Competent hosts and endemicity of multi-host diseases

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Abstract

Keesing, Felicia, et al. discuss in [6] how loss in biodiversity could entail amplification of vector-borne disease. In [6] it is also discussed the relation between resistance of a hosts to difficult conditions and its effectiveness transmitting the disease. In this paper we propose a method to study a simple vector-hosts mathematical model based on a system of ordinary differential equations to show how the loss of biodiversity could influence the dynamics of the disease in different scenarios determined by the resistance of the hosts. Based on our model, we find that if the number of contacts are diluted by the whole amount of hosts, the presence of a competent host is a necessary condition for the existence of an endemic state. In addition, the model also shows how an increase in resilient and competent hosts has a greater impact in the amplification of the disease. This result fits with the amplification observed in several diseases where the most competent hosts tend to be also the most resilient.

1 Introduction

The abundance of hosts of a vector-borne disease could influence the amplification or dilution of the infection. In [6] it is discussed several examples of this influence. For instance, West Nile virus is a mosquito-transmitted disease and it has been shown that there is a correlation between low bird density and amplification of the disease in humans [10]. Another example is the Lyme disease in North America, which is transmitted by the blacklegged tick *Ixodes pacificus*. This disease has the white-footed mouse *Peromyscus leucopus* as competent host, i.e, a host that effectively transmits the disease. Those mice are abundant in either low-diversity or high-diversity ecosystems. On the other hand, the opossum *Didelphis virginiana* is poor in low-diversity forest and is a suboptimal hosts, i.e, it does not effectively transmit the disease. Therefore, loss in biodiversity could entail a drop in the suboptimal reservoirs and because these act as a buffer of the disease by increasing the mortality of the ticks, this loss amplifies the disease. In [3] it is discussed the diluting effect of the individual and collective addition of suboptimal hosts. For example, the transmission of *Schistosoma mansoni* to target snail hosts *Biomphalaria glabrata* is diluted by the inclusion of decoy hosts. These decoy hosts are individually effective to
dilute the infection. However, it is interesting to notice that their combined effects are less than additive [4], [5]. Johnson et al. also discuss how the high diversity of parasites could lead to dilution of a disease. In [4] it is shown how increases in trematode parasites diversity dilutes the infection in an amphibian host. In consequence, the loss in biodiversity of hosts and parasites could have an amplifying effect on the spread of the infection. In this paper, we give a theoretical support to this hypothesis regarding the host diversity.

In [6] it is hypothesized that a greater resistance to a poor biodiversity could entail a greater susceptibility to parasites. For example, plants that are fast growing and nutrient-rich, such as weedy plants, are also more resilient to poor environments [1]. In addition, these plants are optimal hosts for arthropod vectors and pathogens. In poor environments, those plants are likely to increase in number and amplify the disease. In vertebrates organisms, species with adaptation from their high reproduction rates in poor environments tend to invest minimally in immune adaptations. In that way, those species are more susceptible and effective to transmit the infection (17).

The objective of this work is to study the behavior of a vector-borne disease with multiple hosts when changes in biodiversity occur. We use in our study a dynamical system to model a vector-borne disease with multiple hosts that was created taking ideas from [2]. We define competent and suboptimal host using the basic reproductive number of the cycle formed by the host and the vector. We also assume that the abundances of the hosts follow a conservation law and from this law we define what a resilient species is. The existence of a conservation law is observed in dynamics such as Lotka-Volterra [8], and it attempts to capture how a disturbance of the ecosystem leads to changes in the density of the hosts in different proportions. In this way, we can measure the effect on the dynamics of the disease due to different changes in the biodiversity. These scenarios would be determined by the effectiveness of the hosts to transmit the disease and the resistance of the hosts to biodiversity changes. We are interested in the case where the disease is endemic. In particular, in this case the disease free equilibrium is not stable and therefore the basic reproductive number is at least one [11].

