 3. Published in a modified format, as: Devenish-Nelson, E. S., Stephens, P. A., Harris, S., Soulsbury, C., Richards, S. A. (2013) Does litter size variation affect models of terrestrial carnivore extinction risk and management? *PLoS ONE* 8(2): e58060.

Chapter 4. In press in a modified format, as: Devenish-Nelson, E. S., Harris, S., Soulsbury, C. D., Richards, S. A., & Stephens, P. A. (2012) Demography of a carnivore, the red fox, *Vulpes vulpes*: what have we learnt from 70 years of published studies? *Oikos.* DOI: 10.1111/j.1600-0706.2012.20706.x