

# The role of population structure and size in determining bat pathogen richness

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*I, Tim C. D. Lucas, confirm that the work presented in this thesis is my own.  
Where information has been derived from other sources, I confirm that this has  
been indicated in the thesis.*

# Abstract

*The huge diversity of pathogens strongly affects human health and ecological systems. I examine the role of population structure and size in maintaining this diversity. Bats are important reservoirs for zoonotic viruses such as Ebola, SARS, Hendra and Nipah and so are used as a case study throughout. Firstly I test whether population structure is associated with high viral richness across wild bat species. I find evidence that more structured bat populations have more virus species. As this type of study cannot distinguish between specific mechanisms, I formulate epidemiological models to test whether structured populations may allow invading pathogens to avoid competition. These models show that population structure does not affect the rate of pathogen invasion by this mechanism. Rather, in these models only the disease dynamics within the local group matter. As both global population structure and local group size appear to be important for disease invasion, I use the same modelling framework to compare the importance of group size and number of groups. I find that group size has a stronger affect than number of groups. There are very few population size estimates for bats to directly test the importance of population size on pathogen richness. Therefore I develop a method for estimating bat population sizes from acoustic surveys. Overall I show that the structure and size of populations can affect their ability to maintain many pathogen species and provide a method to measure population sizes of bats. These findings increase our understanding of the ecological process of pathogen community construction and can help optimise surveillance for zoonotic pathogens.*

# Acknowledgements

Acknowledge all the things!

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# 1 Introduction

## 1.1 Pathogen richness and the impacts of zoonotic diseases

60% of newly emerged diseases are zoonotic (acquired from animals) with wild animals being the predominant source (Jones et al. 2008, Taylor et al. 2001, Woolhouse & Gowtage-Sequeria 2006). Zoonotic diseases can be extremely virulent with viruses such as Nipah and Ebola having case fatality rates over 50% (Lefebvre et al. 2014, Luby et al. 2009). Furthermore these pathogens can have large economic costs (e.g., SARS is estimated to have cost \$40 billion (Knobler et al. 2004)). In particular these impacts can have huge effects on developing economies. For example, the 2014 Ebola epidemic caused both Liberia and Guinea to fall from positive to negative per capita growth rates (World Bank 2014, World Bank 2015) while death rates per 1,000 people living with AIDS are up to ten times higher in developing countries than in Europe and North America (Granich et al. 2015).

Surveillance of zoonotic diseases is crucial to the health impacts of these diseases. In particular it is important to categorise and describe diseases before they spill over into humans (SARS was not identified until months into the pandemic for example, Drosten et al. (2003)) It is also important to improve our ability to predict when outbreaks will occur. For example, if it is known that there is *i*) a disease prevalence in a given host species than normal, or *ii*) a greater-than-usual abundance of a species that is a known reservoir of a high risk zoonotic disease, or *iii*) increased contacts between humans and a pathogen reservoir, preparations can be made for a potential outbreak in that area.

However, funds for zoonotic disease surveillance are limited and so efforts must be optimised. Knowing which species are likely to have many pathogens allows us to sample and identify potentially zoonotic viruses efficiently. Suggested factors that might control pathogen richness include individual, environmental and population level traits. Individual traits that have been studied include body mass and longevity. Increased body mass is expected to increase pathogen diversity as large bodies provide more resource for pathogens to consume and potentially more niches for them to occupy (Arneberg 2002, Kamiya et al. 2014, Poulin 1995). Increased longevity is also expected to increase pathogen richness by increasing the number of pathogens a host encounters in its lifetime (Ezenwa et al. 2006, Nunn et al. 2003). Environmental factors may also play a role. Latitude has been studied as a proxy for environmental factors (Kamiya

et al. 2014, Poulin 2010). It is predicted that warmer climates promote species richness via metabolic mechanisms or by increasing the rate of evolution (Brown et al. 2004, Dunn et al. 2010, Rohde 1992). Furthermore, population level traits that affect the dynamics of disease spread have also been studied. Animal density (Arneberg 2002, Kamiya et al. 2014, Nunn et al. 2003), sociality (Altizer et al. 2003, Bordes et al. 2007, Ezenwa et al. 2006, Vitone et al. 2004) and population structure (Gay et al. 2014, Maganga et al. 2014, Nunes et al. 2006, Turmelle & Olival 2009) have both been predicted to increase pathogen richness by increasing the rate of spread of new pathogens. Finally, species with larger range sizes are expected to have higher pathogen richness as they experience a wider range of environments and have more sympatric host species (Kamiya et al. 2014, Nunn et al. 2003)). These relationships provide a basis for predicting which species will have high pathogen richness and should be prioritised for sampling and surveillance. However, without a better mechanistic understanding of how pathogen richness is created and maintained it is difficult to predict how pathogen richness, and therefore zoonotic disease risk, will respond to global change.

The global richness of pathogens is large but mostly unknown (Poulin 2014). Recent large studies have found tens (Anthony et al. 2013) or even hundreds (Anthony et al. 2015) of virus species in a single host species. This suggests that the global number of mammalian virus species is of the order of hundreds of thousands (Anthony et al. 2013) while only 3,000 virus species, across all host groups, are currently described (King et al. 2011). Recent large databases include nearly 2,000 pathogens from approximately 400 wild animal hosts (Wardeh et al. 2015). Given that there are over 5,000 named mammal species (Wilson & Reeder 2005), the undiscovered diversity of pathogens is likely huge.

Competition between pathogens can occur by different mechanisms: immunological mechanisms such as cross-immunity or shared immune response (Fenton & Perkins 2010) and ecological mechanisms such as removal of susceptible hosts by death (Rohani et al. 2003) or competition for internal host resources (Griffiths et al. 2014). Like ecological systems this competition leads us to expect competitive exclusions so the diversity of parasites needs an explanation (Ackleh & Allen 2003, Ackleh & Salceanu 2014, Bremermann & Thieme 1989, Martcheva & Li 2013, Turner & Garnett 2002).

## 1.2 Influence of population structure and size on pathogen richness

The role of population size and density in disease dynamics is well established (Anderson & May 1979, Heesterbeek 2002, Lloyd-Smith et al. 2005, May & Anderson 1979). Broadly, larger populations can maintain diseases more easily, due to having a larger pool of susceptible individuals (individuals without acquired immunity) and having a greater number of new susceptible individuals enter the population by birth or immigration (Anderson & May 1979, May & Anderson 1979). High density populations are expected to have a greater number of contacts between individuals and so promote disease spread (though there is much discussion on when the number of contacts might scale independently of density (McCallum et al. 2001)).

There is a large literature on the role of population structure on disease dynamics (see review by Pastor-Satorras et al. (2015)) driven by applications to computer viruses (Pastor-Satorras & Vespignani 2001) and the social spread of information (Goffman & Newill 1964), as well as health applications. In particular, work has concentrated on how structure affects the invasion threshold,  $R_0$  (Barthélemy et al. 2010, Colizza & Vespignani 2007, May & Lloyd 2001, Pastor-Satorras & Vespignani 2001, Wu et al. 2013). This value combines relevant parameters to yield a threshold above which a disease is expected to infect a significant proportion of the population (Anderson & May 1979, May & Anderson 1979). Below the threshold, only small outbreaks that quickly die out are expected.

However, the majority of theoretical work considers single pathogens with models examining whether a pathogen can spread and persist in a population, ignoring all other pathogens. A number of studies examine competing epidemics — when two pathogens spread at the same time, which pathogen infects more individuals? — and have found increasing population structure reduces dominance of one strain (Poletto et al. 2013, Poletto et al. 2015, van de Bovenkamp et al. 2014). However, this again tells us little about how pathogen communities form and what factors control total pathogen richness. Far fewer papers explicitly study the longer term competition between two or more pathogens. Those that do commonly find that competitive exclusion is likely (Ackleh & Allen 2003, Ackleh & Salceanu 2014, Bremermann & Thieme 1989, Castillo-Chavez et al. 1995, Martcheva & Li 2013, Turner & Garnett 2002). Mechanisms that have been shown to allow pathogen coexistence include superinfection (Li et al. 2010, May & Nowak

1994), density-dependent deaths (Ackleh & Allen 2003, Kirupaharan & Allen 2004) and differing transmission routes (Allen et al. 2003).

The specific role of density on the ability of pathogens to coexist has not been theoretically studied though it is commonly found to promote pathogen richness in comparative empirical studies (Arneberg 2002, Kamiya et al. 2014, Nunn et al. 2003). The few papers that have directly studied how coexistence of pathogens responds to population structure have found that population structure can allow pathogens to coexist when competitive exclusion would occur in a fully mixed population (Allen et al. 2004, Nunes et al. 2006, Qiu et al. 2013). Furthermore, genetic diversity has been shown to be maximised at intermediate levels of population structure (Campos & Gordo 2006). The roles of population structure and social group size have been examined in comparative studies (Altizer et al. 2003, Bordes et al. 2007, Ezenwa et al. 2006, Gay et al. 2014, Maganga et al. 2014, Rifkin et al. 2012, Turmelle & Olival 2009, Vitone et al. 2004). There is much disagreement between these studies with population structure being shown to promote (Maganga et al. 2014, Turmelle & Olival 2009) and inhibit pathogen richness (Gay et al. 2014) and similarly group size being shown to promote (Bordes et al. 2007, Rifkin et al. 2012) and inhibit (Ezenwa et al. 2006) richness. While increased group size should generally decrease population structure the literature is rarely clear on how these variables relate.

### 1.3 Bats as reservoirs of zoonotic diseases

In recent decades bats have been implicated in a number of high profile zoonotic outbreaks including Nipah (Field et al. 2001, Halpin et al. 2011), Ebola (Leroy et al. 2005), SARS (Li et al. 2005) and Hendra (Field et al. 2001). This has lead to much research on whether bats are a particular source of zoonotic disease (Luis et al. 2013, Olival et al. 2015, Wang et al. 2011) and examinations of factors, such as flight, social living and longevity, that might predispose them to being reservoirs of zoonotic viruses (Calisher et al. 2006, Dobson 2005, Kuzmin et al. 2011, O'Shea et al. 2014, Racey 2015). Given that bats are the second largest order of mammals (Wilson & Reeder 2005), we may expect them to be the source of many viruses simply through weight of numbers (Luis et al. 2013). The broad conclusions are that while bat do host more zoonotic viruses than other groups (Luis et al. 2013) they do not host more virus species per host species (Olival et al. 2015).

Many factors of bat populations make them epidemiologically interesting.



They have highly varied and sometimes complex social structures (Kerth 2008). While some species are largely solitary or live in very small groups (e.g., *Lasiurus borealis* (Shump & Shump 1982)) some species live in colonies of millions of individuals (e.g., *Pteropus scapulatus* (Birt et al. 2008)). These groups can be very stable (Kerth et al. 2011, McCracken & Bradbury 1981). Further complexity arises due to their propensity for seasonal migration (Cryan et al. 2014, Fleming et al. 2003, Richter & Cumming 2008) and seasonally changing social organisation including maternity roosts, hibernation roosts and swarming sites (Kerth 2008). Finally, their ability to fly means populations can be well mixed across large distances (Peel et al. 2013, Petit & Mayer 1999) but this is highly variable with some species having limited dispersal (Wilmer et al. 1994).

However, the population density of many bat species, particularly tree roosting species, is unknown (Clement & Castleberry 2013). As they are small, nocturnal and difficult to identify in flight, estimating their density is incredibly difficult without disruptive and time-consuming roost surveys (Humphrey 1971, Kloepper et al. 2016, Sabol & Hudson 1995). Furthermore, bat densities are generally estimated by counting bats in roosts and dividing this number by area which assumes all roosts have been surveyed (Moreno-Valdez et al. 2004, Speakman et al. 1991, Zahn et al. 2006). As density is tightly linked to pathogen richness (Kamiya et al. 2014) and central to epidemiological models (Anderson & May 1979, May & Anderson 1979) this leaves large gaps in our understanding of disease processes in this group.

## 1.4 Thesis overview

In this thesis I examined the role of population structure and density on pathogen richness. I used bats as a case study throughout due to their interesting social structure and importance as zoonotic reservoirs. I combined empirical, comparative studies with simulation models. This allowed me to study specific mechanisms while linking my theoretical insights to real-world, empirical tests of hypotheses.

First, in Chapter 2, I empirically test the hypothesis that population structure is associated with pathogen richness in wild bat populations. I used two measures of population structure — the number of subspecies and gene flow — and a larger data set than previous studies to ensure robust results and used viral richness as a proxy for overall pathogen richness. For both measures I found that

bat species with more structured populations have more known viruses. This relationship is robust to controlling for study bias and phylogenetic nonindependence. I also tested for relationships between body mass and pathogen richness, and range size and pathogen richness, finding strong support for larger bodied bats carrying more viruses and mixed support for range size promoting pathogen richness.

In Chapter 3, I examined one specific mechanism by which population structure may promote increased pathogen richness. I tested whether increased population structure can allow newly evolved pathogen strains to invade and persist more easily. I modelled bat populations as individual-based, stochastic metapopulations and examined the competition dynamics of two identical pathogen strains. I tested two factors related to population structure: dispersal rate and the number of links between subpopulations. I found that neither of these factors significantly affected the probability of newly evolved pathogens invading and persisting in the population.

Next, I examined the relationships between a number of elements of population structure (Chapter 4). I clarified the interdependence between range size, population size and density. I also noted that population size can be decomposed into colony size and the number of colonies. Using the same model as in Chapter 3, I then tested which of these factors are most important in promoting pathogen richness. Specifically I tested which factor most strongly promotes the invasion and establishment of newly evolved pathogens. I found that population size is more important than population density and that colony size is the important component of population size.

Given the importance of population size on pathogen richness it is important to have good population estimates for wild bat populations. However, there are currently very few measurements of bat population size due to their small size, nocturnal habit and difficulties in identification. Therefore I aimed to develop a method for estimating bat population size from acoustic data, specifically data collected by the iBats project (Jones et al. 2011). In Chapter 5 I present a generally applicable method — based on random encounter models (Rowcliffe et al. 2008, Yapp 1956) — for estimating population sizes of animal populations using camera traps or acoustic detectors. I used spatial simulations to test the method for biases and to assess its precision. I found that the method is unbiased and precise as long as a reasonable amount of data is collected.

Finally, in Chapter 6, I discuss broader conclusions, applications and implic-

ations of my results. I also discuss potential future directions for research.

## 2 The role of population structure in determining pathogen richness in wild bat populations

This work was conducted in collaboration with Kate Jones and Hilde Wilkinson-Herbots.

## 2.1 Abstract

Zoonotic diseases make up a majority of human infectious diseases and are a major drain on healthcare resources and economies. Species that host many pathogen species are more likely to be the source of a novel zoonotic disease. Unfortunately, the factors that influence pathogen richness in animal species are still poorly understood. The pattern of contacts between individuals (i.e. population structure) can be influenced by habitat fragmentation, sociality and dispersal behaviour. Epidemiological theory suggests that population structure can promote pathogen richness by reducing competition. Conversely, it is often assumed that as population structure slows the spread of a new pathogen less structured populations should have greater pathogen richness. Previous studies have had contradictory results and different measures of population structure have been used complicating the interpretation. Here I use comparative data across 203 bat species using phylogenetic linear models, controlling for body mass, range size and study effort, to test whether population structure correlates with pathogen richness. I use bats as a case study as they have been associated with a number of important, recent zoonotic outbreaks. Unlike previous studies I used two measures of population structure: a novel measure, number of subspecies, and a more careful application of genetic measures which have been used previously. Both of these measures are positively associated with pathogen richness. My results add more robust support to the hypothesis that population structure promotes pathogen richness in bats and lends clarity to the contradictory results previously published. The results support predictions from theory. They contradict the assumption commonly made in the ecological literature that factors that increase  $R_0$  should increase pathogen richness. This implies that competitive processes amongst pathogens are stronger than previously thought. Although my analysis implies that population structure does promote pathogen richness in bats, the weakness of the relationship and the difficulty in obtaining some measurements means this is probably not a useful, predictive factor on its own for optimising zoonotic surveillance. However, the relationship has implications for global change, implying that increased habitat fragmentation might promote greater viral richness in bats.

## 2.2 Introduction

Zoonotic pathogens make up the majority of newly emerging diseases and have profound consequences for public health, economics and international development (Jones et al. 2008, Smith et al. 2014, World Bank 2014). A better understanding of the factors that control which wild host species are potential reservoirs of zoonotic diseases would allow us to effectively optimise zoonotic disease surveillance and anticipate how the risks of disease spillover might change with global change. The chance that a host species will be the source of an outbreak depends on a number of factors including its proximity and interactions with humans and the prevalence and diversity of pathogens it carries (Wolfe et al. 2000). However, our understanding of the factors that control the diversity of pathogens in a wild species is still poor.

Models of single pathogens have shown that population structure can allow persistence of a pathogen where a well-mixed population would experience a single, large epidemic followed by pathogen extinction (Blackwood et al. 2013, Plowright et al. 2011, Pons-Salort et al. 2014). Single pathogen models also show that increasing population structure slows disease spread and makes establishment of a new pathogen less likely (Colizza & Vespignani 2007, Vespignani 2008). In the ecological literature this is often taken as a prediction that increased population structure will decrease pathogen richness (Altizer et al. 2003, Morand 2000, Nunn et al. 2003, Poulin 2014, Poulin & Morand 2000) but this interpretation ignores inter-pathogen competition. Models of competition between multiple pathogens show that in unstructured populations a competitive exclusion process occurs but that adding population structure allows coexistence (Allen et al. 2004, Nunes et al. 2006, Qiu et al. 2013).

Three studies have used comparative data to test for an association between population structure and viral richness. A study on 15 African bat species found a positive relationship between the extent of distribution fragmentation and viral richness (Maganga et al. 2014). Conversely, a study on 20 South-East Asian bat species found the opposite relationship (Gay et al. 2014). These studies used the ratio between the perimeter and area of the species' geographic range as their measure of population structure. Range maps are very coarse for many species. Furthermore there is a potential bias with island living species being given sea based edges where continental species might be assumed to live across their entire range, without considering the different terrestrial habitats in these areas.

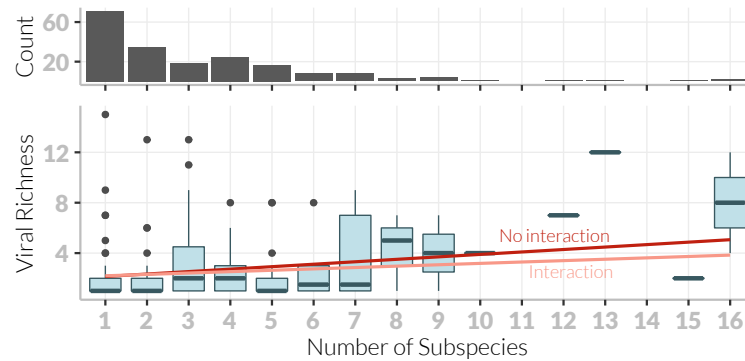
A global study on 33 bat species found a positive relationship between  $F_{ST}$

— a measure of genetic structure — and viral richness (Turmelle & Olival 2009). However, this study included measures using mtDNA which only measures female dispersal which may have biased the results as many bat species show female philopatry (Hulva et al. 2010, Kerth et al. 2002). Furthermore, this study used measures of  $F_{ST}$  irrespective of the study scale including studies covering from tens (McCracken & Bradbury 1981) to thousands (Petit & Mayer 1999) of kilometres. As isolation by distance has been shown in a number of bat species (Burland et al. 1999, Hulva et al. 2010, O'Donnell et al. 2015, Vonhof et al. 2015) this could bias results further. Finally, when a global  $F_{ST}$  value is not given they used the mean of all pairwise  $F_{ST}$  values between sites. This is not correct as pairwise and global  $F_{ST}$  values have different relationships with effective migration rates.

There is a lack of studies using multiple measures of population structure and larger data sets to robustly estimate the importance of population structure. Here I have used two measures of population structure — the number of subspecies and effective gene flow — to robustly test for an association between population structure and pathogen richness in bats. Furthermore, I have used a data set that is much larger than previous studies for one of these analyses, further promoting robustness of results. I found that both measures of population structure are positively associated with viral richness and are included as explanatory variables in the best models for describing viral richness. Furthermore, I found that the role of phylogeny is very weak in the models and in the distribution of viral richness amongst taxa.

## 2.3 Methods

To test for an association between pathogen richness and population structure I have performed multiple regression using a model selection framework to establish whether or not two measures of pathogen richness are likely to be in a 'best model' and therefore important. As species cannot be considered independent due to shared evolutionary history, phylogeny was controlled for in all regressions. A number of other factors that have previously been found to be important were included as additional independent variables: body mass (Gay et al. 2014, Han et al. 2015, Kamiya et al. 2014, Maganga et al. 2014, Turmelle & Olival 2009), range size (Kamiya et al. 2014, Maganga et al. 2014, Turmelle & Olival 2009) and study effort (Gay et al. 2014, Maganga et al. 2014, Turmelle & Olival



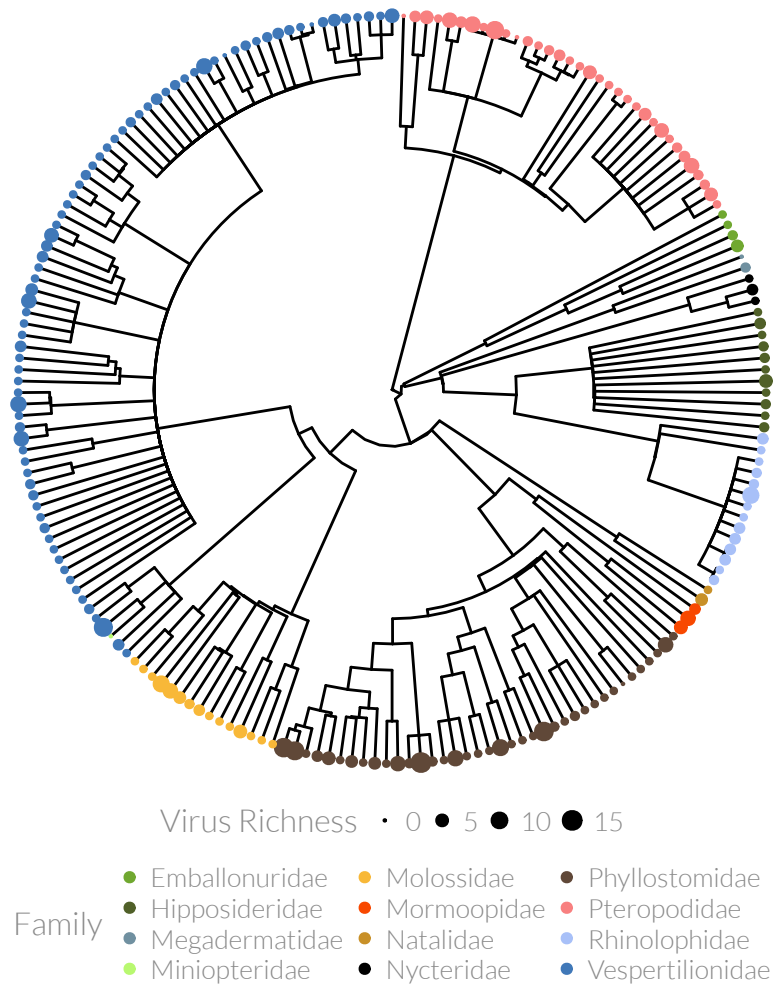
**Figure 2.1** Number of virus species against number of subspecies. The top panel shows the histogram of the data with most species having few subspecies. Data within the same number of subspecies are plotted as boxplots with the dark bar showing the median, the box showing the interquartile range, vertical lines showing the range and outliers shown as separate points. Regression lines are from phylogenetic multiple regressions with all other independent variables set at their median value. The models shown are those with (pink) and without (red) an interaction between study effort and number of subspecies.

2009). This was to attempt to avoid spurious positive results occurring simply due to correlation between pathogen richness and a different, causal factor. Despite commonly being associated with pathogen richness (Arneberg 2002, Kamiya et al. 2014, Nunn et al. 2003), population density is not included in the analysis as there is very little data for bat densities — however Chapter 4 examines the relationship between density and population structure and Chapter 5 presents a method that allows the estimation of density from acoustic surveys. I used both the number of subspecies a bat species has and estimates of gene flow (analysed separately) as measures of population structure. All analyses were run in R (R Development Core Team 2010)

### 2.3.1 Pathogen richness

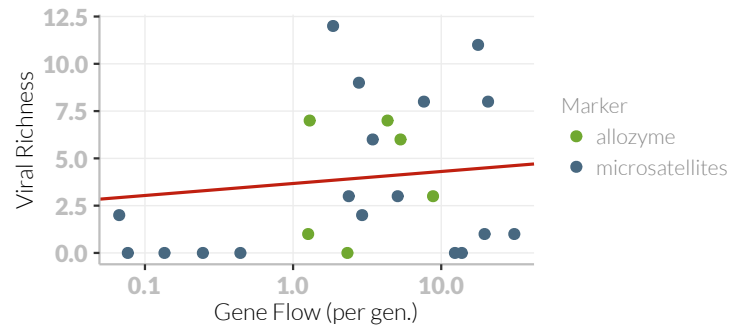
To measure pathogen richness I used data from (Luis et al. 2013). These simply include known infections of a bat species with a pathogen species. Rows with host species that were not identified to species level were removed. Many viruses were not identified to species level or their specified species names were not in the ICTV virus taxonomy (King et al. 2011). I counted a virus if it was the only virus, for that host species, in the lowest taxonomic level identified (present in





**Figure 2.2** Phylogeny from (Bininda-Emonds et al. 2007) pruned to include all species used in either the number of subspecies or gene flow analysis. Dot size shows the number of known viruses for that species and colour shows family.

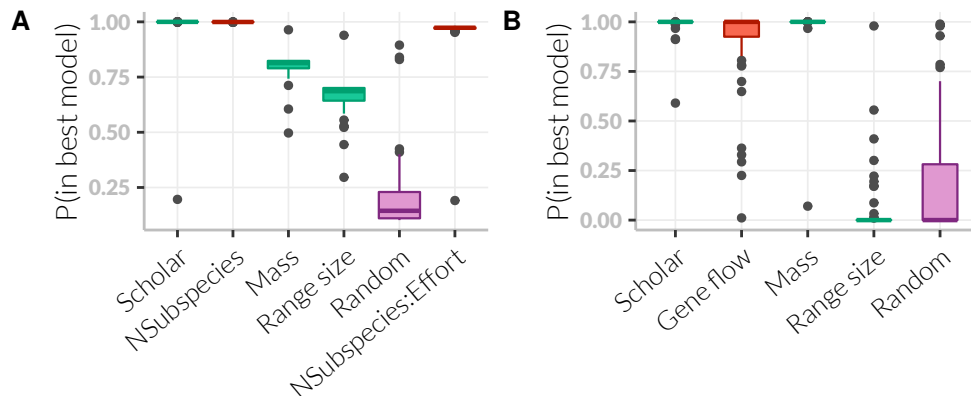
the ICTV taxonomy). That is, if a host is recorded as harbouring an unknown Paramyxoviridae virus, then it must carry at least one Paramyxoviridae virus. If a host carries an unknown Paramyxoviridae virus and a known Paramyxoviridae virus, then it is hard to confirm that the unknown virus is not another record of the known virus. In this case, this would be counted as one virus species.



**Figure 2.3** Gene flow per generation (on a log scale) against viral richness. The genetic marker used is shown with colour. The red line shows the univariate phylogenetic model.

