A null model of community disassembly effects on vector-borne disease risk

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Abstract:

Community structure is heterogeneous at a variety of spatial and temporal scales, and this variation has been shown to influence the risk of zoonotic diseases such as West Nile Virus and Lyme disease. Theoretical models and most empirical evidence suggest that the greatest influence of host diversity occurs when transmission is frequency-dependent (i.e., the rate of contact is constant). These theoretical models are generally based on ordinary differential equations and become intractable when considering more than a few species. This makes it particularly difficult to predict how we might expect the transmission of infectious diseases to change as community structure changes in space or in time. Here we develop a model in which we construct a network of interactions between hosts and vectors to quantify the change in risk under different scenarios of community disassembly. Decreased vector biodiversity always reduced mean risk, while a change in host community structure led to increased or decreased mean risk depending on the manner in which community disassembly altered mean competence of the "new" community. These trends in mean risk can be generalized across a multitude of natural systems because they do not depend on the distribution of host quality, though simulation suggests that variation around the mean can be very high. The primary value of model is that it can be used to establish upper and lower bounds on the expected change in disease risk with decreasing biodiversity.

Keywords: Biodiversity-disease | Diversity gradient | Network model

Article:

1. Introduction

We are just beginning to understand how host community structure can influence the dynamics of multi-host pathogens. Theoretical models have been developed by a number of authors to address the question of when we might expect to see diversity influence epidemiology (e.g., Bowers and Turner, 1997; Holt et al., 2003; Dobson, 2004). These authors have specifically been interested in determining when diversity should lead to amplification or dilution of the overall risk in the system. This work suggests that the effects of community composition are likely to be much more dramatic for pathogens transmitted in a frequency-dependent manner (e.g., sexually transmitted or vector-borne pathogens) than for those whose transmission rate scales with density (Dobson, 2004 and Rudolf and Antonovics, 2005). These models further suggest that high levels of host biodiversity in these systems should dilute risk. The mechanism by which host biodiversity can lower the risk of vector-borne disease transmission arises from a constraint on the number of potentially infectious bites that a vector can give in its lifetime (e.g., Dobson et al., 2006). Given that vectors have a finite lifespan and that there is a non-zero handling time associated with feeding on each host, we can divide the infectious lifespan of a vector into a finite number of searching and biting "events." As a result, there exists a maximum number of potentially infectious bites that can occur over the lifetime of any vector. Increases in the numerical dominance of hosts which have a low competence (defined as the ability of a species to both acquire and pass on infection), through changes in species richness or evenness will reduce the risk of infection across the host community because a greater number of those bites will be "absorbed" by poor hosts.

The unstated assumption of this is that vector feeding is random with respect to species, though we know that feeding biases exist (Lyimo and Ferguson, 2009). In reality, any mechanism that increases the relative rates of "sampling" of inefficient hosts for a pathogen (low competence) should reduce risk because inefficient hosts will absorb some of these potentially infectious contacts (Keesing et al., 2006). The hallmark of random feeding behavior in vectors is that the proportion of bites on a particular host will be proportional to its relative abundance in the community. More likely is that vectors sample from the available hosts in the community based on a behavioral preference for particular hosts (Kilpatrick et al., 2005). Thus, changes in the mean, community-wide force of infection can result from either shifts in vector feeding biases towards low competence hosts, or an increase in the relative abundance of low competence hosts in the face of random feeding. We have little detailed empirical knowledge of the feeding biases in most vector-host communities (but see Lyimo and Ferguson, 2009), though they likely play a role in transmission (Irwin and Thresh, 1988, but see McElhany et al., 1995 and Real et al., 1992). Changes in vector and host infection have been shown to change as a function of changes in both species richness and evenness in host communities (Pitre and Boyd, 1970, Power, 1987, Farrell, 1976, Ostfeld and Keesing, 2000, LoGiudice et al., 2003 and Ezenwa et al., 2006).

Thus, the dilution effect is based on the assumption that increased host diversity will decrease disease risk as a function of an increased proportion of contacts occurring with poor hosts. In

reality, such changes will only occur when changes in host community structure cause large shifts in the mean competence of the host community. In the two best studied empirical examples, Lyme disease in rodents (e.g., Ostfeld and Keesing, 2000) and West Nile Virus in birds (e.g., Ezenwa et al., 2006), changes in community structure that induce a shift in risk are always biased towards or away from species of high competence. Ostfeld and Keesing (2000) found evidence that the highest competence hosts are generally present in low richness communities for Lyme disease and zoonotic cutaneous leishmaniasis (i.e., they are among the first included or last lost in most communities). The recognition that host species identity is critical in governing diversity effects on disease has been previously acknowledged (see Keesing et al., 2006). As the evidence for the effect in other host-parasite systems continues to grow (e.g., Ruedas et al., 2004, Telfer et al., 2005,Estrada-Peña et al., 2008 and Johnson et al., 2009) there is an increasing need for the development of theoretical models that can estimate the change in risk across gradients in host diversity such as those that often arise in suburban and urban landscapes.

While the importance of host community structure on risk has been widely explored, the role of vector community structure has been less well-studied. Shifts in the overall feeding preferences of a vector community should have similar effects as changes in the relative frequency of poor hosts, especially when feeding biases are strongly non-random (e.g., Kilpatrick et al., 2005). As a result, the loss or gain of vector species may induce changes in risk even when host community structure is stationary.