In Section 2 we present the variables and the equations of the model. Section 3 is divided in Subsection 3.1 and Subsection 3.2. Subsection 3.1 presents how an endemic state entails the existence of competent hosts. In this subsection we also present other properties of the basic reproductive number of the system. Subsection 3.2 presents the community constraints that leads us to the our definition of resilient host. We also present the scenario where there is a single competent host that is resilient. In Section 4 we present the discussion and conclusions of the results presented in the Section 3. In Section 5 we present the mathematical justification of the results in Section 3.
2 The model

We propose a mathematical model of a vector-borne disease that is spread among a vector $V$ and hosts $H_i, i = 1, \ldots, k$. Our model is a system of ordinary differential equations that describes the abundance of infectious hosts $I_{h_i}, i = 1, \ldots, k$ and the abundance of infectious vectors $I_v$. We suppose that all the populations are divided in susceptible individuals ($S_v$ susceptible vectors and $S_{h_i}, i = 1, \ldots, k$ susceptible hosts) and infectious individuals ($I_v$ infectious vectors and $I_{h_i}, i = 1, \ldots, k$ infectious hosts). If $N_v$ and $N_{h_i}, i = 1, \ldots, k$ respectively represent the total abundances of vectors and hosts, we also assume that all these abundances are constant in time, i.e., $\dot{N}_v = \dot{S}_v + \dot{I}_v = 0$ and $\dot{N}_{h_i} = \dot{S}_{h_i} + \dot{I}_{h_i} = 0, i = 1, \ldots, k$. In that way, it only suffices to consider as state variables the number of infectious species. We define the total number of hosts as $N_t := \sum_{i=1}^{k} N_{h_i}$. We assume that there are no intra-species infections. In addition, we assume that there is no interspecies infection between hosts; therefore, the only mean of infection is through contact with the vectors as Fig. 1 shows. The parameters of the model are presented in Table 1 and (1) is the system of differential equations for the infectious populations of hosts and vectors.

\[ \begin{align*}
\dot{I}_{h_i} &= \beta_{vhi} I_v \frac{S_{h_i}}{N_t} - \delta_{h_i} I_{h_i}, i = 1, \ldots, k \\
\dot{I}_v &= \sum_{i=1}^{k} \beta_{hi} I_{h_i} \frac{N_{h_i} S_v}{N_v} - \delta_v I_v 
\end{align*} \]  

(1)

Figure 1: The node $V$ represents the infectious vector and the nodes $H_i, i = 1, \ldots, k$ represent the infectious reservoirs. We assume that the number of contacts between the vector and a hosts are diluted by the total population of hosts. This assumes that the vector does not have preference for a specific host.
Table 1: Parameters of the model described by equations [1].

| Parameter | meaning                                      | Units                        |
|-----------|----------------------------------------------|------------------------------|
| $\beta_{vh_i}$ | Transmission rate from $V$ to $H_i$ in the cycle formed by $V$ and $H_i$. | $[H_i]/([time] \ast [V])$ |
| $\beta_{hi_v}$ | Transmission rate from $H_i$ to $V$ in the cycle formed by $V$ and $H_i$. | $[V]/([time] \ast [H_i])$ |
| $\delta_v$   | Mortality rate of vectors                    | $1/[time]$                  |
| $\delta_{hi}$ | Mortality rate of host $H_i$                 | $1/[time]$                  |

3 Results

3.1 Existence of competent hosts

Let us define

$$(R^h_i)^2 := \frac{\beta_{vh_i} \beta_{hi_v}}{\delta_v \delta_{hi}}$$ (2)

The number $R^h_i$ in (2) is the basic reproductive number of the cycle formed by host $H_i$ and the vector $V$ when $N_{h_i} = N_t$. We will say that a host $i$ is competent if $R^h_i \geq 1$, and the host $i$ is suboptimal if $R^h_i < 1$. In consequence, the host $H_i$ is suboptimal (competent) if the disease free equilibrium is (not) stable when $N_{h_i} = N_t$.