### 2.3.2 Population structure data

I used two measures of population structure: gene flow and the number of subspecies. The number of subspecies was counted using the Wilson and Reeder taxonomy (Wilson & Reeder 2005). Gene flow was calculated from estimates of  $F_{ST}$  collated from the literature. The studies were from a wide range of spatial scales, from local ( $\sim 10$  km) to continental. As  $F_{ST}$  often increases with spatial scale (Burland et al. 1999, Hulva et al. 2010, O'Donnell et al. 2015, Vonhof et al. 2015) I controlled for this by only using data from studies where a large proportion of the species range was studied. I used the ratio of the furthest distance between  $F_{ST}$  samples (measured with <http://www.distancefromto.net/> if not stated) to the width of the IUCN species range (IUCN 2010) and only used studies if this ratio was greater than 0.2. This is an arbitrary value that was a compromise between retaining a reasonable number of data points and controlling for the bias in spatial scale. I converted all  $F_{ST}$  value to migration using  $M = (1 - F_{ST})/8F_{ST}$ . This removes the (0,1) bounds of  $F_{ST}$  and is more easily interpretable (though the final results are unaffected). These two measures of population structure were analysed separately as the number of subspecies has 196 data points while there is only  $F_{ST}$  data for 22 bat species. For the subspecies analysis all bat species in Luis et al. (2013) were used (i.e. all species with at least one known virus species). However, for the gene flow analysis, all bat species with suitable  $F_{ST}$  estimates were used. As this included some species not present in Luis et al. (2013) this includes some bat species with zero known virus species.



**Figure 2.4** Akaike variable weights for both analyses. The probability that each variable will be in the best model if the data were recollected is shown for each of the bootstrap analyses. The purple “Random” box is a uniform random variable used as a null. Population structure (Number of subspecies and Gene flow), shown in red, is likely to be in the best model in both analyses.

### 2.3.3 Other independent variables

To control for study bias I collected the number of PubMed and Google Scholar citations for each bat species name including synonyms from ITIS (ITIS 2015) via the taxize package (Chamberlain & Szöcs 2013). The counts were scraped using the rvest package (Wickham 2015). I log transformed these variables as they were strongly right skewed. The log number of citations on PubMed and Google scholar were highly correlated (pgls:  $t = 19.32$ ,  $df = 194$ ,  $p < 10^{-5}$ ). As this correlation is strong, only results for analyses using only Google Scholar citations are presented.

Measures of body mass were taken from Pantheria (Jones et al. 2009) and primary literature (Aldridge 1987, Arita 1993, Canals et al. 2005, Heaney et al. 2012, Henderson & Broders 2008, Lim & Engstrom 2001, López-Baucells et al. 2014, Ma et al. 2003, Oleksy et al. 2015, Orr & Zuk 2013, Owen et al. 2003, Zhang et al. 2009). *Pipistrellus pygmaeus* was assigned the same mass as *P. pipistrellus* as they are indistinguishable by mass. Body mass measurements were log transformed as they were strongly right skewed. Distribution size was estimated by downloading range maps for all species from IUCN (IUCN 2010) and were also logged due to right skew.

### 2.3.4 Phylogenetic nonindependence

To control for phylogenetic nonindependence I used the best-supported phylogeny from Fritz et al. (2009) (shown in Figure 2.2) which is the supertree from (Bininda-Emonds et al. 2007) with names updated to match the Wilson & Reeder taxonomy (Wilson & Reeder 2005). Phylogenetic manipulation was performed using the ape package (Paradis et al. 2004). The importance of the phylogeny on each variable separately (the  $\lambda$  parameter of the variable regressed against an intercept) was estimated and tested against the null of  $\lambda = 0$  with log-likelihood ratio tests using caper (Orme et al. 2012). I also performed the analysis using the tree from (Jones et al. 2005) as this has some broad changes with families in different places. However the phylogeny did not affect the analysis.

### 2.3.5 Statistical analysis

Statistical analysis for both dependent variables was conducted using an information theoretical/model averaging approach (Burnham & Anderson 2002), specifically following Whittingham et al. (2006) and Whittingham et al. (2005). I chose a credible set of models including all combinations of independent variables and a model with just an intercept. In the analysis using the number of subspecies dependent variable I also included an interaction term between study effort and number of subspecies. This interaction was included as I believed *a priori* that this interaction may be present as subspecies in well studied species are more likely to be identified. The interaction was only included in models with both study effort and number of subspecies as individual terms.

I fitted phylogenetic regressions of all models using nlme (Pinheiro et al. 2015). The independent variables were centered and scaled to allow direct comparison of the coefficients (Schiele 2010). In each case I simultaneously fitted the  $\lambda$  parameter as this avoids misspecifying the model (Revell 2010).  $\kappa$  and  $\delta$  parameters were constrained to one as they are more concerned with when along a branch evolution occurs and because fitting multiple parameters makes interpretation difficult.

To establish the importance of variables I calculated the probability,  $Pr$ , that each variable would be in the best model if the data were recollected. For each variable the mean of its coefficient,  $b$ , in all models that contained that variable was also calculated to determine the direction and strength of the variables. In the subspecies analysis, this mean of  $b$  was calculated for: a) all models b) only

models with a interaction term and c) only models without an interaction. As the interaction term greatly affects the estimated value of  $b$ , considering these value separately aids interpretation. Following (Whittingham et al. 2005) I included a uniformly random variable as a null variable as even unimportant variables can have Akaike weights notably greater than zero. The whole analysis was run 50 times, resampling the random variable each time. I calculated AICc for each model. I calculated the average AICc,  $\bar{AICc}$ , by averaging AICc scores within models.  $\Delta AICc$  was calculated as  $\min(\bar{AICc}) - \bar{AICc}$ , not the mean of the individual  $\Delta AICc$  scores, to guarantee that the best model has  $\Delta AICc = 0$ . From these  $\Delta AICc$  values I calculated Akaike weights,  $w$ . This value can be interpreted as the probability that a model would be the best model if the data were recollected. For each variable, the sum of the Akaike weights of models containing that variable are summed to give  $Pr$ . This value can be interpreted as the probability that the given variable is in the best model.

## 2.4 Results

### 2.4.1 Number of Subspecies

After data cleaning there was data for 196 bat species in 11 families. There appears to be a positive relationship between the number of subspecies and viral richness (Figure 2.1) though few species have more than four subspecies. The number of described virus species for a bat host ranged up to 15 viruses in *Carollia perspicillata*. Figure 2.2 shows the phylogeny used and the number of viruses for each species. The mean number of viruses across families is fairly constant with a lower range of 1.67 for Nycteridae. The highest mean is Mormoopidae with 5 virus species per bat species, but this is based on a sample size of 3. The Phyllostomidae have the second highest mean of 3.49 ( $n = 37$ ).

The small change in mean pathogen richness across families and the lack of clear pattern in Figure 2.2 implies that viral richness is not strongly phylogenetic. This is corroborated by the small estimated size of  $\lambda$  ( $\lambda = 0.04$ ,  $p = 0.21$ ). This fact implies that other factors must control pathogen richness. It also implies that pathogens are not directly inherited down the phylogeny, although this is to be expected by the fast evolution of viruses.

Of the explanatory variables, the number of subspecies has no phylogenetic autocorrelation ( $\lambda = 10^{-6}$ ,  $p > 0.999$ ), study effort and distribution size have weak

but significant autocorrelation (Study Effort:  $\lambda = 0.1, p = 9.12 \times 10^{-3}$ , Distribution size:  $\lambda = 0.46, p < 10^{-5}$ ) and mass is strongly phylogenetic ( $\lambda = 0.93, p < 10^{-5}$ ). Across all models the mean value of  $\lambda$  is 0.08 implying the residuals from the model are weakly phylogenetic. A small number of models (0.4%) had negatively phylogenetically distributed residuals.

The top seven models all had  $\Delta\text{AICc} < 4$  meaning there was no clear best model (Table 2.2). These top seven models had a combined weight of 0.96 meaning that there was a 96% chance that one of these models would be the best model if the data was recollected. However these top seven models all contained study effort, number of subspecies and the interaction between these two variables.  $\log(\text{Mass})$  and  $\log(\text{Range Size})$  and the random variable are all in three of the top seven models.

Summing the Akaike weights of all models that contain a given variable gives a probability,  $Pr$ , that the variable would be in the best model (Figure 2.4A) if the data were recollected (Whittingham et al. 2006). The number of subspecies is very likely in the best model ( $Pr > 0.99$ ) as is the interaction between number of species and study effort ( $Pr = 0.96$ ) compared to the benchmark random variable which has  $Pr = 0.21$  (see Figure 2.4A and Table 2.1). When models with the interaction term are removed, on average (mean weighted by Akaike weights) there is a positive relationship between the number of subspecies and viral richness ( $b = 0.63$ , variance = 0.02). Models with an interaction between number of subspecies and study effort have a positive interaction term ( $b = 0.5$ , variance =  $5.11 \times 10^{-5}$ ) and linear term ( $b = 0.31$ , variance =  $2.13 \times 10^{-4}$ ). This supports the hypothesis that population structure promotes pathogen richness. The strong support for a positive interaction term implies that population structure has a stronger relationship with known pathogen richness in the presence of study effort. One interpretation of this is that population structure alone does not predict high known richness; reasonable study effort is also needed to turn the expected high richness into known and recorded viral richness. Another interpretation is that having few subspecies does not predict low viral richness unless the species has been adequately studied as otherwise the low number of subspecies is probably due to a lack of study rather than an accurate measurement.

As seen in Figure 2.4A, study effort is very likely in the best model ( $b = 0.99$ ,  $Pr > 0.98$ ). Body mass and range size are also probably in the best model ( $b = 0.48$ ,  $Pr = 0.8$  and  $b = 0.35$ ,  $Pr = 0.66$ ) with positive relationships of slightly lower strength than the number of subspecies in models without an interaction term

**Table 2.1** Estimated variable weights (probability that a variable is in the best model) and their estimated coefficients for both number of subspecies and gene flow analyses. The coefficients for the number of subspecies variable are also separated for models with and without the interaction term because this term strongly changes the coefficient and because the coefficient can only be usefully interpreted when estimated without the interaction. However, there are no weights for these separated terms as they are not directly compared in the model selection framework.

Variable	<i>Number of Subspecies</i>		<i>Gene flow</i>	
	<i>Pr</i>	Coefficient	<i>Pr</i>	Coefficient
Number of subspecies				
Total	1.00	0.32		
Models without interaction term		0.63		
Models with interaction term		0.31		
Number of subspecies:log(Scholar)	0.96	0.50		
Gene flow			0.89	−0.67
log(Scholar)	0.98	0.99	0.99	2.49
log(Mass)	0.80	0.48	0.98	−0.35
log(Range size)	0.66	0.35	0.06	1.57
Random	0.21	0.05	0.18	0.23

( $b = 0.63$ , variance = 0.02).

## 2.4.2 Gene Flow

Due to the low number of studies and the restrictive requirements imposed on study design, there was only data for 24 bat species in 7 families. The number of described virus species for a bat host ranged up to 12 viruses in *Miniopterus schreibersii*. Figure 2.3 shows the raw data for the relationship between pathogen richness and log gene flow.

As with the number of subspecies dataset, there was no phylogenetic signal in the number of virus species ( $\lambda = 10^{-6}$ ,  $p > 0.999$ ). Gene flow also had no phylogenetic autocorrelation ( $\lambda = 10^{-6}$ ,  $p > 0.999$ ). Due to the low sample size, significance tests are unlikely to have much power. However, study effort had some phylogenetic autocorrelation ( $\lambda = 0.15$ ,  $p = 0.56$ ) while distribution size and mass seemed to show phylogenetic signal (Distribution size:  $\lambda = 0.67$ ,  $p = 0.53$ , Mass:  $\lambda = 0.79$ ,  $p = 2.69 \times 10^{-3}$ ).

**Table 2.2** Model selection results for number of subspecies and gene flow analysis.  $\bar{AICc}$  is the mean AICc score across 50 resamplings of the null random variable.  $\Delta AICc$  is the model's  $\bar{AICc}$  score minus  $\min(\bar{AICc})$ .  $w$  is the Akaike weight and can be interpreted as the probability that the model is the best model (of those in the plausible set).  $\sum w$  is the cumulative sum of the Akaike weights.  $\log(\text{Scholar}) * \text{NSubspecies}$  implies the interaction term between study effort and number of subspecies as well as both of the individual linear terms. In the number of subspecies analysis there are many models with low  $\Delta AICc$  scores suggesting there is no single 'best model'. In the gene flow analysis, only the top model is supported.

Model	$\bar{AICc}$	$\Delta AICc$	$w$	$\sum w$
<i>Number of Subspecies</i>				
$\log(\text{Scholar}) * \text{NSubspecies} + \log(\text{Mass}) + \log(\text{RangeSize})$	882	0.00	0.38	0.38
$\log(\text{Scholar}) * \text{NSubspecies} + \log(\text{Mass})$	884	1.39	0.19	0.57
$\log(\text{Scholar}) * \text{NSubspecies} + \text{rand} + \log(\text{Mass})$	885	2.24	0.12	0.70
$\log(\text{Scholar}) * \text{NSubspecies}$	885	3.14	0.08	0.78
$\log(\text{Scholar}) * \text{NSubspecies} + \log(\text{RangeSize})$	886	3.18	0.08	0.86
$\log(\text{Scholar}) * \text{NSubspecies} + \text{rand} + \log(\text{RangeSize})$	886	3.94	0.05	0.91
$\log(\text{Scholar}) * \text{NSubspecies} + \text{rand}$	886	3.95	0.05	0.96
$\log(\text{Scholar}) + \text{NSubspecies} + \log(\text{Mass}) + \text{rand}$	889	6.93	0.01	0.97
$\log(\text{Scholar}) + \text{NSubspecies} + \log(\text{Mass}) + \log(\text{RangeSize}) + \text{rand}$	890	7.80	0.01	0.98
<i>Gene flow</i>				
$\log(\text{Scholar}) + \log(\text{Gene flow}) + \log(\text{Mass})$	71	0.00	1.00	1.00
$\log(\text{Range size})$	105	34.09	0.00	1.00
$\log(\text{Mass})$	106	35.06	0.00	1.00

Only the model with study effort, gene flow and mass was well supported with the second model having an  $\Delta AICc$  of 34 (Table 2.2). While less strongly supported than the number of subspecies, gene flow was likely in the best model ( $Pr = 0.89$ ) compared to the benchmark random variable which has  $Pr = 0.18$  (Figure 2.4B and Table 2.1). On average (mean weighted by Akaike weights) there was a negative relationship between gene flow and viral richness ( $b = -0.67$ , variance =  $5.48 \times 10^{-3}$ ) despite the apparent positive relationship (see Figure 2.3) weakly suggested by the single-predictor model (pgls:  $b = 0.63$ ,  $t = 1.16$ ,  $df = 13$ ,  $p = 0.27$ ). This supports the hypothesis that population structure promotes viral richness. Possibly due to the smaller sample size, or a weaker relationship, this coefficient was much more varied than the number of subspecies coefficient with 22% of models estimating a positive relationship.



As in the number of subspecies analysis, study effort was very likely in the best model ( $Pr = 0.99$ ) as was body mass ( $Pr = 0.98$ ). However, body mass has a negative average coefficient ( $b = -0.35$ , variance = 0.04) which is in contrast to the number of subspecies analysis, many studies in the literature (Gay et al. 2014, Kamiya et al. 2014, Maganga et al. 2014, Turmelle & Olival 2009) and the single-predictor model (pgls:  $b = 0.6$ ,  $t = 0.38$ ,  $df = 13$ ,  $p = 0.71$ ). In contrast to the number of subspecies analysis, range size was almost certainly not in the best model with  $Pr = 0.06$  being much less than the random variable. This variable being less supported than the random variable was probably because range size is closely correlated with study effort (pgls:  $b = 0.6$ ,  $t = 4.45$ ,  $df = 13$ ,  $p = 6.58 \times 10^{-4}$ ). Of the three independent variables in the best model, study effort had the largest effect ( $b = 2.49$ , variance = 0.08). The effect size of gene flow ( $b = -0.67$ , variance =  $5.48 \times 10^{-3}$ ) was approximately twice the size of that of body mass ( $b = -0.35$ , variance = 0.04).

Across all models the mean value of  $\lambda$  is  $-1.64$  and a large number of individual models (58%) had negatively phylogenetically distributed residuals implying the residuals from the model are spread more uniformly on the phylogeny than expected by chance. Due to the small sample size this was probably due to a small number of data points with large residuals being distant on the tree.

## 2.5 Discussion

I have tested the hypothesis that population structure promotes pathogen richness in bats. By analysing data on two measures of population structure, and using larger datasets than previous studies, it is hoped that any conclusions may be more robust than the conflicting results in the literature (Gay et al. 2014, Maganga et al. 2014, Turmelle & Olival 2009). I have found that a positive effect of population structure (a positive effect of the number of subspecies and a negative effect of gene flow) are likely to be in the best models for explaining viral richness. Study effort is also clearly supported confirming the expectation that additional study of a bat species yields more known viruses infecting that species and highlighting again that this bias cannot be ignored in studies using known pathogen richness as a proxy for total pathogen richness ().

### 2.5.1 Study limitations

Although I have used measures of study effort to try to control for biases in the viral richness data, this bias could still make the results here unreliable — this is especially true as study effort is by far the strongest predictor of viral richness in both datasets. It is hoped that as untargeted sequencing of viral genetic material (e.g., Anthony et al. (2013)) becomes cheaper and more common this bias can be reduced. The strength of the relationship between study effort and known viral richness also highlights the number of virus species and bat-virus host-pathogen relationships yet to be discovered.

I have used two measures of pathogen richness and the number of subspecies dataset is larger than those used in previous studies. However it is clear that the gene flow dataset is small ( $n = 24$ ). This may explain some unexpected results. While the model averaging approach has given a negative model averaged coefficient for gene flow, the univariate model of gene flow against viral richness gave a positive coefficient. Furthermore body mass has a negative average coefficient. This is in contrast to the number of subspecies analysis, many studies in the literature (Gay et al. 2014, Kamiya et al. 2014, Maganga et al. 2014, Turmelle & Olival 2009) and the single-predictor model. It is not easy to interpret these contradictions but it is clear that the results from the gene flow analysis alone should not be considered strong evidence for a relationship between pathogen richness and population structure. These contradiction also reiterates the need to use large datasets where possible and use multiple measures of population structure to promote robust conclusions.

The results here suggest that there is a positive relationship between population structure and pathogen richness in bats. This is in agreement with (Maganga et al. 2014, Turmelle & Olival 2009) but in disagreement with (Gay et al. 2014). Furthermore it contradicts the assumption that factors that promote high  $R_0$  will automatically promote high pathogen richness (Morand 2000, Nunn et al. 2003). This relationship implies that direct or indirect competitive mechanisms are acting such that population structure is needed in order to allow escape from competition.

The relationship between population structure and pathogen richness suggests that population structure has at least some potential as being predictive of high pathogen richness and therefore of a species' likelihood of being a reservoir of a potentially zoonotic pathogen. However given that it is difficult to measure population structure and given that the relationship appears to be weak at best,

this trait on its own is unlikely to be useful in predicting zoonotic risk. However, as a number of other factors are also associated with pathogen richness (body mass and to a lesser extent range size as shown here but also other traits studied elsewhere), using a combination traits in a predictive (i.e. machine learning) framework has potential to be used in prioritising zoonotic disease surveillance. The main hurdle in this approach is finding a way to validate models — due to the study effort bias in current data, predictive models will also be biased. As unbiased pathogen surveys (e.g. Anthony et al. (2013)) become more common this may become possible. Alternatively, predictive models could be trained on all available — and therefore biased — data and validated by predicting smaller, unbiased datasets such as the data collected in Maganga et al. (2014)

The relationship between pathogen richness and population structure also has implications for habitat fragmentation and range shifts due to global change. In short habitat, fragmentation and range shifts that reduce movement between populations would be predicted to increase pathogen richness. However, depending on the mechanisms by which population structure increases pathogen richness this may not be a cause for concern. If the main mechanism is one that reduces pathogen extinction rates, a newly fragmented population is unlikely to increase its pathogen richness over any appreciable timescale. If however population structure actively promotes the evolution of new pathogen strains or allows the persistence of more virulent strains (Blackwood et al. 2013, Plowright et al. 2011, Pons-Salort et al. 2014) this could have important public health implications. Therefore further study on the exact mechanisms by which population structure affects pathogen richness is needed.

## 2.5.2 Conclusions

In conclusion, this study adds to the evidence that population structure may promote pathogen richness. It does not support the view that factors that increase  $R_0$  will increase pathogen richness. Using larger datasets and multiple measurements makes the weight of the evidence here stronger than in previous studies. However, caution must still be taken in interpreting these results as the data is biased and sparse in one of the analyses.

### 3 Population structure does not increase pathogen richness in theoretical bat populations

This work was conducted in collaboration with Kate Jones and Hilde Wilkinson-Herbots.

### 3.1 Abstract

An increasingly large fraction of emerging diseases come from wild animals and these diseases have a huge impact on human health, healthcare systems and economic development. The chance that a new zoonosis will come from any particular wild host species increases with the diversity of pathogens in that species. However, the factors that control pathogen diversity in wild populations are still unclear. Host species traits such as population density, longevity, body size and population structure have been shown to correlate with pathogen diversity. Typically it is assumed that well-connected, unstructured populations promote the invasion of new pathogens and therefore increase pathogen diversity. However, this assumption is largely untested; in particular our mechanistic understanding of how population structure affects pathogen diversity, in the presence of inter-pathogen competition, is poor. Greater understanding is needed to clarify by which exact mechanisms population structure affects pathogen diversity. It is unknown whether population structure allows invading pathogens to escape from competition by stochastically creating areas of low pathogen prevalence. I hypothesised that low dispersal rates and a low number of connections in a metapopulation network will allow invading pathogens to establish more readily, thus increasing pathogen diversity. I tested these hypotheses using population networks parameterised to mimic wild bat populations as bats have highly varied social structures and have recently been implicated in a number of high profile diseases such as Ebola, SARS, Hendra and Nipah. I found that neither population connectedness nor dispersal rate affect the probability that a new pathogen will invade into a population. The assumption that factors causing high  $R_0$  allow new pathogens to invade and therefore increase pathogen diversity is not supported. Instead I found that changes in population structure do not affect the probability of invasion of a new pathogen. This is in contrast to much theoretical literature which predicts that increased population structure may promote coexistence of pathogens. It is also in contrast to the common assumption that more connected populations increase  $R_0$  and therefore increase pathogen richness. This result implies that population structure does not control pathogen diversity. Instead it seems that local dynamics, that affect the short term branching process at the very start of the epidemic control the chance of pathogen invasion. This also implies that population structure is not a useful proxy for pathogen diversity with respect to zoonotic disease surveillance (in bats for example).

## 3.2 Introduction

Over 50% of emerging infectious diseases have an animal source (Jones et al. 2008, Smith et al. 2014). Zoonotic pathogens can be highly virulent (Lefebvre et al. 2014, Luby et al. 2009) and potentially have huge public health impacts (Granich et al. 2015), economic costs (Knobler et al. 2004) and slow down international development (World Bank 2014). Therefore understanding and predicting changes in the process of zoonotic spillover is a global health priority (Taylor et al. 2001). The number of pathogen species hosted by a wild animal species affects the chance that a disease from that species will infect humans. However, the factors that control the diversity of pathogens in a wild animal population are still unclear (Metcalf et al. 2015); in particular our mechanistic understanding of how population processes inhibit or promote pathogen richness is poor.

A number of host traits have been shown to correlate with pathogen richness including body size (Arneberg 2002, Kamiya et al. 2014), population density (Arneberg 2002, Nunn et al. 2003) and range size (Bordes & Morand 2011, Kamiya et al. 2014). There are few studies that study population structure in a comparative framework. Maganga et al. (2014) found that distribution fragmentation predicts high viral richness, but Gay et al. (2014) finds the opposite relationship. While the data set in Gay et al. (2014) is larger, the analysis in (Maganga et al. 2014) is more focused on fragmentation specifically. Genetic correlates of population structure have also been used. Turmelle & Olival (2009), in a small analysis, found that high  $F_{st}$  (i.e. a structured population) correlates with high richness. However, they did not account for the widely different spatial scales found in population structure studies, nor did they deal with the differences between  $F_{ST}$ ,  $\phi_{ST}$  and other measures appropriately.

Empirical, correlative studies are often contradictory due to small sample sizes, noisy data and because empirical relationships often do not extrapolate well to other taxa (though see Kamiya et al. (2014) for a meta-analysis). The correlation between many traits (e.g., Nunn et al. (2015)) also makes it hard to clearly distinguish which factors are important. Furthermore, knowing *which* factors correlate with pathogen richness does not tell us *how* they control richness. Mechanisms by which a trait could increase pathogen diversity include promoting the evolution of new strains within a species (Buckee et al. 2004), reduction of the rate of pathogen extinction (Rand et al. 1995) and an increased probability of pathogen invasion from other host species (Nunes et al. 2006). These separate mechanisms have not been examined and it is difficult to see how they could be

approached through comparative methods. We need explicit mechanistic models in order to tease apart these factors. Furthermore, a solid mechanistic understanding of these processes will allow us to predict how wild populations will respond to increased human pressure and global change. As habitats fragment we expect wild populations to become smaller and less well connected. Understanding how these population changes interact requires a mechanistic understanding of disease dynamics. Different specific mechanisms predict responses to climate change at different rates — the extinction of parasites could occur relatively rapidly, whereas processes that increase the rate of pathogen acquisition are likely to change pathogen community relatively slowly.

In analytical models of well mixed populations competitive exclusion has been predicted (Ackleh & Allen 2003, Allen et al. 2004, Bremermann & Thieme 1989, Martcheva & Li 2013, Qiu et al. 2013). When competitive exclusion occurs, population structure has sometimes been shown to allow coexistence (Allen et al. 2004, Garmer et al. 2016, Nunes et al. 2006, Qiu et al. 2013). Competing epidemics, or two pathogens spreading at the same time in a population, is a well studied area (Karrer & Newman 2011, Poletto et al. 2013, Poletto et al. 2015). This area is related to the study of pathogen richness in that they indicate that dynamics of multiple pathogens in a population do depend on population structure. However, the results for short term epidemic competition do not directly transfer to the study of long term disease persistence.

One commonly taken assumption in the ecological literature is that factors that promote high disease spread automatically promotes high diversity (Altizer et al. 2003, Morand 2000, Nunn et al. 2003, Poulin 2014, Poulin & Morand 2000); this is contrary to the predictions from analytical models (Allen et al. 2004, Nunes et al. 2006, Qiu et al. 2013). Furthermore, this assumption ignores competitive mechanisms such as cross-immunity and depletion of susceptible hosts. If competitive mechanisms are strong, pathogens in populations structured such that  $R_0$  will be high will be able to easily out-compete invading pathogens. Only if competitive mechanisms are weak will high  $R_0$  enable the invasion of new pathogens and allow higher pathogen diversity.

How structured a population is can be defined in many ways on many scales. Social group size is often examined as an indicator of population in large because the data are readily available. However, group size is only one aspect of population structure. Dispersal between colonies is also an integral component of population structure. Furthermore, many species exhibit fission-fusion social struc-

tures or other less clearly defined groupings.

The most relevant scale to study is that of an epidemiological population. This is the population within which a pathogen can spread in an epidemiologically relevant time period (years or decades). It is therefore closely related to a population as defined by population genetics, but with movement defined on a shorter time scale.

The epidemiological contacts within the population can be examined at the individual level (as in contact network epidemiology) or larger scales. I consider the metapopulation network the most appropriate. Ignoring the metapopulation assumes a fully mixed population which is unlikely. Trying to study the contact network relies on detailed individual level detail which is not available. Metapopulation models consider a network of small subpopulations. Within subpopulations, epidemiological contacts are fully mixed and relatively fast. Between subpopulations, epidemiological contacts are dependent on an underlying network structure and relatively slow. The network underlying the metapopulation is made up of nodes representing the subpopulations, and edges which represent movement between subpopulations. Animals, and therefore infection, can only move between two subpopulations if they are connected by an edge.

There are two factors that affect how structured a population is given this model framework. Firstly, dispersal is the rate at which individuals move between subpopulations. Secondly, the metapopulation network structure controls the spatial extent of dispersal. The simplest way to measure the structure of the network is to calculate the average number of edges each node has. In the extremes, all subpopulations could be either connected to all other subpopulations or only connected to one or two other subpopulations. However, other measures that take into account second-order structure in the network are also often used.

