A MECHANISTIC MODEL TO COMPARE THE IMPORTANCE OF INTERRELATED POPULATION MEASURES ON PATHOGEN RICHNESS: HOST POPULATION SIZE, DENSITY AND COLONY SIZE

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Running header: A comparison of the importance of population measures on pathogen richness

Abstract

Zoonotic diseases are an increasingly important source of human infectious diseases, and reservoir host pathogen richness is a critical driver of spill-over risk. Host population-level traits such as population size and density, geographic range size and population structure have all been shown to be important determinants of host pathogen richness. However, empirically identifying the independent influences of these traits has proven difficult as many of these traits directly depend on each other. Here we develop a mechanistic, metapopulation, susceptible-infected-recovered model to identify the influences of independent population-level traits on the ability of a newly evolved pathogen to invade and persist in host populations in the presence of an endemic pathogen, using a case study of bats; a highly social mammalian order. We show that larger population and group sizes had a greater influence on the chances of pathogen invasion and persistence than increased population densities (and therefore decreased population structure) and number of groups. As anthropogenic change affects these traits to different extents, this increased understanding of how traits independently determine pathogen richness will aid in predicting future zoonotic spill-over risk.

Keywords. Pathogen competition, zoonotic disease, metapopulations, host pathogen richness, bats, emerging infectious disease

1. Introduction

Zoonotic diseases are a major source of human infectious disease [1–3]. The chance that a new zoonotic disease will come from a particular reservoir-host depends on a number of factors including the number of pathogen species it carries [4]. Much attention has been devoted to comparatively assessing the factors that are associated with high or low pathogen richness in wild animal species [5–7]. Many empirical, comparative studies have examined morphological or life history traits [6,7], but factors related to reservoir-host population biology are also expected to affect disease dynamics and therefore affect pathogen richness. Population-level traits that have already been shown to correlate with pathogen richness include: increased host density [6,8–11], increased range size [6,9,10,12,13] and increased population structure [12,14]. Other traits such as group size have been well studied but results have been equivocal [10,15–18]. However, some important population-level traits, such as population size, have not been included in comparative analyses despite it being the natural way to describe epidemiological populations [19].

Collinearity between explanatory variables is a common problem in correlative studies. However, this issue is exacerbated when there are clear, causal relationships between explanatory variables. There are two particularly clear relationships between the population-level factors associated with pathogen richness. Firstly, host density, d, host population size, N, and geographic range size, a, are, by definition, linked by $d = N/a$ (see Table S1 for all parameters used). Furthermore, the relationship $N \propto a$ has broad empirical support [20,21]. Secondly, host population size can be decomposed into two components, the number of groups, m, and the average size of a group, n, with $N = mn$. Therefore, correlative comparative studies will be especially poor at identifying which of these factors are closely correlated with pathogen richness. This lack of discriminatory power is particularly important with respect to global change and its effects

on zoonotic disease emergence. Population-level factors such as host population size and geographic range size, although interrelated, will respond differently to global change and the response will be species specific. Some host species may suffer large range contractions, and therefore large falls in population size, while their density remains fairly constant [22]. Other host species might retain their distribution but have a depressed population density [23]. Therefore, only by knowing which of these interrelated factors controls pathogen richness will we be able to predict future changes in pathogen richness.

Mechanistic models provide one method for comparing the importance of intrinsically related factors and can provide a deeper understanding of the system than correlative approaches. Theoretical studies have established that a number of host population factors are important for epidemiological dynamics generally and for the maintenance of pathogen richness specifically. Host density and structure are well established as having central roles in pathogen dynamics [24–29]. However, it has been noted that host population size is a more natural measure than population density and that particularly in comparative settings, population size should be preferred [19]. Host group size is also known to strongly affect disease dynamics with disease spreading more quickly through populations made up of larger groups [24]. Fewer studies specifically study how these factors affect pathogen coexistence. A number of studies find that increased host population structure can promote pathogen coexistence [30–32]. While these studies have examined whether these population-level factors can promote pathogen richness, none have attempted to distinguish which might be the most important.

