Community phylogenetic structure shapes pathogen outbreak potential in an epidemiological multi-host model

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Abstract

Biodiversity loss and anthropogenic alterations to species communities are impacting disease emergence events. These trends may be related through mechanisms in which biodiversity either increases (amplification) or decreases (dilution) diseases among hosts. Biodiversity effects can be direct, when contacts with competent hosts are replaced by sink hosts, or indirect through regulation of abundances and depend on the disease-competence of the added host. Here, we introduce a multi-host compartmental disease model, weighting host competence by their evolutionary history. We explore two scenarios: (1) where the probability of transmission depends on the evolutionary distance between the transmitting and recipient hosts, and (2) where transmission depends on the evolutionary distance from the receiving host to the primary host of the pathogen. Using simulations, and estimating the host community outbreak potential (R_0), we show how differences in phylogenetic structure can switch host communities from diluting to amplifying a disease, even when species richness is unchanged. Common ecophylogenetic metrics of community structure that capture variation in the pairwise distances among hosts, are able to explain >90% of the variation in R_0 across simulations. Our study provides a simple illustration of how host evolutionary histories can drive disease dynamics.

Word count: 4588

Key words: Dilution, Amplification, Multi-host model, Phylogeny, Epidemiology, Pathogens, Disease-ecology

1 Introduction

Over the past century, anthropogenic pressures on the environment have accelerated (Steffen et al., 2015), leaving many species on the verge of extinction (IPBES, 2020; Barrett et al., 2018; IUCN, 2021). Coinciding with the increase in biodiversity loss has been an increase in the number and frequency of disease outbreaks (Daszak et al., 2001; UN, 2020) and it has been suggested that these two trends may be linked (Halliday and Rohr, 2019; Rohr et al., 2020). Similar patterns are seen for plants and animals (Daszak et al., 2000; Woolhouse et al., 2005; Jones et al., 2008), suggesting a common process. However, there remains debate about how shifts in the composition and richness of host communities may translate to changes in disease prevalence and outbreak potential (LoGiudice et al., 2003; Joseph et al., 2013). While much attention has been given to the relationship between biodiversity and disease (e.g. Halliday and Rohr, 2019; Rohr et al., 2020; Keesing et al., 2006, 2010; Han et al., 2020), understanding the mechanism by which changes in host species diversity within a community alters disease prevalence requires knowledge of multi-host disease dynamics (Woolhouse et al., 2001; Dobson, 2004; Luis et al., 2018).

Theoretical models suggest that an increase in host species richness can either decrease disease prevalence (dilution effect: Table 1) or increase disease prevalence (amplification effect: Table 1) (Keesing et al., 2006, 2010; Ostfeld and Keesing, 2012). Both dilution and amplification can occur via changes in transmission, either directly through modifying encounter rates between competent (Table 1) and less-competent hosts or indirectly through changing densities of competent hosts (Rudolf and Antonovics, 2005; Ostfeld and Keesing, 2000). A dilution effect is more likely when added species are low competent hosts, whereas amplification is more likely when added species have high competence. In practice, the distinction between the processes of amplification and dilution is not straightforward. Some diseases appear to be diluted by diversity in some instances but amplified by diversity in other instances, depending on the host community species composition, shedding rate or relative abundance (Barasona et al., 2019; Streicker et al., 2013; Stewart Merrill et al., 2022). It is also possible for both dilution and amplification to occur simultaneously in one system, for example, depending on the density and composition of the host community, as has been suggested in rodents infected by hantavirus (Luis et al., 2018) and for RNA viruses in honey and bumble bees, where species abundance and richness separately had amplifying and diluting effects, respectively (Cohen et al., 2022).

Ultimately, it is the composition and relative competence (Table 1) of hosts within a community that drive dilution/amplification dynamics. Species that are able to maintain endemic prevalence, reservoir hosts (Table 1, Haydon et al., 2002), have high competence. Species (or individuals) that are highly competent can dominate transmission,

a phenomenon often referred to as *super-spreaders*. Less competent hosts, however, may function as dead-end hosts if they either do not maintain the disease, or do not contribute to onward transmission, thereby reducing disease spread. Data on host competence would allow us to make more accurate predictions about how changes in host diversity would impact disease prevalence; however, such data are frequently lacking, especially for low-competent hosts and hosts with no prior exposure. One approach to bridge this data gap is to approximate host competence from life history data and species' physiology, such as body size and the position of hosts on the fast-slow continuum (Dobson, 2004; Joseph et al., 2013; Cohen et al., 2022). Here, we suggest that if relevant traits are evolutionarily conserved, species phylogeny might provide another useful and more general proxy.

There is a growing weight of evidence showing that diseases are more frequently shared between closely related hosts (Gilbert and Webb, 2007; Davies and Pedersen, 2008; Streicker et al., 2010; Parker et al., 2015; Farrell and Davies, 2019a). This phylogenetic structure in the distribution of pathogens among hosts likely reflects phylogenetic conservatism in evolved defense mechanisms (Streicker et al., 2010; Mollentze and Streicker, 2020). We might therefore expect the evolutionary distance between species to be correlated with both the probability of sharing a disease and disease severity following infection. For example, in grassland communities, disease prevalence was higher in species with many close relatives in the community (Parker et al., 2015), whereas hosts that were phylogenetically isolated (Table 1) in their communities obtained a *rare-species advantage* and were less likely to share pathogens with co-occurring species. Phylogenetic isolation—Evolutionary Distinctness (ED, Table 1)—from co-occurring hosts might, therefore, be an indicator of low competent or dead-end hosts (sinks). We suggest that the phylogenetic composition of hosts may thus be an additional component in the establishment probability and disease dynamics of a pathogen in the community.