Currently the literature contains very abstract, simplified models (Allen et al. 2004, Garmer et al. 2016, Nunes et al. 2006, Qiu et al. 2013). These cannot be easily applied to real data. They also do not easily give quantitative predictions of pathogen richness; typically they predict either no pathogen coexistence (Bremermann & Thieme 1989, Martcheva & Li 2013) or infinite pathogen diversity (May & Nowak 1994). We need models that can give quantitative predictions of pathogen richness in wild populations. While predicting an absolute value of pathogen richness is likely to be impossible, we should aim for models that attempt to rank species from highest to lowest pathogen richness. This requires a middle ground of model complexity.



To this end I have run metapopulation, multipathogen, epidemiological simulations based broadly on real-world bat populations. Although still simplified, the model is complex enough that if good measurements of bat populations could be obtained, simulations of real world bat populations could be run. Specifically I used these models to test the affects of population structure on the ability of a new pathogen to invade a population. I tested two aspects of structure, dispersal rate and connectedness of the metapopulation network.

I have used bats as a case study as they have an unusually large variety of social structures. Colony sizes range from 10 to 1 million (Jones et al. 2009). Many bats also have interesting seasonal behaviour such as migration (Fleming et al. 2003, Richter & Cumming 2008), hibernation, birth pulses and swarming behaviours. A number of traits have been suggested as predisposing bats towards being reservoirs of zoonotic diseases: high sympatry (Luis et al. 2013), flight (Wang et al. 2011), longevity (Wang et al. 2011) as well as population structure (Gay et al. 2014, Maganga et al. 2014, Turmelle & Olival 2009). Furthermore, bats have, over the last decade, become a focus for disease research (Calisher et al. 2006, Hughes et al. 2007). Recently they have been implicated in a number of high profile diseases such as Ebola, SARS, Hendra and Nipah (Calisher et al. 2006, Li et al. 2005).

I have studied the invasion of new pathogens as a mechanisms for increasing pathogen richness. I have studied two metrics for population structure, dispersal rate and metapopulation network topology, to test for effects of population structure on pathogen richness. In particular I have focused on studying the invasion of a newly evolved pathogen that is therefore identical in epidemiological parameters to the endemic pathogen. Furthermore, this close evolutionary relationship means that cross-immunity is strong.

Here I show that, given the assumptions of a metapopulation, population structure does not affect the rate of invasion of new pathogens. This is true whether population structure is altered by changing the dispersal rate or the topology of the metapopulation network.

### 3.3 Methods

#### 3.3.1 Metapopulation model

**Two pathogen SIR model** I examined a multipathogen SIR model with vital dynamics. This is a compartment model with individuals being classed as suscep-

ible, infected or recovered with immunity (Figure 3.1). Susceptible individuals are counted in class  $S$ . There are three infected classes,  $I_1$ ,  $I_2$  and  $I_{12}$ , being individuals infected with pathogen 1, pathogen 2 or both respectively (see Table 3.1 for a list of symbols and values used). Recovered individuals,  $R$ , are immune to both pathogens, even if they have only been infected with one. Furthermore, recovery from one pathogen moves an individual straight into the recovered class, even if the individual is infected with both pathogen. This modelling choice allows the model to be easily expanded to include more than two pathogens. The assumption of immediate recovery from all other diseases is likely to be quite accurate for very closely related pathogens as is being studied here as once an

**Table 3.1** All symbols used in Chapters 3 and 4 along with their units and default values.

Symbol	Explanation	Units	Value
$S$	Number of susceptible individuals		
$I_q$	N. individuals infectious with diseases $q \in \{1, 2, 12\}$		
$R$	Number of recovered/immune individuals		
$N$	Total Population		30,000
$m$	Number of colonies		10
$n$	Colony starting size		3,000
$a$	Area	km <sup>2</sup>	10,000
$\beta$	Transmission rate	year <sup>-1</sup> .individual <sup>-1</sup>	2, 5, 10
$\gamma$	Recovery rate	year <sup>-1</sup> .individual <sup>-1</sup>	1
$\lambda$	Dispersal	year <sup>-1</sup> .individual <sup>-1</sup>	0.001–0.1
$\Lambda$	Birth rate	year <sup>-1</sup> .individual <sup>-1</sup>	0.05
$\mu$	Death rate	year <sup>-1</sup> .individual <sup>-1</sup>	0.05
$\rho$	No. pathogens		2
$p$	Pathogen index i.e. $p \in \{1, 2\}$ for pathogens 1 and 2		
$q$	Disease class i.e., $q \in \{1, 2, 12\}$		
$t, t'$	Time and time plus waiting time i.e., $t + \delta$	years	
$k_i$	Degree of node $i$		
$\delta$	Waiting time until next event	years	
$\alpha$	Cross-immunity	Proportion	0.1
$n, m$	Colony index		
$\sigma$	Invading pathogen seed size		10
$r_i$	The rate that event $i$ occurs	year <sup>-1</sup>	

acquired immune response is activated, all infections are likely to be cleared quickly.

The coinfection rate (the rate at which an infected individual is infected with a second pathogen) is adjusted compared to the infection rate by a factor  $\alpha$  (here I used  $\alpha = 0.1$  which means coinfection happens at a tenth the rate of first infections). In the application of long term existence of pathogens it is vital to include vital dynamics (births and deaths) as the SIR without vital dynamics has no endemic state. Birth and death rates are assumed to be equal,  $\mu = \Lambda$ . The time scale of the simulations are set by setting  $\mu = 0.05$  per year, yielding an average host generation time of 20 years. Infection is assumed to cause no extra mortality as for a number of viruses, bats show no clinical signs of infection (de Thoisy et al. 2016, Halpin et al. 2011).

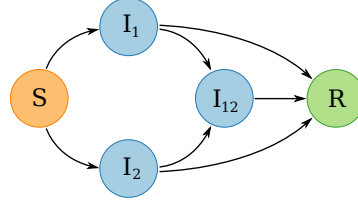
**Metapopulation** The population is divided into a number of subpopulations. This metapopulation is modelled as a network with subpopulations being nodes and dispersal between subpopulations being indicated by edges (Figure 3.2). Individuals within a subpopulation interact randomly so that the subpopulation is fully mixed. Dispersal between subpopulations occurs at a rate  $\lambda$ . Individuals can only disperse to subpopulations connected to theirs in the network. The rate of dispersal is not affected by the number of edges a subpopulation has (the degree of the subpopulation). So the dispersal rate from a subpopulation  $m$  with degree  $k_m$  to subpopulation  $n$  is  $\frac{\lambda}{k_m}$ . Note this rate is independent of the degree and size of subpopulation  $n$ .

**Stochastic simulations** I examined this model using stochastic, continuous time simulations (using the Gillespie algorithm) implemented in R (R Development Core Team 2010). At each step in the simulation we calculate the rate that each possible event might occur. One event is then randomly chosen, weighted by its rate

$$p(\text{event } i) = \frac{r_i}{\sum_{j \neq i} r_j} \quad (3.1)$$

where  $r_i$  is the rate that event  $i$  occurs. Finally, the length of the time step,  $\delta$ , is drawn from an exponential distribution

$$\delta \sim \text{Exp} \left( \sum_i r_i \right). \quad (3.2)$$



**Figure 3.1** Schematic of the SIR model used. Individuals are in one five classes, susceptible (orange, S), infectious with pathogen 1, pathogen 2 or both (blue,  $I_1, I_2, I_{12}$ ) or recovered and immune from further infection (green, R). Transitions between classes occurs only as indicated by arrows. Note that individuals in  $I_{12}$  move into R, not back to  $I_1$  or  $I_2$ . That is, recovery from one pathogen causes immediate recovery from the other pathogen.

We define the number of events we wish to simulate. This means that the total length of each simulation is stochastic.

We can now write down the rates of all events. Assuming asexual reproduction, that all classes reproduce at the same rate and that individuals are born into the susceptible class we get

$$P(S_{nt'} = S_{nt} + 1) = \Lambda \left( S_{nt} + \sum_q I_{qnt} + R_{nt} \right) \quad (3.3)$$

where  $P(S_{nt'} = S_{nt} + 1)$  is the probability that the number of susceptibles in subpopulation  $n$  will increase by 1 (a single birth) in the time interval  $t$  to  $t'$  and  $\sum_q I_{qnt}$  is the sum of all infection classes  $q \in 1, 2, 12$ . The rates of death, given a death rate  $\mu$  are given by

$$P(S_{nt'} = S_{nt} - 1) = \mu S_{nt}, \quad (3.4)$$

$$P(I_{qnt'} = I_{qnt} - 1) = \mu I_{qnt}, \quad (3.5)$$

$$P(R_{nt'} = R_{nt} - 1) = \mu R_{nt}. \quad (3.6)$$

I modelled transmission as being density-dependent. This assumption was more suitable than frequency-dependent transmission as I am modelling a disease transmitted by saliva or urine in highly dense populations confined to caves, building or potentially a small number of tree roosts. I was notably not modelling an STD as these diseases are not expected to be commonly zoonotic. Infection of a susceptible with either pathogen 1 or 2,  $S \rightarrow I_p$  where  $p \in \{1, 2\}$ , is therefore

given by

$$P(I_{1nt'} = I_{1nt} + 1, S_{nt'} = S_{nt} - 1) = \beta S_{nt} (I_{1nt} + I_{12nt}), \quad (3.7)$$

$$P(I_{2nt'} = I_{2nt} + 1, S_{nt'} = S_{nt} - 1) = \beta S_{nt} (I_{2nt} + I_{12nt}), \quad (3.8)$$

while coinfection, given a cross-immunity factor  $\alpha$ , is given by

$$P(I_{12,nt'} = I_{12,nt} + 1, I_{1nt'} = I_{1nt} - 1) = \alpha \beta I_{1nt} (I_{2nt} + I_{12nt}), \quad (3.9)$$

$$P(I_{12,nt'} = I_{12,nt} + 1, I_{2nt'} = I_{2nt} - 1) = \alpha \beta I_{2nt} (I_{1nt} + I_{12nt}). \quad (3.10)$$

The probability of migration from colony  $m$  (with degree  $k_m$ ) to colony  $n$ , given a dispersal rate  $\lambda$  is given by

$$P(S_{nt'} = S_{nt} + 1, S_{mt'} = S_{mt} - 1) = \frac{\lambda S_{mt}}{k_m}, \quad (3.11)$$

$$P(I_{qnt'} = I_{qnt} + 1, I_{qmt'} = I_{qmt} - 1) = \frac{\lambda I_{qmt}}{k_m}, \quad (3.12)$$

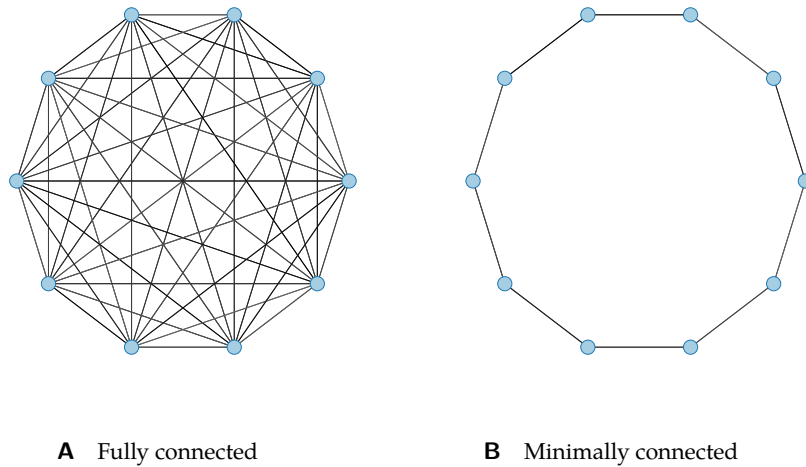
$$P(R_{nt'} = S_{nt} + 1, R_{mt'} = R_{mt} - 1) = \frac{\lambda R_{mt}}{k_m}. \quad (3.13)$$

Finally, recovery from any infectious class occurs at a rate  $\gamma$

$$P(I_{qnt'} = I_{qnt} - 1, R_{nt'} = R_{nt} + 1) = \gamma I_{qnt}. \quad (3.14)$$

### 3.3.2 Experimental setup

The metapopulation contains 10 subpopulations. In each simulation the population was seeded with 10 sets of 200 infected individuals of pathogen 1. These groups were seeded into randomly selected colonies with replacement. For each 200 infected individuals added, 200 susceptible individuals were removed to keep starting colony sizes constant. Pathogen 1 was then allowed to spread and reach equilibrium. After  $3 \times 10^5$  events, 5 individuals infected with pathogen 2 were added to one randomly selected colony. Visual inspection of preliminary simulations was used to decide on  $3 \times 10^5$  as being long enough for the epidemic to reach an equilibrium state. After another  $5 \times 10^5$  events the invasion of pathogen 2 was considered successful if any individuals are still infected with pathogen 2. Again visual inspection of preliminary simulations was used to determine that after  $5 \times 10^5$  events, if an invading pathogen was still present, it was well established.



**Figure 3.2** The two network topologies used to test whether network connectedness influences a pathogen's ability to invade. Blue circles are subpopulations of 3,000 individuals. Dispersal only occurs between subpopulations connected by an edge (black line). The dispersal rate is held constant between the two topologies. A) Dispersal can occur between any subpopulation. B) Animals can only disperse to neighbouring subpopulations.

### 3.3.3 Dispersal

The values used for the independent variables are chosen to highlight the affects of these variables. Dispersal values are  $\lambda = 0.1, 0.01$  and  $0.001$  dispersals per individual per year.  $\lambda = 0.1$  relates to individuals moving between colonies on average twice per lifetime. Therefore exclusively juvenile dispersal would have dispersal rates similar to this. Otherwise it relates to dispersal being a rare event with animals often staying in a colony for many years.  $\lambda = 0.01$  relates to 20% of individuals dispersing once in their lifetime. This value is therefore close to male-biased dispersal, with female philopatry. Finally,  $\lambda = 0.001$  relates to 2% of individuals dispersing in their lifetime. This therefore relates to a population that does not habitually disperse.

### 3.3.4 Network structure

The network structure was synthetically created to be either fully or minimally connected (See Figure 3.2). 10 subpopulations was selected as a trade off between computation time and a network complicated enough that structure might have an effect. This value is artificially small compared to wildlife populations.

### 3.3.5 Parameter selection

The fixed parameters were chosen to roughly reflect realistic wild bat populations. The death rate  $\mu$  was set as 0.05 per year giving a generation time of 20 years. The birth rate  $\Lambda$  was set to be equal to  $\mu$  so that the population size was stable. The recovery rate  $\gamma$  was set to 1 giving a average infection duration of 1 years. This is therefore a long lasting infection but not a chronic infection. It is very difficult to directly estimate infection durations in wild populations but it seems that these infections might sometimes be long lasting (Peel et al. 2012, Plowright et al. 2015). However, other studies have found much shorter infectious periods (Amengual et al. 2007). These shorter lived infections are not studied further here.

Cross-immunity was set to 0.1 so that an individual infected with one pathogen is 90% less likely to be infected with another. This is a rather arbitrary value. However, the rationale of the model was that the invading species might be a newly speciated strain of the endemic species. Furthermore, the model assumes complete cross-immunity after recovery from infection. Therefore cross-immunity to coinfection is likely to be very strong as well.

The population size of each subpopulation was set to 3,000. This is appropriate for many bat species (Jones et al. 2009), especially the large, frugivorous *Pteropodidae* that have been particularly associated with recent zoonotic diseases.

Four values of the transmission rate  $\beta$  are used, 0.1, 0.2, 0.3 and 0.4. All simulations are run under all four transmission rates as this is such a fundamental parameter.

## 3.4 Results

The rates of invasion range from zero out of 100 simulations up to 100 out of 100 (Figure 3.3). Increased transmission clearly increases the rate of invasion even though the endemic pathogen has the same transmission rate as the invading pathogen.

### 3.4.1 Population structure

**Dispersal** The proportion of invasions was not different across dispersal rates for three of four  $\beta$  values ( $\chi^2$  test.  $\beta = 0.1$ :  $\chi^2 = 1.98$ ,  $df = 2$ ,  $p = 0.37$ .  $\beta = 0.3$ :  $\chi^2 = 0.29$ ,  $df = 2$ ,  $p = 0.87$ ,  $\beta = 0.4$ :  $\chi^2 = 0.83$ ,  $df = 2$ ,  $p = 0.66$ ). For  $\beta = 0.2$  the rates of invasion were significantly different ( $\beta = 0.2$ :  $\chi^2 = 9.44$ ,  $df = 2$ ,  $p =$

$8.9 \times 10^{-3}$ ). The middle dispersal rate ( $\lambda = 0.01$ ) has the lowest invasion rate (0.32) with the top and bottom dispersal rates have higher invasion rates ( $\lambda = 0.001$ : invasion rate = 0.38.  $\lambda = 0.1$ : invasion rate = 0.53). Given the 13 hypotheses tested in this study, if a Bonferroni correction is applied this difference would be insignificant at the 95% confidence level ( $8.9 \times 10^{-3} > 0.05/12 = 6.8 \times 10^{-4}$ ). Due to this U-shape relationship and small differences in proportions of invasions I think this is probably not a real effect. Overall, there is no support for the hypothesis that population structure increases the rate at which pathogens invade into the population.

**Network structure** I ran 800 simulations over 4 transmission values ( $\beta = 0.1, 0.2, 0.3, 0.4$ ). The proportion of invasions was not different between highly connected and largely unconnected metapopulations (Figure 3.3B). This was true at all transmission levels ( $\chi^2$  test.  $\beta = 0.1$ :  $\chi^2 < 10^{-5}$ ,  $df = 1$ ,  $p > 0.9999$ .  $\beta = 0.2$ :  $\chi^2 = 0.02$ ,  $df = 1$ ,  $p = 0.88$ .  $\beta = 0.3$ :  $\chi^2 = 1.53$ ,  $df = 1$ ,  $p = 0.22$ .  $\beta = 0.4$ :  $\chi^2 = 0.26$ ,  $df = 1$ ,  $p = 0.61$ ).

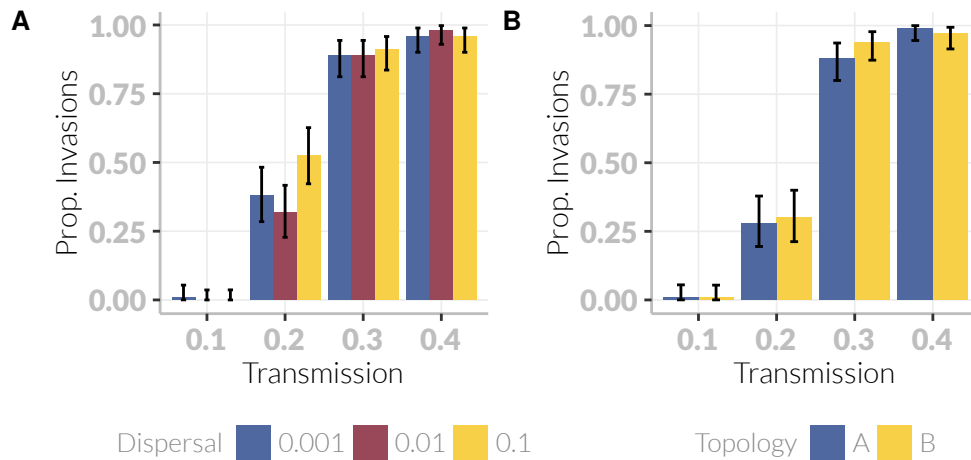
### 3.4.2 Transmission

In line with theory, increasing the transmission rate increased the probability of invasion (Figure 3.3). This is true for all three dispersal values ( $\chi^2$  test.  $\lambda = 0.001$ :  $\chi^2 = 243.56$ ,  $df = 3$ ,  $p < 10^{-5}$ .  $\lambda = 0.01$ :  $\chi^2 = 265.4$ ,  $df = 3$ ,  $p < 10^{-5}$ .  $\lambda = 0.1$ :  $\chi^2 = 244.95$ ,  $df = 3$ ,  $p < 10^{-5}$ ) and both network structures ( $\chi^2$  test. Fully connected:  $\chi^2 = 267.3$ ,  $df = 3$ ,  $p < 10^{-5}$ . Minimally connected:  $\chi^2 = 267.3$ ,  $df = 3$ ,  $p < 10^{-5}$ ).

## 3.5 Discussion

Empirical studies on the role of population structure on pathogen richness have contradictory results (Gay et al. 2014, Maganga et al. 2014, Turmelle & Olival 2009) and cannot examine the specific mechanisms by which pathogen communities are created and maintained. I have used mechanistic, metapopulation models to test whether increased population structure can promote pathogen richness by facilitating invasion of new pathogens. I find that population structure does not affect the ability of a new pathogen to invade and persist in a population. Instead I find that only transmission rate affects the chance of pathogen invasion. That population structure does not affect pathogen richness goes against many predictions that increasing  $R_0$  increases pathogen richness (Altizer et al. 2003, Morand





**Figure 3.3** The probability of successful invasion. For three different transmission rates, the probability of invasion does not change between different A) dispersal rates or B) network topologies (with network topologies A and B referring to the fully and minimally connected topologies in Figure 3.2). Error bars are 95% confidence intervals. 100 simulations were run for each treatment. Other parameters were kept constant at:  $N = 10$ ,  $\mu = \Lambda = 0.05$ ,  $\gamma = 0.1$ ,  $\alpha = 0.1$ . When dispersal is varied, the population structure is fully connected. When population structure is varied,  $\lambda = 0.01$ .

2000, Nunn et al. 2003, Poulin 2014, Poulin & Morand 2000). However, simple analytical models suggest that population structure should increase pathogen richness (Allen et al. 2004, Nunes et al. 2006, Qiu et al. 2013) and I find no evidence of this either.

I have found no evidence that population structure aids the invasion and establishment of newly evolved pathogen species. Instead it seems that invasion relies solely on the local dynamics of the disease. If the transmission rate is high, a pathogen has a good chance of spreading immediately after its introduction when it is at low density. Once the pathogen has infected a large number of individuals in the local metapopulation, it is bound to spread throughout the metapopulation without going extinct.

These results imply that if population structure does in fact affect pathogen richness (Gay et al. 2014, Maganga et al. 2014, Turmelle & Olival 2009) it must occur by a mechanism other than the one studied here. Therefore it is not the spread and persistence of a newly evolved pathogen that is facilitated by population structure. Other mechanisms that should be examined include reduced

competitive exclusion of already established pathogens or increased invasion of less closely and less strongly competing pathogens, perhaps mediated by ecological competition of pathogens (i.e. reduction of the susceptible pool by disease induced mortality). Furthermore, single pathogen dynamics could have an important role such as population structure causing a much slower, asynchronous epidemic preventing acquired herd immunity (Plowright et al. 2011).

Given that I found no affect of population structure on the ability of a new pathogen to invade, this host trait is not useful for predicting the probability that a wild species has many pathogens. This implies it should not be used as a proxy for the probability that a species carries a zoonotic pathogen.

However, my simulations do highlight the importance of competition for the spread of a new pathogen. All parameters used correspond to pathogens with  $R_0 > 1$ . However, the competition with the endemic pathogen means that for some transmission rates the chance of epidemic spread and persistence is close to zero. This has implications for human epidemics as well — if there is strong competition between a newly evolved strain and an endemic strain, we are unlikely to see the new strain spread, irregardless of population structure.

### 3.5.1 Model assumptions

**Complete cross-immunity** I have assumed that once recovered, individuals are immune to both pathogens. Furthermore, when a coinfecting individual recovers from one pathogen, it immediately recovers from the other as well. This is probably a reasonable assumption given that I am modelling a newly evolved strain. However, further work could relax this assumption using a model similar to (Poletto et al. 2015) which contains additional classes for ‘infected with pathogen one, immune to pathogen two’ and ‘infected with pathogen two, immune to pathogen one’. The model here was formulated such that the study of systems with greater than two pathogens is still computationally feasible while a model such as used in (Poletto et al. 2015) contains  $3^p$  classes for a system with  $p$  pathogen species. This quickly becomes computationally restrictive.

**Identical strains** Many papers on pathogen richness have focused on the evolution of pathogen traits and have considered a trade off between transmission rate and virulence (Nowak & May 1994) or infectious period (Poletto et al. 2013). However, here I am interested in host traits. Therefore we have assumed that pathogen strains are identical. It is clear however that there are a number of

factors that affect pathogen richness and our focus on host population structure does not imply that pathogen traits are not important.

**Complex social structure and behaviour** With the models here I have aimed to tread a middle ground between the overly simple models employed in analytical studies (Allen et al. 2004) and the full complexity and variety of true bat social systems (Kerth 2008). Omissions include seasonal migration, maternity roosts, hibernation roosts and swarming sites (Cryan et al. 2014, Fleming et al. 2003, Kerth 2008, Richter & Cumming 2008). While future models might aim to model this complexity more fully the number of parameters that are required to be estimated and varied becomes very large. Furthermore, not all of these social complexities exist in all bat species, so in limiting my analysis to the simpler end of bat social systems it is hoped that the results are more broadly representative of the order.

Furthermore, I have considered a single host species in isolation. In some cases, treating multiple host species as identical could be appropriate. However, more generally, it seems likely that sympatry in bats is epidemiologically important (Brierley et al. 2016, Luis et al. 2013) but was beyond the scope of this study. There is potential for this to be effectively modelled as a multi-layered network (Funk & Jansen 2010, Wang et al. 2016) and this would be expected to act to reduce population structure.

Finally, many species of bat exhibit strong seasonal birth pulses which are known to affect disease dynamics (Amman et al. 2012, Hayman 2015, Peel et al. 2014). This would be expected to facilitate the invasion of new pathogen species; if a new strain evolved or entered the population by migration during a period of low population immunity, it would have a higher chance of invading and establishing in the population.

### 3.5.2 Conclusions

In conclusion I have found no evidence that population structure aids the invasion and establishment of newly evolved pathogen species. This suggests that if population structure does have a role in shaping pathogen communities, it is not by this specific mechanism. Practically, this implies that population structure is not a useful metric for predicting pathogen richness in wild bats. Furthermore it implies that population fragmentation by global change will not promote an increase in pathogen richness.

## 4 A comparison of interrelated population measures: population size, density and colony size

This work was conducted in collaboration with Kate Jones and Hilde Wilkinson-Herbots.

## 4.1 Abstract

An increasingly large fraction of emerging diseases come from wild animals and these diseases have a huge impact on human health. The chance that a new disease will come from any particular host species increases with the diversity of pathogens in that species. However, the factors that control pathogen diversity in wild populations are still poorly understood.

Population density is thought to increase pathogen richness while theory suggests that population structure and population size may also play a role. However, these factors are intrinsically linked — reducing density reduces contacts between individuals and directly reduces population size. In group living species group size and the total number of groups both contribute to total population size. As these factors are all completely interdependent, it is very difficult to study them empirically e.g., in a comparative frame work.

It is unknown whether it is specifically density that controls pathogen diversity or whether density merely correlates with other causal factors such as population structure, group size or population size. Here I use metapopulation SIR models to test whether it is density *per se* that increases the ability of a newly evolved pathogen to invade and persist in a population as apposed to colony size, population size or population structure.

I found that increased group size increases the chance that a new pathogen will invade into a population to the largest extent. Both group size and the number of groups (i.e. the components of population size) promote pathogen richness more than population density. This implies that, in comparative studies, population density is merely a correlate of group size or population size. As these factors are not equally important it is expected that the pathogen communities of different host species will respond to climate change in different ways. Species which experience changes in group size are expected to have larger changes in pathogen richness than other species.

This study helps clarify both the inter-relationships between, and relative importance of, a number of population level factors affecting pathogen richness. It also highlights the necessity for studying the mechanisms underlying pathogen community construction as comparative approaches do not have the specificity to do so.

## 4.2 Introduction

Zoonotic diseases are an increasingly important source of human infectious diseases (Jones et al. 2008, Taylor et al. 2001, Woolhouse & Gowtage-Sequeria 2006). The diversity of pathogens in wild animal populations is huge and largely unknown (Poulin 2014). Furthermore, the factors that allow large numbers of pathogen species to coexist in a host (e.g., Anthony et al. (2013)) are still unclear. It is well known that population level factors such as population density, range size and population structure have an important role in controlling pathogen community dynamics (Anderson & May 1979, Colizza & Vespignani 2007, May & Anderson 1979, May & Lloyd 2001). Global change is strongly perturbing wild animal populations (Craigie et al. 2010, Thomas et al. 2004), but without clear mechanistic models of how these populations maintain pathogen species richness, we can not predict how pathogen communities, and the risks of zoonotic outbreaks, will change in the coming decades.