There is great need for mechanistic models that try to disentangle the interplay between population-level factors including host density, population size, range size, population structure, group size and the number of groups. Here, we have used multipathogen, metapopulation models to individually vary these host population parameters. The metapopulations were parameterised to broadly mimic wild bat populations. We used bats as a case study as the size of bat groups (colonies) is very variable and bat colonies are often very stable [33,34,44]. Furthermore, bats are particularly relevant in the context of zoonotic disease as they are thought to be reservoirs for a number of important, recent outbreaks [35,36]. We examined how the interrelated population 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. We used these simulations to test two specific hypotheses. First, we tested whether host population size or density more strongly promotes the invasion of a new pathogen. Secondly, we tested whether the invasion of a new pathogen is more strongly promoted by colony size or the number of colonies. We found that population size has a much stronger affect on the invasion of a new pathogen than host density and that increasing population size by increasing group size promotes pathogen invasion much more than increasing population size by increasing the number of groups.

2. Methods

(a). Two pathogen SIR model. We developed a multipathogen, susceptible-infected-recovered (SIR) compartment model. We examined the ability of a newly evolved pathogen to invade and persist into a population given the presence of an identical, endemic pathogen. Individuals were classed as susceptible, infected or recovered with immunity (Figure 1). Susceptible individuals are counted in class S. There

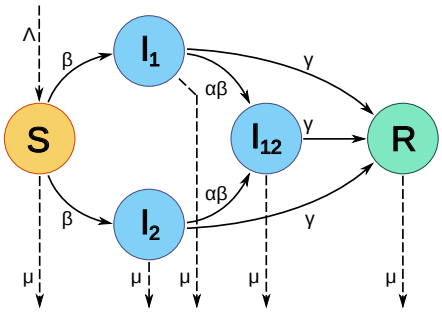

FIGURE 1. Schematic of the SIR model used. Individuals are in one of five classes, susceptible (orange, S), infected with Pathogen 1, Pathogen 2 or both (blue, I_1, I_2, I_{12}) or recovered and immune from further infection (green, R). Transitions between epidemiological classes occur as indicated by solid arrows and depend on transmission rate (β) and coinfection adjustment factor (α). Births (Λ) and deaths (μ) are indicated by dashed 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.

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 (i.e. there is complete cross-immunity). Furthermore, recovery from one pathogen moves an individual straight into the recovered class, even if the individual is infected with both pathogens (Figure 1). This modelling choice allows the model to be easily expanded to include more than two pathogens, though this study is restricted to two pathogens. The assumption of immediate recovery from all other diseases is likely to be reasonable [37]. Any up-regulation of innate immune response will affect both pathogens equally. Furthermore, as the pathogens are identical, any acquired immunity would also affect both pathogens equally. 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.

It is necessary to include births and deaths as the SIR model without population dynamics has no endemic state. Birth and death rates (Λ and μ) are set as being equal meaning the population does not systematically increase or decrease. New born individuals enter the susceptible class. Infection and coinfection were assumed to cause no extra mortality as for a number of viruses, bats show no clinical signs of infection [38,39].

The population is modelled as a metapopulation, being divided into a number of subpopulations (colonies). This model is an intermediate level of complexity between fully-mixed populations and contact networks. There is ample evidence that bat populations are structured to some extent. This evidence

comes from the existence of subspecies, measurements of genetic dissimilarity and ecological studies [33,34,40,41]. Therefore a fully mixed population is a large oversimplification. However, trying to study the contact network relies on detailed knowledge of individual behaviour which is rarely available. The metapopulation is modelled as a network with colonies being nodes and dispersal between colonies being indicated by edges (Figure 2A–D). Individuals within a colony interact randomly so that the colony is fully mixed. Dispersal between colonies occurs at a rate ξ. Individuals can only disperse to colonies connected to theirs by an edge in the network. The total rate of dispersal is not affected by the number of edges a colony has (known as the degree of the colony and denoted k). Therefore, the dispersal rate from a colony y with degree k_y to colony x is ξ/k_y. Note this rate is not affected by the degree and size of colony x. We examined the model using stochastic, continuous-time simulations which we implemented in R [42]. The full details of the model are given in Supplementary Methods S1. The code is available as an R package on GitHub [43].