In addition to the effect of species identity of both the hosts and vectors, the strength of any diversity effect should be constrained by the distributions of competence and contact rates in the host and vector communities, respectively. When host competence is highly heterogeneous or long-tailed (e.g., a lognormal distribution) we expect to see a large disparity between the best and worst hosts in the community. Thus, any bias in the process of community assembly or disassembly over time (or space) should result in a greater amplification or dilution of overall risk in highly heterogeneous communities. This is the inherent assumption behind the idea of managing for higher abundances for very poor hosts to control outbreaks (LoGiudice et al., 2003). By increasing the skew in the distribution of competence in a community, a greater number of vector bites will be absorbed by the poorest hosts, leading to increased dilution of risk.

The goal of this paper is to expand on existing theory associated with diversity-disease effects to better understand how diversity gradients (and the potential biases in community assembly/disassembly which generate them) influence the dilution (and amplification) of risk associated with vector-borne zoonoses. In particular, we establish the upper and lower bounds on the potential change in disease risk along a diversity gradient due to the sampling effect and elucidate the shape of the diversity–risk relationship along gradients induced by local community disassembly. We predict that the shape of the relationship between species richness and disease

risk will be determined by the direction of the bias in community assembly/disassembly. Further, we predict that the magnitude of the change in risk across the richness gradient will be governed by the degree of heterogeneity in the regional species pool.

We differ from previous authors who have generally simulated a specific system (e.g., LoGiudice et al., 2008; Ostfeld and LoGiudice, 2003) or adopted an ordinary differential equations (ODE) framework for exploring diversity-disease dynamics by explicitly considering the network of pairwise interactions between host and vector populations. The use of networks to describe interactions in complex species assemblages is not novel. There is an extensive literature in trophic ecology which employs network-based approaches to model the flow of energy in a community and identify critical guilds of species (see Pascual and Dunne, 2006). More recently, network models have been used in the study of disease transmission in an effort to evaluate how individual-level variation among hosts influences disease transmission (e.g., Meyers et al., 2005, Keeling and Eames, 2005, Lloyd-Smith et al., 2005 and Brooks et al., 2008). The use of a network-based framework here is analogous to these applications, allowing us to evaluate the role that heterogeneity in the community plays in determining overall disease risk. This approach allows us to calculate the change in human risk using the entomological inoculation rate (EIR) along the β -diversity gradient in a manner that makes the derivation of analytical expressions for mean change in risk for large communities tractable.

2. The model

In order to assess the change in vector-borne disease risk along a gradient in species richness, we assembled an interaction network between a community of hosts via a community of multi-host feeding vectors (Fig. 1). This graph-based approach allows us to use matrix products to calculate risk to an arbitrarily chosen focal species as the entomological innoculation rate (EIR)−the number of potentially infectious bites· host^{-1}· day^{-1}. This measure can be derived directly from the classic Ross‑McDonald malaria model or it can be derived from first-principles. At its simplest, EIR is the product of the biting rate (number of bites· host^{-1}· day^{-1}) on the focal host (usually humans in this context) and the incidence of infection in the mosquito population (Birley and Charlewood, 1987). For a vector-borne zoonotic pathogen the vast majority of mosquito infections occur as a function of their contact with infectious hosts and the ability of those hosts to both acquire and transmit infection (reservoir capacity). Given this, we calculate vector incidence as the product of two terms: the reservoir capacity of each host population Y_i (i= 1,2, …, m), and the relative preference of vector species j to feed on host i (p_{ij}). These preferences are represented by the proportion of bites from vector j on host i that would occur in a community of evenly distributed host species such that Σp_j =1. Formally, capacity is defined here as the per contact rate of a new vector infection arising from contact with the population of host i . This value includes the mean, per capita host‑vector contact rate (c), the prevalence of infection in host species i (ρ_i), the relative abundance of

host species i (a_i), and the probability of infection given contact (θ_i) such that $Y_i = c\rho_i a_i \theta_i$. The incorporation of relative abundance is of particular note as it allows us to quantify the effects of both species richness and species evenness (the two components of alpha diversity) directly. The product $Y_i p_{ij}$ is then the per contact rate of infection for vectors of species j that result from contact with host i, or vector incidence. If we then multiply incidence by the biting rate on our focal host for vector species j, U_j ($j = 1,2, \cdots, n$), we can quantify the risk of infection in this focal host as a function of each host-vector-host pathway in a network of interactions (Fig. 1). The total risk for the focal host based on network structure, R, is a simple matrix product of a $1 \times m$ vector of host capacities ($\overline{Y}^T$), an $m \times n$ matrix of vector-host feeding preferences (P), and a $n \times 1$ vector of human biting rates per vector ($\overline{U}$).