If $D_i := \frac{N_{h_i}}{N_t}$ is the density of the host $H_i$ in the total population of hosts, we obtain the basic reproductive number of the whole system in the equation (3) (see Appendix).

$$R^2_0 = \sum_{i=1}^{k} (R^h_i)^2 D_i^2.$$ (3)

This is a convex function of $D_1, \ldots D_k$. Using the restrictions $\sum_{i=1}^{k} D_i = 1$, $D_i \geq 0, i = 1, \ldots, k$ and Lagrange Multipliers, we get that a minimum for $R_0$ is attained in $(D^*_1, \ldots, D^*_k)$ where

$$(R^h_1)^2 D^*_1 = \ldots = (R^h_k)^2 D^*_k$$

Therefore, we get that

$$D^*_i = \frac{\frac{1}{(R^h_i)^2}}{\sum_{j=1}^{k} \frac{1}{(R^h_j)^2}}$$

and

4
\[(R_0)^2_{\text{min}} = \frac{1}{\sum_{j=1}^{k} \frac{1}{(R_{0j}^h)^2}} = \frac{1}{k} H((R_0^h_1)^2, \ldots, (R_0^h_k)^2) \quad (4)\]

where \(H((R_0^h_1)^2, \ldots, (R_0^h_k)^2)\) is the harmonic mean of \((R_0^h_1)^2, \ldots, (R_0^h_k)^2\). In general, the harmonic mean satisfies

\[\min_{i=1, \ldots, k} \{(R_0^h_i)^2\} \leq H((R_0^h_1)^2, \ldots, (R_0^h_k)^2) \leq \frac{1}{k} \min_{i=1, \ldots, k} \{(R_0^h_i)^2\} \]

In consequence,

\[\frac{1}{k} \min_{i=1, \ldots, k} \{(R_0^h_i)^2\} \leq (R_0)^2 \leq \min_{i=1, \ldots, k} \{(R_0^h_i)^2\} \quad (5)\]

From the inequality (5) we obtain that the existence of a reservoir with \(R_0^h_i < 1\) implies that it is possible to get \(R_0 < 1\). However, if all the reservoirs are effectively transmitting the disease \((R_0^h_i >> 1, i = 1, \ldots, k)\) and there are few host \((k\text{ is small})\), inequality (5) could imply that \(R_0\) does not have values below 1.

Moreover, as the function \((R_0)^2(D_1, \ldots, D_k)\) is convex, we get that

\[R_0^2 \leq \max_{i=1, \ldots, k} \{(R_0^h_i)^2\} \quad (6)\]

The inequality (6) implies that the disease can not be amplified beyond the basic reproductive number of the most competent host. From inequality (6) we also get the Theorem 1.

**Theorem 1** There exist values of \(D_1, \ldots, D_k\) for which \(R_0 \geq 1\) if and only if

\[(R_0)_{\text{min}} < 1 < R_0^h_i\]

for some \(i\). In particular, in model \(\hat{1}\) the endemity of a disease implies the existence of a competent host.

In the case of two host, Fig. 2 represents a contour plot of \(R_0\).
Figure 2: Contour plot for different values of $R_0$ for a two hosts system where there is a competent host (horizontal axis) and a suboptimal host (vertical axis). In the red line $R_0$ takes its minimum value, and as we move away from the red line, $R_0$ increases.

3.2 Community constraints

3.2.1 Linear case

Let us assume that the abundances of all species follow $k-1$ linear constraints as in (7).

$$\sum_{i=1}^{k} \delta_{ij} N_i + \Delta_i = 0, i = 1, \ldots, k-1.$$  \hspace{1cm} (7)

If the matrix $(\delta_{ij})_{i,j\leq k-1}$ is nonsingular, the abundance of all hosts can be explained by the abundance of the host $H_k$ as in (8).

$$N_i = -\delta_i N_k + \eta_i, i = 1, \ldots, k-1$$ \hspace{1cm} (8)

When $\delta_i > 0$ and $\eta_i > 0$, the definition in (8) implies that $N_k$ increases as any of the $N_i$ decreases for $i = 1, \ldots, k-1$.

We say that the host $k$ is the resilient if $\delta_i > 1$ for $i = 1, \ldots, k-1$ and it is not-resilient if $0 < \delta_i < 1$ for $i = 1, \ldots, k-1$. We have that
\[
\frac{dR_0}{dN_k} = D_\pi R_0 = \sum_{i=1}^{k} u_i r_i \tag{9}
\]

where \( u := (-\delta_1, \ldots, -\delta_{k-1}, 1) \) and \( r_i := \frac{\partial R_0}{\partial N_i} = \frac{1}{N_i R_0}((R_0^{h_i})^2 D_i - R_0^2) \).

