Biodiversity and the Ecology of Emerging Infectious Diseases

M. G. Roberts and J. A. P. Heesterbeek

Abstract The question of how biodiversity influences the emergence of infectious diseases is the subject of ongoing research. A set of nonlinear differential equations is been used to explore the interactions between ecology and epidemiology. The model allows for frequency-dependent transmission of infection within host species, and density-dependent transmission between species, via the environment or a vector. Three examples are discussed. It is shown that removing a pathogen may increase a consumer population, decreasing its resource. It is then shown that the presence of a pathogen could enable a predator and a prey species to coexist. Finally the dilution effect, by which increasing biodiversity reduces the transmission of an infectious disease, is investigated.

Keywords Biodiversity · Ecology · Epidemiology · Infectious diseases

1 Introduction

Emerging infection diseases present a major threat to world health. On average, two new species of human virus are reported each year [1], most having an animal origin [2–5]. Recent examples are SARS [6], swine flu [7] and avian influenza [8, 9]. In 2014, Ebola virus re-emerged from a bat reservoir [10, 11], causing a major epidemic [12–15]. Climate change could lead to *Aedes* mosquitoes establishing in New Zealand [16, 17], and with them dengue and Zika viruses [18].

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Large complex ecosystems interacting at random are almost certain to be unstable [19]. Adding structure to the community matrix produces a different picture [20–23], competitive interactions are stabilising, whereas mutualism is destabilising [20]. A major component of an ecosystem is the food web: the network of feeding inter-actions among species. Adding pathogens increases the web's complexity [24–28], and parasites have been described as the *dominant* or *missing* links [29, 30]. An infection may make prey easier to catch, or unpalatable to a predator, or reduce a predator's hunting ability, but the overall influence of pathogens on an ecosystem may be unexpected [31]. The influence of ecosystem dynamics on epidemiology can also be unexpected [32] and may lead to a pathogen *jumping* host species causing a pandemic.

We present a model that describes how an infectious disease can modify the dynamics of host and non-host species, and how changes in ecosystem dynamics can modify the epidemiology of a pathogen. We illustrate our model with three examples. In the first, eliminating a pathogen led to an increase in biodiversity, whereas in the second the presence of a pathogen is necessary to maintain a prey–predator relationship. The third example directly addresses the dilution effect—how a change in biodiversity may result in a change in the dynamics of an infectious disease.

2 The Model

To model an infectious disease on a food web infected with a single pathogen of interest, we define N_i to be the abundance of species $i \in \Omega$, I_i/N_i to be the proportion of species i infected, and $S_i = N_i - I_i$ the abundance of susceptible hosts of species i. The equations for the population dynamics of the food web are

$$
\begin{aligned}
\frac{\mathrm{d}N_i}{\mathrm{d}t} = {} & \nu_i(N_i)N_i - \mu_i(N_i)N_i - \alpha_i I_i - \sum_{j \in \mathscr{N}_i} \phi_{ij} \left(S_i + n_{ij}I_i\right)\left(S_j + o_{ij}I_j\right) \\
& - \sum_{k \in \mathscr{P}_i} \psi_{ik} \left(S_i + q_{ik}I_i\right)\left(S_k + p_{ik}I_k\right) + \sum_{\ell \in \mathscr{Q}_i} \pi_{\ell i}\psi_{\ell i}\left(S_i + p_{\ell i}I_i\right)\left(S_\ell + q_{\ell i}I_\ell\right),
\end{aligned}
$$

where species i is born at the rate ν_i and dies at the rate μ_i, both functions of N_i, with increased death rate due to infection α_i. Species i competes for resources with species j when $j \in \mathscr{N}_i$, is eaten by species k when $k \in \mathscr{P}_i$ and eats species ℓ when $\ell \in \mathscr{Q}_i$. The variables ϕ_{ij}, ψ_{ik} and $\pi_{\ell i}$ account for competition for resources between species i and j, consumption of species i by species k and the benefit to species ℓ of consuming species i, respectively. All of these interactions may be modified if one or other of the species is infected with the pathogen.

The dynamics of the pathogen are expressed by

$$\frac{dI_i}{dt} = \beta_i \frac{S_i I_i}{N_i} - \mu_i(N_i)I_i - \alpha_i I_i - I_i \sum_{j \in \mathscr{N}_i} \phi_{ij} n_{ij} \left(S_j + o_{ij} I_j \right)$$

$$- I_i \sum_{k \in \mathscr{P}_i} \psi_{ik} q_{ik} \left(S_k + p_{ik} I_k \right) + S_i \sum_{\ell \in \mathscr{Q}_i} \gamma_{\ell i} q_{\ell i} \psi_{\ell i} I_\ell + \kappa_i S_i W$$

The model allows for three modes of transmission: frequency-dependent intra-species transmission at rate β_i, density-dependent transmission via the environment or an infected vector $\left(W = \sum_{m \in \Omega} r_m I_m \right)$ and transmission from prey to predator while feeding.