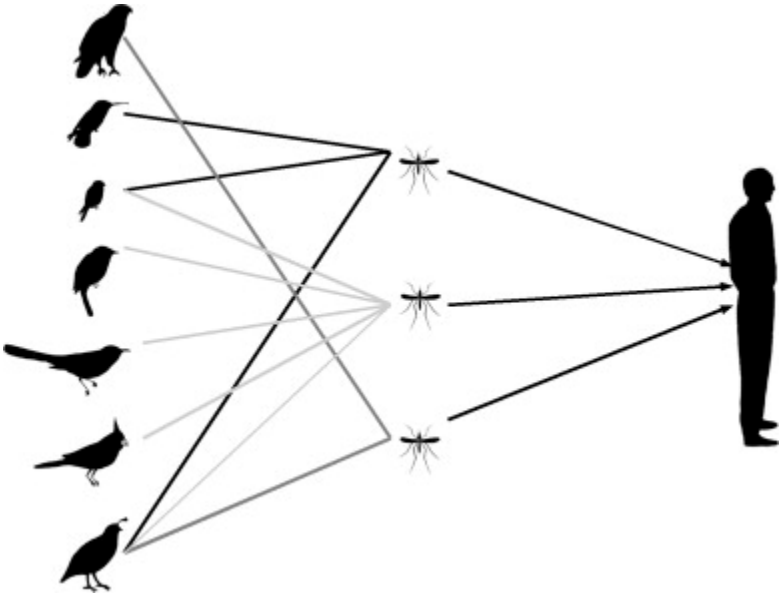

Fig. 1. West Nile Virus as an example of the host-vector-host network used to calculate entomological inoculation rate in our models.

2.1. Mathematical formulation

In an epidemiological context, the community of potential wildlife hosts for a particular pathogen in a particular landscape can be represented by a distribution of host capacities, Y_i. The vector community can likewise be characterized by a distribution of biting rates on the focal species, U_j. We can show that for any distribution of capacities that the mean risk is constant when the community of hosts is disassembled at random

$$E(\tilde{R}_{host}) = nE(U)E(Y)$$

where n is the vector species richness of the community and $E(Y)$ and $E(U)$ are the means of the distributions of host capacity and focal host biting rates, respectively (see Appendix A).

The upper and lower bounds on mean risk across the host diversity gradient is set by stepwise removal of the lowest or highest capacity host from the local community, respectively. Given a ranked list of host species (descending order) we can show that risk for removing the best hosts is

$$R_{upper} = nE(U)\frac{\sum_{i=1}^{m-r} E(Y_i)}{m-r}$$

and that the biased removal of the poorest hosts is

$$R_{lower} = nE(U)\frac{\sum_{i=1+r}^{m} E(Y_i)}{m-r}$$

where r is the number of hosts removed. The significance is that in both cases, human risk is directly proportional to the mean of the capacities of hosts in the community, a term which is weighted by the relative abundances of each reservoir host.

Following a similar approach, we can calculate the change in risk as a function of the random disassembly of the vector community

$$R=(n-w)E(U)E(Y)$$

where w is the number of vector species removed such that $w \leq n$. Variance (calculation not shown) around this mean risk is also proportional to $(n-w)$ so that both the mean risk and the variance around it decrease as $w \to n$. For the change in risk in the vector community the upper and lower bounds on mean risk are calculated in the same manner as for the hosts. After ranking the vectors in descending order based on their host biting rates, both the upper bound

$$R_{upper} = E(Y)\sum_{j=1}^{n-w} E(U_j)$$

And the lower bound on the mean

$$R_{lower} = E(Y)\sum_{j=w+1}^{n} E(U_j)$$

are functions of the sum of host biting rates and will clearly decrease with the removal of any vector.

In order to evaluate our analytical results we conducted a series of simulations, each representing a unique community. We focus on the lognormal distribution for these simulations because it provides the flexibility to vary between a symmetrical distribution and the long-tailed pattern of variation that we frequently observe in natural systems. This does not influence the generality of our analytical results, but because order statistics for the lognormal distribution have not been

fully worked out, we are restricted to the use of tables (Balakrishnan and Chen, 1999) to calculate numerical estimates of mean risk to a community of no more than 25 non-focal hosts and/or vectors.

2.2. Simulation

Using the graph-based framework outlined above to model host‑vector‑host interactions we generated 10,000 unique communities. Simulations presented here were initialized with 25 species of hosts and vectors. This scenario was generated for comparison with analytical results and predictions of biased assembly/disassembly. Simulation involved the removal of a single species at each step, re-normalizing the mosquito feeding preference matrix and re-calculating EIR. Although our approach to simulation involved disassembly, the results should also hold for community assembly using the appropriate constraints for each scenario. We built our simulation on the assumption that host capacity, host‑vector contact probabilities and human biting rates were all lognormally distributed. This assumption allowed us to control the variation in each character from long-tailed (σ large) to more symmetrical (σ small). Note that because we have defined competence as a function of relative abundance, changes in the distribution of the Y_i can likewise be viewed as changes in the distribution of relative abundances in the host community instead of changes in contact or prevalence. To evaluate the upper and lower bounds on mean risk along the gradients, we simulate filtering of the regional species pool as the disassembly of communities given each of three scenarios: a+1, −1, or zero correlation between host capacity and probability of removal. That is, we removed the host with the highest capacity, lowest capacity, or we removed hosts at random, respectively. With each species removal it was necessary to re-normalize the elements of P to maintain relative proportionality in contact preferences for the remaining hosts. A similar approach was used to explore the role of vector diversity on risk, with correlations based on human biting rates for each species. All simulations were conducted using the *R* Statistical Language (R Development Core Team, 2009).