We define the index
\[
\Gamma_k := \frac{N_k}{R_0} \frac{dR_0}{dN_k} \tag{10}
\]

This index \( \Gamma_k \) measures the sensitivity of \( R_0 \) to changes of the population \( N_k \).

### 3.2.2 General constraints

Let us assume that we have \( k \) hosts and their abundances follow the conservation law given by
\[
\overline{F}(N_1, \ldots, N_k) = (F_1(N_1, \ldots, N_k), \ldots, F_m(N_1, \ldots, N_k)) = (0, \ldots, 0) \tag{11}
\]

where \( m < k \). In consequence, the abundances of all species are in
\[
E := \{ \overline{N} \in \mathbb{R}^k | \overline{F}(\overline{N}) = \overline{0} \}
\]

Under suitable conditions (Implicit function theorem, see [16] in Appendix), we have that
\[
N_i = g_i(N_{m+1}, \ldots, N_k)
\]

for some \( m \) functions \( g_1, \ldots, g_m \) and the derivatives \( \frac{\partial g_i}{\partial N_j} \) can be computed in terms of the derivatives of the functions \( F_1, \ldots, F_m \), for \( i = 1, \ldots, m \) and \( m < j \leq k \) in a neighborhood of \( \overline{N}_0 = (N_1^0, \ldots, N_k^0) \).

If we assume that \( m = k - 1 \), and \( \frac{\partial R_0}{\partial N_k}(\overline{N}_0) \neq 0 \) in a neighborhood of \( \overline{N}_0 \) we have that
\[
N_i = g_i(N_k), i = 1, \ldots, k - 1
\]

Moreover, close to the point \( \overline{N}_0 \) we have the approximation
\[
N_i = g_i(N_k) \approx -\delta_i N_k + \eta_i
\]

where \( \delta_i \) and \( \eta_i \) depend on \( \overline{N}_0 \) and the derivatives of \( F_1, \ldots, F_{k-1} \) in \( \overline{N}_0 \) for \( i = 1, \ldots, k - 1 \) (see Appendix). In consequence, we could locally consider the linear restrictions as in [8].

### 3.3 The case of a single competent host

In Appendix we prove the Theorem 2.
Theorem 2 If $R_0^{h_k} > 1$ and $R_0^{h_i} < 1, i = 1, \ldots, k - 1$, we have that
\[ \frac{\partial R_0^2}{\partial D_k} > 0 \]
and
\[ \frac{\partial R_0^2}{\partial D_i} < 0 \]
for $i = 1, \ldots, k - 1$ when $D_1, \ldots, D_k$ are such that $R_0 \geq 1$. In consequence, if there is a unique competent host and its density increases in an endemic state, the disease will always be amplified.

Considering linear constraints as in (8), for simplicity let us assume that
\[ N_i = -\delta N_k + \eta_i, i = 1, \ldots, k - 1. \]  \hspace{1cm} (12)

In that way, if $\delta < 1$ the host $k$ would not be resilient host. On the other hand, if $\delta > 1$, the host $k$ would be the resilient host. Moreover, the larger $\delta$, the more resilient the host $k$. In Appendix we show that $\Gamma_k$ is an increasing function of $\delta$. Furthermore, taking $D_k$ and $\delta$ large, we have that
\[ \Gamma_k \approx \delta(k - 1). \]  \hspace{1cm} (13)

In consequence, $\Gamma_k$ is larger as $k$, $D_k$ and $\delta$ are large. This means that the more resilient the host $k$ is, the bigger the effect on $R_0$ when host $k$ is abundant. This is represented in Fig. 3 when we only consider two hosts.
Figure 3: In figure (a) the host $H_2$ is the competent host (the slope of the red line is less than one). The blue and the black lines represent the community linear constraints. In the blue line the host $H_2$ is not-resilient, whereas in the black line the host $H_2$ is resilient. In figure (b) the blue line represents $R_0$ when host $H_2$ is the competent and not-resilient and in the black line $H_2$ is competent and resilient. Close to the intersection of both lines (where $D_2 = 0.9$ and $D_1 = 0.1$) the derivative $\frac{dR_0}{dN_2}$ in the black line is greater than in the blue lines. Moreover, $\Gamma_2$ is greater in the black line than in the blue one, as equation (13) states. In this simulation $R_{h1}^0 = 2/3$, $R_{h1}^1 = 4/3$. In figure (c), on the left side of the dashed red line (where the minimum of $R_0$ is attained), there is dilution of the disease if $D_2$ increases. On the right side of the red line, there is amplification of the disease as $D_2$ increases. Moreover, if $R_0 \geq 1$, this amplification is ensured by Theorem 2.
4 Conclusions

