Supporting information for
"Spatial structure, host heterogeneity and parasite virulence: implications for vaccine-driven evolution"

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Appendix S1: Theory

S1.1 Spatial invasion fitness

The dynamics of the mutant parasite are given by the following equations

$$\begin{aligned}\frac{dp_{I'_N}}{dt} &= \beta'_{NN}[S_N|I'_N]p_{I'_N} + \beta'_{TN}[S_N|I'_T]p_{I'_T} - (d+\alpha'_N)p_{I'_N}\\ \frac{dp_{I'_T}}{dt} &= \beta'_{NT}[S_T|I'_N]p_{I'_N} + \beta'_{TT}[S_T|I'_T]p_{I'_T} - (d+\alpha'_T)p_{I'_T}\end{aligned}$$

or, in matrix form,

$$\frac{d}{dt}\begin{pmatrix}p_{I'_N}\\ p_{I'_T}\end{pmatrix} = M\begin{pmatrix}p_{I'_N}\\ p_{I'_T}\end{pmatrix} \tag{S1.1}$$

where

$$\boldsymbol{M} = \begin{pmatrix}\beta'_{NN}[S_N|I'_N] - (d+\alpha'_N) & \beta'_{TN}[S_N|I'_T]\\ \beta'_{NT}[S_T|I'_N] & \beta'_{TT}[S_T|I'_T] - (d+\alpha'_T)\end{pmatrix}$$

We can rewrite $\boldsymbol{M}$ as $\boldsymbol{M} = \boldsymbol{F} - \boldsymbol{V}$, where

$$\boldsymbol{F} = \begin{pmatrix}\beta'_{NN}[S_N|I'_N] & \beta'_{TN}[S_N|I'_T]\\ \beta'_{NT}[S_T|I'_N] & \beta'_{TT}[S_T|I'_T]\end{pmatrix}$$

and

$$\boldsymbol{V} = \begin{pmatrix}d+\alpha'_N & 0\\ 0 & d+\alpha'_T\end{pmatrix}$$

All the entries of $\boldsymbol{F}$ and $\boldsymbol{V}^{-1}$ are positive, and the dominant eigenvalue of $-\boldsymbol{V}$ is cleary negative, so we can use the Next-Generation Theorem. Thus, the mutant invades if the dominant eigenvalue of $\boldsymbol{A} = \boldsymbol{F}.\boldsymbol{V}^{-1}$ is greater than 1. With the notations

$$\begin{aligned}R'_{NN} &= \beta'_{NN}/\delta'_N\\ R'_{TN} &= \beta'_{TN}/\delta'_N\\ R'_{NT} &= \beta'_{NT}/\delta'_T\\ R'_{TT} &= \beta'_{TT}/\delta'_T\end{aligned}$$

we have

$$\boldsymbol{A} = \begin{pmatrix}R'_{NN}[S_N|I'_N] & R'_{TN}[S_N|I'_T]\\ R'_{NT}[S_T|I'_N] & R'_{TT}[S_T|I'_T]\end{pmatrix}$$

Some straightforward algebra shows that the dominant eigenvalue of this matrix is

$$\mathcal{R} = \frac{1}{2}\left(R'_{NN}[S_N|I'_N] + R'_{TT}[S_T|I'_T]\right) + \frac{1}{2}\sqrt{(R'_{NN}[S_N|I'_N] + R'_{TT}[S_T|I'_T])^2 + 4(R'_{NT}R'_{TN}[S_N|I'_T][S_T|I'_N] - R'_{NN}R'_{TT}[S_N|I'_N][S_T|I'_T])}$$

When $g_P = 1$ (global dispersal), we recover the expression found by Gandon (2004) for a well-mixed population.

Noting a_{ij} the elements of $\boldsymbol{A}$, we have

$$\boldsymbol{A} = \begin{pmatrix} a_{NN} & a_{TN} \\ a_{NT} & a_{TT} \end{pmatrix}$$

At equilibrium, the dominant eigenvalue is unity, $\mathcal{R} = 1$. An associated right eigenvector is the vector of densities of each class of infected hosts at equilibrum, $\boldsymbol{u} = \begin{pmatrix} \hat{p}_{I_N} & \hat{p}_{I_T} \end{pmatrix}^T$. We therefore have

$$\frac{p_{I_T}}{p_{I_N}} = \frac{1 - a_{NN}}{a_{TN}} = \frac{a_{NT}}{1 - a_{TT}} \tag{S1.2}$$