(b). Deterministic model. We can get some initial insights into the behaviour of the system by examining a simplified, deterministic model with a single subpopulation (for details see Supplementary Methods S2). If we first consider the endemic pathogen (Pathogen 1) we have a typical SIR model with equilibrium values $S^* = \frac{\mu+\gamma}{\beta}$ and $I_1^* = \frac{\beta g n}{\gamma+\mu}$ where g is the population growth rate (here $g = 1$ as the population size is constant). When Pathogen 2 is introduced, its rate of change can be written as

$$\frac{dI_2}{dt} = \beta S^* I_2 + \alpha\beta I_1^* I_2 - (\mu+\gamma)I_2 \tag{1}$$

which is greater than zero when $\frac{\alpha\beta g n}{\gamma+\mu} I_2 > 0$ or $\alpha g R_0 I_2 > 0$ (with R_0 being the basic reproduction number and being equal for the two identical pathogens). R_0 is greater than one (as proved by the fact that Pathogen 1 is endemic), g equals one and I_2 is positive when Pathogen 2 is first introduced. Therefore, this inequality holds as long as α is greater than zero. That is, as long as cross-immunity is not complete, Pathogen 2 will always invade in this deterministic model.

(c). Parameter selection. The fixed parameters were chosen to roughly reflect realistic wild bat populations. The death rate μ was set as 0.05 per year giving a generation time of 20 years [44]. The birth rate Λ was set to be equal to μ. This yields a population that does not systematically increase or decrease. However, the size of each colony changes stochastically. Given the length of the simulations, colonies were very unlikely to go extinct. The recovery rate γ was set to one, giving an average infection duration of one year. 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 [45,46]. However, other studies have found much shorter infectious periods [47]. These shorter infections are not studied further here. The coinfection adjustment parameter, α, was set to 0.1. Therefore, an individual with a single infection is 90% less likely to gain a second infection. Given the deterministic model, $\alpha = 0$ and $\alpha > 0$ are the two qualitatively different conditions. The case where Pathogen 2 does not invade and spread ($\alpha = 0$) is unlikely to be important for pathogen richness so we chose a small, non-zero value for α. Dispersal was only allowed to occur between two colonies if

they were connected nodes in the metapopulation network. The metapopulation network was created for each simulation by randomly placing colonies in a square space (Figure 2A–D), the size of which varied between 2500 and 40000 km^2. If colonies were with 100km of each other, they were connected in the metapopulation network. The dispersal rate ξ was set to 0.01 which yields 17% of individuals dispersing in their lifetime.

(d). Population factors. The effect of range size on disease dynamics occurred through changes in the metapopulation network. Range size was varied between 2500 and 40000 km^2. This corresponds to square areas with sides of 50 to 200 km. The number of connections each colony has is called its degree, k. The mean degree, $\bar{k}$ is a measure of how well connected the metapopulation network is overall. The metapopulation network was not necessarily connected (i.e. made up of a single connected component) as the network was created by randomly placing colonies and only connecting colonies within 100 km of each other. To ensure connected metapopulation networks would have required repeatedly resampling of the colony locations until a connected metapopulation population occurred. However, this would bias $\bar{k}$. Therefore, it was considered preferential to keep the unconnected networks. The threshold of 100 km was arbitrary but we aimed to maximise the range of $\bar{k}$ (Figure S1) while not having many simulations with networks that were unconnected. Given this setup, populations with low densities had relatively unconnected metapopulation networks while high density populations had fully connected networks (Figure S1).

(e). Experimental setup. We let two identical pathogens compete: an endemic pathogen (Pathogen 1) and an invading pathogen (Pathogen 2). We used persistence (coded as 1) or extinction (coded as 0) of Pathogen 2 as a binomial response variable. We tested whether host population size had s strongrt affect on pathogen persistence than host density and then tested whether colony size or the number of colonies was the more important component of population size. We used three values of the transmission rate, β: 0.1, 0.2 and 0.3. This yielded very high values of R_0 which was required so that a reasonable number of simulations experienced invasion of Pathogen 2.

In each simulation the host population was seeded with 20 individuals infected with Pathogen 1 in each colony. Pathogen 1 was then allowed to spread and reach equilibrium. After 6×10^5 events, five host individuals infected with Pathogen 2 were added to one randomly selected colony. The simulation was then run until a further 75 years had elapsed. The invasion of Pathogen 2 was considered successful if any individuals infected with Pathogen 2 remained at the end of the simulation.

Three sets of simulations were run. This set of three simulations was used to compare two pairs of population factors: *i*) population size and host density, *ii*) colony size and the number of groups. The population parameters that were directly varied were colony size, the number of groups and range size. In each case these parameters were assigned their default value multiplied by 0.25, 0.5, 1, 2 and 4. The default colony size was 400, the default number of groups was 20 and the default range size was 10000 km^2.