In [6] and [4] there is a discussion of the biological mechanisms that make the losses of biodiversity amplify a disease. One of them consists in the decoys and alternative hosts that could dilute the spread of the pathogens as a buffer of the disease. The model of Section 2 implicitly assumes this kind of mechanism by dividing the contact rates by the total amount of hosts.

We found that there is an additivity relation between the basic reproductive number of the whole ecosystem and the cycles between the vector and the hosts (through the harmonic mean). From this relation, we conclude that a necessary condition for the endemicity of a disease is the presence of a competent host. The amplification of the disease depends on the initial conditions and it is not intrinsic of one type of species. Moreover, the high density of a host could lead to amplify the disease if the abundance of the host increases (see Fig. 2). We can find this phenomena when livestock is used as decoy to dilute diseases transmitted by mosquitoes. The high abundance of livestock could eventually increase the density of the vector and in that way the disease is amplified [4]. We also conclude that if the number of host $k$ is greater, the basic reproductive number that could be attained is smaller (see equation (4)). This fits with the fact that suboptimal species could serve as decoys for the pathogens and could lead to dilute the disease.

We conclude that the amplification is greater when a competent host that is resilient and abundant increases (see Fig. 3). This type of species (competent and resilient) tend to be present in many diseases [6], and in many of these diseases there is amplification due to loss of biodiversity. This model confirms this amplification phenomena. Moreover, in the scenario where the competent host $H_k$ is resilient and abundant, if the increase of the number of suboptimal species $k - 1$ makes $N_k$ smaller, there is also dilution of the disease. However, if the increase of the number of suboptimal species makes $N_k$ greater, for example in a mutualistic relationship, then the disease is amplified. In the same scenario, the collective effect of the increase of the number of suboptimal species on the sensitivity of $R_0$ of $N_k$ is more than additive (see equation (13)).

5 Appendix

5.0.1 Next generation matrix

We compute $R_0$ using the NGM method from [11]. In our model presented in Section 2 we obtain that if we obtain the matrices $F$ and $V$ that define the NGM are:
\[
F = \begin{pmatrix}
0 & \beta_{h1}vD_1 & \beta_{h2}vD_2 & \cdots & \beta_{hk}vD_k \\
\beta_{vh1}D_1 & 0 & 0 & 0 & 0 \\
\beta_{vh2}D_2 & 0 & 0 & 0 & 0 \\
\ddots & \ddots & \ddots & \ddots & \ddots \\
\beta_{vhk}D_k & 0 & 0 & 0 & 0
\end{pmatrix},
V = \begin{pmatrix}
\delta_v & 0 & 0 & 0 & 0 \\
0 & \delta_{h1} & 0 & 0 & 0 \\
0 & 0 & \delta_{h2} & \cdots & 0 \\
\ddots & \ddots & \ddots & \ddots & \ddots \\
0 & 0 & 0 & \cdots & \delta_{hk}
\end{pmatrix}
\]

In consequence, the NGM of the system is:
\[
G = FV^{-1} = \begin{pmatrix}
0 & \frac{\beta_{h1}v}{\delta_v}D_1 & \frac{\beta_{h2}v}{\delta_v}D_2 & \cdots & \frac{\beta_{hk}v}{\delta_v}D_k \\
\frac{\beta_{vh1}}{\delta_{h1}}D_1 & 0 & 0 & \cdots & 0 \\
\frac{\beta_{vh2}}{\delta_{h2}}D_2 & 0 & 0 & \cdots & 0 \\
\ddots & \ddots & \ddots & \ddots & \ddots \\
\frac{\beta_{vhk}}{\delta_{hk}}D_k & 0 & 0 & \cdots & 0
\end{pmatrix}
\tag{14}
\]

Computing the spectral radius of the matrix in (14) we get that the basic reproductive number of the whole system is given by (15).
\[
R_0 = \rho(FV^{-1}) = \sqrt[k]{\sum_{i=1}^{k} \frac{\beta_{vh1} \beta_{h1}v D_1^2}{\delta_v \delta_{ri}}}
\tag{15}
\]

We say that an equilibrium \( \bar{N}^* \) of (1) is the disease free equilibrium (DFE) if the infectious populations of all species are zero. The following theorem tells us how the basic reproductive number is related to the stability of the disease free equilibrium in our model [11, Theorem 2].