There are usually multiple steady states. The structure of the Jacobian matrix at the infection-free steady state decouples criteria for **ecological stability** and **epidemiological stability** [33].

$$\mathbf{J} = \begin{pmatrix} \mathbf{C} & \mathbf{D} \\ \mathbf{0} & \mathbf{H} \end{pmatrix}$$

The steady state is ecologically stable if the maximum real part of the eigenvalues of the community matrix is negative, $s(\mathbf{C}) < 0$. The steady state is epidemiologically stable if $s(\mathbf{H}) < 0$. The matrix $\mathbf{H}$ determines the stability of an ecological equilibrium to invasion by an infectious disease in *chronological time*. It can be decomposed $\mathbf{H} = \mathbf{T} + \mathbf{\Sigma}$ where $\mathbf{T}$ is the transmission matrix for the pathogen and $\mathbf{\Sigma}$ is the transition matrix. The **next-generation matrix** is $\mathbf{K} = -\mathbf{T}\mathbf{\Sigma}^{-1}$, and the basic reproduction number $\mathscr{R}_0$ is the spectral radius of $\mathbf{K}$ [34, 35]. If $\mathscr{R}_0 > 1$, the pathogen can invade the food web, and hence $\mathbf{K}$ determines epidemiological stability of the ecosystem *in generation time*.

Example 1 **A resource–consumer–pathogen system**. For this simple example, we assume that the pathogen only infects the consumer. The equations are

$$\frac{dN_1}{dt} = \nu_1(N_1)N_1 - \mu_1 N_1 - \psi N_1 (S_2 + pI_2)$$

$$\frac{dN_2}{dt} = \nu_2(N_2)N_2 + \pi \psi N_1 (S_2 + pI_2) - \mu_2 N_2 - \alpha I_2$$

$$\frac{dI_2}{dt} = \beta \frac{S_2 I_2}{N_2} - (\mu_2 + \alpha) I_2$$

The Jacobian matrix at any infection-free state $(N_1, N_2, 0)$ simplifies to

$$\mathbf{J} = \begin{pmatrix} N_1 \nu_1'(N_1) & -\phi N_1 & \psi(1-p)N_1 \\ \pi \psi N_2 & N_2 \nu_2'(N_2) & -\pi \psi (1-p) N_1 - \alpha \\ 0 & 0 & \beta - \mu_2 - \alpha \end{pmatrix}$$

