THE INTERACTIONS BETWEEN POPULATION STRUCTURE AND DENSITY IN PATHOGEN DIVERSITY

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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 particularly 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.* components of population size) promote pathogen richness more than population density. This implies that 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.

2. INTRODUCTION

Zoonotic diseases are an increasingly important source of human infectious diseases (Jones et al. 2008; Taylor et al. 2001; Woolhouse and 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 clear that population level factors such as population density, range size and population structure have an important role in controlling pathogen community dynamics (Anderson and May 1979; Colizza and Vespignani 2007; May and Anderson 1979; May and 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 and May 1979; Barthélemy et al. 2010; Colizza and Vespignani 2007; May and 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 and 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 and 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 and 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 1 for all parameters used) or that the relationship $N \propto a$ has broad empirical support (Blackburn et al. 2006; Borregaard and 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 and Olival (2009) and see Chapter **??**). 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 dependant on the distance between them (Jenkins et al. 2010; Le Galliard et al. 2012; Schooley and 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 dependant. 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
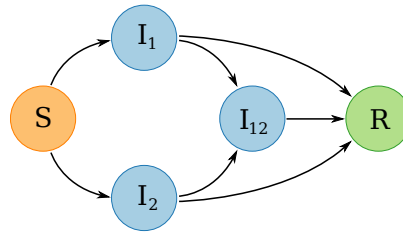
FIGURE 1. Schematic of the SIR model used. Individuals are in one five classes, susceptible (orange, S), infectious with pathogen 1, pathogen 2 or both (blue, I_1, I_2, I_{12}) or recovered and immune from further infection (green, R). Transitions between classes occurs only as indicated by arrows. Note 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.