Here, using simulations and a simple compartmental (Susceptible-Infected) disease model, we explore the importance of the phylogenetic structure of host communities on outbreak potential (R_0) for a multi-host disease, assuming frequency dependent (FD) dynamics, where the contact rate is constant. For simplicity, our model assumes no recovery, describing diseases such as in *Mycobacterium bovis* infection causing bovine Tuberculosis (bTB) (Jolles et al., 2005; Ezenwa et al., 2010), but could easily be extended. Scaling transmission probability in proportion to the inverse of the phylogenetic distance between hosts, such that transmission is more likely between closely related hosts, we consider two alternative scenarios: 1) transmission is proportional to the evolutionary distance between infected and susceptible hosts - *Distance from transmitting host*; 2) transmission is proportional to the distance between the

receiving (secondary) and primary (co-evolved) host, irrespective of the identity of the donating host - *Distance from primary host*. Using simulations, we examine how R_0 varies with host community phylogeny and host richness, and the phylogenetic placement of the primary host. We suggest that our model may help explain the conflicting evidence for dilution and amplification effects in empirical systems, and allows us to better understand how future changes to biodiversity may alter local disease dynamics.

Term	**Abbr**	**Definition**	Source
Reservoir	-	A single or group of host species able to maintain the prevalence of a disease	Haydon et al. (2002)
Primary host	-	A single host with which the pathogen has undergone substantial co-evolution	
Disease competence	-	Susceptibility to and the ability to transmit onward a disease	Ostfeld and Keesing (2000)
Dilution effect	-	Reduction in disease prevalence with increasing biodiversity	Ostfeld and Keesing (2000)
Amplification effect	-	Increased disease prevalence with increasing biodiversity	Ostfeld and Keesing (2000)
Phylogenetic diversity	PD	The summed evolutionary history of a species set	Faith (1992)
Pairwise Phylogenetic distance	PPd	Evolutionary history separating species	Cadotte and Davies (2016)
Evolutionary Distinctiveness	ED	Measure of phylogenetic isolation of a species	Isaac et al. (2007)
Basic reproduction number	R_0	Expected number of infections arising from a single infected individual in a naive population	Kermack and McKendrick (19

Table 1: Glossary of terms for the introduction of this manuscript.

2 Methods

The outbreak potential of a disease is calculated as the number of susceptible infections a single infected individual can infect in its lifetime and is represented by R_0. When this value is greater than unity, an epidemic can occur. In a single host species system, R_0 may be calculated from the ordinary differential equations describing the susceptible (S) and infected (I) individuals (Kermack and McKendrick, 1927; May and Anderson, 1984; Dobson, 2004):

$$\frac{dS}{dt} = -\beta(N_T)SI - \mu S \tag{1}$$

$$\frac{dI}{dt} = \beta(N_T)SI - \delta I - \mu I \tag{2}$$

where $\beta(N_T)$ describes the transmission of infection from S to I, per time unit, δ and μ are the species-specific disease-induced and natural mortality rates, respectively. $N_T = S_T + I_T$, the T representing *all* species in the community combined.

Following Keeling and Rohani (2011), we can decompose the transmission parameter, β as:

$$\beta_{ij}(N_T) = \kappa_{ij}(N_T) \cdot c_{ij} \tag{3}$$

In this equation, transmission rate β is a function of the probability of transmission given a contact, c_{ij}, times the contact rate κ. We can, in turn, define κ as a function of the total number of individuals in the community, N_T, allowing this model to describe both frequency-dependent disease transmission (e.g., transmission occurs through contact at a watering hole) or density-dependent transmission, such that κ is either a constant or varies with N_T, respectively. This is captured in the Kronecker function as described in SI section 3. Because our focus is on the reproductive number, R_0, of the disease in the initial (naive) population, the community size is treated as a constant, allowing us to simply assume a constant κ (and therefore also constant β), as we would for frequency-dependent transmission.

We can calculate R_0 by setting equation (2) to zero and using $I = 1$ while solving for $\frac{1}{S}$, which returns returns a R_0 independent of N:

$$R_0 = \frac{\beta}{\delta + \mu} \tag{4}$$

In a multi-host model with n species, following Dobson (2004), the community R_0 can be calculated from matrix $\mathbf{R}$, also known as the Next Generation Matrix (Diekmann and Kretzschmar, 1991). This is derived from the so-called

WAIFW (Who Acquires Infection from Whom) matrix, $\mathbf{W}$, which includes the intra- and interspecific transmission rates (β_{ij}) of all n species:

$$W = \begin{bmatrix} \beta_{11} & \dots & \beta_{1n} \\ \vdots & \ddots & \vdots \\ \beta_{n1} & \dots & \beta_{nn} \end{bmatrix} \tag{5}$$

where β_{ij} is the transmission rate from host species j to i, given a contact. We can calculate the community R_0 by multiplying each i, j index of matrix $\mathbf{W}$ with the duration of time an individual of this species is infected ($\frac{1}{\delta+\mu}$, as in the single-host system Eqn (4)). All species are initiated with the same number of susceptible individuals.