Variables that describe populations, such as population density and structure, are well established as having a central role in pathogen dynamics (Anderson & May 1979, Barthélemy et al. 2010, Colizza & Vespignani 2007, May & Anderson 1979, Wu et al. 2013). More recently, the role of the population has been examined with respect to pathogen richness and the coexistence of competing pathogens (Allen et al. 2004, Nunes et al. 2006, Qiu et al. 2013). Yet even in theoretical studies there is confusion as to how exactly we should measure populations. There is disagreement on whether population density (individuals per unit area) should be preferred over population size (number of individuals) and how exactly area should be incorporated (Begon et al. 2002).

With the increase of novel zoonotic pathogens (Jones et al. 2008) attention has turned to comparatively assessing the factors that are associated with high or low pathogen richness in wild animal species (Poulin & Morand 2000). Here again there is little clarity on the relationship between a number of species measurements. Population density is commonly studied (Arneberg 2002, Kamiya et al. 2014, Lindenfors et al. 2007, Morand & Poulin 1998, Nunn et al. 2003) as is range size (Huang et al. 2015, Kamiya et al. 2014, Lindenfors et al. 2007, Nunn et al. 2003, Turmelle & Olival 2009). However it is rarely if ever acknowledge that these two values are intrinsically linked by  $d = N/a$  where  $d$  is density,  $N$  is the population size and  $a$  is area (See Table 3.1 for all parameters used) or that the relationship  $N \propto a$  has broad empirical support (Blackburn et al. 2006, Borregaard & Rahbek 2010). In contrast, population size has never been directly studied as

a predictor of pathogen richness — although confusingly, population range size is sometimes used as a measure of size e.g., (Vögeli et al. 2011). Furthermore, population size is considered the more relevant measure in terms of pathogen dynamics, especially when area cannot be assumed to be constant (Begon et al. 2002) as is commonly the case in wild populations, especially in the face of global warming and habitat degradation.

It is clear that animals are neither randomly distributed in space nor epidemiologically ‘will-mixed’: social groups are common (Kerth 2008) and distance and geographic boundaries reduce contacts between isolated populations (Jenkins et al. 2010, Peel et al. 2012). In social species, measures such as global population density are largely meaningless with respect to the number of infectious contacts individuals may have. Rather, contacts are based on group size and rates of movements between groups. Two aspects of non-random transmission have been studied in particular: group size (Ezenwa et al. 2006, Gay et al. 2014, Nunn et al. 2003, Rifkin et al. 2012, Vitone et al. 2004) and global measures of population structure including genetic measures and measures derived from geographic distribution shapes (Gay et al. (2014), Maganga et al. (2014), Turmelle & Olival (2009) and see Chapter 2). Again however, the relationships between these terms and range size, population size and density are rarely examined. Population size can be decomposed into two components, the number of groups and the average size of a group with  $N = nm$  where  $n$  is group size and  $m$  is the number of groups. The amount of movement between groups is at least partially dependent on the distance between them (Jenkins et al. 2010, Le Galliard et al. 2012, Schooley & Branch 2009). The distance between neighbouring groups decreases with the number of groups per area  $m/a$  or  $N/na$ .

Importantly, these factors, although interrelated, will respond differently to global change and the response will be species dependent. Some species may suffer large range contractions, and therefore large falls in population size, while their density remains fairly constant. Other species might retain their distribution but have a depressed population density. Similarly with population structure, species particularly affected by habitat fragmentation can expect increased reduced movement of individuals between groups, while other species may be most affected by a reduction in group size. Furthermore, different mechanisms of maintenance and creation of pathogen richness will respond to changes in these factors differently as well. If pathogen richness ultimately depends on the “island size” of the host population, then falls in population size will reduce pathogen

richness the most. If local group size affects the ability of new pathogens to invade (Nunn et al. (2003), Chapter 3) then changes in group size are likely to be more important. Finally, if increased population structure allows pathogens to coexist (Allen et al. (2004), Nunes et al. (2006) and Qiu et al. (2013) and Chapter 2) increased habitat fragmentation could be expected to increase pathogen richness.

As these population factors — population size and density, range size and group size — are likely to be intercorrelated, correlative comparative studies will struggle to distinguish between them. Furthermore, even if some factors are statistically supported or rejected, the specific mechanisms by which they promote pathogen richness will remain unknown, and these may suggest different responses to global change. Finally, mechanistic models are expected to be more predictive into the future and into hitherto unseen population regimes.

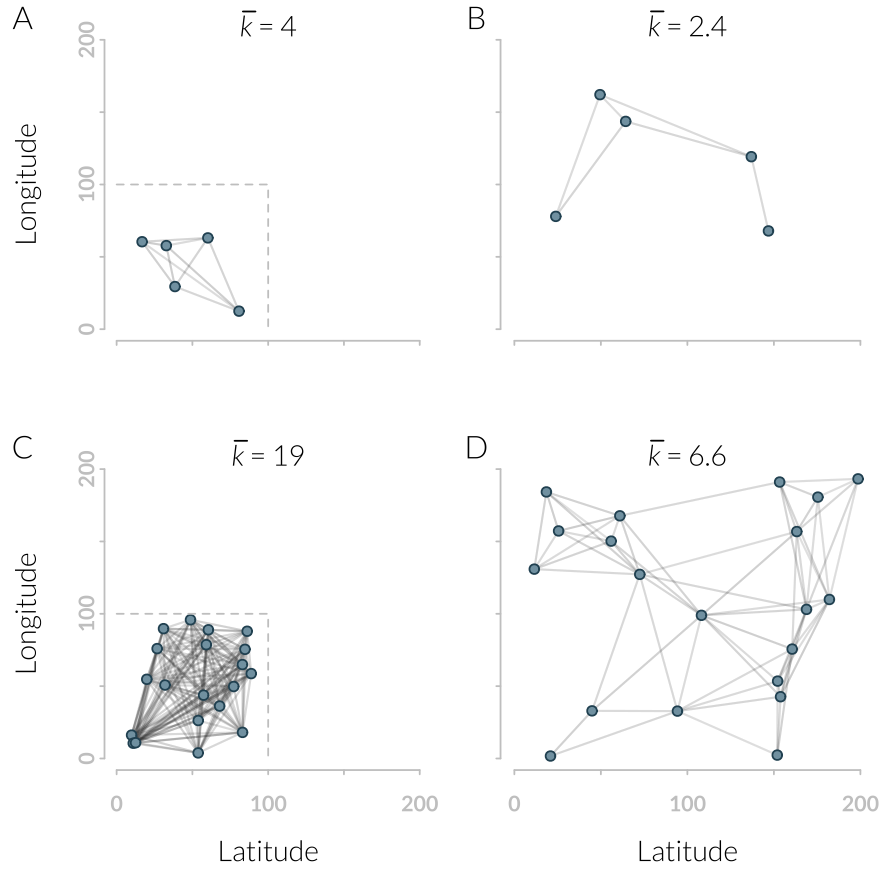
Therefore there is great need for mechanistic models that try to disentangle the interplay between these many factors: density, population size, range size, population structure, group size and the number of groups. Here, I have used multipathogen, metapopulation models to individually vary these population parameters. I examined how these factors affect the ability of a newly evolved pathogen to invade and persist in a population in the presence of strong competition from an endemic pathogen strain. I used these simulations to test two specific hypotheses. First I tested whether population size or population density more strongly promotes the invasion of a new pathogen. Secondly I tested whether the invasion of a new pathogen is more strongly promoted by colony size or the number of colonies.

## 4.3 Methods

### 4.3.1 Metapopulation model

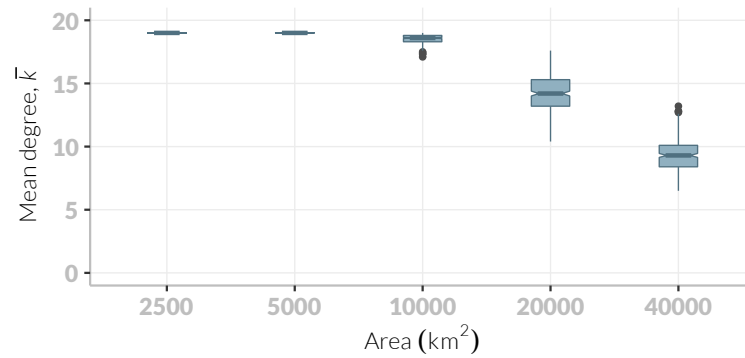
I used a two-pathogen, metapopulation SIR model to compare the roles of demographic parameters on pathogen species richness. Specifically I let two identical pathogens — an endemic pathogen and an invading pathogen — compete and used persistence or not of the second pathogen as my response variable. I tested whether population size is more important than population density. I then tested whether colony size or the number of colonies is the more important component of population size. The multipathogen SIR model is identical to that in Chapter 3 and is implemented in R (R Development Core Team 2010).





**Figure 4.1** Examples of the metapopulation networks used. They include the smallest number of colonies (five, A and B) and the default (20, C and D). They also include the default area ( $10^4 \text{ km}^2$ , grey dashed lines, A and C) and the largest area ( $4 \times 10^4 \text{ km}^2$ , full plot, B and C), though all networks are plotted on the same spatial scale. Colonies are connected if they are within 100km. As area increases, the number of connections each subpopulation has decreases as seen by the changes in mean degree,  $\bar{k}$ .

In each simulation the population is seeded with 20 individuals infected with pathogen 1 in each colony. Pathogen 1 is then allowed to spread and reach equilibrium. After  $7 \times 10^5$  events, 5 individuals infected with pathogen 2 are added to one randomly selected colony. After another  $3 \times 10^5$  events the invasion of pathogen 2 is considered successful if any individuals with pathogen 2 still remain.



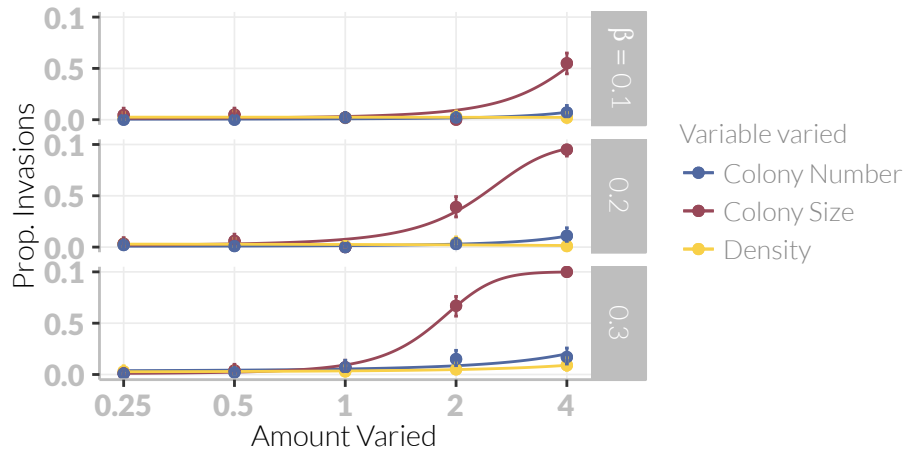
**Figure 4.2** Change in average metapopulation network degree ( $\bar{k}$ ) with increasing area. Bars show the median, boxes show the interquartile range, vertical lines show the range and grey dots indicate outlier values. Notches indicate the 95% confidence interval of the mean. All simulations had 20 colonies, meaning 19 is the maximum value of  $\bar{k}$ .

### 4.3.2 Independent variables

Three independent variables were varied: colony size, number of colonies and area. From these parameters, population size and population density can be calculated. The default values of these parameters was a population size of 8000 individuals split into 20 colonies of 400 individuals. The default area of the simulations was  $10^4 \text{ km}^2$  (space is given in square kilometres for simplicity even though they are in fact arbitrary units).

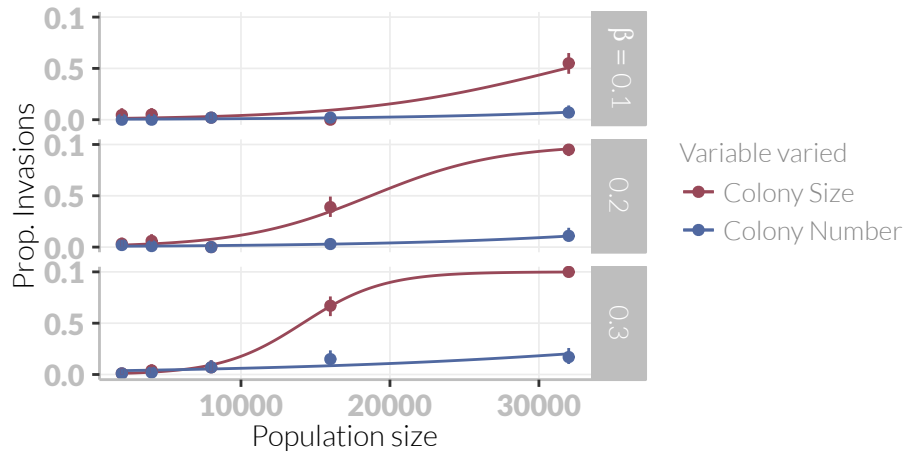
Three sets of simulations were run. First, colony size was varied using values 100, 200, 400, 800 and 1600. The number of colonies was kept constant and so population size varied directly proportionally with colony size. Area was scaled to keep population density constant. Secondly, number of colonies (and therefore population size) was varied and again area was varied to keep density constant. 5, 10, 20, 40 and 80 colonies were used. Finally, colony size and number of colonies were kept constant (therefore keeping population size constant) and area was varied alone to alter population density. The values of area used were  $4 \times 10^4$ ,  $2 \times 10^4$ ,  $10^4$ ,  $5 \times 10^3$  and  $2.5 \times 10^3 \text{ km}^2$  which gave density values of 0.2, 0.4, 0.8, 1.6 and  $3.2 \text{ animals} \cdot \text{km}^{-2}$ .

The affects of area occur through changing the metapopulation network. The metapopulation structure was created for each simulation by randomly placing colonies in space (Figure 4.1). The spatial scale of the simulations vary between  $2.5 \times 10^3$  and  $4 \times 10^4 \text{ km}^2$ . This corresponds to square areas with sides



**Figure 4.3** Comparison of the effect of colony size, colony number and area on probability of invasion. Default values are: colony number = 20, colony size = 400 and density =  $0.8 \text{ animals} \cdot \text{km}^{-2}$ . The  $x$ -axis shows the relative change in each of these values ( $\times 0.25, 0.5, 1, 2$  and  $4$ ). For colony size and number, area is altered so that density remains constant. For density, population size is constant at 8,000 and area is altered. Each point is the mean of 100 simulations and bars are 95% confidence intervals. Curves are simple logistic regression fits for each independent variable. Relationships are shown separately for each transmission value.

of 50 to 200km. Dispersal can only occur between two colonies if they are within 100 kilometres of each other i.e. they are then counted as connected nodes in the metapopulation network. The number of connections each colony has is called its degree,  $k$ . How well connected the metapopulation network is overall is measured by the mean degree,  $\bar{k}$ . This random placement does not guarantee that the population is connected (i.e. made up of a single connected component) but as the endemic pathogen is seeded in all colonies, the invading pathogen cannot be seeded into a fully susceptible colony. This was considered more realistic than repeatedly resampling the population until a fully connected population occurred. The threshold of 100 kilometres was arbitrary but I aimed to maximise the range of  $\bar{k}$  (Figure 4.2) while not having many simulations with networks that were not fully connected. Given this setup, simulations with low densities had relatively unconnected metapopulation networks while high density populations had fully connected networks.



**Figure 4.4** Comparison of the effect of population size on probability of invasion when population size is altered by changing colony size or colony number. Relationship is shown separately for each transmission value. It can be seen that changes in colony size give a much greater increase in invasion probability than changes in colony number. Note that this is the same data as Figure 4.3 but with the  $x$ -axis scaled by population size, not parameter change.

### 4.3.3 Other Parameters

The fixed parameters used are chosen to roughly reflect realistic wild bat populations. The death rate  $\Lambda$  is set as 0.05 per year giving a generation time of 20 years. The birth rate is set to be equal to  $\mu$  so that the population size is stable. The recovery rate  $\gamma$  is set to 1 giving a average infection duration of 1 years. This is therefore a long lasting infection but not a chronic infection. It is difficult to estimate the duration of infections in wild bats but it seems that these infections may often be long lasting (Peel et al. 2012, Plowright et al. 2015). However, much shorter infectious periods have also been identified (Amengual et al. 2007). These shorter lived infections are not studied further here.

Cross-immunity is set to 0.1 so that coinfection is 90% less likely than an initial infection. This is an arbitrary value that is based on the fact that the rationale of the model is that the invading species is a newly speciated strain of the endemic species. Furthermore, the model assumes complete cross-immunity after recovery from infection. Therefore cross-immunity to coinfection is also likely to be strong. Three values, 0.1, 0.2 and 0.3, of the transmission rate  $\beta$  are used. All simulations are run under all three transmission rates.

#### 4.3.4 Statistical comparisons

I tested two hypotheses. Firstly I tested the hypothesis that an increase in population size creates a stronger increase in invasion probability (of the second pathogen) than an equal increase in population density. Secondly, I tested the hypothesis that an increase in colony size creates a stronger increase in invasion probability than a proportionally equal increase in number of colonies. To statistically test these hypotheses I combined the results from different simulations and fitted multiple logistic regressions, centering and scaling the independent variables. Specifically, I fitted the model

$$\text{Invasion} = b_1d + b_2n + b_3m + c + \epsilon \quad (4.1)$$

where  $d, n$  and  $m$  are density, colony size and number of colonies respectively and  $b_i$  are the regression coefficients.  $c$  is a fitted intercept and  $\epsilon$  is a binomially distributed error term. To test the first hypothesis I compared the size (and 95% confidence intervals) of  $b_1$  to  $b_2$  and  $b_3$ . To test the second hypothesis I compared  $b_2$  to  $b_3$ .

### 4.4 Results

At the default parameter settings, the probability of invasion and establishment of the second pathogen,  $P(I)$ , was rare ( $\beta = 0.1$ ,  $P(I) = 0.02$ ;  $\beta = 0.2$ ,  $P(I) = 3.33 \times 10^{-3}$ ;  $\beta = 0.3$ ,  $P(I) = 0.06$ ). Although there is no clear, directional relationship, these proportions are significantly different ( $\chi^2$  test:  $\chi^2 = 17.21$ ,  $df = 2$ ,  $p = 1.83 \times 10^{-4}$ ).

In 37 simulations, both of the pathogens went extinct. This did not depend on transmission rate ( $\chi^2$  test:  $\chi^2 = 1.51$ ,  $df = 2$ ,  $p = 0.47$ ). However they were all either in simulations with the smallest colony size (colony size = 100, 29 simulations) or with the fewest colonies (5 colonies, 8 simulations). Results from these simulations were removed before further analyses.

#### 4.4.1 Population density or size

To test whether population density or size has a stronger affect on invasion probability I compared the regression coefficients of the multiple regressions fitted to simulation results (Figure 4.3). Increasing population size, either by increasing colony size or number of colonies, increased the probability of invasion (Table 4.1).

**Table 4.1** Regression results comparing effects of colony size, colony number and density. Coefficients are from multiple logistic regressions with invasion as the dependent variable and all independent variables being scaled and centred. Colony size and colony number were varied while keeping density equal while density was varied by changing area while keeping population size equal.  $p$  is for the test against the null hypothesis that  $b = 0$ .

$\beta$	Variable	Estimate ( $b$ )	(95% CI)	$p$
0.1	Intercept	-3.52	(-3.87, -3.2)	$< 10^{-5}$
	Colony Size	1.07	(0.75, 1.49)	$< 10^{-5}$
	Colony Number	0.35	(-0.02, 0.79)	0.08
	Density	0.01	(-0.66, 0.52)	0.97
0.2	Intercept	-2.84	(-3.12, -2.58)	$< 10^{-5}$
	Colony Size	2.11	(1.71, 2.6)	$< 10^{-5}$
	Colony Number	0.51	(0.16, 0.95)	0.009
	Density	-0.31	(-0.96, 0.19)	0.29
0.3	Intercept	-2.11	(-2.34, -1.9)	$< 10^{-5}$
	Colony Size	2.74	(2.35, 3.16)	$< 10^{-5}$
	Colony Number	0.25	(0.04, 0.48)	0.02
	Density	0.27	(-0.06, 0.57)	0.09

The relationship between colony size and invasion is strong and significant at all transmission rates, while the relationship between colony number and invasion is weaker and more marginally significant. In contrast, varying population density does not alter invasion probability. Therefore the simulations support the hypothesis that population size affects invasion more strongly than population density.

#### 4.4.2 Colony size or number of groups

To test whether colony size or the number of colonies is the more important component of population size, I compared the regression coefficients,  $b_2$  and  $b_3$ , of the multiple regressions fitted to simulation results (Figure 4.4). Increasing colony size or the number of colonies increases the probability of invasion but this affect is much stronger and more statistically significant, for colony size (Table 4.1). Therefore the simulations support the hypothesis that colony size is the more important component of population size.

## 4.5 Discussion

Overall, my results suggest that population size promotes pathogen richness significantly more than population density in the context of metapopulations or group living. Furthermore, the component of population size that is important is group size.

These results lead to a number of other conclusions. All else being equal, increasing range size (with density remaining constant) will not increase pathogen richness significantly unless the increased range size promotes larger groups. Furthermore, social species that live in large groups are likely to harbour more pathogen species, even if sociality promotes reduced interactions between groups due to territory defence or simply because of larger distances between groups due to groups needing larger home ranges than solitary individuals.

For related, strongly competing strains, the factor that most strongly allows new pathogens to invade is the number of susceptible individuals in the local group. As long as there are enough susceptible individuals that the new pathogen species persists through the stochastic, early stages of the epidemic, the new pathogen will persist. As dispersal is a very slow process compared to infection, the global pool of susceptibles is not important. This is why increasing the number of colonies does not increase pathogen invasion as quickly as the size of a colony does. Similarly, the density — at the global scale — of the species has little affect. In these simulations, increasing density without increasing population size implies a reduction in range size, which simply increases the number of colonies which are connected to the colony experiencing the invading pathogen. This increases the pool of susceptibles that are within one dispersal of the invading pathogen. However, again, this affect is very weak compared to the strong changes in local disease dynamics caused by increasing colony size.

### 4.5.1 Global change

It is clear that many species are suffering strong population changes due to climate change (Thomas et al. 2004). However these changes might affect range size (Thomas et al. 2004), population size (Craigie et al. 2010), population connectivity (Fontúrbel et al. 2014, Rivera-Ortíz et al. 2015, Wasserman et al. 2013) or group size (Atwood 2006, Lehmann et al. 2010, Manor & Saltz 2003, Zunino et al. 2007) to different extents. My results suggest that pathogen communities will respond differently depending on which factors are affected, although it should be

noted that the mechanism here — invasion of a new pathogen — is possibly more relevant over longer, multi-generation time scales than decade-long time scales. In short, species suffering reductions in groups size (Atwood 2006, Lehmann et al. 2010, Manor & Saltz 2003, Zunino et al. 2007) are predicted to experience decreases in pathogen richness in the long term. Species that are experiencing increases in group size (Lehmann et al. 2010) would be expected gain new pathogen species more quickly. In contrast, species suffering range contractions (Thomas et al. 2004) and decreases in population size (Craigie et al. 2010) are expected to experience smaller changes in pathogen richness.

#### 4.5.2 Comparative studies

Many comparative studies measure some aspect of a species population size or structure, yet it is rarely discussed how these relate. Instead most studies use the data that are available, without considering *a priori* how it may depend on other factors (though statistical correlations between independent variables is usually considered and dealt with using PCA or by removing collinear variables). Population density is often measured (Arneberg 2002, Lindenfors et al. 2007, Morand & Poulin 1998, Nunn et al. 2003) yet density is directly associated with population size. This study suggests that it is in fact population size that is important (in the context of social species as studied here). Therefore, the density measures in these comparative studies are more likely to be proxies for population size than the true causal factor. Similarly, this study suggests that host range size does not promote pathogen richness by the mechanism studied here, yet a number of studies have found evidence of this relationship (Kamiya et al. 2014, Nunn et al. 2003). This suggests that either the relationship found in comparative studies is in fact due to a correlation with another factor, or that mechanisms other than rate of invasion of new pathogens are important. Range size has been suggested to affect pathogen richness by a number of mechanisms such as increasing the diversity of sympatric species and these other mechanisms should be specifically tested.

The studies that have tested specifically the affect of group size have in fact found both positive (Vitone et al. 2004) and negative associations (Gay et al. 2014) or no relationship (Ezenwa et al. 2006). Meta-analyses suggest that the relationship between social group size and pathogen richness is weak (Rifkin et al. 2012). This suggests that the mechanism studied here — invasion of recently evolved pathogens — is not the major cause of pathogen richness in wild populations.



### 4.5.3 Assumptions and limitation

Being based on the same model as used earlier, the work presented here relies on many of the same assumptions (see Section 3.5). Furthermore, as a comparison is being made between the effects of area and population size, the exact specifications of how the metapopulation is affected by area is important. I have conducted this study at one rate of dispersal, 0.01 dispersals per individual per year. In practice this relates to only 20% of individuals dispersing in their lifetime. This low rate of dispersal is expected to exaggerate the effect of area; at high rates of dispersal the population is essentially well-mixed, despite the metapopulation.

Also, I have assumed that dispersal only occurs between colonies a certain distance apart. Based on *a priori* considerations such as the time and energy required to disperse long distances this is a reasonable assumption. The exact threshold was chosen to attempt to maximise the range of  $\bar{k}$  studied (Figure 4.2). However, a similar assumption could be made in other ways. Instead of a threshold distance, individuals could be expected to disperse in a random direction and stop at the first colony they encounter; this could create some long distance links in the network and increase network connectivity, potentially reducing the effects of area. Alternatively, the metapopulation could have been modelled as a weighted network with dispersal occurring at a higher rate to nearby colonies. Depending on the parameterisation of this distance-dispersal relationship this could serve to increase the affect of area — by exaggerating dispersal to very nearby colonies — or decrease the affect of area by allowing rare, but significant, global dispersal creating a small-world network structure. Ultimately, the modelling choices could increase or decrease the affects of area relative to colony size and the number of colonies but I have aimed to make the effect of area as strong as possible.

I have used the simple relationships between demographic factors — density = population size / area for example — to illustrate that these are tightly linked. In order to isolate the effects of these factors I have assumed these simple relationships hold; to examine density without altering population size I have fixed population size and manipulated area. However, in reality, these are likely to co-vary both within species across time and also between species. Therefore, while these quantities are certainly linked, they cannot be assumed to have simple linear relationships and should not be used as proxies of each other without further examination. For example, rates and distances of dispersal — which affect the influence of space — may be related to local density (Marjamäki et al. 2013). Sim-

ilarly it is unlikely that a species whose range size decreases will not experience a decrease in total population size as well; the range contraction is likely to occur over generations rather than a simple squeezing of the existing individuals into a smaller area.

#### 4.5.4 Conclusions

Overall I have shown that while a number of demographic factors are intrinsically linked, they have different effects on the rate at which new pathogens will invade. I found that population size, not density, has the stronger impact on the ability of a pathogen to invade. Furthermore, species with large groups are likely to harbour more pathogens than species with many, smaller groups. Due to the correlations between these factors, they are particularly hard to study within a comparative framework; this highlights the utility of mechanistic models.

## 5 A generalised random encounter model for estimating animal density with remote sensor data

This work was conducted in collaboration with Elizabeth Moorcroft, Robin Freeman, Marcus Rowcliffe and Kate Jones and is now published in *Methods in Ecology and Evolution* (Lucas et al. 2015). The text here is almost completely reproduced from Lucas et al. (2015). I formulated and analysed the analytical model. Elizabeth Moorcroft wrote the code for and carried out the simulations. I led the writing of the manuscript with coauthors.