size affects the ability of new pathogens to invade (Chapter **??**) 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; Qiu et al. 2013) and Chapter **??**) increase 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.
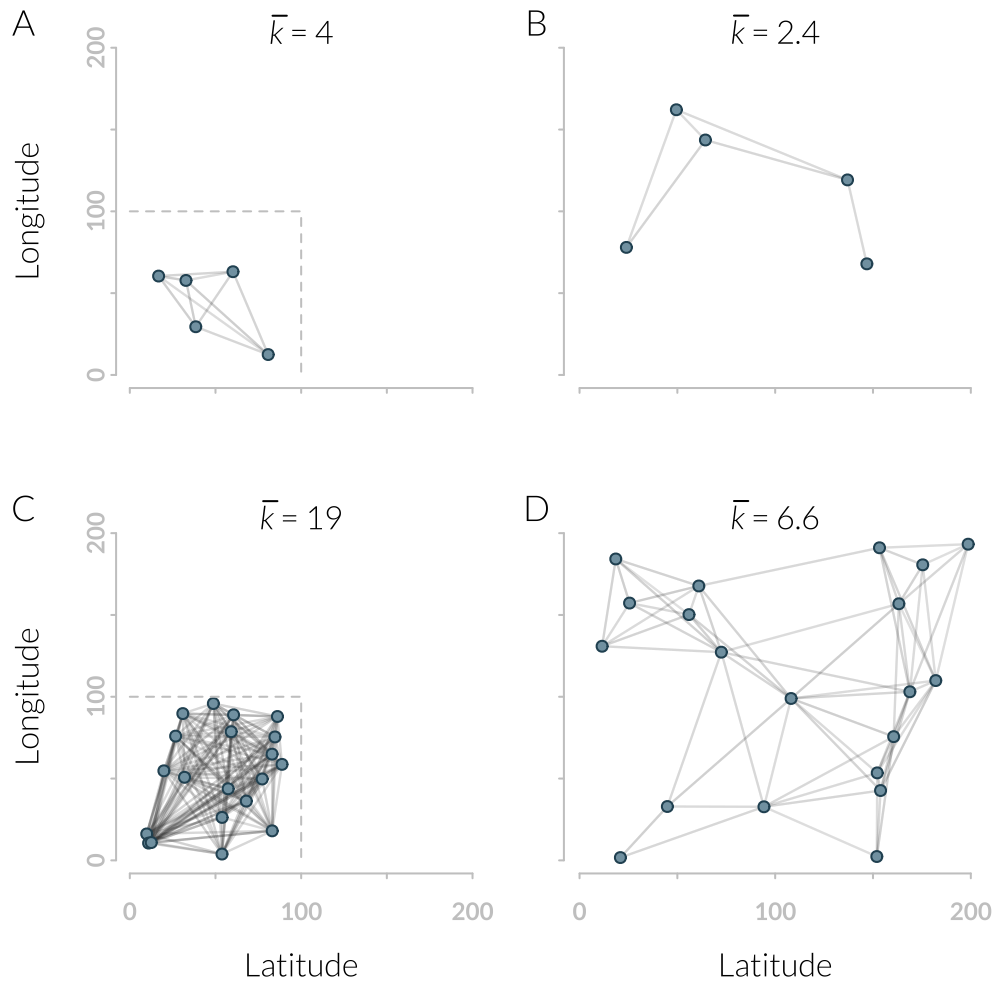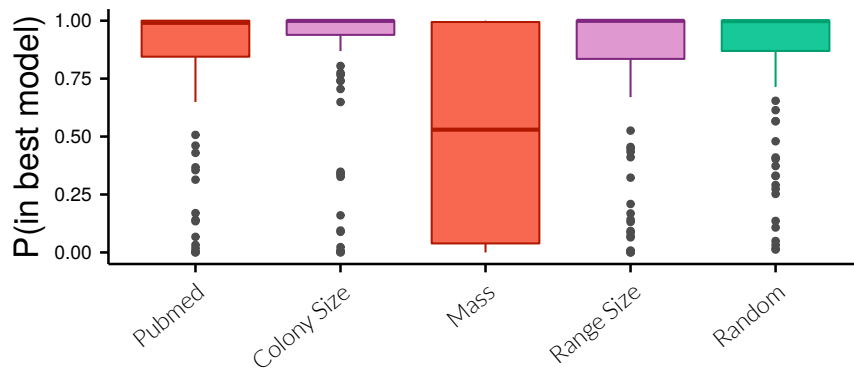
FIGURE 2. Examples of the metapopulation networks used. They include the smallest number of colonies (five, A and B) and the default (20, C and D). They also include the default area ($10^4 \, \text{km}^2$, grey dashed lines, A and C) and the largest area ($4 \times 10^4 \, \text{km}^2$, full plot, B 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, $\bar{k}$.
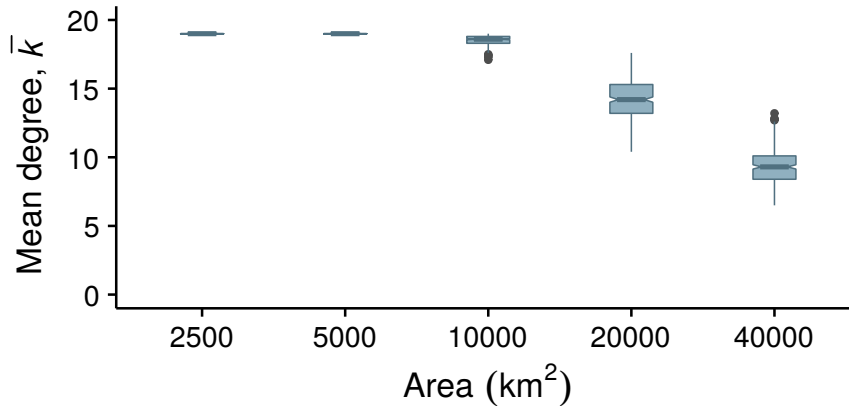
FIGURE 3. Change in average metapopulation network degree ($\bar{k}$) with increasing area. Bars show the median, boxes show the interquartile range, vertical lines show and range and grey dots indicate outlier values. Notches indicate the 95% confidence interval of the mean. All simulations had 20 colonies, meaning 19 is the maximum value of $\bar{k}$.