3. Results

Simulation of random disassembly suggests that mean risk decreases linearly with decreasing species richness of bridging vectors with a slope consistent with our analytical calculation (Fig. 2a). Decreasing host richness, as predicted, resulted in no change in the mean risk of infection (Fig. 3a), though there was a non-linear increase in variance around the mean with decreasing richness. This change in variance can be interpreted as the unbiased sampling effect that results directly from the size of the sample drawn from the original distribution of host capacities.

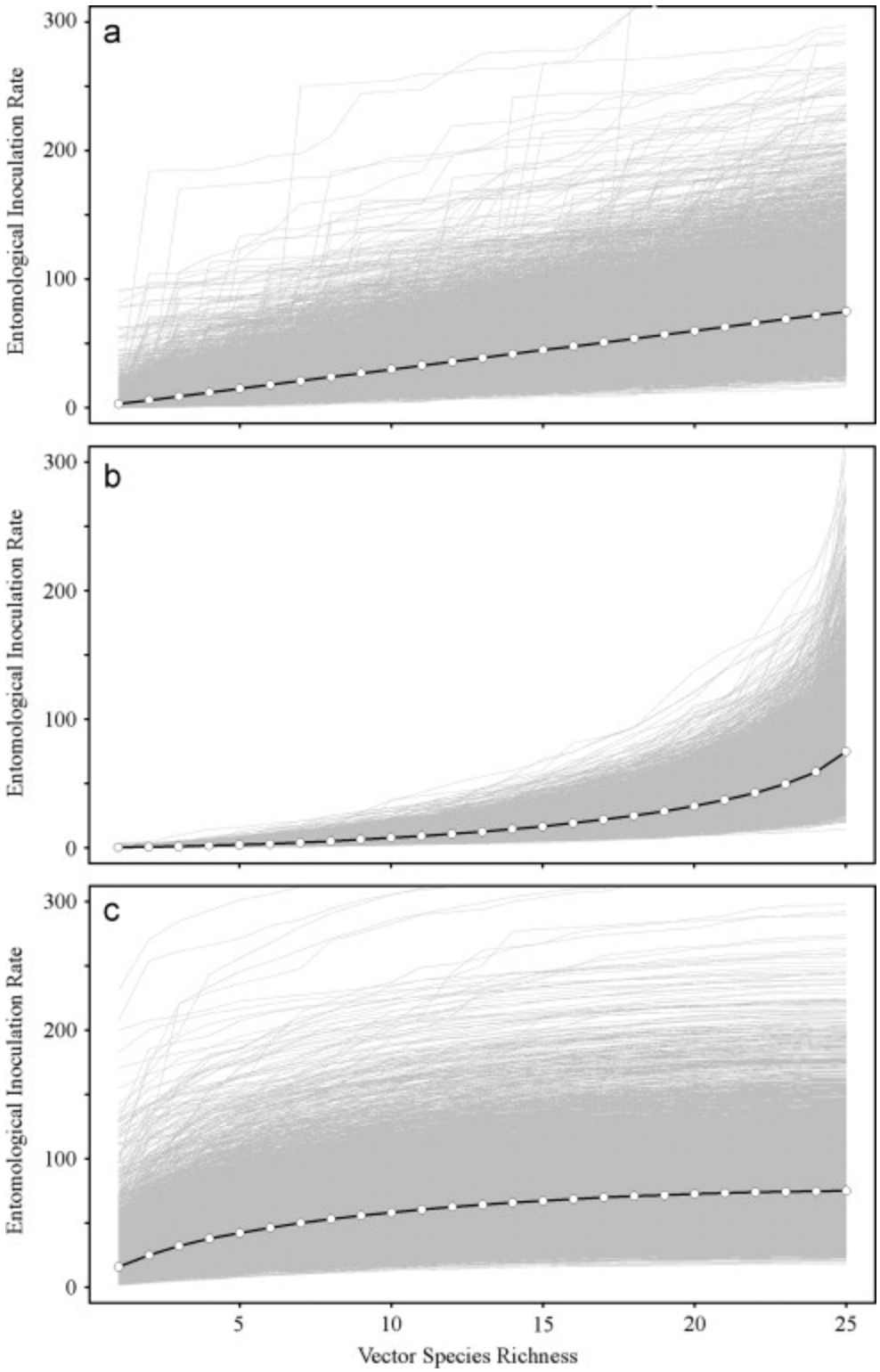

Fig. 2. The change in EIR as a function of changing vector richness where removal is (a) random, (b) biased such that the species with the highest human biting rate is removed at each step in the simulation, or (c) biased such that the species with the lowest biting rate is removed at

each step. Grey lines indicate individual iterations of the simulation and the dark black line shows the mean of those runs. Circles show the predicted values from the analytical expressions.