In the first set of simulations, host density was varied by keeping population size constant while varying range size. Colony size was kept at a constant value of 400 while the number of groups was fixed at 20

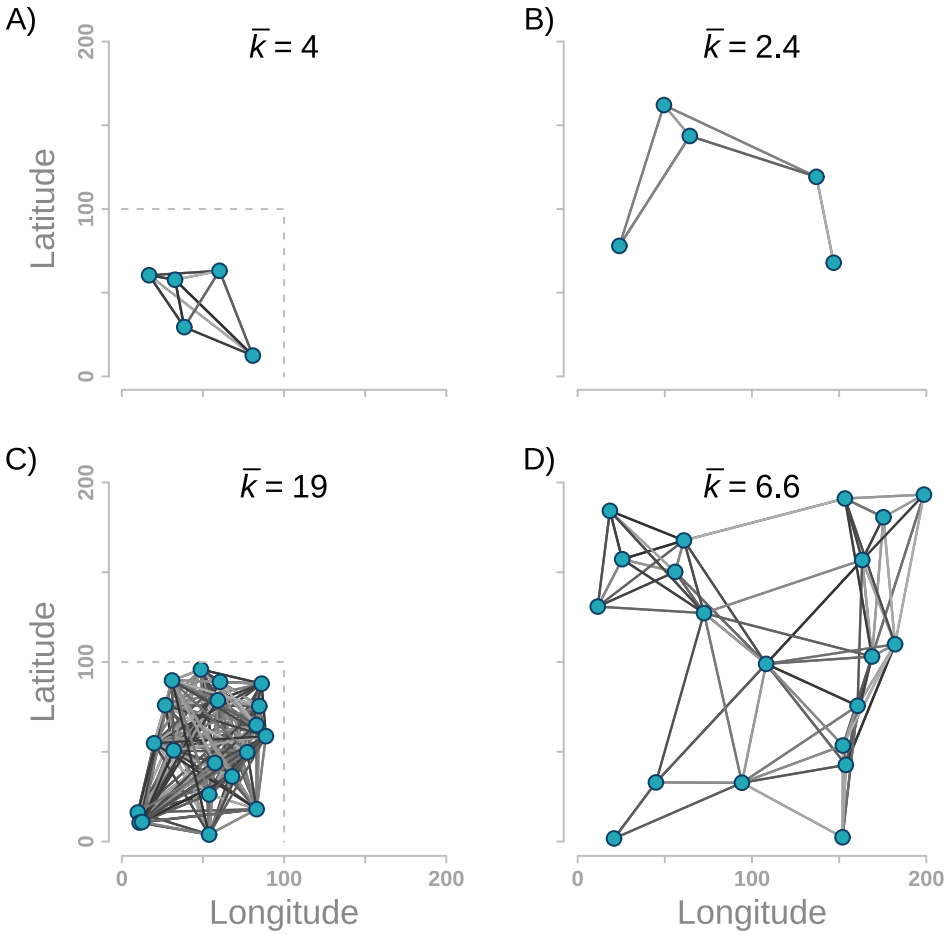

FIGURE 2. The relationship between range size and metapopulation network structure. Colonies are shown by circles. Colonies that are close enough for animals to disperse between (i.e. within 100 km of each other) are joined by a line. Colonies are placed randomly in spaces of various sizes (grey dashed lines). A and C) the default range size (10000 km^2). B and D) the largest range size (40000 km^2). A and B) the smallest number of colonies (five). C and D) the default number of colonies (20). The mean number of connections per subpopulation, $\bar{k}$, is shown for each metapopulation.

giving a population size of 8000. The values of range size used were 40000, 20000, 10000, 5000 and 2500 km^2 which gave density values of 0.2, 0.4, 0.8, 1.6 and 3.2 animals per km^2.

In the second set of simulations, population size was varied by changing colony size while the number of groups was kept constant. To keep host density constant, range size was increased as population size increased. The values of colony size used were 100, 200, 400, 800 and 600 while range size was set to 40000, 20000, 10000, 5000 and 2500 km^2. This gave population size values of 2000, 4000, 8000, 16000 and 32000 while host density remained at 0.8 hosts per km^2.