3. METHODS

3.1. Metapopulation model.

3.1.1. *Two pathogen SIR model.* I examine a multipathogen SIR model with vital dynamics. This is a compartment model with individuals being classed as susceptible, infected or recovered with immunity (Figure 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. 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 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 α (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, $\mu = \Lambda$ (see Table 1 for a list of symbols and values used). 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).

3.1.2. *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 ??). Individuals within a subpopulation interact randomly so that the subpopulation is fully mixed. Dispersal between subpopulations occurs at a rate λ. 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
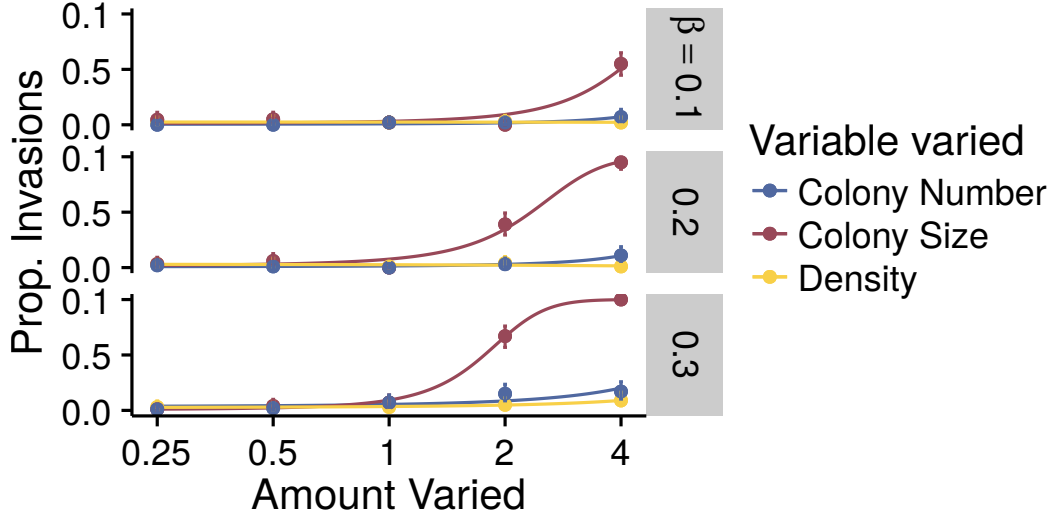
FIGURE 4. Comparison of the effect of colony size, colony number and area on probability of invasion. Default values are: colony number = 20, colony size = 400 and density = 0.8 animals per unit area. The *x*-axis shows the relative change in each of these values (×0.25, 0.5, 1, 2 and 4). For colony 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 bivariate logistic regression fits for each independant variable. Relationships are shown seperately for each transmission value.

from a subpopulation m with degree k_m to subpopulation n is $\frac{\lambda}{k_m}$. Note this rate is independent of the degree of subpopulation n.

3.1.3. *Stochastic simulations.* I examine 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 it's rate

