THESIS INTRODUCTION

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1. Discussion

2. 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 (Leroy et al. 2005; Field et al. 2001; Halpin et al. 2011; Li et al. 2005; Field et al. 2001). 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 be able to also 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 stuctured population did not recover the same relationship implying that this mechanism is not important in wild populations. Subsequently, I examined a number of intrinsically linked factors—population abundance, 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 abundances from acoustic data; as bat abundances are very difficult to estimate, this method fills a great need in bat ecology and zoonotic sirveillance.

2.1. **How I did it (Chapters overview).** In Chapter **??** I tested the hypothesis that bat species with more structured populations harbour more virus species. I test this hypothesis with two measurement of population structure: the number of subspecies (a novel measure and the largest dataset yet used to test this hypothesis) and gene flow. Using both meausres 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 (Kamiya et al. 2014; Arneberg 2002; Gay et al. 2014; Nunn et al. 2003; Turmelle & Olival 2009).

While the results from Chapter **??** suggest that there is a relationship between population structure and pathogen richness, comparative studies like these cannot identify by which specific mechanisms the higher pathogen richness is being maintained. To examine this I developed a model of two recently diverged—and therefore identical—pathogen lineages competing in a metapopulation based on large bat colonies with limited movement between colonies (Chapter **??**). 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 it's spread, it would then survive and

spread throughout the metapopulation irregardless of how structured it was. This imples 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 **??**.

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 (Kamiya et al. 2014; Nunn et al. 2003; Morand & Poulin 1998; Lindenfors et al. 2007; Gay et al. 2014; Ezenwa et al. 2006) yet the intrinsic relationships between these variables are rarely acknowledged or discussed. Therefore in Chapter **??** I used the same model as Chapter **??** to test whether population density or population abundance more strongly promotes pathogen richness and whether a pathogen invades more easily into a population comprising many small colonies or few big colonies. I found that population abundance has a much stronger affect than density and that the component of abundance that has the strongest affect is colony size.

Theory (May & Anderson 1979; Anderson & May 1979), previous literature (Kamiya et al. 2014; Nunn et al. 2003; Morand & Poulin 1998) and Chapters **??** and **??** suggested that population sizes (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 abundance for bats and colony counts are time consuming and costly. I therefore aimed to obtain estimates of abundance from acoustic data such as iBats (Jones et al. 2011). I developed a general method for estimating abundance and density from acoustic detectors (Chapter **??**). I used spatial simulations of animal movement to validate the method and found it to be precise and unbiased.

3. 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 affect 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 (Anthony et al. 2013; Anthony et al. 2015) or using much larger pathogen data sets (Wardeh et al. 2015) predictive models may become a more viable tool. Furthermore, the method provided in Chapter **??** makes the collection of population abundance data more feasible over broad taxonomic, spatial or temporal scales, further increasing the potential of predictive models. Field tests should test its ability to estimate density and abundance 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 (Lehmann et al. 2010; Zunino et al. 2007; Manor & Saltz 2003; Atwood 2006) 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 abundance (Craigie et al. 2010) are expected to experience smaller changes in pathogen richness despite these being the more commonly studied factors. This suggests that further research should study in more detail the affects of climate change on social group size.

Furthermore, I have shown that while population factors such as density, abundance and range size are directly linked, they have very different affects on pathogen richness. Therefore future studies should be careful to acknowledge these relationships and where

data makes it possible, compare multiple demographic measurements to further test which factors are in fact causally affecting pathogen richness.

3.1. **What agreed/disagreed with the literature.** There is a common assumption that factors that increase R_0 should increase pathogen diversity (Nunn et al. 2003; Morand 2000). However, my results imply a more nuanced relationship. I found that populations with large groups 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 affect of population structure and that in wild bat populations, population structure promotes global 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 **??**, Turmelle & Olival (2009) and Maganga et al. (2014)) and range size (Kamiya et al. 2014; Nunn et al. 2003) than group size (Rifkin et al. 2012; Ezenwa et al. 2006). This highlights the distinction between factors that promote the addition of new pathogens to the community and those factors that instead allow a larger overall number of pathogens 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.

Much research in multipathogen systems has been conducted over the short time scales of a single epidemic (van de Bovenkamp et al. 2014; Poletto et al. 2013; Poletto et al. 2015; Funk & Jansen 2010). 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 seem to be often 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 **??** and **??**). This can be understood by considering that at very long time scale, any population is well mixed unless there is complete seperation of subpopulations.

3.2. **Furtherwork.** Colony size has been found to be 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 **??** I found that colony size is particularly important for promoting pathogen richness. I did not include colony size in my comparative analysis (Chapter **??**) for three reasons: the focus of the chapter was broader scale population structure, the lack of evidence of a positive relationship (Gay et al. 2014; Turmelle & Olival 2009) and the lack of data. However, given the results of Chapter **??** filling these data gaps would be a useful avenue for further research. In particularly, testing the relative affects of colony size, population structure and range size would be a useful test of the model used in Chapter **??**.

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 new pathogen, identical to an endemic pathogen and in the presence of strong competition) to invade an persist. However, there are a number of other mechanisms that could equally strongly affect pathogen richness in the wild. Close related to the mechanism 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. This case has been studied in well-mixed populations TODO.

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 occasionaly extinctions, especially in the presence of interpathogen competition. A number of population factors could affect this rate. It is expected that large populations will experience slower rates of pathogen extinction as a stochastic changing number of infections is less likely to drop to zero. Furthermore, populations that support stable levels of infection are likely to have a lower rate of pathogen extinction. This includes populations where epidemic cycles are common. This affect will be exacerbated in the case where an epidemic cycle is synchronous across the whole population. 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; Levin 1974; Hanski 1998), but less so in the epidemiological literature.

- Collect data for colony size and test importance against structure.
- Limitations of poor data, escpecially for gen flow.
- bias of viral data. Perhaps fixed by non-biased sampling or larger datasets.
- Examine other mechanisms for richness
- Examine multi host species more carefully
- Field test gREM
- Use gREM to collect density estimates

3.3. **Conclusions.**

- Population structure does influence pathogen richness but the mechanisms are still unclear.
- Local dynamics (local density) are most important for pathogen invasions not broad scale structure.
- Data on density should be collected using the gREM.

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