**Theorem 3** Let \( \bar{N}^* \) be the DFE of (1). Then, \( R_0 < 1 \) implies \( \bar{N}^* \) is locally asymptotically stable and \( R_0 > 1 \) implies that \( \bar{N}^* \) is unstable.

5.1 Community constraints

Let us assume that the matrix
\[
J_d := \frac{\partial(F_1, \ldots, F_m)}{\partial(N_1, \ldots, N_m)}(\bar{N}_0) := \left( \frac{\partial F_i(\bar{N}_0)}{\partial N_j} \right)_{i,j \leq m}
\tag{16}
\]
is invertible and let us also define
\[
J_i := \frac{\partial(F_1, \ldots, F_m)}{\partial(N_{m+1}, \ldots, N_k)}(\bar{N}_0) := \left( \frac{\partial F_i(\bar{N}_0)}{\partial N_j} \right)_{i \leq m, j > m}
\tag{17}
\]
The implicit function theorem [9, Theorem 2.12] states that in a neighborhood of \( \overline{N}_0 = (N_1^0, \ldots, N_k^0) \) we have that \( N_i = g_i(N_{m+1}, \ldots, N_k) \) for \( i = 1, \ldots, m \) and if \( \overline{g} := (g_1, \ldots, g_m) \), then

\[
\frac{\partial \overline{g}}{\partial N_j} = \begin{pmatrix}
\frac{\partial g_1}{\partial N_j} \\
\vdots \\
\frac{\partial g_m}{\partial N_j}
\end{pmatrix} = -J_d^{-1} \begin{pmatrix}
\frac{\partial F_1}{\partial N_j} \\
\vdots \\
\frac{\partial F_m}{\partial N_j}
\end{pmatrix}
\]

where \( m < j \leq k \).

Let us define

\[
J := \frac{\partial (F_1, \ldots, F_m)}{\partial (N_1, \ldots, N_k)}(\overline{N}_0) := \left( \frac{\partial F_i(\overline{N}_0)}{\partial N_j} \right)_{i \leq m, j \leq k}
\]

We are interested in computing \( D_\pi \mathcal{R}_0 \) for \( \overline{\pi} \in T_{\overline{N}_0} E \), where

\[
T_{\overline{N}_0} E := \{ \overline{\pi} \in \mathcal{R}^k | J \overline{\pi} = \overline{0} \} = \text{Ker}(J)
\]

If \( \overline{\pi} = (u_1, \ldots, u_k) \), using \( u_{m+1}, \ldots, u_k \) as free variables, we have that

\[
\begin{pmatrix}
u_1 \\
\vdots \\
u_m
\end{pmatrix} = -\sum_{j=m+1}^k u_j J_d^{-1} \begin{pmatrix}
\frac{\partial F_1}{\partial N_j} \\
\vdots \\
\frac{\partial F_m}{\partial N_j}
\end{pmatrix} = -J_d^{-1} J_i \begin{pmatrix}
u_{m+1} \\
\vdots \\
u_k
\end{pmatrix}
\]

Therefore, for a given set of values \( u_{m+1}, \ldots, u_k \), we can obtain the values \( u_1, \ldots, u_m \) and

\[
D_\pi \mathcal{R}_0 = \sum_{i=1}^k u_i r_i
\]

where \( r_i = \frac{1}{N_i \cdot R_0} ((R_0^h)^2 D_i - R_0^2) \) is computed in \( \overline{N}_0 \).