$$p(\text{event } i) = \frac{r_i}{\sum_i r_i} \tag{1}$$

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

$$\delta \sim \text{Exp}\left(\sum_i r_i\right). \tag{2}$$

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

We can now write down the rates of all events. I defined I_p^+ to be the sum of all classes that are infectious with pathogen p, for example $I_1^+ = I_1 + I_{12}$. Assuming asexual reproduction, that all classes
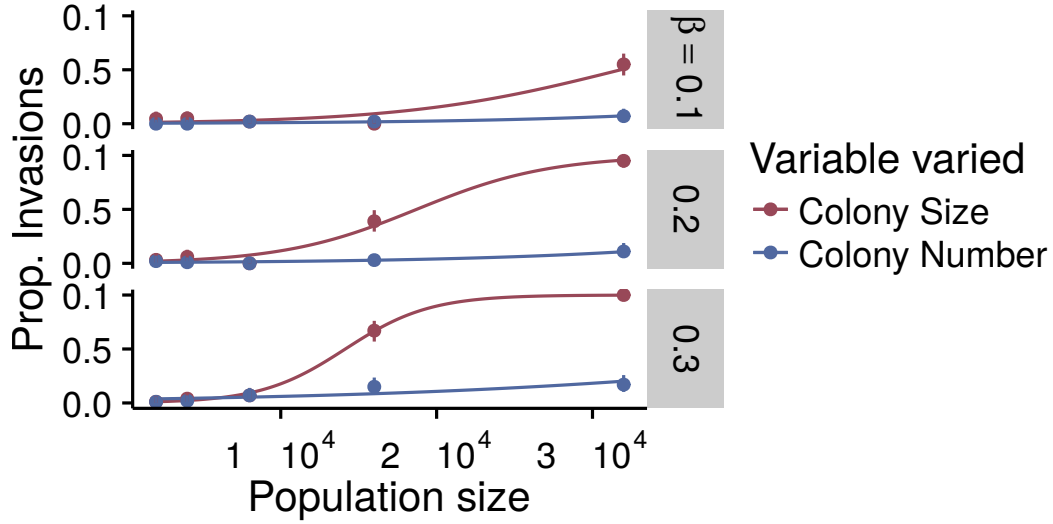
FIGURE 5. Comparison of the effect of population size on probability of invasion when population size is altered by changing colony size or colony number. Relationship is shown seperately for each transmission value. It can be seen that changes in colony size give a much greater increase in invasion probability than changes in colony number. Note that this is the same data as Figure 4 but with the x-axis scaled by population size, not parameter change.

reproduce at the same rate and that individuals are born into the susceptible class we get

$$P\left(S_{nt'} = S_{nt} + 1\right) = \Lambda\left(S_{nt} + \sum_q I_{qnt} + R_{nt}\right) \tag{3}$$

where $P\left(S_{nt'} = S_{nt} + 1\right)$ 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 d are given by

$$P\left(S_{nt'} = S_{nt} - 1\right) = \mu S_{nt}, \tag{4}$$

$$P\left(I_{qnt'} = I_{qnt} - 1\right) = \mu I_{qnt}, \tag{5}$$

$$P\left(R_{nt'} = R_{nt} - 1\right) = \mu R_{nt}. \tag{6}$$

I modelled transmission as density-dependant. This assumption was more suitable than frequency-dependant 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, $S \to I_p$ where $p \in \{1, 2\}$, is therefore given by

$$P\left(I_{pnt'} = I_{pnt} + 1, S_{nt'} = S_{nt} - 1\right) = \beta S_{nt} I_{pnt}^+, \tag{7}$$

while coinfection, given a crossimmunity factor α, is given by

$$P\left(I_{12,nt'} = I_{12,nt} + 1, I_{1nt'} = I_{1nt} - 1\right) = \alpha\beta I_{1nt} I_{2nt}^+, \tag{8}$$

$$P\left(I_{12,nt'} = I_{12,nt} + 1, I_{2nt'} = I_{2nt} - 1\right) = \alpha\beta I_{2nt} I_{1nt}^+. \tag{9}$$

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

$$P\left(S_{nt'} = S_{nt} + 1,\ S_{mt'} = S_{mt} - 1\right) = \frac{\lambda S_{mt}}{k_m - 1}, \tag{10}$$

$$P\left(I_{qnt'} = I_{qnt} + 1,\ I_{qmt'} = I_{qmt} - 1\right) = \frac{\lambda I_{qmt}}{k_m - 1}, \tag{11}$$

$$P\left(R_{nt'} = S_{nt} + 1,\ R_{mt'} = R_{mt} - 1\right) = \frac{\lambda R_{mt}}{k_m - 1}. \tag{12}$$

Finally, recovery from any infectious class occurs at a rate γ

$$P\left(I_{qnt'} = I_{qnt} - 1,\ R_{nt'} = R_{nt} + 1\right) = \gamma I_{qnt}. \tag{13}$$

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. Visual inspection of preliminary simulations was used to decide on 7×10^5 as being long enough for the epidemic to reach an equilibrium state. After 7×10^5 events, 5 individuals infected with pathogen 2 are added to one randomly selected

TABLE 1. All symbols used in Chapters **??** and **??** along with their units and default values.