To account for each species' contribution to the community R_0, each index of the returned matrix above is multiplied by the relative-contact (within-to-between) term p_{ij}, returning matrix $\mathbf{R}$ (Diekmann and Kretzschmar, 1991). For a system of hosts with frequency-dependent transmission there is a *constant* contact rate, independent of population density, so p_{ij} is described as (Dobson, 2004):

$$p_{ij} = \frac{\kappa_{ij}}{\kappa_{total,j}} \tag{6}$$

where κ_{ij} is the number of contacts that species j has with i, and so describes the proportion of contacts j had with i relative to the total number of contacts for species j ($\kappa_{j,total}$):

$$\kappa_{total,j} = \sum_{x=1}^{n} \kappa_{xj} \tag{7}$$

As contact rates are included in term p_{ij}, transmission is now simply defined as: $\beta^*_{ij} = c_{ij}$. For simplifying purposes here, we assume interspecific and intraspecific contacts are equally probable. However, it may be more realistic to model interspecific contacts as less frequent than intraspecific contacts, as we describe in SI Section 3.

In a 3-host system, matrix R can be represented as:

$$R = \begin{bmatrix} \frac{\beta^*_{11}p_{11}}{\delta_1+\mu_1} & \frac{\beta^*_{12}p_{12}}{\delta_2+\mu_2} & \frac{\beta^*_{13}p_{13}}{\delta_3+\mu_3} \\ \frac{\beta^*_{21}p_{21}}{\delta_1+\mu_1} & \frac{\beta^*_{22}p_{22}}{\delta_2+\mu_2} & \frac{\beta^*_{23}p_{23}}{\delta_3+\mu_3} \\ \frac{\beta^*_{31}p_{31}}{\delta_1+\mu_1} & \frac{\beta^*_{32}p_{32}}{\delta_2+\mu_2} & \frac{\beta^*_{33}p_{33}}{\delta_3+\mu_3} \end{bmatrix} \tag{8}$$

The maximum real eigenvalue of matrix $\mathbf{R}$ is the community R_0 and describes the reproductive success of a generalist

pathogen:

$$R_{0,community} = R_0 = \rho(R) \tag{9}$$

On the diagonal of matrix $\mathbf{R}$ we can find the individual R_0 of species i in isolation, $R_{ii} = R_{0,i}$ (so $p_{ii} = 1$). When $R_{0,i} > 1$, host species i is a reservoir host; when $R_{0,i} < 1$, host species i cannot sustain infection on its own, resulting in obligate multi-host parasitism (Fenton et al., 2015). The sum of column j in $\mathbf{R}$ shows the total received infection of species j from all of the n species. The sum of row i shows the total contribution of species i to community transmission. Hereon, we use R_0 to refer to the community R_0 (Eqn (9)).

We can easily rescale the interspecific values for c_{ij} in the β transmission rate term; here, we assume that probability of successful infection increases with decreasing evolutionary distance (Parker et al., 2015; Farrell and Davies, 2019a). Now, the probability of successful transmission from host j to i is determined by the evolutionary distance separating the two species (Pairwise Phylogenetic distance, PPd, see Table 1). While we assume that phylogenetic distance inversely impacts the probability of transmission given a contact, we may expect that contact rates to also co-vary with phylogeny. However, the weighting by phylogeny can be equally placed on either term. We use simulations to explore two alternative scenarios describing primary-secondary host dynamics:

Scenario 1: No primary host - *Distance from transmitting host*

In this scenario, the probability of successful transmission for species j after contact with species i, is modelled as:

$$c_{ij} = \lambda \cdot \frac{1}{1 + \psi PPd_{ij}} \tag{10}$$

where PPd_{ij} is the pairwise phylogenetic distance between hosts i and j (typically measured in millions of years), and ψ is a scalar, as $\psi = \frac{1}{2} \cdot \psi_0$ (given that PPd represents 2 * the time since divergence). λ is the baseline probability of infection within a species, so here $\lambda = c_{jj}$. Thus the probability of establishing an infection in a novel host simply depends on the distance between the transmitting, j, and receiving, i, host.

This scenario might apply to diseases such as Rabies virus (*Rabies lyssavirus*), a multi-host pathogen affecting a large range of species, where isolation within a species and high mutation rates cause differentiation of the virus in each of the host species (Streicker et al., 2010; Mollentze et al., 2014).

Scenario 2: Single primary host - *Distance from primary host*

Here, we scale the interspecific probability of successful transmission by the distance between the secondary, recipient host (i) and the primary host, regardless of the identity of the transmitting host. In this scenario, we assume the pathogen is best adapted to the primary host P, and so the probability of disease transmission will decreases with the evolutionary distance between the recipient host and the primary host, such that:

$$c_{ij} = \lambda \cdot \frac{1}{1 + \psi P P d_{iP}} \tag{11}$$

In a three-species system, there are two unique phylogenetic placements of the primary host, nested within the in-group sister pair, or as outgroup to the sister pair, we refer to these alternative placements as ingroup and outgroup, respectively. Both scenarios are portrayed in Figure 1, including the two possibilities for Scenario 2.

One well-described empirical example of a disease systems with primary-secondary host dynamics is bovine tuberculosis (bTB), caused by *M.bovis*. In sub-Saharan Africa, bTB is thought to be endemic in African Buffalo (*Syncerus caffer*), which function as a reservoir, and primary host, together with greater kudu (*Tragelaphus strepsiceros*) and warthogs (*Phacochoerus africanus*) (Bengis and Erasmus, 1988; Jolles et al., 2005; De Garine-Wichatitsky et al., 2013; Ezenwa and Jolles, 2015). As it spread from cattle, the pathogen is most adapted to bovines such as cows and buffalo (their primary host in sub-Saharan Africa). However, bTB frequently spills over to secondary hosts, including other ungulates, where it may establish persistent infections, and, rarely, to more phylogenetically distant species, such as lions (*Panthera leo*) and honey badgers (*Mellivora capensis*), which act as dead-end hosts with no forward transmission (Michel et al., 2006).