The community matrix $\mathbf{C}$ is the leading 2×2 sub-matrix of $\mathbf{J}$. It has negative trace and positive determinant, and hence it is stable. The infection-free equilibrium is unstable if $\mathscr{R}_0 = \frac{\beta}{\mu_2 + \alpha} > 1$. The steady states of the system are plotted as functions
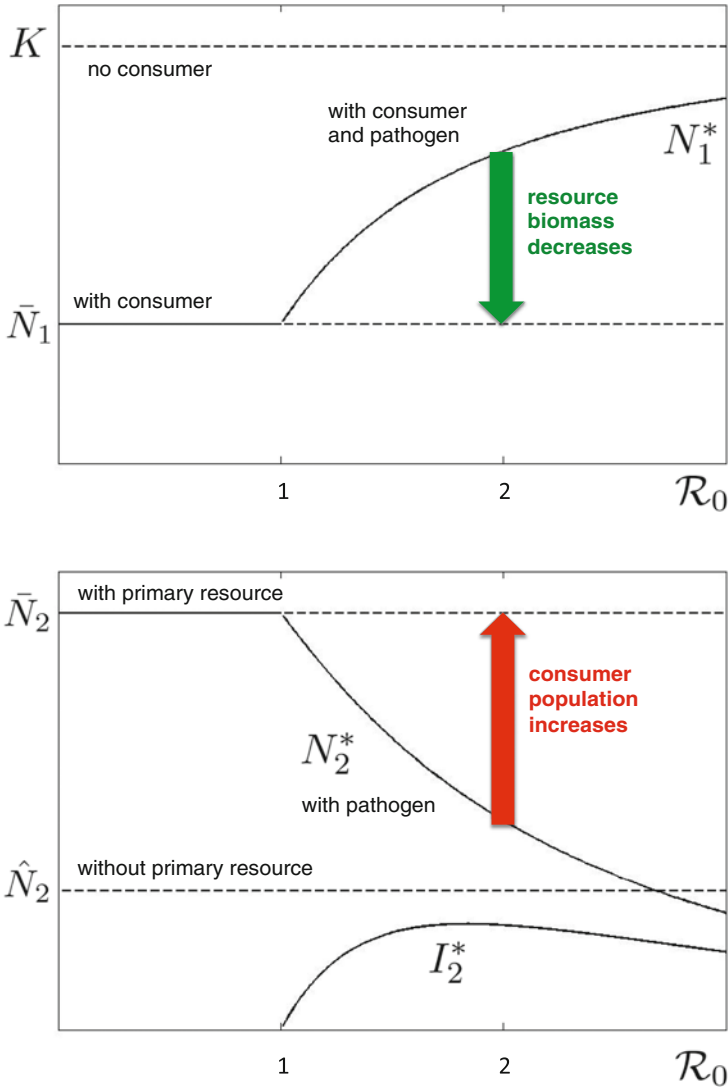
Fig. 1 Bifurcation diagram for the model presented in Example 1. Top: steady states for the resource plotted against the basic reproduction number $\mathcal{R}_0$. K is the steady state in the absence of the consumer, $\bar{N}_1$ in the presence of the consumer and N_1^* in the presence of consumer and pathogen. Bottom: steady states for the consumer plotted against $\mathcal{R}_0$. $\hat{N}_2$ is the steady state in the absence of the primary resource, $\bar{N}_2$ in the presence of the resource and N_2^* in the presence of resource and pathogen. I_2^* is the abundance of the pathogen. The effect of removing the pathogen is indicated by the arrows

of $\mathscr{R}_0$ in Fig. 1. Eliminating the pathogen from the consumer increases its abundance from N_2^* to $\bar{N}_2$. As a consequence, the resource biomass decreases from N_1^* to $\bar{N}_1$. This is consistent with observations in the Serengeti. Following the eradication of rinderpest, wildebeest numbers increased and the grass biomass decreased. As a consequence there were fewer fires, more trees, more giraffes and more predators [36]. These are further interactions that could have been included in a larger model.

Example 2 **A prey–predator–pathogen system**. For this example, the pathogen infects both prey (species 1) and predator (species 2), with transmission from predator to prey via environmental contamination. The model is

$$
\frac{dN_1}{dt} = v_1(N_1)N_1 - \mu_1 N_1 - \alpha_1 I_1 - \psi\,(S_1 + q I_1)\,(S_2 + p I_2)
$$

$$
\frac{dN_2}{dt} = v_2(N_2)N_2 + \pi\,\psi\,(S_1 + q I_1)\,(S_2 + p I_2) - \mu_2 N_2 - \alpha_2 I_2
$$

$$
\frac{dI_1}{dt} = \beta_1 \frac{S_1 I_1}{N_1} - (\mu_1 + \alpha_1)\,I_1 - \psi q I_1\,(S_2 + p I_2) + \kappa S_1\,(I_1 + r I_2)
$$

$$
\frac{dI_2}{dt} = \gamma q \psi I_1 S_2 + \beta_2 \frac{S_2 I_2}{N_2} - (\mu_2 + \alpha_2)\,I_2
$$

The basic reproduction number is the spectral radius of the next-generation matrix, $\mathscr{R}_0 = \rho\,(\mathbf{K})$, where

$$
\mathbf{K} = \begin{pmatrix} \frac{\beta_1}{\mu_1 + \alpha_1 + \psi q \bar{N}_2} + \frac{\kappa \bar{N}_1}{\mu_1 + \alpha_1} & \frac{\kappa \bar{N}_1}{\mu_2 + \alpha_2} \\ \frac{\gamma q \psi \bar{N}_2}{\mu_1 + \alpha_1 + \psi q \bar{N}_2} & \frac{\beta_2}{\mu_2 + \alpha_2} \end{pmatrix}
$$

In the absence of prey–predator interaction, $\psi = 0$. The basic reproduction number in the prey is then $\mathscr{R}_0 = \frac{\beta_1 + \kappa \widehat{N}_1}{\mu_1 + \alpha_1}$, and in the predator $\mathscr{R}_0 = \frac{\beta_2}{\mu_2 + \alpha_2}$. The possible steady states of the prey species are $\widehat{N}_1$ without predators; $\bar{N}_1$ with predators; N_1^* without predators with pathogen; and N_1^{**} with predators and pathogen. These are plotted as functions of the feeding rate ψ in Fig. 2. When the feeding rate of the predator is greater than a critical value ($\psi > \psi_{\text{crit}}$), the prey species is driven to extinction unless the pathogen is present. Hence, the presence of the pathogen is necessary to keep the prey population viable.