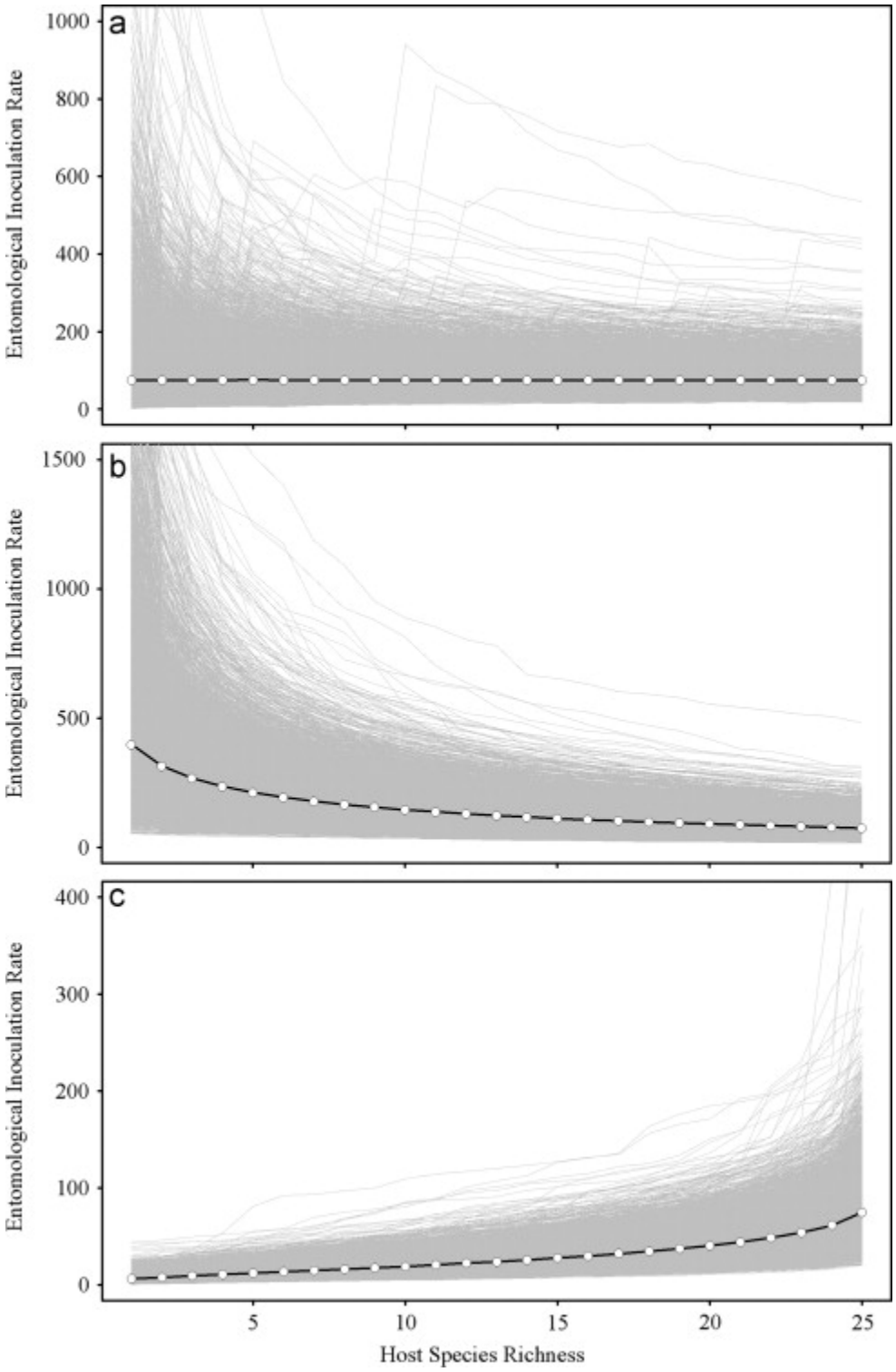

Fig. 3. The change in EIR as a function of changing host richness where removal is (a) random, (b) biased such that the species with the lowest competence/relative abundance is always

removed at each step in the simulation, or (c) biased such that the species with the highest competence/relative abundance is always removed at each step. Note the variation in the range of the y-axes on each panel as an indication of the degree to which each scenario influences overall risk. Grey lines indicate individual iterations of the simulation and the dark black line shows the mean of those runs. Circles show the predicted values from the analytical expressions.

Decreasing vector species richness in the community always led to a decrease in risk of infection (Fig. 2). The shape of the relationship between host richness and risk differed between scenarios such that removal of vectors having the highest biting rate on the focal host induced a more rapid initial decline in risk (Fig. 2b). Variance in risk also dropped sharply when vectors with high host biting rates were preferentially removed, while it remained relatively constant across moderate richness levels when low biting rate vectors were removed first.

The type of bias in the removal of host species was important in determining the direction and magnitude of the change in the predictability of risk with decreasing host richness. When highly competent host species were preferentially removed, there was an exponential decrease in human risk with decreasing host richness (Fig. 3c). The total change in risk across the richness gradient (between a "full" community and one including one species) declined exponentially as a function of the coefficient of variation in capacity across the "full" host community (r^2=0.999, p<0.0001). In contrast, preferential removal of low competence (or low relative abundance) hosts induced a non-linear increase in human risk (Fig. 3b). It is of particular note that the range in risk across the gradient in diversity was an order of magnitude greater than the total decrease in risk that occurred under preferential removal of highly competent hosts (Fig. 3). The change in risk is especially rapid as low competence (or abundance) hosts are removed from large communities. The CV of the "full" community also accurately predicts the total change in risk across the gradient. When removal preferentially targets low competence (or low relative abundance) hosts, the total change in risk increases linearly with increasing CV (r^2=0.992, p<0.0001).