## 5.1 Abstract

Wildlife monitoring technology is advancing rapidly and the use of remote sensors such as camera traps and acoustic detectors is becoming common in both the terrestrial and marine environments. Current methods to estimate population size or density require individual recognition of animals or knowing the distance of the animal from the sensor, which is often difficult. A method without these requirements, the random encounter model (REM), has been successfully applied to estimate animal densities from count data generated from camera traps. However, count data from acoustic detectors do not fit the assumptions of the REM due to the directionality of animal signals.

We developed a generalised REM (gREM), to estimate absolute animal density from count data from both camera traps and acoustic detectors. We derived the gREM for different combinations of sensor detection widths and animal signal widths (a measure of directionality). We tested the accuracy and precision of this model using simulations of different combinations of sensor detection widths and animal signal widths, number of captures, and models of animal movement.

We find that the gREM produces accurate estimates of absolute animal density for all combinations of sensor detection widths and animal signal widths. However, larger sensor detection and animal signal widths were found to be more precise. While the model is accurate for all capture efforts tested, the precision of the estimate increases with the number of captures. We found no effect of different animal movement models on the accuracy and precision of the gREM.

We conclude that the gREM provides an effective method to estimate absolute animal densities from remote sensor count data over a range of sensor and animal signal widths. The gREM is applicable for count data obtained in both marine and terrestrial environments, visually or acoustically (e.g., big cats, sharks, birds, echolocating bats and cetaceans). As sensors such as camera traps and acoustic detectors become more ubiquitous, the gREM will be increasingly useful for monitoring unmarked animal populations across broad spatial, temporal and taxonomic scales.

## 5.2 Introduction

The density of animal populations is one of the fundamental measures in ecology and conservation and has important implications for a range of issues, such as sensitivity to stochastic fluctuations (Wright & Hubbell 1983) and extinction risk (Purvis et al. 2000). Monitoring animal population changes in response to anthropogenic pressure is becoming increasingly important as humans rapidly modify habitats and change climates (Everatt et al. 2014). Sensor technology, such as camera traps (Karanth 1995, Rowcliffe & Carbone 2008) and acoustic detectors (Acevedo & Villanueva-Rivera 2006, Walters et al. 2012) are widely used to monitor changes in animal populations as they are efficient, relatively cheap and non-invasive, allowing for surveys over large areas and long periods (Kessel et al. 2014, Rowcliffe & Carbone 2008, Walters et al. 2013). However, converting sampled count data into estimates of density is problematic as detectability of animals needs to be accounted for (Anderson 2001).

Existing methods for estimating animal density often require additional information that is often unavailable. For example, capture-mark-recapture methods (Borchers et al. 2014, Karanth 1995) require recognition of individuals, and distance methods (Harris et al. 2013) require estimates of how far away individuals are from the sensor (Barlow & Taylor 2005, Marques et al. 2011). When individuals cannot be told apart, an extension of occupancy modelling can be used to estimate absolute population size (Royle & Nichols 2003). However, as the model is originally formulated to estimate occupancy, count information is simplified to presence-absence data. Assumptions about the distribution of individuals (e.g., a Poisson distribution) must also be made (Royle & Nichols 2003) which may be a poor assumption for nonrandomly distributed species. Furthermore repeat, independent surveys must be performed and the definition of a site can be difficult, especially for wide-ranging species (MacKenzie & Royle 2005).

The REM method has been successfully applied to estimate animal densities from camera trap surveys (Zero et al. 2013). However, extending the REM method to other types of sensors (e.g., acoustic detectors) is more problematic, because the original derivation assumes a relatively narrow sensor width (up to  $\pi/2$  radians) and that the animal is equally detectable irrespective of its heading (Rowcliffe et al. 2008).

Whilst these restrictions are not problematic for most camera trap makes (e.g., Reconyx, Cuddeback), the REM cannot be used to estimate densities from camera traps with a wider sensor width (e.g., canopy monitoring with fish eye

lenses, (Brusa & Bunker 2014)). Additionally, the REM method is not useful in estimating densities from acoustic survey data as acoustic detector angles are often wider than  $\pi/2$  radians. Acoustic detectors are designed for a range of diverse tasks and environments (Kessel et al. 2014), which naturally leads to a wide range of sensor detection widths and detection distances. In addition to this, calls emitted by many animals are directional (Blumstein et al. 2011), breaking the assumption of the REM method.

There has been a sharp rise in interest around passive acoustic detectors in recent years, with a 10 fold increase in publications in the decade between 2000 and 2010 (Kessel et al. 2014). Acoustic monitoring is being developed to study many aspects of ecology, including the interactions of animals and their environments (Blumstein et al. 2011, Rogers et al. 2013), the presence and relative abundances of species (Marcoux et al. 2011), biodiversity of an area (Depraetere et al. 2012), and monitoring population trends (Walters et al. 2013).

Acoustic data suffers from many of the problems associated with data from camera trap surveys in that individuals are often unmarked, making capture-mark-recapture methods more difficult to use (Marques et al. 2013). In some cases the distance between the animal and the sensor is known, for example, when an array of sensors is deployed and the position of the animal is estimated by triangulation (Lewis et al. 2007). In these situations distance-sampling methods can be applied (Buckland et al. 2008). However, in many cases distance estimation is not possible, for example, when single sensors are deployed, a situation typical in the majority of terrestrial acoustic surveys (Buckland et al. 2008). In these cases, only relative measures of local abundance can be calculated, and not absolute densities. This means that comparison of populations between species and sites is problematic without assuming equal detectability (Schmidt 2003, Walters et al. 2013). Equal detectability is unlikely because of differences in environmental conditions, sensor type, habitat, and species biology.

In this study, we create a generalised REM (gREM) as an extension to the camera trap model of (Rowcliffe et al. 2008), to estimate absolute density from count data from acoustic detectors, or camera traps, where the sensor width can vary from 0 to  $2\pi$  radians, and the signal given from the animal can be directional. We assessed the accuracy and precision of the gREM within a simulated environment, by varying the sensor detection widths, animal signal widths, number of captures and models of animal movement. We use the simulation results to recommend best survey practice for estimating animal densities from remote

sensors.

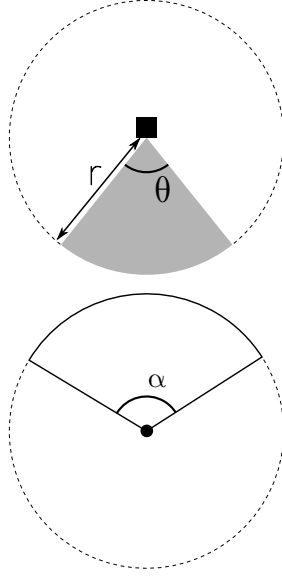
## 5.3 Methods

### 5.3.1 Analytical Model

The REM presented by (Rowcliffe et al. 2008) adapts the gas model to count data collected from camera trap surveys. The REM is derived assuming a stationary sensor with a detection width less than  $\pi/2$  radians. However, in order to apply this approach more generally, and in particular to stationary acoustic detectors, we need both to relax the constraint on sensor detection width, and allow for animals with directional signals. Consequently, we derive the gREM for any detection width,  $\theta$ , between 0 and  $2\pi$  with a detection distance  $r$  giving a circular sector within which animals can be captured (the detection zone) (Figure 5.1). Additionally, we model the animal as having an associated signal width  $\alpha$  between 0 and  $2\pi$  (Figure 5.1, see Appendix S1 for a list of symbols). We start deriving the gREM with the simplest situation, the gas model where  $\theta = 2\pi$  and  $\alpha = 2\pi$ .

**Gas Model** Following (Yapp 1956), we derive the gas model where sensors can capture animals in any direction and animal signals are detectable from any direction ( $\theta = 2\pi$  and  $\alpha = 2\pi$ ). We assume that animals are in a homogeneous environment, and move in straight lines of random direction with velocity  $v$ . We allow that our stationary sensor can capture animals at a detection distance  $r$  and that if an animal moves within this detection zone they are captured with a probability of one; while outside this zone, animals are never captured.

In order to derive animal density, we need to consider relative velocity from the reference frame of the animals. Conceptually, this requires us to imagine that all animals are stationary and randomly distributed in space, while the sensor moves with velocity  $v$ . If we calculate the area covered by the sensor during the survey period, we can estimate the number of animals the sensor should capture. As a circle moving across a plane, the area covered by the sensor per unit time is  $2rv$ . The expected number of captures,  $z$ , for a survey period of  $t$ , with an animal density of  $D$  is  $z = 2rvtD$ . To estimate the density we rearrange to get  $D = z/2rvt$ . Note that as  $z$  is the number of encounters, not individuals, the possibility of repeated detections of the same individual is accounted for (Hutchinson & Waser 2007).

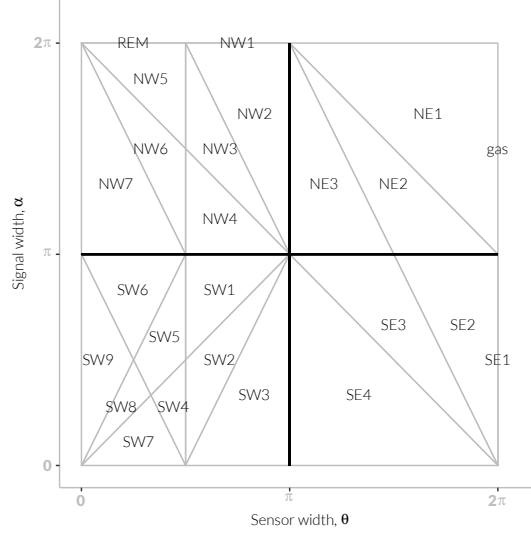


**Figure 5.1** Representation of sensor detection width and animal signal width. The filled square and circle represent a sensor and an animal, respectively;  $\theta$ , sensor detection width (radians);  $r$ , sensor detection distance; dark grey shaded area, sensor detection zone;  $\alpha$ , animal signal width (radians). Dashed lines around the filled square and circle represents the maximum extent of  $\theta$  and  $\alpha$ , respectively.

**gREM derivations for different detection and signal widths** Different combinations of  $\theta$  and  $\alpha$  would be expected to occur (e.g., sensors have different detection widths and animals have different signal widths). For different combinations  $\theta$  and  $\alpha$ , the area covered per unit time is no longer given by  $2rv$ . Instead of the size of the sensor detection zone having a diameter of  $2r$ , the size changes with the approach angle between the sensor and the animal. The width of the area within which an animal can be detected is called the profile,  $p$ . The size of  $p$  depends on the signal width, detector width and the angle that the animal approaches the sensor. The size of the profile (averaged across all approach angles) is defined as the average profile  $\bar{p}$ . However, different combinations of  $\theta$  and  $\alpha$  need different equations to calculate  $\bar{p}$ .

We have identified the parameter space for the combinations of  $\theta$  and  $\alpha$  for which the derivation of the equations are the same (defined as sub-models in the gREM) (Figure 5.2). For example, the gas model becomes the simplest gREM sub-model (upper right in Figure 5.2) and the REM from (Rowcliffe et al. 2008) is another gREM sub-model where  $\theta < \pi/2$  and  $\alpha = 2\pi$ . We derive one gREM



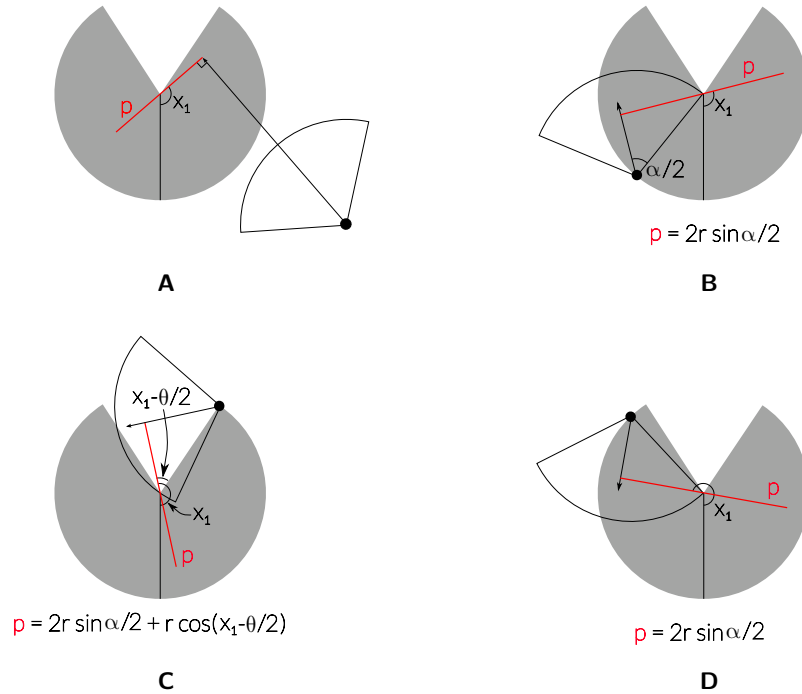


**Figure 5.2** Locations where derivation of the average profile  $\bar{p}$  is the same for different combinations of sensor detection and animal signal widths. Symbols within each polygon refer to each gREM submodel named after their compass point, except for Gas and REM which highlight the position of these previously derived models within the gREM. Symbols on the edge of the plot are for submodels where  $\alpha, \theta = 2\pi$

sub-model SE2 as an example below, where  $2\pi - \alpha/2 < \theta < 2\pi$ ,  $0 < \alpha < \pi$  (see Appendix S2 for derivations of all gREM sub-models). Any estimate of density would require prior knowledge of animal velocity,  $v$  and animal signal width,  $\alpha$  taken from other sources, for example, existing literature (Brinkløv et al. 2011, Carbone et al. 2005). Sensor width,  $\theta$ , and detection distance,  $r$  would also need to be measured or obtained from manufacturer specifications (Adams et al. 2012, Holderied & Von Helversen 2003).

**Example derivation of SE2** In order to calculate  $\bar{p}$ , we have to integrate over the focal angle,  $x_1$  (Figure 5.3a). This is the angle taken from the centre line of the sensor. Other focal angles are possible ( $x_2, x_3, x_4$ ) and are used in other gREM sub-models (see Appendix S2). As the size of the profile depends on the approach angle, we present the derivation across all approach angles. When the sensor is directly approaching the animal  $x_1 = \pi/2$ .

Starting from  $x_1 = \pi/2$  until  $\theta/2 + \pi/2 - \alpha/2$ , the size of the profile is  $2r \sin \alpha/2$  (Figure 5.3b). During this first interval, the size of  $\alpha$  limits the width of the profile.



**Figure 5.3** An overview of the derivation of the average profile  $\bar{p}$  for the gREM submodel SE2, where (a) shows the location of the profile  $p$  (the line an animal must pass through in order to be captured) in red and the focal angle,  $x_1$ , for an animal (filled circle), its signal (unfilled sector), and direction of movement (shown as an arrow). The detection zone of the sensor is shown as a filled grey sector with a detection distance of  $r$ . The vertical black line within the circle shows the direction the sensor is facing. The derivation of  $p$  changes as the animal approaches the sensor from different directions (shown in b-d), where (b) is the derivation of  $p$  when  $x_1$  is in the interval  $[\frac{\pi}{2}, \frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}]$ , (c)  $p$  when  $x_1$  is in the interval  $[\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}, \frac{5\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}]$  and (d)  $p$  when  $x_1$  is in the interval  $[\frac{5\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}, \frac{3\pi}{2}]$ , where  $\theta$ , sensor detection width;  $\alpha$ , animal signal width. The resultant equation for  $p$  is shown beneath b-d. The average profile  $\bar{p}$  is the size of the profile averaged across all approach angles.

When the animal reaches  $x_1 = \theta/2 + \pi/2 - \alpha/2$  (Figure 5.3c), the size of the profile is  $r \sin(\alpha/2) + r \cos(x_1 - \theta/2)$  and the size of  $\theta$  and  $\alpha$  both limit the width of the profile (Figure 5.3c). Finally, at  $x_1 = 5\pi/2 - \theta/2 - \alpha/2$  until  $x_1 = 3\pi/2$ , the width of the profile is again  $2r \sin \alpha/2$  (Figure 5.3d) and the size of  $\alpha$  again limits the width of the profile.

The profile width  $p$  for  $\pi$  radians of rotation (from directly towards the sensor to directly behind the sensor) is completely characterised by the three intervals (Figure 5.3b–d). Average profile width  $\bar{p}$  is calculated by integrating these profiles over their appropriate intervals of  $x_1$  and dividing by  $\pi$  which gives

$$\bar{p} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2}}^{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}} 2r \sin \frac{\alpha}{2} dx_1 + \int_{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}}^{\frac{5\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}} r \sin \frac{\alpha}{2} + r \cos \left( x_1 - \frac{\theta}{2} \right) dx_1 + \int_{\frac{5\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}}^{\frac{3\pi}{2}} 2r \sin \frac{\alpha}{2} dx_1 \right) \quad (5.1)$$

$$= \frac{r}{\pi} \left( \theta \sin \frac{\alpha}{2} - \cos \frac{\alpha}{2} + \cos \left( \frac{\alpha}{2} + \theta \right) \right) \quad (5.2)$$

We then use this expression to calculate density

$$D = z/vt\bar{p}. \quad (5.3)$$

Rather than having one equation that describes  $\bar{p}$  globally, the gREM must be split into submodels due to discontinuous changes in  $p$  as  $\alpha$  and  $\beta$  change. These discontinuities can occur for a number of reasons such as a profile switching between being limited by  $\alpha$  and  $\theta$ , the difference between very small profiles and profiles of size zero, and the fact that the width of a sector stops increasing once the central angle reaches  $\pi$  radians (i.e., a semi-circle is just as wide as a full circle). As an example, if  $\alpha$  is small, there is an interval between Figure 5.3c and 5.3d where the ‘blind spot’ would prevent animals being detected giving  $p = 0$ . This would require an extra integral in our equation, as simply putting our small value of  $\alpha$  into 5.1 would not give us this integral of  $p = 0$ .

gREM submodel specifications were done by hand, and the integration was done using SymPy (SymPy Development Team 2014) in Python (Appendix S3). The gREM submodels were checked by confirming that: (1) submodels adjacent in parameter space were equal at the boundary between them; (2) submodels that border  $\alpha = 0$  had  $p = 0$  when  $\alpha = 0$ ; (3) average profile widths  $\bar{p}$  were between 0 and  $2r$  and; (4) each integral, divided by the range of angles that it was integrated over, was between 0 and  $2r$ . The scripts for these tests are included in Appendix S3 and the R (R Development Core Team 2010) implementation of the gREM is given in Appendix S4.

### 5.3.2 Simulation Model

We tested the accuracy and precision of the gREM by developing a spatially explicit simulation of the interaction of sensors and animals using different combinations of sensor detection widths, animal signal widths, number of captures, and models of animal movement. One hundred simulations were run where each consisted of a 7.5 km by 7.5 km square with periodic boundaries. A stationary sensor of radius  $r$ , 10 m, was set up in the exact centre of each simulated study area, covering seven sensor detection widths  $\theta$ , between 0 and  $2\pi$  ( $2/9\pi$ ,  $4/9\pi$ ,  $6/9\pi$ ,  $8/9\pi$ ,  $10/9\pi$ ,  $14/9\pi$ , and  $2\pi$ ). Each sensor was set to record continuously and to capture animal signals instantaneously from emission. Each simulation was populated with a density of 70 animals  $\text{km}^{-2}$ , calculated from the equation in (Damuth 1981) as the expected density of mammals weighing 1 g. This density therefore represents a reasonable estimate of density of individuals, given that the smallest mammal is around 2 g (Jones et al. 2009). A total of 3937 individuals per simulation were created which were placed randomly at the start of the simulation. 11 signal widths  $\alpha$  between 0 and  $\pi$  were used ( $1/11\pi$ ,  $2/11\pi$ ,  $3/11\pi$ ,  $4/11\pi$ ,  $5/11\pi$ ,  $6/11\pi$ ,  $7/11\pi$ ,  $8/11\pi$ ,  $9/11\pi$ ,  $10/11\pi$ ,  $\pi$ ).

Each simulation lasted for  $N$  steps (14400) of duration  $T$  (15 minutes) giving a total duration of 150 days. The individuals moved within each step with a distance  $d$ , with an average speed,  $v$ . The distance,  $d$ , was sampled from a normal distribution with mean distance,  $\mu_d = vT$ , and standard deviation,  $\sigma_d = vT/10$ , where the standard deviation was chosen to scale with the average distance travelled. An average speed,  $v = 40 \text{ km day}^{-1}$ , was chosen based on the largest day range of terrestrial animals (Carbone et al. 2005), and represents the upper limit of realistic speeds. At the end of each step, individuals were allowed to either remain stationary for a time step (with a given probability,  $S$ ), or change direction where the change in direction has a uniform distribution in the interval  $[-A, A]$ . This resulted in seven different movement models where: (1) simple movement, where  $S$  and  $A = 0$ ; (2) stop-start movement, where (i)  $S = 0.25$ ,  $A = 0$ , (ii)  $S = 0.5$ ,  $A = 0$ , (iii)  $S = 0.75$ ,  $A = 0$ ; (3) correlated random walk movement, where (i)  $S = 0$ ,  $A = \pi/3$ , (ii)  $S = 0$ ,  $A = 2\pi/3$ , (iii)  $S = 0$ ,  $A = \pi$ . Individuals were counted as they moved into the detection zone of the sensor per simulation.

We calculated the estimated animal density from the gREM by summing the number of captures per simulation and inputting these values into the correct gREM submodel. The accuracy of the gREM was determined by comparing the true simulation density with the estimated density. Precision of the gREM

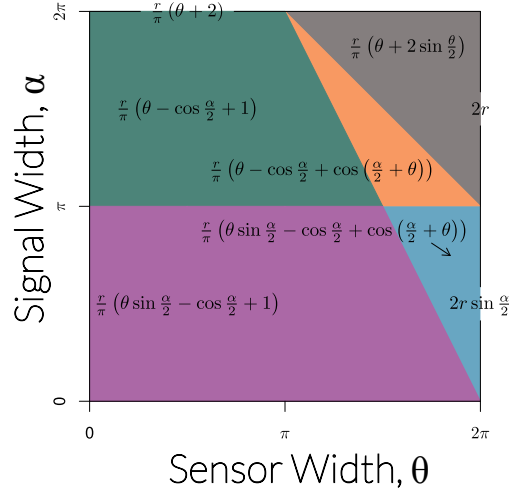
was determined by the standard deviation of estimated densities. We used this method to compare the accuracy and precision of all the gREM submodels. As these submodels are derived for different combinations of  $\alpha$  and  $\theta$ , the accuracy and precision of the submodels was used to determine the impact of different values of  $\alpha$  and  $\theta$ .

The influence of the number of captures and animal movement models on accuracy and precision was investigated using four different gREM submodels representative of the range  $\alpha$  and  $\theta$  values (submodels NW1, SW1, NE1, and SE3, Figure 5.2). From a random starting point we ran the simulation until a range of different capture numbers were recorded (from 10 to 100 captures), recorded the length of time this took, and estimated the animal density for each of the four sub-models. These estimated densities were compared to the true density to assess the impact on the accuracy and precision of the gREM. We calculated the coefficient of variation in order to compare the precision of the density estimates from simulations with different expected numbers of captures. The gREM also assumes that individuals move continuously with straight-line movement (simple movement model) and we therefore assessed the impact of breaking the gREM assumptions. We used the four submodels to compare the accuracy and precision of a simple movement model, stop-start movement models (using different average amounts of time spent stationary), and random walk movement models. Finally, as the parameters ( $\alpha$ ,  $\beta$ ,  $r$  and  $v$ ) are likely to be measured with error, we compared true simulation densities to densities estimated with parameters with errors of 0%,  $\pm 5\%$  and  $\pm 10\%$ , for all gREM submodels.

## 5.4 Results

### 5.4.1 Analytical model

The equation for  $\bar{p}$  has been newly derived for each submodel in the gREM, except for the gas model and REM which have been calculated previously. However, many models, although derived separately, have the same expression for  $\bar{p}$ . Figure 5.4 shows the expression for  $\bar{p}$  in each case. The general equation for density, 5.3, is used with the correct value of  $\bar{p}$  substituted. Although more thorough checks are performed in Appendix S3, it can be seen that all adjacent expressions in Figure 5.4 are equal when expressions for the boundaries between them are substituted in.

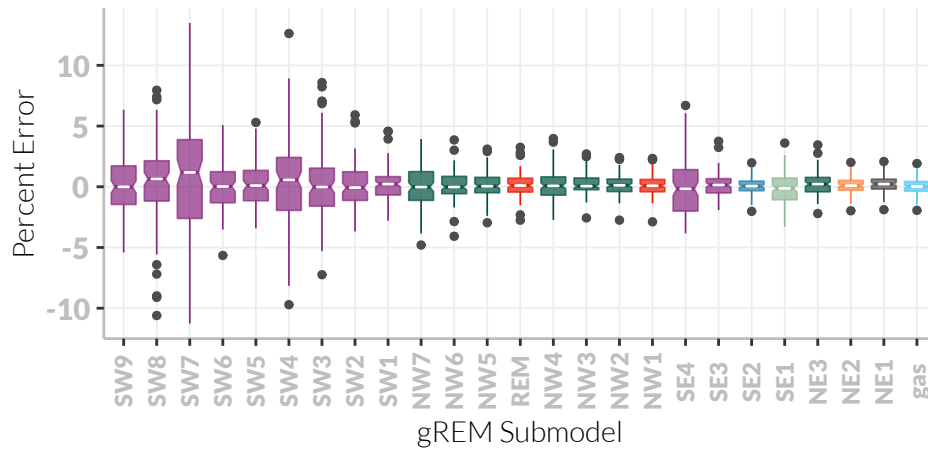


**Figure 5.4** Expressions for the average profile width,  $\bar{p}$ , given a range of sensor and signal widths. Despite independent derivation within each block, many models result in the same expression. These are collected together and presented as one block of colour. Expressions on the edge of the plot are for submodels with  $\alpha, \theta = 2\pi$ .

### 5.4.2 Simulation model

**gREM submodels** All gREM submodels showed a high accuracy, i.e., the median difference between the estimated and true values was less than 2% across all models (Figure 5.5). However, the precision of the submodels do vary, where the gas model is the most precise and the SW7 sub model the least precise, having the smallest and the largest interquartile range, respectively (Figure 5.5). The standard deviation of the error between the estimated and true densities is strongly related to both the sensor and signal widths (Appendix S5), such that larger widths have lower standard deviations (greater precision) due to the increased capture rate of these models.

**Number of captures** Within the four gREM submodels tested (NW1, SW1, SE3, NE1), the accuracy was not strongly affected by the number of captures. The median difference between the estimated and true values was less than 15% across all capture rates (Figure 5.6). However, the precision was dependent on the number of captures across all four of the gREM submodels, where precision increases as number of captures increases, as would be expected for any statistical estimate (Figure 5.6). For all gREM submodels, the the coefficient of variation falls to 10%

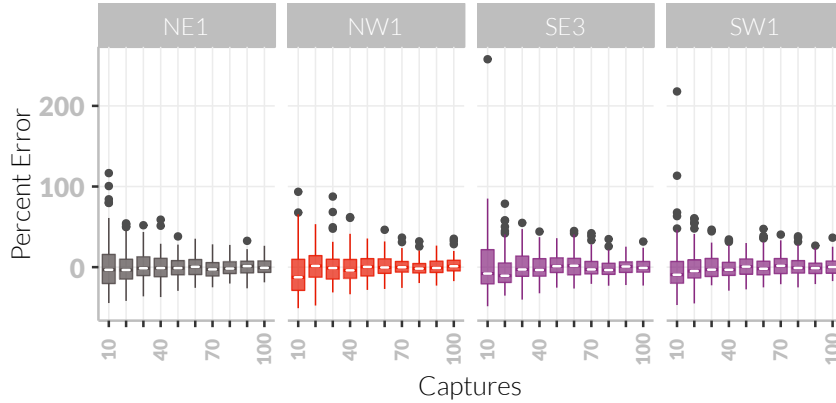


**Figure 5.5** Simulation model results of the accuracy and precision for gREM submodels. The percentage error between estimated and true density for each gREM sub model is shown within each box plot, where the white line represents the median percentage error across all simulations, boxes represent the middle 50% of the data, whiskers represent variability outside the upper and lower quartiles with outliers plotted as individual points. Notches indicate 95% confidence intervals. Box colours correspond to the expressions for average profile width  $\bar{p}$  given in Figure 5.4.

at 100 captures.