In the third set of simulations, population size was varied by changing the number of groups while colony size was kept constant. Again, to keep host density constant, range size was increased as population size increased. The numbers of groups used were 5, 10, 20, 40 and 80 while range size was set to 40000,

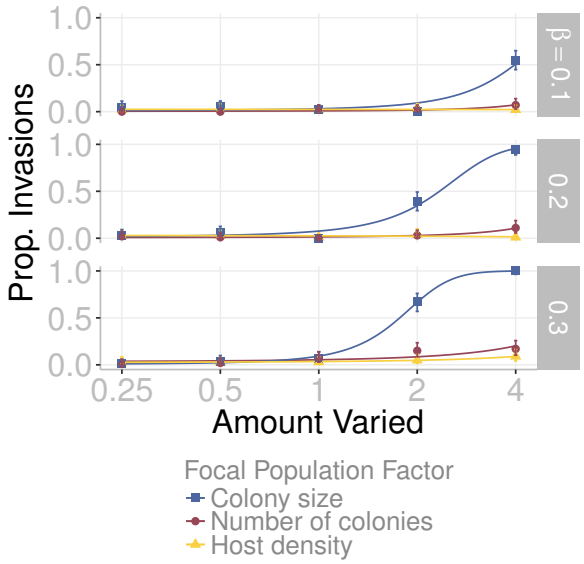

FIGURE 3. Comparison of the effect of colony size, number of colonies and host density on probability of invasion. The x-axis shows the change ($\times$0.25, 0.5, 1, 2 and 4) in each of these factors relative to the default value. Default values are: colony number = 20, colony size = 400 and density = 0.8 animals per km^2. Blue lines, squares: population size is altered by changing colony size. Red lines, circles: population size is altered by changing number of colonies. Yellow lines, triangles: population density is altered by changing range size. 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, β.

20000, 10000, 5000 and 2500 km^2. Again, this gave population size values of 2000, 4000, 8000, 16000 and 32000 while host density remained at 0.8 hosts per km^2.

To compare colony size and the number of groups, only the second and third set of simulations above were used. However, colony size and the number of groups were directly used as independent variables instead of using the derived values of host population size and density. It can be seen that population density and range size are identical in the two sets of simulations. Therefore, the only difference between these two sets of simulations was the factor used to increase population size; this factor being either colony size or the number of groups.

(f). Statistical analysis. We combined the results from different simulations and fitted a logistic multipleregression model, centering and scaling the independent variables. In 37 simulations, both of the

TABLE 1. Regression results comparing effects of colony size, colony number and density. Coefficient estimates (b) and 95% confidence intervals of each variable 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 range size while keeping population size equal. p is for the test against the null hypothesis that $b = 0$. Results are given for three transmission values (β).

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)	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

pathogens went extinct. The number of simulations where both pathogens went extinct did not depend on transmission rate (GLM: $b = -6.67 \times 10^{-3}$, $p = 0.69$). However all of the simulations where extinction of both pathogens occurred had either the smallest colony size (colony size = 100, 29 simulations) or the fewest number of groups (5 groups, 8 simulations). Results from these simulations were removed before further analyses. We fitted the model

$$\text{Invasion} = b_1 d + b_2 n + b_3 m + c + \epsilon \tag{2}$$

where d, n and m are density, colony size and number of groups, respectively, and b_i are the regression coefficients. c is a fitted intercept and ϵ is a binomially distributed error term. To test the hypothesis that an increase in host population size creates a stronger increase in invasion probability (of the second pathogen) than an equal increase in host density we compared the size (and 95% confidence intervals) of b_1 to b_2 and b_3. To test the hypothesis that an increase in colony size creates a stronger increase in invasion probability than a proportionally equal increase in number of groups we compared b_2 to b_3.

3. RESULTS

Increasing host population size, either by increasing colony size or number of groups, increased the probability of invasion (Figure 3, Table 1). The relationship between colony size and invasion was strong

and significant at all transmission rates, while the relationship between colony number and invasion was weaker and more marginally significant. In contrast, varying host density did not alter invasion probability. The 95% confidence intervals for b_1 do not overlap those for b_2 for the simulations with $\beta = 0.2$ or 0.3 but the confidence intervals do overlap for the simulations where $\beta = 0.1$. The 95% confidence intervals for b_1 overlaps those for b_3 at all values of β (Table 1).