Simulating communities

We used simulations to explore the consequence of host dynamics and host phylogenetic structure on disease outbreak potential (R_0). We first simulate host phylogenetic topologies under a Yule (pure birth) process, using the R-package *phytools* (Revell, 2012), with birth rate, b, drawn from an exponential distribution to simulate *waiting times* (the branching times between speciation events) (See SI for more information). Communities simulated with low b have an increased distance between branching events, and are thus composed of more distantly related species than communities simulated with high b (SI Figure 2).

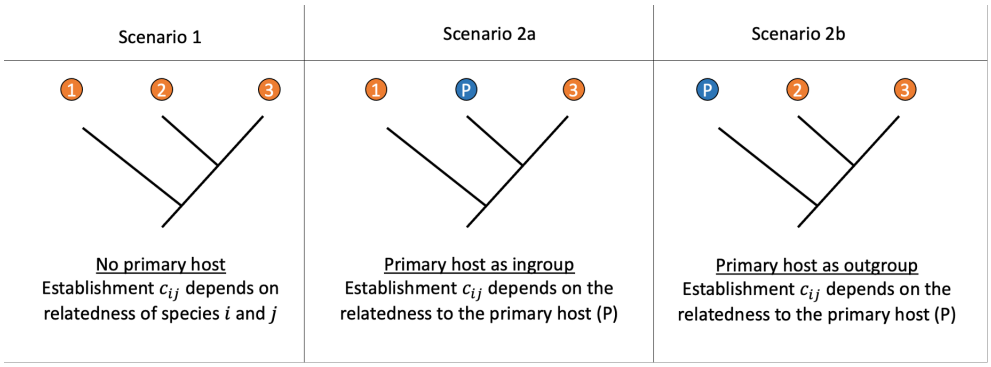

Figure 1: Illustration of alternative primary-secondary host dynamics for a simple three-taxon host system. The first panel shows scenario 1, with no assigned primary host. The second and third panels show scenario 2a (primary host as ingroup) and 2b (primary host as outgroup), respectively, where the blue circles represent the primary host, and the orange circles the secondary hosts.

We simulated 100 host communities for different combinations of richness (S=3,4,5,7,10) and speciation rates ($0 < b < 1$), assuming the distance between species to be measured in million years. For each simulated community, we calculated the Phylogenetic Diversity (PD), Mean Pairwise Distance between hosts (MPD, (Cadotte and Davies, 2016)), allowing the tree depth to vary, and tree Imbalance, Ic, of the phylogenetic tree connecting the community of hosts (Table 2). Because PD is highly correlated with species richness, S, we use MPD as our metric of phylogenetic structure in out statistical models, below. To explore primary-secondary host dynamics in Scenario 2, for communities with > 3 hosts, we additionally recorded primary host Evolutionary Distinctiveness (ED), a metric describing the phylogenetic isolation of a tip on the phylogeny (Isaac et al., 2007). For each simulated host community, we then calculated the community R_0, with for all species a natural and disease-induced mortality of $\delta = 0.1$ and $\mu = 0.2$, respectively. We use a value of $\lambda = 1$, $\kappa_{ij} = 1$ and $\psi = 1$ to obtain realistic values of R_0 ($0 < R_0 < 4$). We also set contact rates, $\kappa_{ij} = 1$, such that interspecific and intraspecific contacts are assumed equally likely. We explored alternative scenarios in SI section 3. The phylogenetic placement of the primary host (Scenario 2) was assigned randomly. We then generated a series of regression models to explore how host phylogeny could mediate disease outbreak potential. MPD and ED were log-transformed. All variables were subsequently scaled using a z-transform before model fitting. We note that MPD and ED were highly colinear (VIFs > 10), we thus report results only including either MPD or ED in the main text.

Biodiversity metric	Abbreviation	Definition	Source
Edge length	θ	Length from tip to node (in MY)	-
Pairwise Phylogenetic Distance	PPd_{ij}	Branch length $i \rightarrow j$	See: Cadotte and Davies (2016)
Phylogenetic Diversity	PD	$\sum_{e \subset z(T)} \theta_e$	Faith (1992)
Mean Phylogenetic Distance	MPD	$\frac{1}{D} \sum_i \sum_{i \neq j} PPd_{ij}$	Webb et al. (2002)
Imbalance	Colless's I (Ic)	*colless(tree, norm = "yule")*	Bortolussi et al. (2006)
Evolutionary Distinctiveness	ED	$ED(T,i) = \sum_{e \subset q(T,i,r)} (\theta_e \frac{1}{S_e})$	Isaac et al. (2007)

Table 2: Biodiversity metrics used in the analyses. θ_i is the branch length from species i to the root of the tree. S is the number of species in the tree. *tree* is the phylogenetic tree generated using the *Phytools* package under a Yule (no death) process. $e \subset z(T)$ denotes the edges, e, in a set z for tree T, D is the total number of species combinations, and the set $q(T,i,r)$ connecting species i to the root r, and S_e is the number of species that descend from edge e. This latter is used for the calculation of *Fair proportions* ED.

3 Results

We used simulations to explore the relationship between host community phylogenetic structure and disease outbreak potential when transmission success is modelled as a function of phylogenetic distance between hosts. We show that both the phylogenetic structure of the host community and the phylogenetic placement of the primary host influence the community R_0 (SI Figure 3).

Scenario 1: No primary host - *Distance from transmitting host*

In the absence of a single primary host, the establishment probability of the disease (c) simply depends on the phylogenetic distance between the receiving and donating hosts, as described in Eqn (10). We modelled R_0 as a function of MPD, species richness (S) and Imbalance (Ic), capturing the key axes of variation in our PCAs (SI Figure 3). There was strong covariation between MPD and PD, we report multivariate models for MPD here as it is independent of species richness, S; however, models with PD were qualitatively similar.

