S1 Appendix

Contents

1 Overview

The following sections present calculations of when the equilibria of our model are feasible and stable in the case when the bacteria do not affect the host. We also discuss the connection to the disease ecology literature in more detail and show how the parameters of our model map to epidemiological processes.

2 Feasibility criteria

Any equilibrium of Equation 1 in the main text where all three species have positive abundance must satisfy the equations

$$\begin{aligned} p^* &= h^* - R_p, \\ m^* &= R_p + R_m - \frac{c_p}{c_m} h^*, \\ 0 &= A h^{*2} + B h^* + C, \end{aligned} \tag{A1}$$

where $R_p = \frac{d_p + d_h + d_{hp}}{c_p}$, $R_m = \frac{d_p + d_{hp} - d_m}{c_m}$ and

$$\begin{aligned} A &= -c_h + \frac{c_p}{c_m} \left(c_{hm} - c_h \right), \\ B &= c_h - (d_h + d_{hp}) + (c_{hm} - c_h) \left(R_p + R_m + \frac{c_p}{c_m} \right), \\ C &= (c_{hm} - c_h)(R_p + R_m) + d_{hp} R_p \ . \end{aligned} \tag{A2}$$

To determine feasibility in the main text, we solved these equations numerically and checked the signs of their solutions.

2.1 No effects on host

Consider the situation where the bacteria do not impact the host: $c_{hm} = c_h$ and $d_{hp} = 0$. In this scenario, $A = -c_h$, $B = c_h - d_h$ and $C = 0$ so that $h^* = 1 - \frac{d_h}{c_h}$ if it is non-zero. We find that

$$\begin{aligned} p^* &= h^* - \frac{d_p + d_h}{c_p} \\ &= 1 - \frac{d_h}{c_h} - \frac{d_p + d_h}{c_p} \end{aligned} \tag{A3}$$

which is positive when

$$c_p > \frac{d_p + d_h}{c_h - d_h} c_h \ . \tag{A4}$$

As $d_h \to 0$, this inequality reduces to $c_p > d_p$ as we expect. On the other hand, increasing d_h from below c_h always makes this inequality harder to satisfy, as we found in the main text. We also find

$$\begin{aligned} m^* &= \frac{d_p + d_h}{c_p} + \frac{d_p - d_m}{c_m} - \frac{c_p}{c_m} h^* \\ &= \frac{d_p + d_h}{c_p} + \frac{d_p - d_m}{c_m} - \frac{c_p}{c_m}\left(1 - \frac{d_h}{c_h}\right) \end{aligned} \tag{A5}$$

which is positive when

$$c_m > \frac{d_m - d_p + c_p\left(1 - \frac{d_h}{c_h}\right)}{d_p + d_h} c_p \ . \tag{A6}$$

Once again, as $d_h \to 0$, we get $c_m > \frac{d_m - d_p + c_p}{d_p} c_p$ which is the standard formula for the competition-colonization tradeoff. In this case, however, increasing d_h actually increases the proportion of mutualists at equilibrium, unlike in the case of the pathogen.

2.2 Pathogen-induced host mortality

Now, we still take $c_{hm} = c_h$, but we let $d_{hp} > 0$. Because the host dynamics now depend on the pathogen, its equilibrium fraction is given by a quadratic whose solution is

$$h^* = \frac{c_h - (d_h + d_{hp}) \pm \sqrt{(c_h - (d_h + d_{hp}))^2 + 4 d_{hp} c_h R_p}}{2 c_h} \ . \tag{A7}$$

By assumption, $4 d_{hp} c_h R_p > 0$, so we know that the term in the square root is larger in absolute value than $|c_h - (d_h + d_{hp})|$. As a result, there is at most one positive root for h^*, which is given by (A7) when the plus-minus is a plus.

3 Stability analysis

After some rewriting, the Jacobian of our model evaluated at any non-trivial and feasible fixed point (h^*, p^*, m^*) can be written as

$$J(h^*,p^*,m^*) = \begin{bmatrix} -c_h h^* + d_{hp}\frac{p^*}{h^*} + (c_h - c_{hm})\frac{m^*}{h^*} & -d_{hp} & -(c_h - c_{hm})(1-h^*) \\ c_p p^* & -c_p p^* & 0 \\ c_m m^* & -(c_p + c_m) m^* & -c_m m^* \end{bmatrix}. \quad \text{(A8)}$$

To determine stability in the main text, we evaluated this Jacobian at a given feasible equilibrium and then compute its eigenvalues numerically.

3.1 No effects on host

When there is no effect of the bacteria on the host, the Jacobian simplifies to

$$J(h^*,p^*,m^*) = \begin{bmatrix} -c_h h^* & 0 & 0 \\ c_p p^* & -c_p p^* & 0 \\ c_m & -(c_p + c_m) m^* & -c_m m^* \end{bmatrix} \quad \text{(A9)}$$

The eigenvalues of this lower-triangular matrix are simply

$$\lambda_1 = -c_h h^* \quad \text{(A10)}$$
$$\lambda_2 = -c_p p^*, \quad \text{(A11)}$$
$$\lambda_3 = -c_m m^*. \quad \text{(A12)}$$

Therefore, in this case, if a fixed point is feasible it must also be stable.

4 Reducing to a single differential equation

When τ is very large, the bacterial frequencies converge to their equilibrium solutions as a function of h, which we reported in the Feasibility criteria section above. Note that, because the d_h term in the bacterial dynamics does not have a τ factor (because it is not a fast, bacterial process), host death rate does not enter into the bacterial frequencies in this limit. Plugging these equilibrium solutions into the host dynamics then yields a single differential equation for the host dynamics.

5 Connection to SI model

Rewriting our model in terms of unoccupied hosts yields

$$\begin{aligned}
\frac{d\bar{h}}{dt} &= ((\bar{h}+p)c_h + c_{hm}m)(1-(\bar{h}+p+m)) - d_h h + d_p p + d_m m - \bar{h}(c_p p + c_m m),\\
\frac{dp}{dt} &= c_p(\bar{h}+m)p - (d_p + d_h + d_{hp})p,\\
\frac{dm}{dt} &= c_m \bar{h} m - (d_m + c_p p + d_h)m
\end{aligned} \tag{A13}$$

where $\bar{h}$ is the frequency of unoccupied hosts. In this formulation, bacterial deaths increase the frequency of unoccupied hosts, while bacterial colonization decreases it. Interestingly, this model is mathematically equivalent to an SI model with two disease strains and a constant population size. In the epidemiological interpretation, $\bar{h}$ is the frequency of healthy hosts, p is the frequency of hosts infected with the "pathogen" disease while m is the frequency of hosts infected with the "mutualist" disease. Coinfection is not possible, and instead the competitively superior pathogen displaces the mutualist. Unlike the dynamics in most disease ecology models, the mutualist disease strain actually increases the healthy host population growth rate. Further connections between epidemiological theory and the theory of holobionts could prove to be a promising avenue for future research.