Symbol	Explanation	Units	Value
S	Number of susceptible individuals		
I_q	N. individuals infectious with diseases q		
I_p^+	Sum of classes infected with pathogen p		
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^2	10,000
β	Transmission rate	Per year per individual	2, 5, 10
γ	Recovery rate	Per year per individual.	1
λ	Dispersal	Per day per individual	0.001–0.1
Λ	Birth rate	Per year per individual	0.05
μ	Death rate	Per year per individual	0.05
ρ	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.*, $t + \delta$	Days	
k_i	Degree of node i		
δ	Waiting time until next event	Days	
α	Cross immunity	Proportion	0.1
n, m	Colony index		
σ	Invading pathogen seed size		10
r_i	The rate that event i occurs.	Days^{-1}	

colony. After another 3×10^5 events the invasion of pathogen 2 is considered successful if any individuals with pathogen 2 still remain. Again visual inspection of preliminary simulations was used to determine that after 3×10^5 events, if an invading pathogen was still present, it was well established.

3.2. **Dependant variables.** Three dependant 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. The default area of the simulations was 10^4km^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 1.6×10^3. The number of colonies was kept constant and so population size varied 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 4×10^4, 2×10^4, 10^4, 5×10^3 and 2.5×10^3km^2 which gave density values of 0.2, 0.4, 0.8, 1.6 and 3.2animals·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 2). The spatial scale of the simulations vary between 2.5×10^3 and 4×10^4 km^2. This corresponds to square areas with sides of 50 to 200km. Dispersal can only occur between two colonies if they are within 100 kilometres of each other *i.e.* they are 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 does not guarantee that the population is fully connected 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 3) 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.

3.3. **Other Parameters.** The fixed parameters used are chosen to roughly reflect realistic wild bat populations. The death rate Λ is set as 0.05 per year giving a generation time of 20 years. The birth rate is set to be equal to μ so that the population size is stable. The recovery rate γ 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 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 not infections are studied further here.

Cross immunity is 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 is 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.

Three values of the transmission rate β are used, 0.1, 0.2 and 0.3. All simulations are run under all three transmission rates as this is a fundamental parameter that changes the broad dynamics of the pathogens.

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 an 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 dependant variables. Specifically, I fitted the model

$$\text{invasion} = b_1 d + b_2 n + b_3 m + c + \epsilon \tag{14}$$

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, ϵ 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.

3.5. **Comparative Analysis.** To test for an association between pathogen richness 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 independant 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 independant variables: body mass (Gay et al. 2014; Han et al. 2015; Kamiya et al. 2014; Maganga et al. 2014; Turmelle and Olival 2009), range size (Kamiya et al. 2014; Maganga et al. 2014; Turmelle and Olival 2009) and study effort (Gay et al. 2014; Maganga et al. 2014; Turmelle and Olival 2009). This was to attempt to avoid spurious positive results occuring 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 **??** examines the relationship between density and population structure and Chapter **??** 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)

3.6. **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. Only species with at least one pathogen were included in the analysis. 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 (present in the ICTV taxonomy) identified. That is, if a host carries 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.

3.7. **Other independant 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 and Szöcs 2013). The counts were scraped using the rvest package (Wickham 2015). I log transformed these variables as they were strongly right skewed. As this correlation is strong, the results here are for analyses using only Google Scholar citations.