**Movement models** Within the four gREM submodels tested (NW1, SW1, SE3, NE1), neither the accuracy or precision was affected by the average amount of time spent stationary. The median difference between the estimated and true values was less than 2% for each category of stationary time (0, 0.25, 0.5 and 0.75) (Figure 5.7). Altering the maximum change in direction in each step (0,  $\pi/3$ ,  $2\pi/3$ , and  $\pi$ ) did not affect the accuracy or precision of the four gREM submodels (Figure 5.7).

**Impact of parameter error** The percentage error in the density estimates across all parameters and gREM submodels shows a similar response for under and over estimated parameters, suggesting the accuracy is reasonable with respect to parameter error (Appendix S6). The impact of parameter error on the precision of the density estimate varies across gREM submodels and parameters, where  $\alpha$  shows the largest variation including the largest values. However, in all cases



**Figure 5.6** Simulation model results of the accuracy and precision of four gREM submodels (NW1, SW1, SE3 and NE1) given different numbers of captures. The percentage error between estimated and true density within each gREM sub model for capture rate is shown within each box plot, where the white line represents the median percentage error across all simulations, boxes represent the middle 50% of the data, whiskers represent variability outside the upper and lower quartiles with outliers plotted as individual points. Notches show the 95% confidence interval. Sensor and signal widths vary between submodels. The numbers beneath each plot represent the coefficient of variation. The colour of each box plot corresponds to the expressions for average profile width  $\bar{p}$  given in Figure 5.4.

the percentage error in the density estimate is not more than 5% greater than the error in the parameter estimate (Appendix S6).

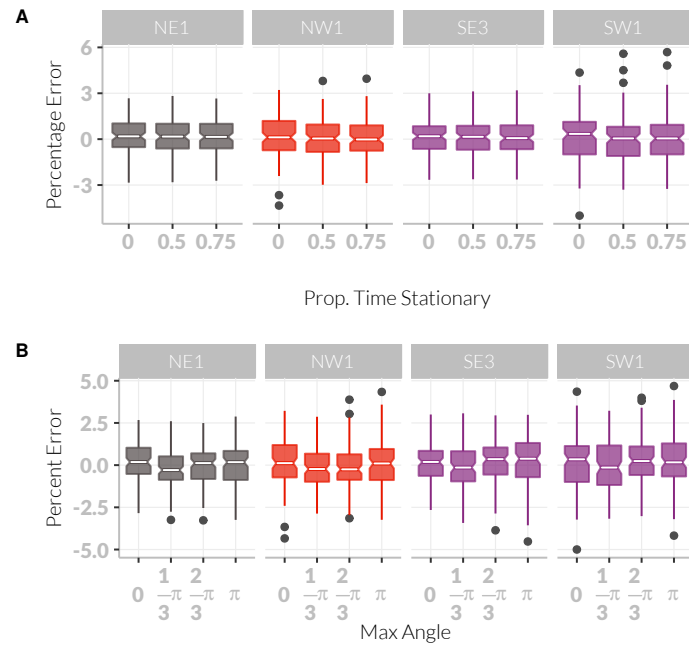
## 5.5 Discussion

### 5.5.1 Analytical model

We have developed the gREM such that it can be used to estimate density from acoustic sensors and camera traps. This has entailed a generalisation of the gas model and the REM in (Rowcliffe et al. 2008) to be applicable to any combination of sensor width  $\theta$  and signal directionality  $\alpha$ . We emphasise that the approach is robust to multiple detections of the same individual. We have used simulations to show, as a proof of principle, that these models are accurate and precise.

There are a number of possible extensions to the gREM which could be developed in the future. The original gas model was formulated for the case where





**Figure 5.7** Simulation model results of the accuracy and precision of four gREM submodels (NW1, SW1, SE3 and NE1) given different movement models where (a) average amount of time spent stationary (stop-start movement) and (b) maximum change in direction at each step (correlated random walk model). The percentage error between estimated and true density within each gREM sub model for the different movement models is shown within each box plot, where the white line represents the median percentage error across all simulations, boxes represent the middle 50% of the data, whiskers represent variability outside the upper and lower quartiles with outliers plotted as individual points. Notches in boxplots show the 95% confidence for the median. The simple model is represented where time and maximum change in direction equals 0. The colour of each box plot corresponds to the expressions for average profile width  $\bar{p}$  given in Figure 4.

both animals and sensor are moving (Hutchinson & Waser 2007). Indeed any of the models which have animals that are equally detectable in all directions ( $\alpha = 2\pi$ ) can be trivially expanded by replacing animal speed  $v$  with  $v + v_s$  where  $v_s$  is the speed of the sensor. However, when the animal has a directional call the extension becomes less simple. The approach would be to calculate again the mean profile width. However, for each angle of approach, one would have to average the profile width for an animal facing in any direction (i.e., not necessarily moving towards the sensor) weighted by the relative velocity of that direction. There are a number of situations where a moving detector and an-

imal could occur, e.g., an acoustic detector towed from a boat when studying porpoises (Kimura et al. 2014) or surveying echolocating bats from a moving car (Jones et al. 2011).

Interesting but unstudied problems impacting the gREM are firstly, edge effects caused by sensor trigger delays (the delay between sensing an animal and attempting to record the encounter) (Rovero et al. 2013), and secondly, sensors which repeatedly turn on and off during sampling (Jones et al. 2011). The second problem is particularly relevant to acoustic detectors which record ultrasound by time expansion. Here ultrasound is recorded for a set time period and then slowed down and played back, rendering the sensor 'deaf' periodically during sampling. Both of these problems may cause biases in the gREM, as animals can move through the detection zone without being detected. As the gREM assumes constant surveillance, the error created by switching the sensor on and off quickly will become more important if the sensor is only on for short periods of time. We recommend that the gREM is applied to constantly sampled data, and the impacts of breaking these assumptions on the gREM should be further explored.

### 5.5.2 Accuracy, Precision and Recommendations for Best Practice

Based on our simulations, we believe that the gREM has the potential to produce accurate estimates for many different species, using either camera traps or acoustic detectors. However, the precision of the gREM differed between submodels. For example, when the sensor and signal width were small, the precision of the model was reduced. Therefore when choosing a sensor for use in a gREM study, the sensor detection width should be maximised. If the study species has a narrow signal directionality, other aspects of the study protocol, such as length of the survey, should be used to compensate.

The precision of the gREM is greatly affected by the number of captures. The coefficient of variation falls dramatically between 10 and 60 captures and then after this continues to slowly reduce. At 100 captures the submodels reach 10% coefficient of variation, considered to be a very good level of precision and better than many previous studies (Foster & Harmsen 2012, O'Brien et al. 2003, Thomas & Marques 2012). The length of surveys in the field will need to be adjusted so that enough data can be collected to reach this precision level. Populations of fast moving animals or populations with high densities will require less survey effort than those species that are slow moving or have populations with low densities.

We found that the sensitivity of the gREM to inaccurate parameter estimates was both predictable and reasonable (Appendix S6), although this varies between different parameters and gREM submodels. Whilst care should be taken in parameter estimation when analysing both acoustic and camera trap data, acoustic data poses particular problems. For acoustic surveys, estimates of  $r$  (detection distance) can be measured directly or calculated using sound attenuation models (Holderied & Von Helversen 2003), while the sensor angle is often easily measured (Adams et al. 2012) or found in the manufacturer’s specifications. When estimating animal movement speed  $v$ , only the speed of movement during the survey period should be used. The signal width is the most sensitive parameter to inaccurate estimates (Appendix S6) and is also the most difficult to measure. While this parameter will typically be assumed to be  $2\pi$  for camera trap surveys, fewer estimates exist for acoustic signal widths. Although signal width has been measured for echolocating bats using arrays of microphones (Brinkløv et al. 2011), more work should be done on obtaining estimates for a range of acoustically surveyed species.

### 5.5.3 Limitations

Although the REM has been found to be effective in field tests (Rowcliffe et al. 2008, Zero et al. 2013), the gREM requires further validation by both field tests and simulations. For example, capture-mark-recapture methods could be used alongside the gREM to test the accuracy under field conditions (Rowcliffe et al. 2008). While we found no effect of the movement model on the accuracy or precision of the gREM, the models we have used in our simulations to validate the gREM are still simple representations of true animal movement. Animal movement may be highly nonlinear and often dependent on multiple factors such as behavioural state and existence of home ranges (Smouse et al. 2010). Therefore testing the gREM against real animal data, or further simulations with more complex movement models, would be beneficial.

The assumptions of our simulations may require further consideration, for example, we have assumed an equal density across the study area. However, in a field environment the situation may be more complex, with additional variation coming from local changes in density between sensor sites. Although unequal densities should theoretically not affect accuracy (Hutchinson & Waser 2007), it will affect precision and further simulations should be used to quantify this effect. Additionally, we allowed the sensor to be stationary and continuously de-

tecting, negating the triggering, and non-continuous recording issues that could exist with some sensors and reduce precision or accuracy. Finally, in the simulation animals moved at the equivalent of the largest day range of terrestrial animals (Carbone et al. 2005). Slower speed values should not alter the accuracy of the gREM, but precision would be affected since slower speeds produce fewer records. The gREM was both accurate and precise for all the movement models we tested (stop-start movement and correlated random walks).

A feature of the gREM is that it does not fit a statistical model to estimate detection probability as occupancy models and distance sampling do (Barlow & Taylor 2005, Marques et al. 2011, Royle & Nichols 2003). Instead it explicitly models the process, with animals only being detected if they approach the sensor from a suitable direction. Other processes that affect detection probability could be included in the model to improve realism.

#### 5.5.4 Implications for ecology and conservation

The gREM is applicable for count data obtained either visually or acoustically in both marine and terrestrial environments, and is suitable for taxa including echolocating bats (Walters et al. 2012), songbirds (Buckland & Handel 2006), whales (Marques et al. 2011) and forest primates (Hassel-Finnegan et al. 2008). Many of these taxa contain critically endangered species and monitoring their populations is of conservation interest. For example, current methods of density estimation for the threatened Franciscana dolphin (*Pontoporia blainvillei*) may result in underestimation of their numbers (Crespo et al. 2010). In addition, using gREM may be easier than other methods for measuring the density of animals which may be useful in quantifying ecosystem services, such as songbirds with a known positive influence on pest control (Jirinec et al. 2011).

The gREM will aid researchers to study species with non-invasive methods such as remote sensors, which allows for large, continuous monitoring projects with limited human resources (Kelly et al. 2012). The gREM is also suitable for species that are sensitive to human contact or are difficult or dangerous to catch (Thomas & Marques 2012). As sensors such as camera traps and acoustic detectors become more ubiquitous, the gREM will be increasingly useful for monitoring unmarked animal populations across broad spatial, temporal and taxonomic scales.

## 6 Discussion

## 6.1 Overview

In this thesis I have aimed to examine the importance of population size and structure on the accumulation of pathogen richness. I used bats as a case study throughout due to their interesting and varied social structure (Kerth 2008) and their association with a number of important, recent zoonoses (Field et al. 2001, Halpin et al. 2011, Leroy et al. 2005, Li et al. 2005). I have studied the role of these population factors using both simulation studies and empirical, comparative approaches in order to both examine the specific, epidemiological mechanisms involved in a controlled and interpretable *in silico* environment, while also being able to link these results back to real-world data. I have found the most robust evidence so far that population structure does relate to higher pathogen richness in bats. However, my simulation study testing whether newly evolved pathogens would invade more easily in a structured population did not recover the same relationship. This implies that it is not this specific mechanism that is important in wild populations. Subsequently, I examined a number of intrinsically linked factors — population size, density and range size as well as colony size and the number of colonies — and found that contrary to beliefs commonly held in the literature, only colony size strongly promotes the invasion of newly evolved pathogens. Finally, I derived and validated a method for estimating bat population sizes from acoustic data; as bat population sizes are very difficult to estimate, this method fills a great need in bat ecology and zoonotic surveillance.

In Chapter 2 I tested the hypothesis that bat species with more structured populations harbour more virus species. I tested this hypothesis with two measurements of population structure: the number of subspecies (a novel measure and the largest dataset yet used to test this hypothesis) and gene flow. With both measures I found that, after controlling for phylogeny and study bias, a positive relationship between population structure and pathogen richness was very likely in the best model. This relationship was of similar strength, and at least as likely to be in the best model, as other measures (body mass and range size) which have been thought to promote pathogen richness in bats and other mammals (Arneberg 2002, Gay et al. 2014, Kamiya et al. 2014, Nunn et al. 2003, Turmelle & Olival 2009).

While the results from Chapter 2 suggest that there is a relationship between population structure and pathogen richness, comparative studies like these cannot identify which specific mechanisms maintain high pathogen richness. To examine this I developed a model of two recently diverged — and therefore

identical — pathogen lineages competing in a metapopulation i.e. a population of large bat colonies with limited movement between them (Chapter 3). I tested whether population structure (specifically network topology and dispersal rate) allowed a second pathogen to invade and persist in the presence of strong competition from the first, endemic pathogen. However, I found no relationship between probability of invasion and population structure. Instead it appeared that if transmission rate was high enough for the invading pathogen to survive the initial, highly stochastic part of its spread, it would then survive and spread throughout the metapopulation irregardless of how structured it was. This implies that local dynamics, defined in part by colony size, are controlling disease invasion and that a different mechanism must be causing the relationship seen in Chapter 2.

Group (or colony) size is one of many demographic parameters measured in comparative studies of pathogen richness. Other commonly measured parameters include population density and range size (Ezenwa et al. 2006, Gay et al. 2014, Kamiya et al. 2014, Lindenfors et al. 2007, Morand & Poulin 1998, Nunn et al. 2003) yet the intrinsic relationships between these variables are rarely acknowledged or discussed. Therefore in Chapter 4 I used the same model as Chapter 3 to test whether population density or population size more strongly promoted pathogen richness and whether a pathogen invaded more easily into a population comprising many small colonies or few big colonies. I found that population size has a much stronger affect than density and that the component of population size that has the strongest affect is colony size.

Theory (Anderson & May 1979, May & Anderson 1979), previous literature (Kamiya et al. 2014, Morand & Poulin 1998, Nunn et al. 2003) and Chapters 3 and 4 suggested that population size (either local group size or global population size) strongly influences the dynamics of disease and pathogen richness. However, there are very few estimates of population size for bats and colony counts are time consuming and costly (Kloepper et al. 2016). I therefore aimed to obtain estimates of population size from acoustic data such as the iBats program (Jones et al. 2011). I developed a general method for estimating population size and density from acoustic detectors (Chapter 5). I used spatial simulations of animal movement to validate the method and found it to be precise and unbiased.

## 6.2 Applications and implications for research

I have found evidence, both empirical and theoretical, that demographic parameters can influence pathogen richness. However it seems likely that this effect alone is not strong enough to be a useful predictor of viral richness with respect to surveillance for zoonotic diseases. While there is potential for population structure and colony size to be useful variables when combined with other variables in a predictive framework, the biases in all pathogen richness datasets makes these approaches difficult. However, as more unbiased data is collected — as in Anthony et al. (2013) and Anthony et al. (2015) — or using much larger pathogen data sets — such as Wardeh et al. (2015) — predictive models may become a more viable tool. Furthermore, the method provided in Chapter 5 makes the collection of population size data more feasible over broad taxonomic, spatial or temporal scales, further increasing the potential of predictive models. Field trials should test the gREM's ability to estimate population size and density and to ensure it is not strongly biased by species specific factors; only if it is unbiased can it be effectively used in predictive models and other applications.

While predictive models are difficult to build due to a lack of data and strong biases in pathogen richness data, the mechanistic understanding obtained by the theoretical chapters here can suggest how pathogen richness may respond to global change. Firstly, when global change acts to reduce group size (Atwood 2006, Lehmann et al. 2010, Manor & Saltz 2003, Zunino et al. 2007) pathogen richness is expected to decrease while in species where group size is increasing (Lehmann et al. 2010) pathogen richness is expected to increase. In contrast, species suffering range contractions (Thomas et al. 2004) and decreases in population size (Craigie et al. 2010) are expected to experience smaller changes in pathogen richness despite these being the more commonly studied effects of global change. This suggests that further research should study in more detail the effects of climate change on social group size.

Furthermore, I have shown that while population factors such as population size, density and range size are directly linked, they have very different effects on pathogen richness. Therefore future studies should be careful to acknowledge these relationships and, where possible, compare multiple demographic measurements to further test which factors are in fact causally affecting pathogen richness.

There is a common assumption that factors that increase  $R_0$  should increase pathogen diversity (Morand 2000, Nunn et al. 2003). However, my results im-



ply a more nuanced relationship. In Chapters 3 and 4 I found that populations with large group sizes, and therefore many localised contacts (i.e. high  $R_0$ ), promote the invasion of new pathogen species, but that at the global level there is little or no effect of population structure. However, in Chapter 2 I found that in wild bat populations, global population structure does promote pathogen species richness. This implies that there are two distinct phases or scales to pathogen competition. When a new pathogen first enters a population, the local scale is important, and many contacts (i.e. a highly connected population) allows the pathogen to spread and avoid stochastic extinction. However, after this initial spread, the global scale may be more important as shown by the stronger support for mechanisms such as population structure (Chapter 2, Maganga et al. (2014) and Turmelle & Olival (2009)) and range size (Kamiya et al. 2014, Nunn et al. 2003) than group size (Ezenwa et al. 2006, Rifkin et al. 2012). This highlights the distinction between factors that promote the addition of new pathogens to the community and those factors that instead allow a larger number of pathogens to coexist or reduce the rate of extinction of pathogens due to competition or other processes. Little research has so far been conducted contrasting these different processes and examining which mechanisms could promote high pathogen richness at each scale.

Much research in multipathogen systems has been conducted over the short time scales of a single epidemic (Funk & Jansen 2010, Poletto et al. 2013, Poletto et al. 2015, van de Bovenkamp et al. 2014). While this time scale has important human health consequences, when examining the slow process of the accumulation of pathogen species, a longer term view needs to be examined. Interestingly, my results, along with previously published studies show quite strong differences between these timescales. Competing epidemics are strongly affected by population structure with structure promoting coexistence of pathogens and allowing less competitive pathogens to persist (Poletto et al. 2013, Poletto et al. 2015). In contrast, in the longer time scales studied here, I have found that population structure does not seem to allow an invading pathogen to escape competition (Chapters 3 and 4). This can be understood by considering that at very long time scales, any population is well mixed unless there is complete separation of sub-populations.

### 6.2.1 Further work

**Other mechanisms controlling pathogen richness** Colony size has been found to have a negative relationship (Gay et al. 2014) and no relationship (Turmelle & Olival 2009) with parasite richness in previous comparative studies using relatively small datasets. However, in Chapter 4 I found that colony size is particularly important for promoting pathogen richness. I did not include colony size in my comparative analysis (Chapter 2) for three reasons: the focus of the chapter was broad-scale population structure, the lack of previous evidence of a positive relationship (Gay et al. 2014, Turmelle & Olival 2009) and the lack of data. However, given the results of Chapter 4, filling these data gaps would be a useful avenue for further research. In particular, testing the relative effects of colony size, population structure and range size would be a useful test of the model used in Chapter 4.

In this thesis I have only examined one mechanism by which demographic attributes may affect pathogen richness. I have only examined the ability of a newly evolved pathogen (i.e. a pathogen, identical to an endemic pathogen and in the presence of strong competition) to invade and persist.

However, there are a number of other mechanisms that could equally strongly affect pathogen richness in the wild. Closely related to the mechanism studied here is the case of pathogens invading from other host species. These pathogens are likely to have different epidemiological parameters (transmission rate, virulence, recovery rate) to the endemic pathogen. Furthermore, the competition between pathogens is expected to be less strong. Alternatively, host population traits could affect the rate of pathogen extinction. Once a number of pathogens are established in a population, there is still likely to be occasional extinctions, especially in the presence of inter-pathogen competition. A number of population factors could affect this rate. It is expected that large populations will experience slower rates of pathogen loss as stochastic extinction will be more rare. Furthermore, populations with strongly varying disease prevalences are likely to have higher rates of pathogen extinction. This includes populations where epidemic cycles are common (Altizer et al. 2006); subsequent to a large epidemic, and after a time lag, the number of susceptible individuals in the populations will be low due to immunity, host death or low birth rates induced by infection (Hethcote 1994, Scott & Lewis 1987). While the number of susceptibles is low, incidence could stochastically drop to zero for one disease generation, thus causing extinction. This effect will be exacerbated in the case where an epidemic cycle is syn-

chronous across the whole population (Duke-Sylvester et al. 2011). Structured populations with asynchronous epidemic cycles may experience local pathogen extinction but rarely global extinction; this pattern of local extinction and recolonisation has been well studied in the ecological literature (Grenfell et al. 1995, Hanski 1998, Levin 1974), but less so in the epidemiological literature.

**Bat social structure** Finally it is important to note that I have ignored much of the social complexity found in bats. In Chapters 3 and 4 I have modelled bat populations as a metapopulation where the only social structure is the grouping of individuals into subpopulations. There is dispersal between these subpopulations but otherwise they are static. Similarly, information on these other social behaviours was not explicitly included in the regression in Chapter 2. Firstly, I have not modelled the creation of new colonies, or the disbanding of colonies (Metheny et al. 2008). Especially in the face of habitat destruction, it is likely that the number of colonies of a species will be decreasing. Furthermore, in some species, colonies are likely to be more fluid, with groups joining and splitting (August et al. 2014, Kerth & Van Schaik 2012). Secondly, there are a number of behaviours common in bats, particularly in temperate regions, that has been excluded from these models. For example, many species have different types of colonies — maternity colonies, mating colonies and hibernation colonies (Kerth 2008). Epidemiological dynamics are likely to be altered by the physiological differences in bats while in these different colony types (Blehert 2012, George et al. 2011, Langwig et al. 2015) but also due to their role in population structure. The extent to which the individuals move together when switching between these colony types is largely unknown (Kurta & Murray 2002) but if there is a large degree of mixing during the transition between colony types, then there will be considerably less population structure overall. Similarly, swarming behaviour — the coming together of many bats from different colonies — is likely to decrease epidemiological population structure (Kerth & Van Schaik 2012).

Furthermore, many bat species, both temperate and tropical, are migratory (Fleming et al. 2003, Hutterer et al. 2005, Krauel & McCracken 2013, Popa-Lisseanu & Voigt 2009). Again, it is largely unknown whether colonies travel together during migration. It is therefore also unknown whether colony structure is similar before and after migration (Carter & Wilkinson 2013) though Kurta & Murray (2002) find that individuals do not migrate together. There is also little data on whether parameters such as dispersal rate are constant before and after migration, though it is likely that most dispersal between colonies is juvenile

dispersal and so dispersal rate is likely to be much higher in one location than the other. Even if colonies remain fairly constant during migration, the spatial relationships may be different; colonies that were far apart in one area could subsequently be near neighbours after migration. Migratory status has been included in previous comparative analyses and not been found to be a strong predictor of pathogen richness (Maganga et al. 2014, Turmelle & Olival 2009).

Another potentially important factor that has been ignored here is roost sharing by different bat species (de Thoisy et al. 2016, López-Roig et al. 2014, Maganga et al. 2014, Pons-Salort et al. 2014, Serra-Cobo et al. 2002). If the species are very similar in most epidemiological factors, this could potentially be sensibly modelled by ignoring species identity and treating the whole population as one. However, it is more likely that there will be fewer close contact events between individuals of different species even if they roost share. It is also likely that species will have different dispersal patterns between colonies. Therefore, more complex models such as overlay network models might be needed in order to effectively model these populations (Funk & Jansen 2010, Marceau et al. 2011). Roost sharing and the amount of sympatry has been included in comparative studies of bat pathogen richness (Maganga et al. 2014) but was not found to correlate with pathogen richness.

Finally, birth and deaths have been modelled here as occurring randomly through time but many bat species have very tightly controlled birth pulses (Dietrich et al. 2015, George et al. 2011, Greiner et al. 2011, Porter & Wilkinson 2001). This has important epidemiological consequences; there will be a pulse of susceptible individuals each year with very few new susceptibles during the rest of the year (Dietrich et al. 2015). Models of these population dynamics have found that birth pulses can drive pathogen extinction (Peel et al. 2014). Hayman (2015) found that certain Filoviruses were less likely to persist in bat species with an annual birth pulse than a biannual birth pulse. In other mammals, birth pulses have also been shown to reduce synchrony of dynamics (Duke-Sylvester et al. 2011).

Overall, there is much complexity that could be added to epidemiological models of bats. However, there is little data for many species which makes parameterisation difficult. Furthermore, as these factors differ between species, trying to make general models that apply across the order is difficult. Further work should include specific, detailed models of well studied species and further examination of how important these various factors might be.

### 6.2.2 Conclusions

Overall my studies suggest that population size and structure do have consequences for pathogen richness. However, the exact mechanisms by which these effects occur are not clear. I have found that colony size is particularly important in the case of closely related, strongly competing pathogens. I have also provided a tool to facilitate the estimation of population sizes in echolocating bats; data which is currently sparse despite its importance to epidemiology and bat ecology more generally.

## A gREM Appendix

## A.1 Table of symbols

**Table A.1** List of symbols used to describe the gREM and simulations. ‘-’ means the quantity has no units.

Symbol	Description	Units
$\theta$	Sensor width	rad
$\alpha$	Animal signal width	rad
$x_i$	Focal angle, $i \in \{1, 2, 3, 4\}$	rad
$r$	Detection distance	m
$\bar{p}$	Average profile width	m
$p$	A specific profile width	m
$v$	Velocity	$\text{m s}^{-1}$
$t$	Time	s
$z$	Number of detections	-
$D$	Animal density	$\text{m}^{-2}$
$T$	Step length	s
$N$	Number of steps per simulation	-
$d$	Distance moved in a time step	m
$S$	Probability of remaining stationary	-
$A$	Maximum turning angle	rad

## A.2 Supplementary Methods

### A.2.1 Introduction

These supplementary methods derive all the models used. For continuity, the gas model derivation is included here as well as in the main text. The calculation of all integrals used in the gREM is included in the Python script S3.

### A.2.2 Gas model

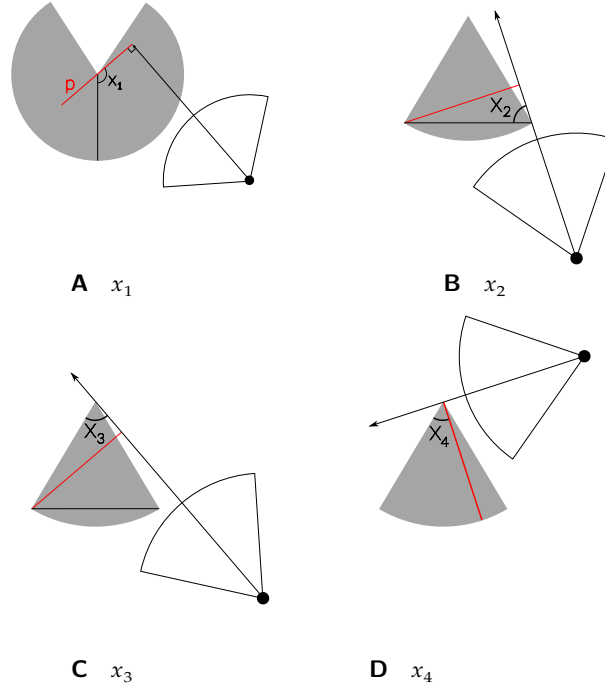
Following (Yapp 1956), we derive the gas model where sensors can capture animals in any direction and animal signals are detectable from any direction ( $\theta = 2\pi$  and  $\alpha = 2\pi$ ). We assume that animals are in a homogeneous environment, and move in straight lines of random direction with velocity  $v$ . We allow that our stationary sensor can capture animals at a detection distance  $r$  and that if an animal moves within this detection zone they are captured with a probability of one, while animals outside the zone are never captured.