An associated left eigenvector is the vector of reproductive values, $\boldsymbol{v}$ (Taylor, 1990; Rousset, 2004). Normalising $\boldsymbol{v}$ such that $\boldsymbol{v}^T\boldsymbol{u} = 1$, we find that the class reproductive values $c_j = v_j u_j$ at equilibrium satisfy $c_N + c_T = 1$, with

$$c_N = \frac{a_{NT}p_{I_N}^2}{a_{NT}p_{I_N}^2 + a_{TN}p_{I_T}^2}. \tag{S1.3}$$

Furthermore, at equilibrium, $\det(\boldsymbol{A} - \boldsymbol{I}) = 0$, which yields the following equilibrium condition

$$1 - a_{NN} - a_{TT} = a_{NT}a_{TN} - a_{NN}a_{TT} \tag{S1.4}$$

For the sake of simplicity, we make now the additional assumption that transmission can be written as the product of infectivity and susceptibility. Hence, we write $\beta_{ij} = \beta_i\sigma_j$, where $\sigma_N = 1$ and σ_T is the relative susceptibility of treated hosts. We then have

$$\begin{aligned} R'_{NN} &= R'_N = \beta'_N/\delta'_N \\ R'_{TT} &= \sigma_T R'_T = \sigma_T\beta'_T/\delta'_T \\ R'_{TN} &= R'_T\frac{\delta'_T}{\delta'_N} \\ R'_{NT} &= \sigma_T R'_N\frac{\delta'_N}{\delta'_T} \end{aligned}$$

and we obtain

$$\mathcal{R} = \frac{1}{2}\left(R'_N[S_N|I'_N] + \sigma_T R'_T[S_T|I'_T]\right) + \frac{1}{2}\sqrt{(R'_N[S_N|I'_N] + \sigma_T R'_T[S_T|I'_T])^2 - 4\sigma_T R'_N R'_T\mathcal{C}'} \tag{S1.5}$$

where

$$\mathcal{C}' = [S_N|I'_N][S_T|I'_T] - [S_N|I'_T][S_T|I'_N] \tag{S1.6}$$

measures the spatial correlation of treatments experienced by mutant hosts. Equation (S1.4) can then be rewritten as

$$1 - (R_N[S_N|I_N] + \sigma_T R_T[S_T|I_T]) = -\sigma_T R_N R_T\mathcal{C} \tag{S1.7}$$

S1.2 Selection gradient

Assuming that selection is weak, we can further calculate the selection gradient.

$$\partial\mathcal{R} = \frac{1}{2}\partial\left(R'_N[S_N|I'_N] + \sigma_T R'_T[S_T|I'_T]\right) + \frac{\frac{1}{4}\partial\left((R'_N[S_N|I'_N] + \sigma_T R'_T[S_T|I'_T])^2 - 4\sigma_T R'_N R'_T \mathcal{C}'\right)}{\sqrt{(R_N[S_N|I_N] + \sigma_T R_T[S_T|I_T])^2 - 4\sigma_T R_N R_T \mathcal{C}}}$$

At neutrality, we have $\mathcal{R} = 1$ and therefore

$$\sqrt{(R_N(S_N|I_N] + \sigma_T R_T[S_T|I_T])^2 - 4\sigma_T R_N R_T \mathcal{C}} = 2 - (R_N[S_N|I_N] + \sigma_T R_T[S_T|I_T]) > 0 \tag{S1.8}$$

Using equation (S1.7), we thus have

$$\sqrt{(R_N(S_N|I_N] + \sigma_T R_T[S_T|I_T])^2 - 4\sigma_T R_N R_T \mathcal{C}} = 1 - \sigma_T R_N R_T \mathcal{C} > 0 \tag{S1.9}$$

Hence

$$\partial\mathcal{R} = \frac{1}{2}\partial\left(R'_N[S_N|I'_N] + \sigma_T R'_T[S_T|I'_T]\right) + \frac{\frac{1}{4}\partial\left((R'_N[S_N|I'_N] + \sigma_T R'_T[S_T|I'_T])^2 - 4\sigma_T R'_N R'_T \mathcal{C}'\right)}{1 - \sigma_T R_N R_T \mathcal{C}}$$