Measures of body mass are taken from Pantheria (Jones et al. 2009) and primary literature (Aldridge 1987; Arita 1993; Canals et al. 2005; Heaney et al. 2012; Henderson and Broders 2008; Lim and Engstrom 2001; López-Baucells et al. 2014; Ma et al. 2003; Oleksy et al. 2015; Orr and 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.

3.8. **Phylogenetic nonindependance.** To control for phylogenetic nonindependance I used the best-supported phylogeny from Fritz et al. (2009) (shown in Figure **??**) which is the supertree from (Bininda-Emonds et al. 2007) with names updated to match the Wilson & Reeder taxonomy (Wilson and Reeder 2005). Phylogenetic manipulation was performed using the ape package (Paradis et al. 2004). The importance of the phylogeny on each variable separately (the λ 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.

3.9. **Statistical analysis.** Statistical analysis for both dependant variables was conducted using a information theoretical/model averaging approach (Burnham and Anderson 2002) specifically following (Whittingham et al. 2006; 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 dependant 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 independant variables were centered and scaled to allow direct comparison of the coefficients (Schielzeth 2010). In each case I simultaneously fitted the λ parameter as this avoids mispecifying the model (Revell 2010). κ and δ 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 it's coefficient, β, 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 β was calculated for all models, only models with a interaction term and only models without an interaction. As the interaction term greatly affects the estimated value of β, considering these value seperately aids interpretation. Following (Whittingham et al. 2005) I included a uniformly random variable as a null variable as even unimportant variables can have Akaiki weights notably greater than zero. The whole analysis was run 100 times, resampling the random variable each time. I calculated AICc for each model. I calculated the average AICc, $\overline{\text{AICc}}$, by averaging AICc scores within models. ΔAICc was calculated as $\min(\overline{\text{AICc}}) - \overline{\text{AICc}}$, not the mean of the individual ΔAICc scores, to guarantee that the best model has ΔAICc $= 0$. From these ΔAICc I calculated Akaiki 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 Akaiki 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.

4. RESULTS

4.1. **Simulation 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 (χ^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 (χ^2 test: $\chi^2 = 1.51$, df $= 2$, $p = 0.47$). However they were all either in simulations with the smallest

TABLE 2. Regression results comparing affects of colony size, colony number and area. Coefficients are from multiple logistic regressions with invasion as the dependant variable and all independant 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 test against the null hypothesis that $b = 0$.

β	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)	9×10^{-3}
	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

colony size (colony size = 100, 29 simulations) or with the fewest colonies (5 colonies, 8 simulations). These simulations were ignored in further analyses.

4.2. **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). Increasing population size, either by increasing colony size or number of colonies, increases the probability of invasion (Table 2). 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.

4.3. **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 5). 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 2). Therefore the simulations support the hypothesis that colony size is the more important component of population size, though colony number does still increase the probability of invasion.

4.4. **Comparative analysis results.**

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 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 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.

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 and Saltz 2003; Zunino et al. 2007) to different extents. My results suggest that pathogen communities will response differently depending on the change 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 decadely time scales. In short, species suffering reductions in groups size (Atwood 2006; Lehmann et al. 2010; Manor and 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.

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 how it may depend on other factors. Population density is often measured (Arneberg 2002; Lindenfors et al. 2007; Morand and 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 a 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). Metaanalyses 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.

5.3. **Assumptions and limitation.** Being based on the same model as Chapter **??**, the work presented here relies on many of the same assumptions. Furthermore, as a comparison is being made between the affects 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 related to only a 20 percent of individuals dispersing at all. This low rate of dispersal is expected to exaggerate the affect of area; at high rates of dispersal the population is essentially well-mixed, despite the metapopulion.

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 3). 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 affects of area. Alternatively, the metapopulation could have been modelled as a weighted network with dispersal occuring 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 affects 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 covary both within species across time and 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). Similarly 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.

5.4. **Conclusions.** Overall I have shown that while a number of demographic factors are intrinsically linked, they have different affects 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.

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