The deterministic model showed that Pathogen 2 can invade even in a single subpopulation but depends on R_0 which in turn depends on colony size. In the stochastic simulations, increasing either colony size or the number of groups increased the probability of invasion but this effect was much stronger and more statistically significant for colony size (Figure S2, Table 1). The 95% confidence intervals for b_2 do not overlap those for b_3 for the simulations with $\beta = 0.2$ or 0.3 but the confidence intervals do overlap for the simulations where $\beta = 0.1$ (Table 1).

At the default parameter settings, the probability of invasion and persistence of the second pathogen, $P(I)$, was rare (Figure 3 and Tables S2–4). These proportions significantly increased with transmission rate (GLM: $b = 0.18$, $p = 5.28 \times 10^{-3}$).

4. Discussion

Overall, our results suggest that population size promotes pathogen richness significantly more than host density does in the context of metapopulations or group living. Furthermore, the results suggest that the most important component of population size is colony size; a large population made up of large groups rather than many, small groups promotes pathogen invasion to the greatest degree. These results lead to a number of other conclusions. All else being equal, increasing range size (with density remaining constant) will not strongly increase pathogen richness 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 the larger groups require more space and therefore dispersal between groups is reduced. The results suggest that, 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 does not go extinct during the stochastic, early stages of the epidemic, the new pathogen will persist. This is explicitely shown by the deterministic model. In the stochasic model, dispersal is a very slow process compared to infection, and therefore the global pool of susceptibles is largely unimportant. This is probably why increasing the number of groups did not increase pathogen invasion rate as quickly as the size of a colony did. Similarly, the global host density of the species had little effect on pathogen invasion rate. In these simulations, increasing density without increasing population size was only achieved by reducing range size; this simply increased the number of connections between groups in the metapopulation network. This, in turn, increased the pool of susceptibles that were could be reached by one dispersal event of the invading pathogen. However, again, this effect was very weak compared to the strong changes in local disease dynamics caused by increasing colony size.

This study is limited to one mechanism by which pathogen richness can be increased; the invasion and persistence of a newly evolved pathogen. Furthermore, we restrict outselves to the context of competition between two pathogens in a social host species.

Many comparative studies measure some aspect of a species population size or structure, yet it is rarely discussed how these are related. Instead most studies use the data that are available, without considering *a priori* how the explanatory variables are causally related (statistical correlations between independent variables are however often handled appropriately). For example, host density is often used in studies [8–11] yet density is directly associated with population size. Our results suggest that it is in fact population size that is important (in the context of social species as studied here) and previous authors have suggested it is the more natural descriptor of population size [19] Therefore, density is likely acting as a proxy for population size in these comparative studies. These causal relationships between population factors should be considered more carefully in future comparative studies. Our results also suggest that host range size does not promote pathogen richness, yet a number of studies have found evidence of this relationship [6,10]. This contradiction suggests that either the relationship found in comparative studies is in fact due to a correlation with another factor, or that mechanisms other than probability of invasion of new pathogens (as studied here) are important. The studies that have specifically tested the effect of group size have in fact found both positive [15] and negative associations [16] or no relationship [17]. A Meta-analysis suggested that the relationship between social group size and pathogen richness is weak [18]. However, we have found that group size is the most important population factor. This suggests that the mechanism studied here — invasion of recently evolved pathogens — may not be the major mechanism by which pathogen richness is created in wild populations.

It is clear that many species are suffering strong population changes due to global change [22]. However these changes might affect range size [22], population size [23], population connectivity [49–51] or group size [52–55] to different extents. Our results suggest that pathogen communities will respond differently depending on which factors are most strongly affected by global change. In short, species suffering reductions in groups size [52–55] are predicted to experience decreases in pathogen richness in the long term and there is some evidence that this process is occurring [12,56]. Species that are experiencing increases in group size [52] would be expected to gain new pathogen species. In contrast, species suffering range contractions [22] and decreases in population size [23] are expected to experience smaller changes in pathogen richness. However, the monitoring of these different aspects of population change, especially in bats, can often be difficult and may require further developments in acoustic monitoring to be effective [57–59].

Data accessibility

The implementation of the model is available as an R package on GitHub [43]. This can be found at https://github.com/timcdlucas/MetapopEpi. All code and simulation output data is available on GitHub at https://github.com/timcdlucas/Abundance-Density-Manuscript.

Competing interests

We have no competing interests.

Author's contributions

TCDL wrote the simulations and performed the analysis. TCDL, HMH and KEJ all helped design the study. TCDL drafted the manuscript TCDL, HMH and KEJ all edited the manuscript and gave final approval for publication.

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