Example 3 **The dilution effect**. Resolving the situations under which the dilution effect applies is an outstanding challenge in epidemiology [37]. The idea is that reducing biodiversity removes species that are hosts of a particular pathogen, hence increasing the risk of transmitting that pathogen to a new host, notably a human. The alternative is that removing hosts from an ecosystem reduces the viability of the pathogen, possibly driving it to extinction. A simple model with two prey species (1 and 2), two predator species (3 and 4) and one pathogen has been used to explore the dilution effect. The host population dynamics are described by
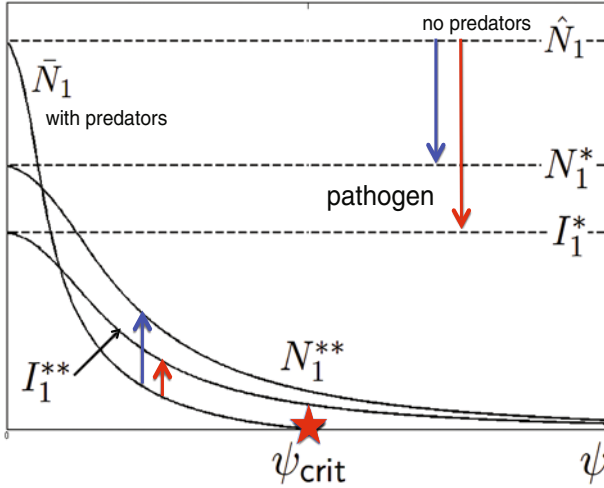
Fig. 2 Steady states of the prey population (species 1) in Example 2 plotted against the feeding rate of the predator ψ. The states are $\hat{N}_1$ without predators; $\bar{N}_1$ with predators; N_1^* without predators with pathogen; and N_1^{**} with predators and pathogen. The abundance of infected prey is I_1^* or I_1^{**} with predators. The effect of introducing the pathogen is indicated by the arrows

$$\left.\frac{\mathrm{d}N_i}{\mathrm{d}t}\right|_{i=1,2} = \nu_i N_i - \mu_i N_i - N_i \sum_{j=1,2} \phi_{ij} N_j - N_i \sum_{k=3,4} \psi_{ik} N_k$$

$$\left.\frac{\mathrm{d}N_i}{\mathrm{d}t}\right|_{i=3,4} = \nu_i N_i - \mu_i N_i - N_i \sum_{j=3,4} \phi_{ij} N_j + N_i \sum_{\ell=1,2} \pi_{\ell i} \psi_{\ell i} N_\ell$$

and the abundance of infected hosts by

$$\left.\frac{\mathrm{d}I_i}{\mathrm{d}t}\right|_{i=1,2} = \beta_i \frac{S_i I_i}{N_i} - \mu_i I_i - I_i \sum_{j=1,2} \phi_{ij} N_j - I_i \sum_{k=3,4} \psi_{ik} N_k + \kappa_i S_i W$$

$$\left.\frac{\mathrm{d}I_i}{\mathrm{d}t}\right|_{i=3,4} = \beta_i \frac{S_i I_i}{N_i} - \mu_i I_i - I_i \sum_{j=3,4} \phi_{ij} N_j + S_i \sum_{\ell=1,2} \gamma_{\ell i} \psi_{\ell i} I_\ell + \kappa_i S_i W$$

Preliminary results based on frequency-dependent transmission only show no dilution effect in prey species and an effect in predator species if increasing the prey population leads to an increased predator population. Adding transmission from prey to predator while feeding means the effects of population increase must exceed the effects of transmission to susceptibles through consuming prey. The results are more complicated with density-dependent transmission. We found a dilution effect in prey species in response to environmental dilution and a dilution effect in predator species under some restricted conditions. Exploring these effects is the subject of ongoing research.

3 Conclusion

A model described by a set of nonlinear differential equations has been used to explore the interactions between ecology and epidemiology. Three examples have been discussed. In the first, it was shown that removing a pathogen increased a consumer population and decreased the resource. The second example showed that the presence of a pathogen could enable a predator and prey species to coexist. Finally, the complex issue of the dilution effect was addressed. The question of how biodiversity influences the emergence of infectious diseases is the subject of ongoing research.

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