In the single-predictor models (Table 3), PD explained the most variation in R_0; more phylogenetically diverse communities generally had lower disease outbreak potentials. MPD was the second strongest predictor of R_0 (Table 3), with communities composed of less closely related species having a lower outbreak potential (Figure 2A). We show that R_0 also decreases with increasing host richness, S, reflecting decreasing contact rates (more species translates into more individuals, thus less contacts per individual). Imbalance (Ic) is positively correlated with R_0, indicating that more balanced trees have a higher outbreak potential, the correlation is relatively weak (Figure 2B).

Of the scaled predictors in our multivariate model, MPD was the best predictor of R_0 (Table 3), but both S and Ic were also significant. Our full model was able to explain a high proportion of the variation ($R^2 = 0.91$) in R_0 across simulations, suggesting that these relatively common phylogenetic indices are able to reliably capture the underlying disease transmission probabilities in our simulations.

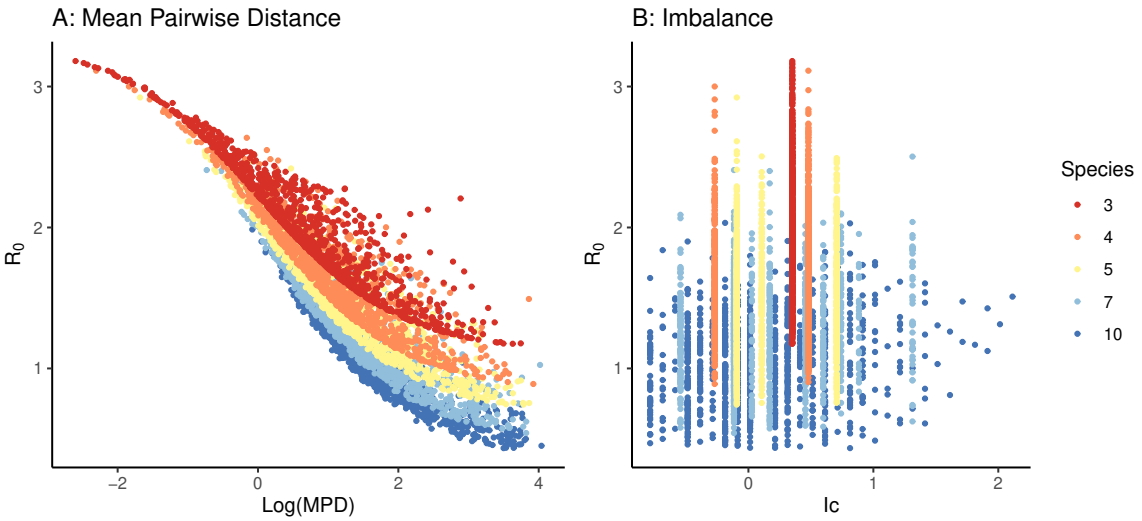

Figure 2: Scenario 1 (no primary host). A: Mean Pairwise Distance (MPD) of the community is inversely related to R_0. B: Imbalance of the phylogenetic tree representing the host species community shows high variation in its effect on R_0, with increasing variation when species richness, S, is increased. Data from simulations with $\psi = 1$, $\delta = 0.1$, $\mu = 0.2$ and $\lambda = 1$. Colours indicate simulated communities with different host richness (S=3,4,5,7,10).

Scenario 2: Single primary host - *Distance from primary host*

When we specify a primary host, the establishment probability of the disease (c) depends on the phylogenetic distance between the receiving and the primary host. Here, we quantify the phylogenetic isolation of the primary using the metric of Evolutionary Distinctiveness (ED) using the fair proportions method (Isaac et al., 2007).

In the 3-taxon case (as illustrated in Figure 1), when the primary host is nested within the ingroup (Scenario 2a), the distance between primary host (P) and the most proximate secondary host (2), is the main driver of disease dynamics (Figures 3A and 3B), whereas the more distant secondary host (1) contributes little to the outbreak potential because its further distance from the primary host reduces transmission probability. As the phylogenetic distance between the primary and proximate secondary host decreases ($PPd_{2P} \rightarrow 0$), the model converges to a two-host system. Similarly, when the primary host is sister to both secondary hosts (Scenario 2b), both secondary hosts are equidistant from the

primary host (Figure 3C), and therefore show little - to no - variation. However, as the distance between the primary host and secondary hosts (PPd_{P2} or PPd_{P3}) increases, the system effectively converges on a one-host system, as successful transmission from the donor (P) to either secondary hosts becomes increasingly improbable.

In our single predictor regressions, PD again explains the most variation in R_0, although marginally less than for Scenario 1 ($R^2 = 0.89$ and 0.84, for Scenario 1 and Scenario 2, respectively); however, ED can also explains a great part of the variation ($R^2 = 0.69$; Table 3), with R_0 decreasing with increasing host ED (Figure 4).

