

# THESIS INTRODUCTION

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## 1. ABSTRACT

**ABSTRACT.** Pathogens make up a huge proportion of global diversity and their role in disease strongly affects human disease, economics and development as well as having an important ecological role. However, the factors that control the number of pathogen species are poorly understood.

The patterns of contacts between individuals — in both human and animal populations — are nonrandom and depend on the density of individuals. Population structure and density have important epidemiological consequences, but their role in the control of pathogen richness is unknown.

It is still unknown whether population structure and density controls pathogen richness in bats. Furthermore, the specific mechanisms by which structure might influence pathogen richness have not been studied.

In this thesis I have studied how population structure and density control pathogen richness using bats as a case study. I have used epidemiological simulations and comparative analyses of data from wild bat populations to show that population structure does not have a strong affect on pathogen richness. Using further simulations I have shown that the interaction between population density and population structure is an important consideration. Finally, I have created a model for estimating bat densities — previously an incredibly challenging task — using acoustic data. Together, these studies clarify the relative roles of population density and structure and facilitate further study of population density in bats as a driver of pathogen richness.

While theory previously predicted that population structure should increase pathogen richness, the expectation in the ecological literature was that population structure would decrease pathogen richness. My studies support neither of these views, instead suggesting that population structure does not have a strong affect in either direction.

## 2. INTRODUCTION

### 3. PATHOGEN RICHNESS AND THE IMPACTS OF ZOO NOTIC DISEASES

The diversity of pathogens is huge and largely unknown (Poulin 2014). Recent large studies have found tens (Anthony et al. 2013) or even hundreds of virus species in a single host species (Anthony et al. 2015). This suggests the number of mammalian viruses globally is of the order of hundreds of thousands of virus species (Anthony et al. 2013) while only 3,000 species of virus, 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 nearly 4,000 named mammal species (Wilson & Reeder 2005), the undiscovered diversity of pathogens is likely huge.

In general we can expect competition between pathogens. 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 (Ackleh & Allen 2003, Ackleh & Salceanu 2014, Bremermann & Thieme 1989, Martcheva & Li 2013, Turner & Garnett 2002) so the diversity of parasites needs an explanation.

This diversity of pathogens presents a risk to human health. 60% of newly emerged disease 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: viruses such as Nipah (Luby et al. 2009), Ebola (Lefebvre et al. 2014) having case fatality rates over 50%. 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).

Surveillance of zoonotic diseases is crucial to mitigate these health impacts. In particular we want to categorise diseases before they spillover into humans (SARS was not identified until months into the pandemic for example (Drosten et al. 2003)) and we want to anticipate outbreaks. If we know there is an increased number of infections in host species, increased numbers of a species that is a known reservoir of a high risk zoonotic disease, or increased contacts between humans and a pathogen reservoir, we can prepare for a potential outbreak in that area.

However, funds for zoonotic 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 traits (body mass (Arneberg 2002, Kamiya et al. 2014, Poulin 1995) and longevity (Ezenwa et al. 2006, Nunn et al. 2003)) as well as environmental factors such as latitude (Kamiya et al. 2014, Poulin 2010). 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), population structure (Gay et al. 2014, Maganga et al. 2014, Nunes et al. 2006, Turmelle & Olival 2009) and species range size (Kamiya et al. 2014, Nunn et al. 2003)). This understanding provides a basis for predicting which species will have high pathogen richness and should be prioritised for sampling and surveillance. Furthermore, given a good mechanistic understanding of how pathogen richness is created and maintained we can start to predict how pathogen richness, and therefore zoonotic disease risk, will respond to global change.

#### 4. BATS AS RESERVOIRES OF ZOO NOTIC DISEASES

In recent decades bats have been implicated in a number of high profile zoonotic outbreaks including Nipah (Field et al. 2001), 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, 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 in general (Olival et al. 2015). Questions remain as to why bat viruses have a tendency to have such high zoonotic potential.

Many factors of bat populations make them epidemiologically interesting. Firstly, the population density of most bat species is largely unknown. As they are small, nocturnal and difficult to identify in flight, estimating there density is incredibly difficult without disruptive and time consuming roost surveys. As this parameter is tightly linked to pathogen richness (Kamiya et al. 2014) and central to epidemiological models (Anderson & May 1979, May & Anderson 1979) these leaves large gaps in our understanding of

disease processes in this group. Secondly 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).

## 5. POPULATION DENSITY AND STRUCTURE AS FACTORS THAT INFLUENCE PATHOGEN RICHNESS

The role of population density and abundance 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 (Daley & Kendall 1964, 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. Single pathogen models suggest 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. Many 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-dependant 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 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). 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.

## 6. THESIS OVERVIEW

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

First, in Chapter 2, I empirically test the hypothesis that population structure is associated with pathogen richness in wild bat populations. I used two measurements of population structure — the number of subspecies and gene flow — and a larger dataset than previous studies. For both measurements 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 a relationship between body mass and range size, finding strong support for larger bodied bats carrying more viruses and mixed support for range size promoting pathogen richness.

In Chapter 3 I examine 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 meta-populations and examine 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, at a number of different transmission rates, neither of these factors altered the probability of newly evolved pathogens invading and persisting in the population.

Next I examine the relationships between a number of elements of population structure (Chapter 4). I clarified the interdependences between range size, and population size and density. I also noted that population size can be decomposed into colony size and colony density (the number of colonies per unit area). Using the same model as 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 estimates of these for wild bat populations. However, there are currently very few measurements of bat abundance due to their small size, nocturnal habit and difficulties in identification. Therefore I aimed to develop a method for estimating bat abundance 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 abundance 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.

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