If we assume that \( m = k - 1 \), in a neighborhood of \( \overline{N}_0 \) we have that

\( N_i = g_i(N_k), i = 1, \ldots, k - 1 \)

We also have that

\[
J_i = \begin{pmatrix}
\frac{\partial F_1(\overline{N}_0)}{\partial N_j} \\
\vdots \\
\frac{\partial F_{k-1}(\overline{N}_0)}{\partial N_j}
\end{pmatrix}
\]

and if \( (u_1, \ldots, u_{k-1}, u_k) \in T_{\overline{N}_0} E \), we have

\[
\begin{pmatrix}
u_1 \\
\vdots \\
u_{k-1}
\end{pmatrix} = -u_k J_d^{-1} \begin{pmatrix}
\frac{\partial F_1(\overline{N}_0)}{\partial N_j} \\
\vdots \\
\frac{\partial F_{k-1}(\overline{N}_0)}{\partial N_j}
\end{pmatrix} = -u_k J_d^{-1} J_i
\]

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Taking \( u_k = 1 \), we have
\[
\begin{pmatrix}
  u_1 \\
  \vdots \\
  u_{k-1}
\end{pmatrix} =
\begin{pmatrix}
  \frac{\partial g_1}{\partial N_k} \\
  \vdots \\
  \frac{\partial g_m}{\partial N_k}
\end{pmatrix}
\]

Therefore, close to the point \((N_0^k, N_i^0)\) we have the approximation
\[
N_i = g_i(N_k) \approx u_i(N_k - N_i^0) + N_i^0 = -\delta_i N_k + \eta_i
\]
where \(\delta_i := -u_i, \eta_i := N_i^0 - u_i N_k^0\) for \(i = 1, \ldots, k-1\).

### 5.2 One competent host

Let us prove that \(\partial R_0^2/\partial D_k > 0\) and \(\partial R_0^2/\partial D_i < 0\) for \(i = 1, \ldots, k-1\) and \(D_1, \ldots, D_k\) such that \(R_0 \geq 1\). Indeed, we have that
\[
\frac{\partial R_0^2}{\partial D_i} = 2((R_0^{h_i})^2 D_i - (R_0^{h_k})^2 D_k)
\]

If \(R_0^{h_j} < 1\) for \(j = 1, \ldots, k-1\) and \(R_0^{h_k} > 1\), using \(D_k = 1 - \sum_{j=1}^{k-1} D_j\) and \(1 = \sum_{j=1}^k (R_0^{h_j})^2 D_j^2\), we obtain
\[
D_k((R_0^{h_k})^2 D_k - 1) \geq \sum_{j=1}^{k-1} D_j (1 - (R_0^{h_j})^2 D_j) \geq 0
\]

Therefore, we must have
\[
(R_0^{h_k})^2 D_k \geq 1 \geq (R_0^{h_j})^2 D_j
\]

for \(j = 1, \ldots, k-1\), and in that way \(\partial R_0^2/\partial D_i \leq 0\) for \(i = 1, \ldots, k-1\). Using that \(D_k = 1 - \sum_{j=1}^{k-1} D_j\), we also obtain that \(\partial R_0^2/\partial D_k \geq 0\).

Now, let us consider the linear restrictions in \([12]\). Computing \([9]\) and \([10]\) with \(\bar{u} = (-\delta, \ldots, -\delta, 1)\), we have
\[
\Gamma_k = \frac{D_k}{R_0^2} \sum_{i=1}^{k} u_i ((R_0^{h_i})^2 D_i - R_0^2)
\]

If \(\hat{r} := -\sum_{i=1}^{k-1} ((R_0^{h_i})^2 D_i - R_0^2)\), we get
\[
\Gamma_k = \frac{D_k}{R_0^2} (\hat{r} + ((R_0^{h_k})^2 D_k - R_0^2)) \quad (18)
\]
We have that \( \hat{r} > 0 \) if we assume that the host \( H_1, \ldots, H_{k-1} \) are suboptimal. Under this assumption, \( \Gamma_k \) is an increasing function of \( \delta \).

Taking \( D_k \) large, \( D_1, \ldots, D_{k-1} \) would be small and we would have \((R_0^{h_i})^2 D_i - R_0^2 \approx -R_0^2 \) for \( i = 1, \ldots, k - 1 \). In that way, \( \hat{r} \approx (k - 1)R_0^2 \). In addition, if \((R_0^{h_k})^2 D_k - R_0^2 \approx 0 \) and \( \delta \) is large,

\[
\Gamma_k \approx \delta(k - 1).
\]

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