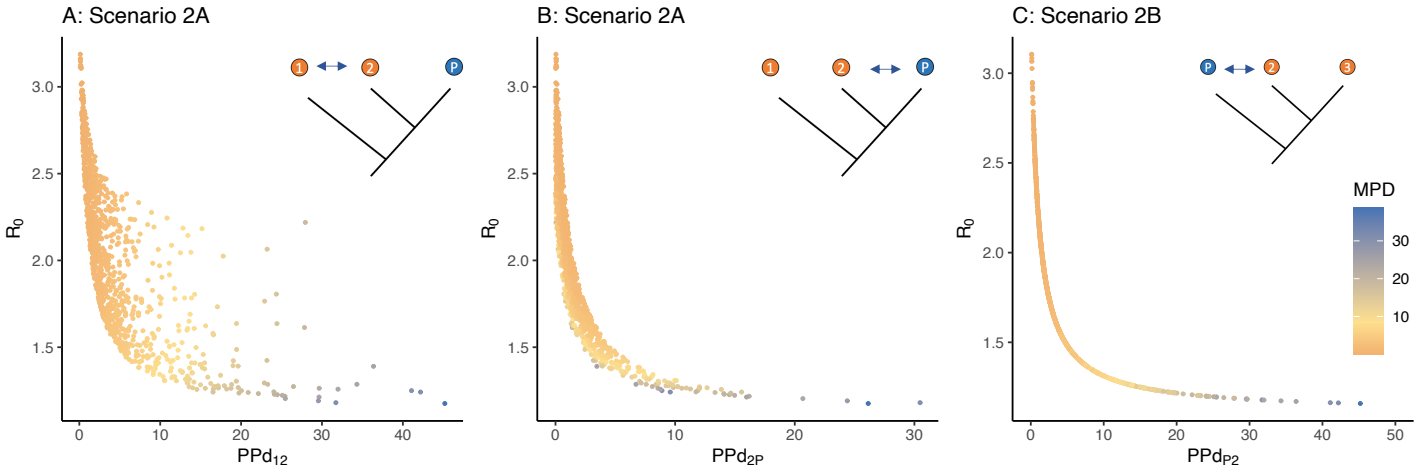

Figure 3: Community disease outbreak potential (R_0) plotted against pairwise phylogenetic distance between hosts. Shading is proportional to MPD. A: Scenario 2a, with the primary host nested within the secondary hosts, showing pairwise distance between the two secondary hosts. Here, there is large variation in R_0 amongst simulations as disease dynamics are determined mainly by the phylogenetic distance between the primary and most proximate secondary host (2), capturing indirectly the effect of the primary host's location. B: Scenario 2a, showing the effect of the distance between the primary and most proximate secondary host (2) affecting R_0 directly. C: Scenario 2b, with the primary host sister to both secondary hosts, illustrating little variation in R_0 across simulated trees, as disease dynamics are determined by the primary host's distance to both secondary hosts, which are equidistant from the primary host, and thus the system converges on a simple two-host model, and R_0 is effectively determined by tree depth. Simulated data from the model with $\psi = 1$, $\delta = 0.1$, $\mu = 0.2$, $\lambda = 1$.

Fitting the matching multiple regression model from Scenario 1 to data from Scenario 2, our explanatory power is notably lower ($R^2 = 0.85$), and Ic is no longer a significant predictor in the model. However, when we substitute MPD for ED, we increase our explained variance to 93%, the highest across either scenario, with all parameters significant (Table 3). Effect sizes for Ic also increase, although only slightly (SI Figure 3 and Table 3). ED has the

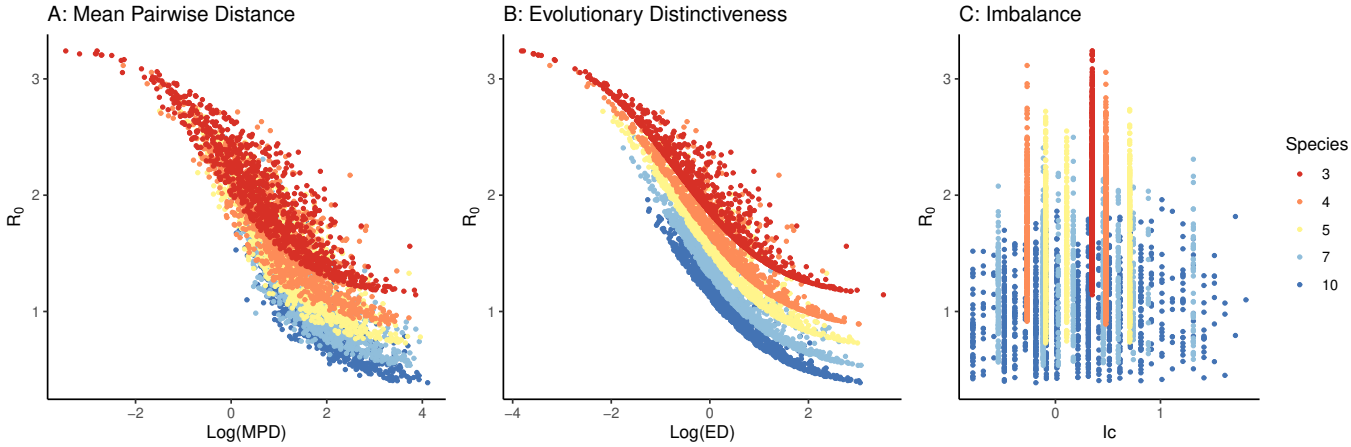

Figure 4: Scenario 2 (single primary host). A: Relationship between R_0 and the Mean Pairwise Phylogenetic Distance (MPD) between host species, illustrating outbreak potential decreasing logistically with MPD. B: Relationship between R_0 and the Evolutionary distinctiveness (ED) of the primary host, demonstrating similar dynamics to MPD. C: Relationship between R_0 and phylogenetic tree Imbalance (Ic), illustrating a weak but significant trend for R_0 to decrease with increasing tree balance, and showing larger variation in Ic with greater species richness (S, in shading). Data from simulated communities with host species richness of $S =$ 3, 4, 5, 7 and 10, using $\psi = 1$, $\delta = 0.1$, $\mu = 0.2$, $\lambda = 1$.

largest standardized effect size.