4. Discussion

The decrease in risk associated with decreasing vector diversity was not surprising. Because we included only vectors fed on our focal host and at least one species in the community (bridging vectors), the removal of each species reduced risk. While we expect that reduced vector diversity will always reduce risk because vectors serve to "sum" across the host community, we cannot rule out the potential for non-bridging vectors to have an effect during the initial epidemic wave in a community. In this transient state, heterogeneity among vectors in feeding preferences or competence may cause some species to reach a peak prevalence prior to others. Empirical evidence for vector diversity effects is limited, largely because few authors have investigated its role in determining risk. In one such study, Barrera et al. (2002)demonstrated that reduced vector diversity would generally lower the risk of equine encephalitis virus in a South American community, but because rodent diversity covaried with vector diversity, the effect of the vector diversity alone could not be determined.

While loss of vector diversity always results in decreased risk, the model predicts that change in risk to humans as a function of host diversity is related to the manner in which changes in community structure alter the mean capacity of the reservoir community. As expected, when the loss of hosts occurred independent of host competence (i.e., random removal), diversity had no effect on mean disease risk. This does not suggest that random loss of species from a community will not alter risk. For any individual iteration of the simulation (analogous to disassembly of a particular regional species pool), slight increases or decreases of risk occur as a function of the stochastic nature of disassembly. As the analytical results show, when there are "strings" of high- or low-competence hosts removed we expect a change in risk that is proportional to the mean competence of the species remaining in the community. Taking this into account, we can interpret the constancy of the mean risk as an indication that risk is just as likely to increase as it is to decrease along the gradient in diversity, and that the magnitude of changes in both directions is the same. The consequence is that the sampling effect (see Tilman (1999) for a discussion of the sampling effect; variance here is an unbiased estimate of this effect) is just as likely to decrease as increase risk. Thus, even in the absence of bias, we expect a small effect on risk along any particular diversity gradient, but the direction of that change would be difficult to predict and the magnitude would be small relative to more biased assembly/disassembly.

When changes in community structure are biased based on each species' ability to acquire and pass on infection or its relative abundance, risk can vary over several orders of magnitude. The effect emerges through an interaction between host species and is mediated by the degree of heterogeneity present in the original host community (as evidenced by the strong relationship between the CV for host competence and mean risk in our simulations). The greatest decrease in risk occurs when the most competent species are also most likely to be lost. Keep in mind that simulation progressed as a function of community disassembly and that this decrease in risk with the preferential loss of highly competent hosts during disassembly is the same as an amplification effect due to preferential inclusion of highly competent hosts during assembly. Because low abundance hosts are most likely to be lost from the community and our measure of competence includes the relative abundance of reservoir hosts in the community, preferential removal of species with large Y_i suggests very strong skew in species' reservoir competence. That is to say that low abundance hosts must be much better hosts for the pathogen than are high abundance hosts in order for this amplification effect to occur.

The necessity of such strong skew in competence may explain why there are a limited number of empirical examples of an amplification effect, as common species are more likely than rare species to be frequently exploited by parasites (Keesing et al., 2006). However, when the host and pathogen do not share a long co-evolutionary history we might expect to see this effect more often. Sauby (2009) recently found that increased host richness led to a higher level of infestation by the invasive cactophagous moth, *Cactoblastis cactorum*. This is similar to the findings of Power and Mitchell (2004) who found a diversity effect on native and invasive grasses based largely on the inclusion of a single invasive grass species. Kilpatrick et al., 2006a and Kilpatrick

et al., 2006b have also suggested that the spike in the newly introduced pathogen, West Nile Virus (WNV), result from the seasonal movement of the most competent avian host in the community, the American Robin.

When hosts with the lowest competence or lowest relative abundance are preferentially lost from the community, the model predicts a mean increase in risk that is directly related to the mean competence of remaining hosts. When Y_i is highly skewed, as in our simulations, we see a non-linear increase in risk that can be an order of magnitude greater than the mean risk predicted by random removal (data not shown). During community assembly, when additional hosts are preferentially poor hosts, we predict a dilution effect similar to that widely cited in the literature (Dobson et al., 2006). Because of the inclusion of relative abundance in the definition of competence here, this is analogous to the preferential loss of low abundance species in the community. Thus, this effect may be the result of preferential loss of low abundance species when communities do not vary strongly in competence, or due to highly skewed competence with respect to the ability of a species to acquire and pass on infection.

Not surprisingly, there are a number of empirical data in both plant (Pitre and Boyd, 1970, Farrell, 1976 and Power, 1987) and animal (Ostfeld and Keesing, 2000 and Ezenwa et al., 2006) systems that are consistent with the prediction of dilution through the addition of low competence hosts. In large monocultures of maize (*Zea mays*), corn stunt disease is transmitted through extended feeding of the corn leafhopper (*Dalbulus maidis*) (Power, 1987). By interspersing maize plants with other crops (e.g., beans)Power (1987) was able to show that *D. maidis* moved between plants less frequently and that disease was less prevalent in polycultures than even low-density monocultures, presumably due to the addition of poor hosts for the vector (and pathogen). Likewise, the presence and relative abundance of highly competent rodent species (e.g., white-footed mice) can alter the risk of infection by *Borrelia burgdorfi*, the causative agent of Lyme Disease (Ostfeld and Keesing, 2000). Similar to the current findings, Ostfeld and LoGiudice (2003) used simulation models of their empirical system to show that these influential species were responsible for the largest proportion of total risk because most other species are poor reservoir hosts for the pathogen. The present work simply allows us to quantify the degree to which skew in reservoir capacity and/or abundance in the host community is expected to influence the change in risk across various communities, and under different scenarios of assembly/disassembly.