In order to derive animal density, we need to consider relative velocity from the reference frame of the animals. Conceptually, this requires us to imagine that all animals are stationary and randomly distributed in space, while the sensor moves with velocity  $v$ . If we calculate the area covered by the sensor during the survey period we can estimate the number of animals the sensor should capture. As a circle moving across a plane, the area covered by the sensor per unit time is  $2rv$ . The number of expected captures,  $z$ , for a survey period of  $t$ , with an animal density of  $D$  is  $z = 2rvtD$ . To estimate the density, we rearrange to get  $D = z/2rvt$ .

#### A.2.2.1 gREM derivations for different detection and signal widths

Different combinations of  $\theta$  and  $\alpha$  would be expected to occur (*e.g.*, sensors have different detection widths and animals have different signal widths). For different combinations  $\theta$  and  $\alpha$ , the area covered per unit time is no longer given by  $2rv$ . Instead of the size of the sensor detection zone having a diameter of  $2r$ , the size changes with the approach angle between the sensor and the animal. For any given signal width and detector width and depending on the angle that the animal approaches the sensor, the width of the area within which an animal can be detected is called the profile,  $p$ . The size of the profile (averaged across all approach angles) is defined as the average profile  $\bar{p}$ . However, different combin-





**Figure A.1** The location of the focal angles  $x_{i \in [1,4]}$ .  $x_1$  is used in SE and NE models (including the gas model).  $x_2 - x_4$  are used in NW and SW models. The sector shaped detection region is shown in grey. Animals are filled black circles and the animal signal is an unfilled sector. The animals direction of movement is indicated with an arrow. The profile  $p$  is shown with a red line. (a) Animal is directly approaching the sensor at  $x_1 = \frac{\pi}{2}$ . (b) Animal is directly approaching the sensor at  $x_2 = \frac{\pi}{2}$ .  $x_2$  then decreases until the profile is perpendicular to the edge of the detection region. (c) When the profile is perpendicular to the edge of the detection region,  $x_3 = \theta$ . (d)  $x_4$  measures the angle between the left side of the detection region and the profile.

ations of  $\theta$  and  $\alpha$  need different equations to calculate  $\bar{p}$ . This  $\bar{p}$  is the only thing that changes

We have identified the parameter space for the combinations of  $\theta$  and  $\alpha$  for which the derivation of the equations are the same (defined as sub-models in the gREM) (Figure 5.2). For example, the gas model becomes the simplest gREM sub-model (upper right in Figure 5.2) and the REM from (Rowcliffe et al. 2008) is another gREM sub-model where  $\theta < \pi/2$  and  $\alpha = 2\pi$ .

Models with  $\theta = 2\pi$  are described first (the gas model described above and SE1). Then models with  $\theta > \pi$  are described (NE then SE). Finally models with

$\theta < \pi$  (NW then SW) are described.

### A.2.3 Model SE1

SE1 is very similar to the gas model except that because  $\alpha \leq \pi$  the profile width is no longer  $2r$  but is instead limited by the width of the animal signal. We therefore get a profile width of  $2r \sin(\alpha/2)$  instead.

$$\bar{p}_{\text{SE1}} = \frac{1}{\pi} \int_{\frac{\pi}{2}}^{\frac{3\pi}{2}} 2r \sin\left(\frac{\alpha}{2}\right) dx_1 \quad (\text{A.1})$$

$$\bar{p}_{\text{SE1}} = 2r \sin\left(\frac{\alpha}{2}\right) \quad (\text{A.2})$$

This profile is integrated over the interval  $[\frac{\pi}{2}, \frac{3\pi}{2}]$  which is  $\pi$  radians of rotation starting with the animal moving directly towards the sensor (Figure A.1a).

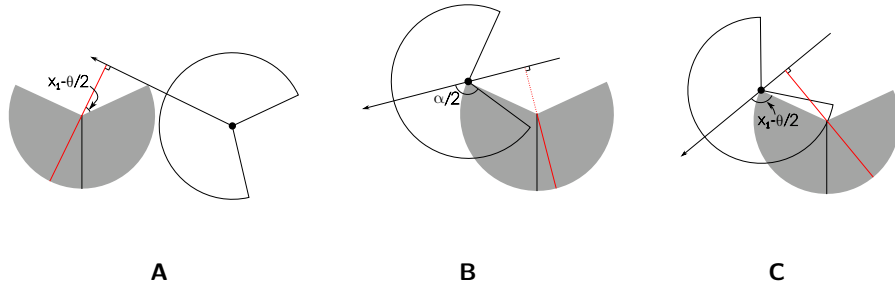
### A.2.4 Models NE1–3

When the detection zone is not a circle, we have more complex profiles and need to explicitly write functions for the width of the profile for every approach angle. We then use these functions to find the average profile width  $\bar{p}$  for all approach angles by integrating across all  $2\pi$  angles of approach and dividing by  $2\pi$ .

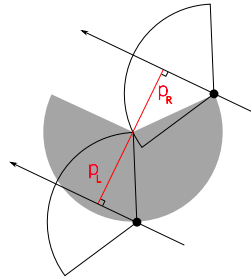
There are three submodels within quadrant NE (Figure 5.2). Note that NE1 covers the area  $\alpha = 2\pi$  as well as the triangle below it as these two models are specified exactly the same, rather than happening to have equal results.

These models have up to five profiles.

1. The profile width starts, from  $x_1 = \frac{\pi}{2}$  as  $2r$ .
2. At  $x_1 = \theta/2$ , the right hand side of the profile cannot be  $r$  wide as the corner of the ‘blind spot’ limits its size to being  $r \cos(x_1 - \theta/2)$  wide (Figure A.2A).
3. The third profile is only found in NE3. If  $\alpha < 4\pi - 2\theta$ , then at  $x_1 = \theta/2 + \pi/2$ , when the profile is perpendicular to the edge of the blind spot, the whole right side of the profile is invisible to the sensor (Figure A.2B). This gives a profile size of just  $r$ .
4. At some point, the sensor can detect animals once they have passed the blind spot giving a profile width of  $r + r \cos(x_1 + \theta/2)$  (Figure A.2C). From



**Figure A.2** Three of the integrals in NE models. The sector shaped detection region is shown in grey. Animals are filled black circles and the animal signal is an unfilled sector. The animals direction of movement is indicated with an arrow. The profile  $p$  is shown with a red line. Dashed red lines indicate areas where animals cannot be detected. (a) The second integral in NE with width  $r + r \cos(x_1 - \theta/2)$ . (b) The third integral in NE3.  $\alpha/2$  is labelled. As it is small, animals to the right of the detector cannot be detected. (c) After further rotation,  $\alpha/2$  is now bigger than the angle shown and animals to the right of the detector can again be detected.



**Figure A.3** The second integral in SE. The right side of the profile ( $p_R$ ) is limited by the size of the sensor region while the left side of the profile ( $p_L$ ) is limited by the size of the signal width. The full profile has width  $p = r \sin(\alpha/2) + r \cos(\theta/2 - x_1)$ . The sector shaped detection region is shown in grey. Animals are filled black circles and the animal signal is an unfilled sector. The animals direction of movement is indicated with an arrow. The profile  $p$  is shown with a red line.

$x_1 = \pi$ , if the animal signal is wide enough to be detected in this area, this is the wider profile. This then defines the split between NE1 and NE2. In NE1, with  $\alpha > 3\pi - \theta$ , the animal signal is wide enough that at  $x_1 = \pi$  the animal can immediately be detected past the blind spot and so this profile is used. In NE2, with  $\alpha < 3\pi - \theta$ , the latter profile is reached at  $5\pi/2 - \theta/2 - \alpha/2$ .

5. Finally, common to all three models, at  $x_1 = 2\pi - \theta/2$  the profile becomes a full  $2r$  once again.

#### A.2.4.1 Model NE1

Submodel NE1 exists within the area bounded by  $\alpha \leq 2\pi$ ,  $\theta \leq 2\pi$  and  $\alpha \geq 3\pi - \theta$  (Figure 5.2). It has four profiles; it does not include the  $r$  profile at  $x_1 = \pi$  (profile described in point (3) in Section A.2.4). Furthermore,  $\theta$  is wide enough that the  $r + r \cos(x_1 + \theta/2)$  profile starts at  $\pi$ . This then gives us

$$\begin{aligned} \bar{p}_{\text{NE1}} = \frac{1}{\pi} & \left( \int_{\frac{\pi}{2}}^{\frac{\theta}{2}} 2r \, dx_1 + \int_{\frac{\theta}{2}}^{\pi} r \cos\left(\frac{\theta}{2} - x_1\right) + r \, dx_1 \right. \\ & \left. + \int_{\pi}^{2\pi - \frac{\theta}{2}} r \cos\left(\frac{\theta}{2} + x_1\right) + r \, dx_1 + \int_{2\pi - \frac{\theta}{2}}^{\frac{3\pi}{2}} 2r \, dx_1 \right) \end{aligned} \quad (\text{A.3})$$

$$\bar{p}_{\text{NE1}} = \frac{r}{\pi} \left( \theta + 2 \sin\left(\frac{\theta}{2}\right) \right) \quad (\text{A.4})$$

#### A.2.4.2 Model NE2

Model NE2 is bounded by  $\alpha \leq 3\pi - \theta$ ,  $\alpha \geq 4\pi - 2\theta$  and  $\alpha \geq \pi$  (Figure 5.2). It is the same as NE1 except that the third profile starts at  $5\pi/2 - \theta/2 - \alpha/2$  instead of at  $\pi$  which is reflected in the different bounds in the second and third integral.

$$\begin{aligned} \bar{p}_{\text{NE2}} = \frac{1}{\pi} & \left( \int_{\frac{\pi}{2}}^{\frac{\theta}{2}} 2r \, dx_1 + \int_{\frac{\theta}{2}}^{\frac{5\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}} r \cos\left(\frac{\theta}{2} - x_1\right) + r \, dx_1 \right. \\ & \left. + \int_{\frac{5\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}}^{2\pi - \frac{\theta}{2}} r \cos\left(\frac{\theta}{2} + x_1\right) + r \, dx_1 + \int_{2\pi - \frac{\theta}{2}}^{\frac{3\pi}{2}} 2r \, dx_1 \right) \end{aligned} \quad (\text{A.5})$$

$$\bar{p}_{\text{NE2}} = \frac{r}{\pi} \left( \theta - \cos\left(\frac{\alpha}{2}\right) + \cos\left(\frac{\alpha}{2} + \theta\right) \right) \quad (\text{A.6})$$

#### A.2.4.3 Model NE3

Model NE3 is bound by  $\alpha \leq 4\pi - 2\theta$ ,  $\alpha \geq \pi$  and  $\theta \geq \pi$  (Figure 5.2). It is the same as NE2 except that it contains the extra profile with width  $r$  (third integral).

$$\bar{p}_{\text{NE3}} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2}}^{\frac{\theta}{2}} 2r \, dx_1 + \int_{\frac{\theta}{2}}^{\frac{\theta}{2} + \frac{\pi}{2}} r \cos\left(\frac{\theta}{2} - x_1\right) + r \, dx_1 \right. \\ \left. + \int_{\frac{\theta}{2} + \frac{\pi}{2}}^{\frac{5\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}} r \, dx_1 + \int_{\frac{5\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}}^{2\pi - \frac{\theta}{2}} r \cos\left(\frac{\theta}{2} + x_1\right) + r \, dx_1 + \int_{2\pi - \frac{\theta}{2}}^{\frac{3\pi}{2}} 2r \, dx_1 \right) \quad (\text{A.7})$$

$$\bar{p}_{\text{NE3}} = \frac{r}{\pi} \left( \theta - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.8})$$

### A.2.5 Models SE2–4

Quadrant SE contains three submodels excluding SE1 (Figure 5.2). The differences between these three models are similar to the differences between the models in NE. There are four possible profiles.

1. As  $\alpha$  is less than  $\pi$  the profile is smaller than  $2r$ , even when the sensor width is a full diameter. The profile width starts as  $2r \sin(\alpha/2)$ .
2. Similar to NE, at a certain point the blind spot of the sensor area limits the profile width on one side. This gives a profile width of  $r \sin(\alpha/2) + r \cos(x_1 - \theta/2)$  (Figure A.3).
3. Also similar to NE, there can be a point where the right side of the profile is 0 giving a profile width of  $r \sin(\alpha/2)$ .
4. If  $\alpha \leq 2\pi - \theta$ , then at  $x_1 = \theta/2 + \pi/2 + \alpha/2$  the profile width becomes 0. This inequality distinguishes between SE3 and SE4.
5. The third profile  $r \sin(\alpha/2)$  starts at  $\theta/2 + \pi/2$  while at  $5\pi/2 - \alpha/2 - \theta/2$  the profile returns to size  $2r \sin(\alpha/2)$ . If  $\theta/2 + \pi/2 \geq 5\pi/2 - \alpha/2 - \theta/2$  we go straight into the  $2r \sin(\alpha/2)$  profile and miss the  $r \sin(\alpha/2)$  profile. SE2 and SE3 are separated by this inequality which simplifies to  $\alpha \leq 4\pi - 2\theta$ .

#### A.2.5.1 Model SE2

SE2 is bounded by  $\alpha \geq 4\pi - 2\theta$ ,  $\alpha \leq \pi$  and  $\theta \leq 2\pi$  (Figure 5.2). As  $\alpha \geq 4\pi - 2\theta$ , there is no  $r \sin(\alpha/2)$  profile. As  $\alpha \leq 4\pi - 2\theta$ , the profile returns to  $2r \sin(\alpha/2)$  rather than going to 0. These integrals relate to profiles (1), (2) and (5) in Section A.2.5.

$$\bar{p}_{\text{SE2}} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2}}^{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}} 2r \sin\left(\frac{\alpha}{2}\right) dx_1 + \int_{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}}^{\frac{5\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}} r \sin\left(\frac{\alpha}{2}\right) + r \cos\left(\frac{\theta}{2} - x_1\right) dx_1 \right. \\ \left. + \int_{\frac{5\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}}^{\frac{3\pi}{2}} 2r \sin\left(\frac{\alpha}{2}\right) dx_1 \right) \quad (\text{A.9})$$

$$\bar{p}_{\text{SE2}} = \frac{r}{\pi} \left( \theta \sin\left(\frac{\alpha}{2}\right) - \cos\left(\frac{\alpha}{2}\right) + \cos\left(\frac{\alpha}{2} + \theta\right) \right) \quad (\text{A.10})$$

### A.2.5.2 Model SE3

SE3 is bounded by  $4\pi - 2\theta \leq \alpha \leq 4\pi - 2\theta$  and  $\alpha \leq \pi$  (Figure 5.2). Therefore there is a  $r \sin(\alpha/2)$  profile but no  $0r$  profile. This relates to profiles (1), (2), (3) and (5) above.

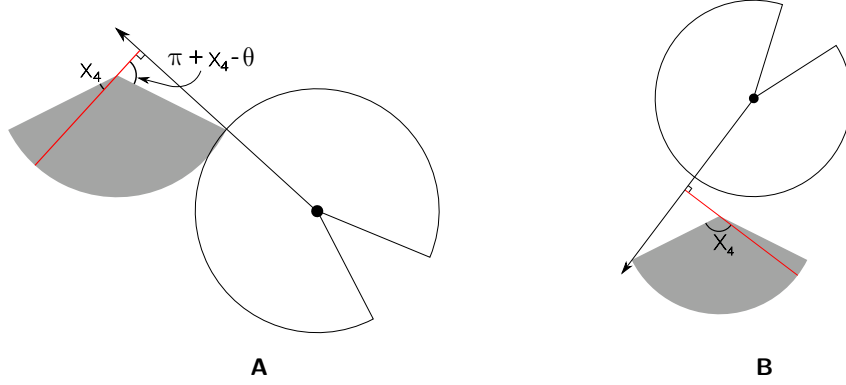
$$\bar{p}_{\text{SE3}} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2}}^{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}} 2r \sin\left(\frac{\alpha}{2}\right) dx_1 + \int_{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}}^{\frac{\theta}{2} + \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) + r \cos\left(\frac{\theta}{2} - x_1\right) dx_1 \right. \\ \left. + \int_{\frac{\theta}{2} + \frac{\pi}{2}}^{\frac{5\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}} r \sin\left(\frac{\alpha}{2}\right) dx_1 + \int_{\frac{5\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}}^{\frac{3\pi}{2}} 2r \sin\left(\frac{\alpha}{2}\right) dx_1 \right) \quad (\text{A.11})$$

$$\bar{p}_{\text{SE3}} = \frac{r}{\pi} \left( \theta \sin\left(\frac{\alpha}{2}\right) - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.12})$$

### A.2.5.3 Model SE4

Finally SE4 is bounded by  $\alpha \leq 4\pi - 2\theta$ ,  $\alpha \leq \pi$  and  $\theta \leq \pi$  (Figure 5.2). It is the same as SE3 except that the profile becomes 0 rather than returning to  $2r \sin(\alpha/2)$ . This relates to profiles (1), (2), (3) and (4) above though profile (4) with width 0 is not shown.

$$\bar{p}_{\text{SE4}} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2}}^{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}} 2r \sin\left(\frac{\alpha}{2}\right) dx_1 + \int_{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}}^{\frac{\theta}{2} + \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) + r \cos\left(\frac{\theta}{2} - x_1\right) dx_1 \right. \\ \left. + \int_{\frac{\theta}{2} + \frac{\pi}{2}}^{\frac{\alpha}{2} + \frac{\theta}{2} + \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) dx_1 \right) \quad (\text{A.13})$$



**Figure A.4** The second and fourth profiles of NW1. The left side of both profiles is of width  $r$  while the right side differs. (a) The right side of the profile is  $r \cos(\pi + x_4 - \theta) = -r \cos(\theta - x_4)$  (b) The right side is  $r \cos(\pi - x_4) = -r \cos x_4$  respectively. In both images the sector shaped detection region is shown in grey. Animals are filled black circles and the animal signal is an unfilled sector. The animal's direction of movement is indicated with an arrow. The profile  $p$  is shown with a red line.

$$\bar{p}_{SE4} = \frac{r}{\pi} \left( \theta \sin\left(\frac{\alpha}{2}\right) - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.14})$$

### A.2.6 Model NW1

NW1 is the first model with  $\theta < \pi$ . Whereas previously the focal angle has always been  $x_1$ , we now use different focal angles.  $x_2$  and  $x_3$  correspond to  $\gamma_1$  and  $\gamma_2$  in (Rowcliffe et al. 2008) while  $x_4$  is new. They are described in Figure A.1b–d.

There are five different profiles in NW1.

1.  $x_2$  has an interval of  $[\pi/2, \theta/2]$  which is from the angle of approach being directly towards the sensor until the profile is parallel to the left hand radius of the sensor sector (Figure A.1B). During this interval the profile width is  $2r \sin(\theta/2) \sin(x_2)$  which is calculated using the equation for the length of a chord. Note that while rotating anti-clockwise (as usual)  $x_2$  decreases in size.

2. From here, we examine focal angle  $x_4$  (note that  $x_3$  is used in later models, but is not relevant here). The left side of the profile is a full radius while the right side is limited to  $-r \cos(x_4 - \theta)$  (Figure A.4A).
3. At  $x_4 = \theta - \pi/2$ , the profile is perpendicular to the edge of the sensor area. Here, the right side of the profile is  $0r$  giving a profile size of  $r$ .
4. When  $x_4 = \pi/2$  the angle of approach is from behind the sensor, but we can once again be detected on the right side of the sensor (Figure A.4B). Therefore the width of the profile is  $r - r \cos(x_4)$ .
5. Finally, we have the  $x_2$  profile, but from behind.

$$\bar{p}_{NW1} = \frac{1}{\pi} \left( \int_{\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) dx_2 + \int_0^{\theta - \frac{\pi}{2}} r - r \cos(-x_4 + \theta) dx_4 \right. \\ \left. + \int_{\theta - \frac{\pi}{2}}^{\frac{\pi}{2}} r dx_4 + \int_{\frac{\pi}{2}}^{\theta} r - r \cos(x_4) dx_4 + \int_{\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) dx_2 \right) \quad (\text{A.15})$$

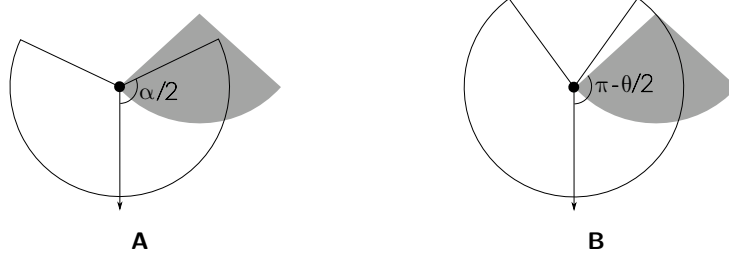
$$\bar{p}_{NW1} = \frac{r}{\pi} (\theta + 2) \quad (\text{A.16})$$

### A.2.7 Models NW2–4

The models NW2–4 have the five potential profiles in NW1 but not all profiles occur in each model, and the angle at which transitions occur are different. Furthermore, there is one extra profile possible.

1. When approaching the sensor from behind, there is a period where the profile is  $r$  wide as in NW1 profile (3).
2. At some point after profile (1) animals to the right of the sensor can be detected again. If this occurs in the  $x_4$  region, the profile width becomes  $r - r \cos(x_4)$  as in NW1.
3. However, as  $\alpha$  is now less than  $2\pi$ , animals to the right of the sensor may be undetectable until we are in the second  $x_2$  region. In this case, when we first enter the second  $x_2$  region, the profile has a width of  $r \cos(x_2 - \theta/2)$ . This occurs only if  $\alpha \leq 3\pi - 2\theta$ . This inequality is found by noting that





**Figure A.5** Profile sizes when an animal approaches from behind in models NW2–4. If  $\alpha$  is relatively large, animals can be detected when approaching from behind. Otherwise animals cannot be detected. The sector shaped detection region is shown in grey. Animals are filled black circles and the animal signal is an unfilled sector. The animals direction of movement is indicated with an arrow. (a) If  $\alpha/2$  is less than  $\pi - \theta/2$ , as is the case here, then the width of the profile when an animal approaches directly from behind is zero. (b) If  $\alpha/2 > \pi - \theta/2$  the profile width from behind is  $2r \sin(\theta/2) \sin(x_2)$ .

animals to the right of the sensor can be detected again at  $x_4 = 3\pi/2 - \alpha$  but the  $x_2$  region starts at  $x_4 = \theta$ . The new profile in  $x_2$  will only occur if  $\theta < 3\pi/2 - \alpha/2$  which is rearranged to find the inequality above. This defines the boundary between NW2 and NW3.

4. As  $\alpha \leq 2\pi$  it is possible that when the angle of approach is from directly behind the sensor the animal will not be detected at all. This is the case if  $\alpha/2 \leq \pi - \theta/2$  (Figure A.5). This inequality (simplified as  $\alpha \leq 2\pi - \theta$ ) defines the boundary between NW3 and NW4.

#### A.2.7.1 Model NW2

NW2 is bounded by  $\alpha \geq 3\pi - 2\theta$ ,  $\alpha \leq 2\pi$  and  $\theta \leq \pi$  (Figure 5.2).

NW2 has all five profiles as found in NW1. However, the change from the  $r$  profile (third integral) to the  $r - r \cos(x_4)$  profile (fourth integral) occurs at  $x_4 = 3\pi/2 - \alpha/2$  instead of at  $x_4 = \theta$ .

$$\bar{p}_{\text{NW2}} = \frac{1}{\pi} \left( \int_{\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) \, dx_2 + \int_0^{\theta - \frac{\pi}{2}} r - r \cos(-x_4 + \theta) \, dx_4 \right)$$

$$+ \int_{\theta - \frac{\pi}{2}}^{\frac{3\pi}{2} - \frac{\alpha}{2}} r \, dx_4 + \int_{\frac{3\pi}{2} - \frac{\alpha}{2}}^{\theta} r - r \cos(x_4) \, dx_4 + \int_{\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) \, dx_2 \Bigg) \quad (\text{A.17})$$

$$\bar{p}_{\text{NW2}} = \frac{r}{\pi} \left( \theta - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.18})$$

### A.2.7.2 Model NW3

NW3 is bounded by  $\alpha \leq 3\pi - 2\theta$ ,  $\alpha \geq 2\pi - \theta$  and  $\theta \geq \pi/2$  (Figure 5.2).

NW3 does not have the fourth integral from NW2 as animals are not detectable to the right of the sensor until after the  $x_4$  region has ended and the  $x_2$  region has begun. Therefore the second  $x_4$  integral has an upper limit of  $\theta$  and the profile after has a width of  $r \cos(x_2 - \theta/2)$  and is integrated with respect to  $x_2$ . The final integral starts at  $x_4 = 3\pi/2 - \alpha/2 - \theta/2$  and has the full width of  $2r \sin(x_2) \sin(\theta/2)$ .

$$\begin{aligned} \bar{p}_{\text{NW3}} = \frac{1}{\pi} \Bigg( & \int_{\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) \, dx_2 + \int_0^{\theta - \frac{\pi}{2}} r - r \cos(-x_4 + \theta) \, dx_4 + \int_{\theta - \frac{\pi}{2}}^{\theta} r \, dx_4 \\ & + \int_{\frac{\theta}{2}}^{\frac{3\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}} r \cos\left(\frac{\theta}{2} - x_2\right) \, dx_2 + \int_{\frac{3\pi}{2} - \frac{\theta}{2} - \frac{\alpha}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) \, dx_2 \Bigg) \end{aligned} \quad (\text{A.19})$$

$$\bar{p}_{\text{NW3}} = \frac{r}{\pi} \left( \theta - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.20})$$

### A.2.7.3 Model NW4

Finally, NW4 is bounded by  $\alpha \geq \pi$ ,  $\theta \geq \pi/2$  and  $\alpha \leq 2\pi - \theta$  (Figure 5.2). NW4 is the same as NW3 except that the final profile width is zero and this profile is reached at  $\alpha/2 + \theta/2 - \pi/2$ .

$$\begin{aligned} \bar{p}_{\text{NW4}} = \frac{1}{\pi} \Bigg( & \int_{\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) \, dx_2 + \int_0^{\theta - \frac{\pi}{2}} r - r \cos(-x_4 + \theta) \, dx_4 \\ & + \int_{\theta - \frac{\pi}{2}}^{\theta} r \, dx_4 + \int_{\frac{\theta}{2}}^{\frac{\alpha}{2} + \frac{\theta}{2} - \frac{\pi}{2}} r \cos\left(\frac{\theta}{2} - x_2\right) \, dx_2 \Bigg) \end{aligned} \quad (\text{A.21})$$

$$\bar{p}_{\text{NW4}} = \frac{r}{\pi} \left( \theta - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.22})$$

### A.2.8 Model REM

REM is the model from (Rowcliffe et al. 2008). It has  $\alpha = 2\pi$  and  $\theta \leq \pi/2$  (Figure 5.2). It has three profile widths, two of which are repeated, once as the animal approaches from in front of the sensor and once as the animal approaches from behind the sensor.

1. Starting with an approach direction of directly towards the sensor, and examining focal angle  $x_2$ , the profile width is  $2r \sin(x_2) \sin(\theta/2)$ .
2. When the profile is perpendicular to the radius on the right hand of the sector sensor region, we instead examine  $x_3$  where the profile width is  $r \sin(x_3)$ .
3. At  $x_3 = \pi/2$  the profile becomes simply  $r$  and this continues for  $\theta$  radians of  $x_4$ .
4. The  $x_3$  profile is then repeated with an approach direction from behind the sensor.
5. Finally the  $x_2$  profile is repeated, again with an approach direction from behind the sensor.

$$\bar{p}_{\text{REM}} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2}-\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) \, dx_2 + \int_{\theta}^{\frac{\pi}{2}} r \sin(x_3) \, dx_3 \right. \\ \left. + \int_0^{\theta} r \, dx_4 + \int_{\theta}^{\frac{\pi}{2}} r \sin(x_3) \, dx_3 + \int_{\frac{\pi}{2}-\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) \, dx_2 \right) \quad (\text{A.23})$$

$$\bar{p}_{\text{REM}} = \frac{r}{\pi} (\theta + 2) \quad (\text{A.24})$$

### A.2.9 Models NW5–7

In the models NW5–7, the sensor has  $\theta \leq \pi/2$  as in the REM. As  $\alpha \geq \pi$  a lot of the profiles are similar to the REM. Specifically, the first three profiles are always the same as the first three profiles of the REM. This is because when an animal is moving towards the sensor, the  $\alpha \geq \pi$  signal is no different to a  $2\pi$  signal. However, when approaching the sensor from behind, things are slightly different. The animal can only be detected by the sensor if the signal width is large enough that it can be detected once it has passed the sensor.