	Scenario 1		Scenario 2	
Variable	Estimate effect on R_0	R^2	Estimate effect on R_0	R^2
log(PD)	-0.44	0.89	-0.45	0.84
log(MPD)	-0.46	0.77	-0.47	0.73
Ic	0.29	0.05	0.21	0.02
S	-0.13	0.40	-0.13	0.37
log(ED)	-	-	-0.47	0.69
Multivariate	1.49-0.38log(MPD)-0.19S+0.02Ic	0.91	1.46-0.41log(ED)-0.26S+0.03Ic	0.93

Table 3: Models, uni- and multivariate, describing the variation in R_0 using various phylogenetic metrics for simulations of Scenario 1 and 2, using $\delta = 0.1$, $\mu = 0.2$ and $\lambda = 1$ in simulated communities with a host species richness of 3, 4, 5, 7 and 10 species. Table shows the standardized effects size of each metric and its explanatory power for R_0. All parameter estimations were significant.

4 Discussion

We are witnessing a substantial rearrangement of biological diversity. Many species are threatened with extinction, while others are expanding their global distributions, sometimes invasively (IPBES, 2020; Barrett et al., 2018; IUCN, 2021). There is an urgent need to understand how these biotic changes might restructure host communities through altering environmental carrying capacities and species range-shifts, and the consequences of these changes on the maintenance and potential spillover of pathogens to novel hosts and to altered host communities (Daszak et al., 2001; Webb et al., 2002; Williams and Jackson, 2007; Faust et al., 2018). The potential dilution and amplification effects of biodiversity on disease prevalence have been a topic of controversy in many systems (Ostfeld and Keesing, 2012; Halliday and Rohr, 2019). Empirical studies provide evidence for both processes, for example, increasing amphibian species richness has been linked to greater fungal pathogen prevalence (Becker and Zamudio, 2011), but reduced flatworm (*Ribeiroia*) prevalence (Johnson et al., 2012). However, the mechanisms underlying both processes are often unclear, and there remains debate on whether increasing biodiversity will more often dilute versus amplify pathogen prevalence.

Part of the explanation for the mixed support for dilution versus amplification effects likely reflects differences in disease competence among host species. The addition of highly competent hosts to the community will generally promote amplification, while the addition of lower-competent hosts or strong interspecific competitors (that can reduce the abundance of high-competent hosts) will tend to promote dilution (Cortez and Duffy, 2021). Thus, generating accurate predictions of how changes in host diversity will impact disease prevalence thus requires data on host competence, which we lack for most species.

Here, we develop a novel framework that builds upon recent observations that disease transmission in multi-host systems is often strongly phylogenetically structured. Using simulation of a compartmental SI disease model, and weighing the probability of successful transmission by the pairwise phylogenetic distance between species, we show that simply by altering the phylogenetic relationships among host species, the community's disease outbreak potential (R_0) can switch between amplifying ($R_0 > 1$) to diluting ($R_0 < 1$) the probability of outbreak, while species richness remains the same. Our model highlights that adding a closely related host to a community will more likely amplify a disease, while adding a distant relative will more likely dilute it. When we include primary-secondary host dynamics, we additionally show that that it is not only the phylogenetic structure of the host community that determines the outbreak potential of the pathogen, but also the phylogenetic placement of the primary host. We

were able to explain up to 93% of the variation in the community R_0 using commonly-used phylogenetic diversity metrics that captured axes of tree topology and phylogenetic clustering. While the contribution of host phylogenetic structure to ecosystem functioning has been well studied (e.g. Cadotte et al., 2008) and there is increasing evidence for phylogenetic signals in disease transmission (Parker et al., 2015), these relationships have largely been explored independently; here, we suggest that including the phylogenetic structure of host communities into disease models could help contribute to disease management.

The assumption implemented here, that the transmission of diseases among host species co-varies with the phylogenetic distance that separates them (Davies and Pedersen, 2008; Parker et al., 2015; Farrell and Davies, 2019b; Stephens et al., 2019; Wang et al., 2019; Gougherty and Davies, 2021), argues that the phylogenetic signal in transmission reflects the tendency for closely related species to share similar immune systems, inherited, with modification, from a common ancestor. In other words, the likelihood of establishment success of a disease in a novel host is correlated with the immuno-compatibility and phylogenetic identity of (donor and recipient) hosts (Davies and Pedersen, 2008; Kuiken et al., 2006; Streicker et al., 2010; Gilbert et al., 2012; Huang et al., 2014). It has already been established that the genetic diversity of host populations can affect disease outbreak potential (Altizer et al., 2003) and that R_0 may be inversely related with the number of host genotypes (Lively, 2010). We extend this reasoning to encompass host phylogenetic diversity and show that under assumptions of phylogenetic conservatism in host competence, community R_0 is lower in communities with higher host species richness and greater host phylogenetic diversity, indexed by the phylogenetic pairwise distances separating hosts in the community.

We investigated two scenarios: First, we assume transmission success is simply weighted by the phylogenetic distance between donor and recipient hosts (Scenario 1), which we argue to resemble Rabies virus dynamics, given its generalism to host species and fast mutation rates, allowing it to co-evolve with its host (Woolhouse et al., 2001; Mollentze et al., 2014). However, the majority of disease systems likely have more complex primary-secondary host dynamics, in which one or a few host species are the primary (or reservoir) hosts, and host competence co-varies with phylogenetic distance between the secondary (recipient) and primary host (see also Farrell and Davies, 2019b). Our example of bTB best describes this phenomenon. The disease most recently spilled over from cattle imported from Europe into sub-Saharan wildlife, where it became endemic in African buffalo, resembling Scenario 2; It established well in closely related ungulates, with occasional spill-overs to distantly related species (Michel et al., 2006).