The overarching result is that when host competence covaries with the likelihood of being lost from or added to the community, assembly and disassembly can induce shifts in disease risk that can range over several orders of magnitude, depending on the degree of heterogeneity in the original community. The consequences for understanding how risk changes along β -diversity gradients are enormous. First, we know little about the relationship between capacity and the processes driving community structure for most natural systems. Even in systems where we have extensive knowledge of the capacity of different reservoir species for a particular pathogen, it is

unclear how those patterns might translate to other closely related pathogens. This means that disassembly which is uncorrelated with capacity for one pathogen might covary strongly with another pathogen. Likewise, the change in risk associated with a diversity gradient in one landscape may differ greatly from the change in risk in another. Ezenwa et al. (2006) and Kilpatrick et al., 2006a and Kilpatrick et al., 2006b found significant effects of diversity on the prevalence of WNV in coastal Louisiana and the Washington, D.C. metro areas, respectively. However, Loss et al. (2009) found no relationship between host diversity and WNV prevalence in mosquitoes in the Chicago, Illinois area. This variation generates a mosaic of unpredictable landscape- and pathogen-specific patterns of risk, though the strong dilution effect predicted in the absence of exceptionally competent, rare hosts suggests that increased diversity will usually decrease risk.

A number of approaches to the management of zoonotic disease have been proposed that rely on the hypothesized commonality of the dilution effect. One proposed strategy is to manage for the presence of one or more exceptionally poor reservoir host species to dilute the risk in the community (LoGiudice et al., 2003 and Keesing et al., 2006). This is the idea of zooprophylaxis (Sota and Mogi, 1989), the best example of which is the epidemiological role that cows play in absorbing bites from the mosquitoes responsible for spreading malaria (Dobson et al., 2006). Our model suggests that this approach will change risk in a manner that is directly proportional to the decrease in mean competence across the new community. While theoretically sound, the implementation of zooprophylaxis is likely to be intractable for most systems. The second approach is to manage wildlife communities so that ecosystem processes such as predation (Ostfeld and Holt, 2004) and competition can keep populations of especially good reservoir hosts at low levels. This effect is likely to be successful only when competence covaries positively with prey palatability or negatively with competitive ability, and it also suffers from the logistical impracticalities of implementation.

In addition to the logistical issues, we suffer from lack of the basic knowledge of which species are competent hosts, what role(s) they play in species interactions, and in the roles they may play in the epidemiology of alternative pathogens. With emerging zoonotic diseases making up over 70% of all emerging infectious diseases (Jones et al., 2008), and with taxonomic patterns in emergence being difficult to discern (Woolhouse and Gowtage-Sequeria, 2005), we cannot currently determine which species are more important than others in buffering human populations from future disease emergence. This uncertainty and the likelihood of diversity leading to the dilution of risk suggest that the most effective protection that we could provide against future zoonotic disease emergence would be the preservation of native biodiversity. While we cannot predict which species will play different roles in any future outbreak, few species are likely to be high competence hosts for any pathogen. As such, the preservation of biodiversity will likely decrease the mean competence of the community of zoonotic reservoirs in most situations. At the same time, we can improve our ability to predict the consequences of zoonotic disease emergence through the continued search for the mechanisms that make certain

species amplifying hosts−including the role of molecular physiology and genetics on ecological interactions and the mechanisms by which human resource use influences patterns and rates of contact with potential wildlife hosts.

Appendix A. Derivation of the mathematical model

Let $\overline{Y}=(Y_1,Y_2,\ldots,Y_m)^T$ (the superscript T denotes the matrix transpose) be a random sample of size m from a population with finite mean $E(Y)$ and variance $Var(Y)$, describing the capacity of wildlife hosts in a community of size m to acquire and pass on infection. Likewise, let $\overline{U}=(U_1,U_2,\ldots,U_n)^T$ be an independent sample of size n drawn from a population with finite mean $E(U)$ which describes the human biting rates of vectors in the community of size n. Then let $P=(p_{ij})$ be a random matrix with dimension $m\times n$ describing the probability of contact between vectors and wildlife hosts such that the column sum satisfies $\Sigma_{i=1}^{m}p_{ij}=1$ for each j. Here each column $\{p_{ij}\}_{i=1}^{m}(j=1,2,\ldots,n)$ of the matrix P is obtained through normalization of an independent matrix (ϕ_{ij}) from the same population with finite mean $E(\phi)$. That is, $p_{ij}=(\varphi_{ij})/(\Sigma_{i=1}^{m}\varphi_{ij})$, for each j=1,2, ⋯, n. Therefore, the total risk is given by the random variable

$$R=\overline{Y}^T P\overline{U}\sum_{j=1}^{n}\left[\sum_{i=1}^{m}p_{ij}Y_i\right]U_j$$

The following result is obvious and will be used throughout the appendix.