1. Starting with an approach direction of directly towards the sensor, and examining focal angle  $x_2$ , the profile width is  $2r \sin(x_2) \sin(\theta/2)$ .
2. When the profile is perpendicular to the radius edge of the sector sensor region, we instead examine  $x_3$  where the profile width is  $r \sin(x_3)$ .
3. At  $x_3 = \pi/2$  the profile becomes simply  $r$  and this continues for  $\theta$  radians of  $x_4$ .
4. If  $\alpha \leq 2\pi + 2\theta$ , the animal becomes undetectable during this profile when  $x_3$  has decreased in size to  $\pi - \alpha/2$ . This inequality marks the boundary between NW7 and NW6.
5. If instead  $\alpha \geq 2\pi + 2\theta$  then the animal does not become undetectable during the  $x_3$  focal angle. Instead the profile has width greater than zero for the whole of the  $x_3$  angle. The  $x_2$  profile starts with width  $r \cos(x_2 - \theta/2)$  as only animals approaching to the left of the sensor are detectable.
6. During this second  $x_2$  profile the signal width needed for animals to be detected to the left of the detector is increasing while the angle needed for animals to be detected to the right of the detector is decreasing. Therefore, either the left side becomes undetectable, making both sides undetectable (this occurs if  $\alpha \leq 2\pi - \theta$  as in NW6)
7. or the right becomes detectable (if  $\alpha \geq 2\pi - \theta$  as in NW5), making both sides detectable and giving a profile width of  $2r \sin(x_2) \sin(\theta/2)$ .

#### A.2.9.1 Model NW5

NW5 is bounded by  $\alpha \geq 2\pi - \theta$ ,  $\alpha \leq 2\pi$  and  $\theta \leq \pi/2$  (Figure 5.2).

It is the same as REM except that it includes the extra profile in  $x_2$  (the fifth integral) where only animals approaching to the left of the profile are detected.

$$\begin{aligned} \bar{p}_{NW5} = \frac{1}{\pi} & \left( \int_{\frac{\pi}{2}-\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) dx_2 + \int_{\theta}^{\frac{\pi}{2}} r \sin(x_3) dx_3 + \int_0^{\theta} r dx_4 \right. \\ & + \int_{\theta}^{\frac{\pi}{2}} r \sin(x_3) dx_3 + \int_{\frac{\pi}{2}-\frac{\theta}{2}}^{\frac{3\pi}{2}-\frac{\theta}{2}-\frac{\alpha}{2}} r \cos\left(\frac{\theta}{2}-x_2\right) dx_2 \\ & \left. + \int_{\frac{3\pi}{2}-\frac{\theta}{2}-\frac{\alpha}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) dx_2 \right) \end{aligned} \quad (A.25)$$

$$\bar{p}_{NW5} = \frac{r}{\pi} \left( \theta - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (A.26)$$

#### A.2.9.2 Model NW6

NW6 is bounded by  $\alpha \leq 2\pi - \theta$ ,  $\alpha \geq 2\pi + 2\theta$  and  $\theta \leq \pi/2$  (Figure 5.2).

NW6 is the same NW5 except that as  $\alpha \leq 2\pi - \theta$ , animals that approach from directly behind the detector are not detected. Therefore at  $x_2 = \alpha/2 + \theta/2 - \pi/2$  the profile width goes to zero and therefore the last integral in NW5 is not included.

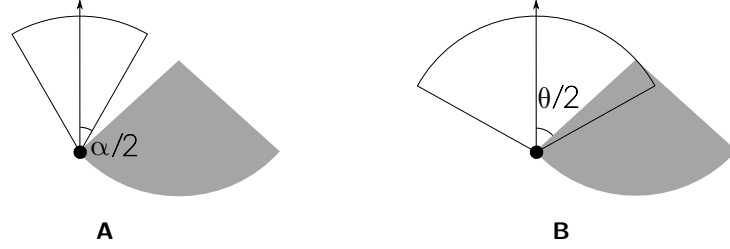
$$\begin{aligned} \bar{p}_{NW6} = \frac{1}{\pi} & \left( \int_{\frac{\pi}{2}-\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) dx_2 + \int_{\theta}^{\frac{\pi}{2}} r \sin(x_3) dx_3 \right. \\ & \left. + \int_0^{\theta} r dx_4 + \int_{\theta}^{\frac{\pi}{2}} r \sin(x_3) dx_3 + \int_{\frac{\pi}{2}-\frac{\theta}{2}}^{\frac{\alpha}{2}+\frac{\theta}{2}-\frac{\pi}{2}} r \cos\left(\frac{\theta}{2}-x_2\right) dx_2 \right) \end{aligned} \quad (A.27)$$

$$\bar{p}_{NW6} = \frac{r}{\pi} \left( \theta - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (A.28)$$

#### A.2.9.3 Model NW7

NW7 is bounded by  $\alpha \geq 2\pi + 2\theta$ ,  $\alpha \geq \pi$  and  $\theta \geq 0$  (Figure 5.2).

It is similar to NW6 but does not include the last integral as during the  $x_3$  profile, at  $x_3 = \pi - \alpha/2$  the signal width is too small for any animals to be detected, so the profile width goes to zero.



**Figure A.6** The first profile in SW models is limited by either  $\alpha$  or  $\beta$  depending on whether  $\alpha < \beta$ . The sector shaped detection region is shown in grey. Animals are filled black circles and the animal signal is an unfilled sector. The animals direction of movement is indicated with an arrow. (a) As  $\alpha/2 < \theta/2$  the profile width is limited by the signal width rather than the sensor region. The profile width is  $2r \sin(\alpha/2)$  (b) As  $\alpha/2 > \theta/2$  the profile width is limited by the sensor region, not the signal width. The profile width is  $2r \sin(\theta/2) \sin(x_2)$ .

$$\bar{p}_{NW7} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2}-\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) dx_2 + \int_{\theta}^{\frac{\pi}{2}} r \sin(x_3) dx_3 + \int_0^{\theta} r dx_4 + \int_{\pi-\frac{\alpha}{2}}^{\frac{\pi}{2}} r \sin(x_3) dx_3 \right) \quad (\text{A.29})$$

$$\bar{p}_{NW7} = \frac{r}{\pi} \left( \theta - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.30})$$

### A.2.10 Model SW1–3

The models in SW1–3 are described with the two focal angles used in models NW2–4,  $x_2$  and  $x_4$ . As  $\alpha \leq \pi$  an animal can never be detected if it is approaching the detector from behind. This makes these models simpler in that they go through the  $x_2$  and  $x_4$  profiles only once each.

There are five potential profile sizes.

1. At the beginning of  $x_2$ , with an approach direction directly towards the sensor, the parameter that limits the width of the profile can either be the sensor width, in which case the profile width is  $2r \sin(\theta/2) \sin(x_2)$ .

2. Or the signal width can be the limiting parameter, in which case the profile width is instead  $2r \sin(\alpha/2)$  (Figure A.6)
3. The next potential profile in  $x_2$  has a width of  $r \sin(\alpha/2) - r \cos(x_2 + \theta/2)$  as the right side of the profile is limited by the width of the sensor region while the left side is limited by the signal width. However, the angle at which the profile starts depends on whether the first profile was 1) or 2) above. If the first profile is profile 1) then the profile is limited on both sides by the sensor region and then the left side of the profile becomes limited by the signal width. This happens at  $x_2 = \pi/2 - \alpha/2 + \theta/2$ . If however the first profile was 2) then the first profile is limited by the signal width. We move into the new profile when the right side of the profile becomes limited by the sensor region. This occurs at  $x_2 = \pi/2 + \alpha/2 - \theta/2$ .
4. In the  $x_4$  region the left side of the profile is always  $r \sin(\alpha/2)$  while the right side is either 0, giving a profile of  $r \sin(\alpha/2)$ .
5. Or limited by the sensor giving a profile of size  $r \sin(\alpha/2) - r \cos(x_4 - \theta)$ .

#### A.2.10.1 Model SW1

SW1 is bounded by  $\alpha \geq \theta$ ,  $\alpha \leq \pi$  and  $\theta \leq \pi$  (Figure 5.2).

As  $\alpha$  is large the first profile is limited by the size of the sensor region giving it a width of  $2r \sin(\theta/2) \sin(x_2)$ . It is the only one of the three SW models to start in this way. Later on, still with  $x_2$  as the focal angle the left side of the profile does become limited by the signal width. So at  $x_2 = \pi/2 - \alpha/2 + \theta/2$  the profile width becomes  $r \sin(\alpha/2) - r \cos(x_2 + \theta/2)$ .

As we enter the  $x_4$  region, the profile remains limited by the signal on the left and by the sensor on the right, giving a profile width of  $r \sin(\alpha/2) - r \cos(x_4 - \theta)$ . Finally, at  $x_4 = \theta - \pi/2$  the right side of the profile becomes zero and the profile is width is  $r \sin(\alpha/2)$ .

$$\bar{p}_{\text{SW1}} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) dx_2 + \int_{\frac{\theta}{2}}^{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}} r \sin\left(\frac{\alpha}{2}\right) - r \cos\left(\frac{\theta}{2} + x_2\right) dx_2 \right. \\ \left. + \int_0^{\theta - \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) - r \cos(\theta - x_4) dx_4 + \int_{\theta - \frac{\pi}{2}}^{\frac{\alpha}{2} + \theta - \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) dx_4 \right) \quad (\text{A.31})$$

$$\bar{p}_{\text{SW1}} = \frac{r}{\pi} \left( \theta \sin\left(\frac{\alpha}{2}\right) - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.32})$$

### A.2.10.2 Model SW2

SW2 is bounded by  $\theta \geq \pi/2$ ,  $\alpha \leq \theta$  and  $\alpha \geq 2\theta - \pi$  (Figure 5.2).

SW2 is largely similar to SW1. However, as  $\alpha \leq \theta$  the first profile is limited by  $\alpha$  and not by the detection region. Therefore the first profile has width  $2r \sin(\alpha/2)$ . This also means the transition to the second profile occurs at  $x_2 = \pi/2 + \alpha/2 - \theta/2$  instead of  $x_2 = \pi/2 - \alpha/2 + \theta/2$ .

$$\begin{aligned} \bar{p}_{\text{SW2}} = \frac{1}{\pi} & \left( \int_{\frac{\alpha}{2} - \frac{\theta}{2} + \frac{\pi}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\alpha}{2}\right) dx_2 + \int_{\frac{\theta}{2}}^{\frac{\alpha}{2} - \frac{\theta}{2} + \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) - r \cos\left(\frac{\theta}{2} + x_2\right) dx_2 \right. \\ & \left. + \int_0^{\theta - \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) - r \cos(\theta - x_4) dx_4 + \int_{\theta - \frac{\pi}{2}}^{\frac{\alpha}{2} + \theta - \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) dx_4 \right) \quad (\text{A.33}) \end{aligned}$$

$$\bar{p}_{\text{SW2}} = \frac{r}{\pi} \left( \theta \sin\left(\frac{\alpha}{2}\right) - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.34})$$

### A.2.10.3 Model SW3

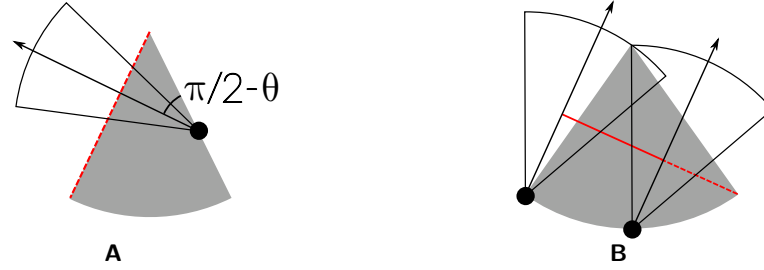
SW3 is bounded by  $\alpha \leq 2\theta - \pi$  and  $\theta \leq \pi$  (Figure 5.2).

SW3 is similar to SW2 except that the profile does not become limited by sensor at all during the the  $x_4$  regions. Therefore, at  $x_4 = 0$  the profile is still of width  $2r \sin(\alpha/2)$ . Only at  $x_4 = \theta - \pi/2 - \alpha/2$  does the profile become limited on the right by the sensor region.

$$\begin{aligned} \bar{p}_{\text{SW3}} = \frac{1}{\pi} & \left( \int_{\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\alpha}{2}\right) dx_2 + \int_0^{-\frac{\pi}{2} + \theta - \frac{\alpha}{2}} 2r \sin\left(\frac{\alpha}{2}\right) dx_4 \right. \\ & \left. + \int_{-\frac{\pi}{2} + \theta - \frac{\alpha}{2}}^{\theta - \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) - r \cos(\theta - x_4) dx_4 + \int_{\theta - \frac{\pi}{2}}^{\frac{\alpha}{2} + \theta - \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) dx_4 \right) \quad (\text{A.35}) \end{aligned}$$

$$\bar{p}_{\text{SW3}} = \frac{r}{\pi} \left( \theta \sin\left(\frac{\alpha}{2}\right) - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.36})$$





**Figure A.7** Description of two profiles in SW models. The sector shaped detection region is shown in grey. Animals are filled black circles and the animal signal is an unfilled sector. The animals direction of movement is indicated with an arrow. The profile  $p$  is shown with a red line. Dashed red lines indicate areas where animals cannot be detected. (a) At  $x_4 = 0$ , if  $\alpha/2 < \pi/2 - \theta$  then  $\alpha/2$  is too small for an animal to be detected at all during the  $x_4$  profile (shown with dashed red). This inequality simplifies to  $\alpha < \pi - 2\theta$ . (b) The right of the profile is limited by the signal width, not the sensor. On the left, the profile is limited by the sensor and not the signal. Overall the profile width is  $r \sin(\alpha/2) - r \cos(x_2 + \theta/2)$ .

### A.2.11 Model SW4–9

As  $\alpha < \pi$ , animals approaching the sensor from behind can never be detected, so unlike REM, the second  $x_2$  and  $x_3$  profiles are always zero. The six models are split by three inequalities that relate to the models as follows.

1. Models with  $\alpha \leq \pi - 2\theta$  have no  $x_4$  profile. This is because at  $x_4 = 0$ , the signal width is already too small to be detected as can be seen in Figure A.7A where  $\alpha/2 < \pi/2 - \theta$  which simplifies to give the previous inequality.
2. Models with  $\alpha \leq \theta$  are limited by  $\alpha$  in the first,  $x_2$  region (Figure A.6), rather than being limited by  $\theta$ . Therefore this first profile is of width  $2r \sin(\alpha/2)$  rather than  $2r \sin(\theta/2) \sin(x_2)$ .
3. Finally, models with  $\alpha \leq 2\theta$  have a second profile in  $x_2$  where to one side of the sensor  $\alpha$  is the limiting factor of profile width, while on the other side  $\theta$  is (Figure A.7B). This gives a width of  $r \sin(\alpha/2) - r \cos(x_2 + \theta/2)$ . This profile does not occur in models with  $\alpha \geq 2\theta$ .

## A.2.11.1 Model SW4

SW4 is bounded by  $\alpha \leq \theta$ ,  $\alpha \geq \pi - 2\theta$  and  $\theta \leq \pi/2$  (Figure 5.2). Therefore it does contain a  $x_4$  profile, starts with an  $\alpha$  limited profile and does contain the  $r \sin(\alpha/2) - r \cos(x_2 + \theta/2)$  profile in  $x_2$ .

$$\bar{p}_{SE4} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2}}^{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}} 2r \sin\left(\frac{\alpha}{2}\right) dx_1 + \int_{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}}^{\frac{\theta}{2} + \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) + r \cos\left(\frac{\theta}{2} - x_1\right) dx_1 + \int_{\frac{\theta}{2} + \frac{\pi}{2}}^{\frac{\alpha}{2} + \frac{\theta}{2} + \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) dx_1 \right) \quad (A.37)$$

$$\bar{p}_{SE4} = \frac{r}{\pi} \left( \theta \sin\left(\frac{\alpha}{2}\right) - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (A.38)$$

## A.2.11.2 Model SW5

SW5 is the only model with a tetrahedral bounding region. It is bounded by  $\alpha \geq \theta$ ,  $\alpha \geq \pi - 2\theta$ ,  $\alpha \leq 2\theta$  and  $\theta \leq \pi/2$  (Figure 5.2). Therefore it does contain a  $x_4$  profile, but starts with a  $\theta$  limited profile. It does contain the  $r \sin(\alpha/2) - r \cos(x_2 + \theta/2)$  profile in  $x_2$ .

$$\bar{p}_{SW5} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) dx_2 + \int_{\frac{\pi}{2} - \frac{\theta}{2}}^{\frac{\pi}{2} + \frac{\theta}{2} - \frac{\alpha}{2}} r \sin\left(\frac{\alpha}{2}\right) - r \cos\left(\frac{\theta}{2} + x_2\right) dx_2 + \int_{\theta}^{\frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) dx_3 + \int_0^{\frac{\alpha}{2} + \theta - \frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) dx_4 \right) \quad (A.39)$$

$$\bar{p}_{SW5} = \frac{r}{\pi} \left( \theta \sin\left(\frac{\alpha}{2}\right) - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (A.40)$$

## A.2.11.3 Model SW6

SW6 is bounded by  $\alpha \geq \pi - 2\theta$ ,  $\alpha \geq 2\theta$  and  $\alpha \leq \pi$  (Figure 5.2). It starts with a  $\theta$  limited profile and has a  $x_4$  profile. However, it does not contain the  $r \sin(\alpha/2) - r \cos(x_2 + \theta/2)$  profile.

$$\bar{p}_{\text{SW6}} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2}-\frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) dx_2 + \int_{\theta}^{\frac{\alpha}{2}} r \sin(x_3) dx_3 \right. \\ \left. + \int_{\frac{\alpha}{2}}^{\frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) dx_3 + \int_0^{\frac{\alpha}{2}+\theta-\frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) dx_4 \right) \quad (\text{A.41})$$

$$\bar{p}_{\text{SW6}} = \frac{r}{\pi} \left( \theta \sin\left(\frac{\alpha}{2}\right) - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.42})$$

#### A.2.11.4 Model SW7

SW7 is bounded by  $\alpha \leq \pi - 2\theta$ ,  $\alpha \leq \theta$  and  $\alpha < 0$  (Figure 5.2). Therefore it does not contain a  $x_4$  profile. It starts with an  $\alpha$  limited profile and contains the  $r \sin(\alpha/2) - r \cos(x_2 + \theta/2)$  profile in  $x_2$ .

$$\bar{p}_{\text{SW7}} = \frac{1}{\pi} \left( \int_{\frac{\alpha}{2}-\frac{\theta}{2}+\frac{\pi}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\alpha}{2}\right) dx_2 + \int_{\frac{\pi}{2}-\frac{\theta}{2}}^{\frac{\alpha}{2}-\frac{\theta}{2}+\frac{\pi}{2}} r \sin\left(\frac{\alpha}{2}\right) - r \cos\left(\frac{\theta}{2} + x_2\right) dx_2 \right. \\ \left. + \int_{\theta}^{\frac{\alpha}{2}+\theta} r \sin\left(\frac{\alpha}{2}\right) dx_3 \right) \quad (\text{A.43})$$

$$\bar{p}_{\text{SW7}} = \frac{r}{\pi} \left( \theta \sin\left(\frac{\alpha}{2}\right) - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.44})$$

#### A.2.11.5 Model SW8

SW8 is bounded by  $\alpha \leq \pi - 2\theta$ ,  $\alpha \geq \theta$  and  $\alpha \leq 2\theta$  (Figure 5.2). It starts with a  $\theta$  limited profile. It does contain the  $r \sin(\alpha/2) - r \cos(x_2 + \theta/2)$  profile in  $x_2$  but does not have a  $x_4$  profile.

$$\bar{p}_{\text{SW8}} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2}+\frac{\theta}{2}-\frac{\alpha}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) dx_2 + \int_{\frac{\pi}{2}-\frac{\theta}{2}}^{\frac{\pi}{2}+\frac{\theta}{2}-\frac{\alpha}{2}} r \sin\left(\frac{\alpha}{2}\right) - r \cos\left(\frac{\theta}{2} + x_2\right) dx_2 \right. \\ \left. + \int_{\theta}^{\frac{\alpha}{2}+\theta} r \sin\left(\frac{\alpha}{2}\right) dx_3 \right) \quad (\text{A.45})$$

$$\bar{p}_{\text{SW8}} = \frac{r}{\pi} \left( \theta \sin\left(\frac{\alpha}{2}\right) - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.46})$$

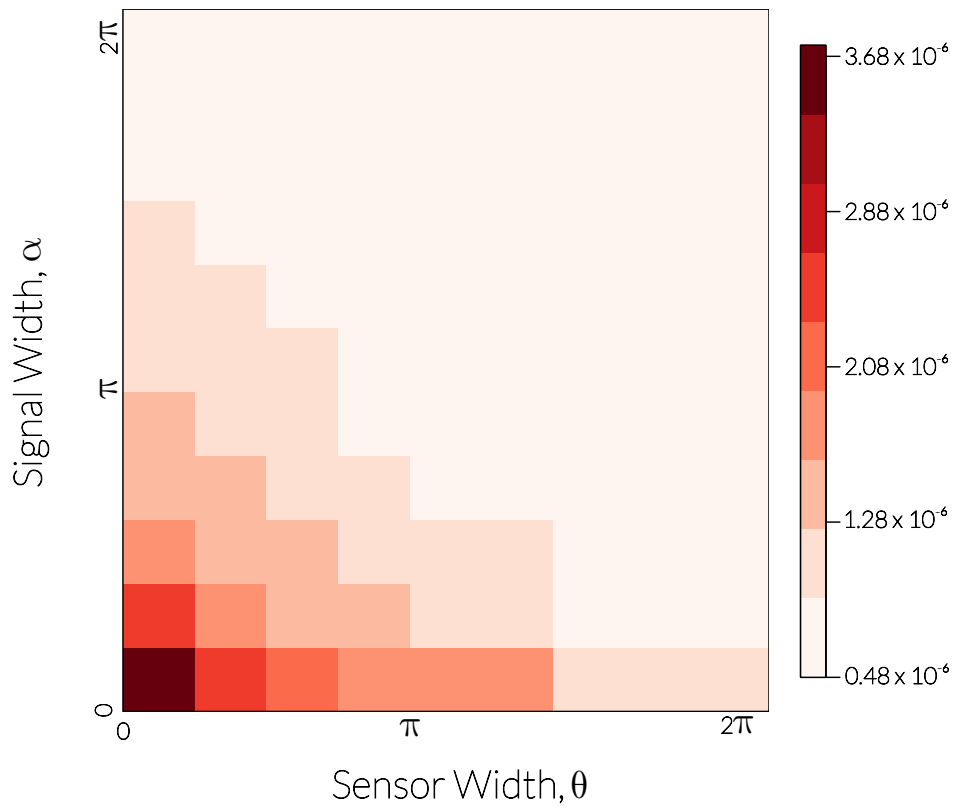
## A.2.11.6 Model SW9

Finally, SW9, the last model, is bounded by  $y \alpha \leq \pi - 2\theta$ ,  $\alpha \geq 2\theta$  and  $\theta \geq 0$  (Figure 5.2). Therefore it starts with a  $\theta$  limited profile. However it does not contain the extra  $x_2$  profile nor a  $x_4$  profile.

$$\bar{p}_{\text{SW9}} = \frac{1}{\pi} \left( \int_{\frac{\pi}{2} - \frac{\theta}{2}}^{\frac{\pi}{2}} 2r \sin\left(\frac{\theta}{2}\right) \sin(x_2) \, dx_2 + \int_{\theta}^{\frac{\alpha}{2}} r \sin(x_3) \, dx_3 + \int_{\frac{\alpha}{2}}^{\frac{\alpha}{2} + \theta} r \sin\left(\frac{\alpha}{2}\right) \, dx_3 \right) \quad (\text{A.47})$$

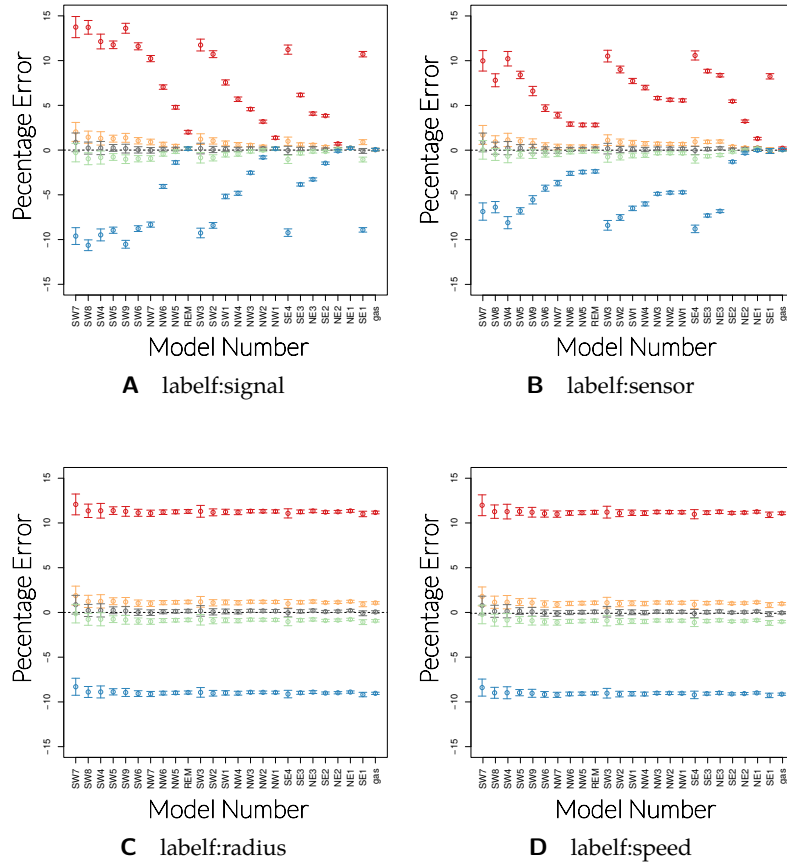
$$\bar{p}_{\text{SW9}} = \frac{r}{\pi} \left( \theta \sin\left(\frac{\alpha}{2}\right) - \cos\left(\frac{\alpha}{2}\right) + 1 \right) \quad (\text{A.48})$$

### A.3 Supplementary Information: Simulation model results of the gREM precision



**Figure A.1** Simulation model results of the gREM precision given a range of sensor and signal widths, shown by the standard deviation of the error between the estimated and true densities. Standard deviations are shown from deep red to pink, representing high to low values between  $0.483 \times 10^{-6}$  to  $3.74 \times 10^{-6}$ .

## A.4 Supplementary Information: Impact of parameter error



**Figure A.2** Model sensitivity (for all gREM submodels) to error in estimates of a) signal width  $\alpha$ , b) sensor width  $\theta$ , c) detection distance  $r$  and d) animal movement speed  $v$ . Estimates are -10% (red), -1% (orange), 0% (grey), +1% (green) and +10% (blue) of the true parameter value. The black dashed line indicates zero error in density estimates. The error bars 95% confidence intervals across all simulations.

## B Colophon

This thesis was set using  $\text{\LaTeX}$ ,  $\text{\XeLaTeX}$  and  $\text{\BibLaTeX}$ . The formatting is defined by the `phdthesis` class by Robert Stanley. The TeX Gyre Pagella typeface is used in the main text while Lato Light and **Lato Black** are used in the figures. Chapters 2, 3 and 4 are entirely reproducible knitr documents (Xie 2015). Code for the simulations in Chapter 5 is not combined into a knitr document but code for creating figures is. All code will be made available on Github at [. Plots were created with a combination of Inkscape, ggplot2 \(Wickham 2009\), palettetown \(Lucas 2015\), ggtree \(Yu 2015\) and base R \(R Development Core Team 2010\). References were handled with JabRef \(JabRef Development Team 2015\).](#)

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