In both scenarios, an increase in species richness (S) decreased the R_0, as is expected in frequency- and density-dependent transmission models (Rudolf and Antonovics, 2005; Dobson, 2004). With a constant contact rate per time unit, the addition of a low competent host may reduce contacts among high competent hosts and thus lower overall disease prevalence (Ostfeld and Keesing, 2000; Dobson, 2004; Rudolf and Antonovics, 2005; Keesing et al., 2006). Often, wildlife diseases are modelled using density-dependence (White et al., 2017); however, at higher host densities contacts tend to saturate (Antonovics et al., 2017), and thus we might expect a shift from density- to frequency-dependence (constant contact rate per time unit) at high population densities, allowing for the possibility of dilution even for pathogens more often described as density-dependent. Phylogenetic tree shape was also weakly correlated with disease outbreak potential in both scenarios (more balanced trees reducing community R_0), indicating that a more balanced tree decreases the outbreak potential. A highly imbalanced tree is characterized by one or a few clades of very closely related species, which are more likely to have similar immune-competence, and thus easily share a disease, we suggest this parallels the *phylogenetic clade effect*, (as described by Wang et al., 2019). However, in the absence of an assigned primary host, by far the greatest predictor of community R_0 was the mean of the pairwise phylogenetic distance between hosts in the community (MPD). Communities composed of more closely related hosts had higher outbreak potentials than communities composed of more distantly related hosts because the average transmission success between recipient and donor hosts will be relatively high in the former and low in the latter. To compare, including a primary host in the model (Scenario 2), we are again able to explain most of the variation in community R_0 using the three variables species richness, tree imbalance, and phylogenetic distance between hosts, but the evolutionary isolation (measured as Evolutionary Distinctiveness; ED) of the primary host served better as a predictor than MPD between all hosts in the community. This also showed that the community R_0 is lower when the primary host is phylogenetically isolated (greater ED) from secondary hosts, and higher when the primary host is phylogenetically close (lower ED) to secondary hosts. We emphasize that both scenarios have high R^2 due to the underlying assumptions of transmission in this model, and could also be explained by the symmetry in communities, due to the equal population sizes and contact rates.

The amplitude of interspecific transmission of hosts plays an important role in the definition of dilution and amplification as well and an increase of interspecific transmission can cause species losses to result more often in amplification than dilution (Dobson, 2004; Joseph et al., 2013). For simplicity, we modelled the intra- and interspecific contact rates equally; however this could be easily modified if different assumptions were a better fit to our understanding of a particular empirical system. We show in SI section 3 how the R^2 increases when we switch to more realistic contact

structures, where the interspecific contact rates are reduced to up to 10% of the intraspecific contacts, relatively. An additional simplification in our model was assuming a deterministic relationship between phylogenetic relationship between hosts and their potential to share a disease, thus ignoring potential for stochastic dynamics, which can have significant impacts on disease outbreaks dynamics(Wolfe et al., 2005), and variation in species densities, which may contribute to dilution and amplification effects (Dobson, 2004; Keesing et al., 2006; Cohen et al., 2022). Although beyond the scope of the present study, extending the models we present here to explore these additional complexities could further elucidate the consequences of shifting host community restructuring on infectious disease dynamics.

We are currently experiencing accelerating rates of species loss, climate change and species invasions, all of which have been linked to changes in disease outbreak potential (Daszak et al., 2001; Barrett et al., 2018; IPBES, 2020; IUCN, 2021; UN, 2020; Rohr et al., 2020; Mora et al., 2022). Here, we have illustrated how shifts in host community structure might also impact disease dynamics. Our simulations, based upon the reasonable assumptions that spillover of pathogens is more likely between closely related hosts, highlight how the phylogenetic structure of host communities might mediate risk of disease outbreaks. Communities that are more phylogenetically diverse are also more likely to reduce disease outbreak potential (see also Davies and Pedersen, 2008; Parker et al., 2015; Farrell and Davies, 2019b; Wang et al., 2019, 2021). However, there is evidence that more evolutionarily distinct specie—those adding the greatest phylogenetic diversity to communities—might also be more at risk of extinction (Daru et al., 2013), suggesting that extinction might shift communities towards increased potential for disease outbreaks. In addition, the non-random distribution of extinction on the tree-of-life suggests that, at least at local scales where disease dynamics play out, we risk losing a disproportionate amount of phylogenetic diversity if currently threatened species are lost to extinction (Huang et al., 2012). Thus, we risk losing the species and community attributes most likely to suppress disease, while the species less at risk of extinction tend to be those associated with faster life-histories, and higher disease competence due to lower investment in pathogen defense (Joseph et al., 2013; Gibb et al., 2020; Halliday et al., 2020; Valenzuela-Sánchez et al., 2021).

Through the increase of livestock numbers and decrease of wildlife abundance communities homogenize (Bar-On et al., 2018). Additionally, communities re-assemble through climate change (Williams and Jackson, 2007). Therefore, we should further investigate how climate change, extreme climate events, and biodiversity decline affect disease outbreaks. Conserving and phylogenetic diversity could prevent future major outbreaks, and we argue that, given the relationship between phylogenetic community structure and disease transmission in wildlife, shifts in community

structure will be of importance in predicting the distribution of disease and potenitally future host shifts.

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