A.1. Random removal

A.1.1. Random removal of hosts: mean risk with changing host biodiversity

Suppose that r ($1\le r\le m-1$) components of Y corresponding to r wildlife hosts have been randomly removed from a community of original size m. Without loss of generality, we assume that the leftover sample is given by $\tilde{Y}=(Y_1,Y_2,\ldots,Y_{m-r})^T$. Let $Q=(q_{ij})$ of size $(m-r)\times n$ be the adjusted random matrix so that for each j =1,2, ⋯ , n , $q_{ij}=(p_{ij})/(\Sigma_{i=1}^{m-r}p_{ij})=(\varphi_{ij})/(\Sigma_{i=1}^{m-r}\varphi_{ij})$ and $\Sigma_{i=1}^{m-r}q_{ij}=1$. Therefore, the new total risk is given by

$$\tilde{R}_{host}=\tilde{Y}^T Q\overline{U}=\sum_{j=1}^{n}\left[\sum_{i=1}^{m-r}q_{ij}Y_i\right]U_j$$

and

$$E(\tilde{R}_{host})=\sum_{j=1}^{n}E(U)E\left(\sum_{i=1}^{m-r}q_{ij}\right)E(Y)=nE(U)E(Y)$$

which is independent of r, i.e., the change in host biodiversity does not change mean risk.

A.1.2. Random removal of vectors: mean risk with changing vector biodiversity

Now we consider the case of randomly removing w vector species from $\overline{U}$. For simplicity, we assume that the leftover sample after w $(1 \leq w \leq n-1)$ removals is given by $\tilde{U} = (U_1, U_2, \ldots, U_{n-w})$. Let $\tilde{P} = (p_{ij})$ of size $m \times (n-w)$ be the adjusted random matrix, and so

$$\tilde{R}_{vector} = Y^T \tilde{P} \tilde{U} = \sum_{j=1}^{n-w} \left(\sum_{i=1}^{m} p_{\bar{ij}} Y_i \right) U_j$$

is the new total risk. Hence

$$E(\tilde{R}_{vector}) = \sum_{j=1}^{n-w} E(U) E\left(\sum_{i=1}^{m} p_{\bar{ij}} \right) E(Y) = (n-w) E(U) E(Y)$$

A.2. Biased removal

A.2.1. Removal of the hosts with the highest capacity: mean in risk with changing host biodiversity

If $\overline{Y} = (Y_1, Y_2, \ldots, Y_m)$ is a random sample, we denote $Y_{(1)} < Y_{(2)} < \ldots Y_{(m)}$ to be the corresponding order statistics of Y. Under this scenario we remove the r $(1 \leq r \leq m-1)$ largest Y_i 's from the sample Y. Here let $\tilde{Y} = (Y_{(1)}, Y_{(2)}, \ldots Y_{(m-r)})$ and $Q=(q_{ij})$ of size $(m-r) \times n$ be the same adjusted random matrix given in A.1.1, therefore the new total risk is given by

$$\tilde{R}_{lower} = \overline{Y}^T Q \overline{U} = \sum_{j=1}^{n} \left[\sum_{i=1}^{m-r} q_{ij} Y_{(i)} \right] U_j$$

Hence

$$E(\tilde{R}_{lower}) = \sum_{j=1}^{n} \left(\sum_{i=1}^{m-r} E(q_{ij}) E(Y_{(i)}) \right) E(U) = \frac{n}{m-r} E(U) \sum_{i=1}^{m-r} E(Y_{(i)})$$
$$= nE(U) \left[\frac{\sum_{i=1}^{m-r} E(Y_{(i)})}{m-r} \right]$$

A.2.2. Removal of the vectors with the highest human biting rates: mean risk with changing vector biodiversity

Let $U_{(1)} < U_{(2)} < \ldots U_{(m)}$ denote the order statistics for the random sample U. Under this scenario we remove the w largest U_i 's (human biting rates) among vectors. For simplicity we assume that the leftover sample after w removals is given by $\tilde{U} = (U_{(1)}, U_{(2)}, \ldots U_{(n-w)})$.
Let $\tilde{P} = (p_{ij})$ of size $m \times (n-w)$ be the same adjusted matrix as given in A.1.2 above, and

$$\tilde{R}_{lower} = Y^T \tilde{P} \tilde{U} = \sum_{j=1}^{n-w} \left(\sum_{i=1}^{m} p_{\bar{ij}} Y_i \right) U_{(j)}$$

is the new total risk. Hence, the mean total risk is given by

$$E(\tilde{R}_{lower}) = \sum_{j=1}^{n-w} \left(\sum_{i=1}^{m} E(p_{ij}) E(U_{(j)}) \right) E(Y) = E(Y) \sum_{j=1}^{n-w} E(U_{(j)})$$

A.2.3. Removal of the hosts with the lowest capacity: mean in risk with changing host biodiversity

Here we remove the r smallest values of Y and calculate the risk as in A.2.1. The mean total risk under this scenario for community disassembly is given as

$$E(\tilde{R}_{upper}) = \sum_{j=1}^{n} \left(\sum_{i=r+1}^{m} E(q_{ij}) E(Y_{(j)}) \right) E(U) = nE(U) \left[\frac{\sum_{i=r+1}^{m} E(Y_{(i)})}{m-r} \right]$$

A.2.2. Removal of the vectors with the lowest human biting rates: mean risk with changing vector biodiversity

Here we remove the smallest w values of U and calculate risk as shown in A.2.2. The mean total risk under this scenario for vector community disassembly is given as

$$E(\tilde{R}_{upper}) = \sum_{j=w+1}^{n} \left(\sum_{i=1}^{m} E(p_{ij}) E(U_{(j)}) \right) E(Y) = E(Y) \sum_{j=r+1}^{n} E(U_{(j)})$$

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