The numerator of the right-hand side of the latter equation can be written as

$$\begin{aligned}
&\frac{1}{2}\left(2 - (R_N[S_N|I_N] + \sigma_T R_T[S_T|I_T])\right)\partial\left(R'_N[S_N|I'_N] + \sigma_T R'_T[S_T|I'_T]\right) \\
&+ \frac{1}{4}\partial\left((R'_N[S_N|I'_N] + \sigma_T R'_T[S_T|I'_T])^2 - 4\sigma_T R'_N R'_T \mathcal{C}'\right) \\
=&\frac{1}{2}\left(2 - (R_N[S_N|I_N] + \sigma_T R_T[S_T|I_T])\right)\partial\left(R'_N[S_N|I'_N] + \sigma_T R'_T[S_T|I'_T]\right) \\
&+ \frac{1}{2}\left(R_N[S_N|I_N] + \sigma_T R_T[S_T|I_T]\right)\partial\left((R'_N[S_N|I'_N] + \sigma_T R'_T[S_T|I'_T])\right) - \sigma_T\partial\left(R'_N R'_T \mathcal{C}'\right)
\end{aligned}$$

which yields the following expression for $\mathcal{R}$

$$\partial\mathcal{R} = \frac{1}{1 - \sigma_T R_N R_T \mathcal{C}}\partial\left(R'_N[S_N|I'_N] + \sigma_T R'_T[S_T|I'_T] - \sigma_T R'_N R'_T \mathcal{C}'\right) \tag{S1.10}$$

S1.3 Simplifications

We can write $\partial\mathcal{R}$ as

$$\partial\mathcal{R} = \frac{\partial W + \partial S}{1 - \sigma_T R_N R_T \mathcal{C}} \tag{S1.11}$$

where ∂W collects all direct selective effects, and ∂S collects all indirect selective effects, i.e. the selective effects on local densities.

Direct effects

We have

$$\partial W = [S_N|I_N]\partial R'_N + \sigma_T[S_T|I_T]\partial R'_T - \sigma_T\mathcal{C}(R_N\partial R'_T + R_T\partial R'_N) \tag{S1.12}$$

Plugging (S1.7) into the expression of ∂W, we obtain

$$\partial W = [S_N|I_N]\partial R'_N + \sigma_T[S_T|I_T]\partial R'_T + (R_N\partial R'_T + R_T\partial R'_N)\left(\frac{1 - (R_N[S_N|I_N] + \sigma_T R_T[S_T|I_T])}{R_N R_T}\right) \tag{S1.13}$$

which gives after simplifications

$$\partial W = \frac{\partial R'_N}{R_N}\left(1 - \sigma_T R_T[S_T|I_T]\right) + \frac{\partial R'_T}{R_T}\left(1 - R_N[S_N|I_N]\right) \tag{S1.14}$$

From the dynamics of I_N and I_T, we have

$$R_N[S_N|I_N] = 1 - \frac{\beta_T[S_N|I_T]p_{I_T}}{\delta_N p_{I_N}} = 1 - \frac{\beta_T[S_N|I_T]p_{I_T}}{h_N p_{S_N}} = 1 - \frac{\beta_T[I_T|S_N]}{h_N} \tag{S1.15}$$

$$\sigma_T R_T[S_T|I_T] = 1 - \sigma_T \frac{\beta_N[S_T|I_N]p_{I_N}}{\delta_T p_{I_T}} = 1 - \sigma_T \frac{\beta_N[S_T|I_N]p_{I_N}}{\sigma_T h_T p_{S_T}} = 1 - \frac{\beta_N[I_N|S_T]}{h_T} \tag{S1.16}$$

so $\tau_T \equiv 1 - R_N[S_N|I_N]$ is the share of the force of infection on naive hosts that is caused by infections from the treated class, and $\tau_N = 1 - \sigma_T R_T[S_T|I_T]$ has the same interpretation for treated hosts. We then have

$$\partial W = \tau_N \frac{\partial R'_N}{R_N} + \tau_T \frac{\partial R'_T}{R_T} \tag{S1.17}$$

Indirect effects

We now turn to the "spatial" component of the selection gradient

$$\begin{aligned}\partial S =& R_N \partial[S_N|I'_N] + \sigma_T R_T \partial[S_T|I'_T] \\ &+ \sigma_T R_N R_T \left([S_N|I_T]\partial[S_T|I'_N] + [S_T|I_N]\partial[S_N|I'_T] - [S_N|I_N]\partial[S_T|I'_T] - [S_T|I_T]\partial[S_N|I'_N]\right)\end{aligned} \tag{S1.18}$$

$$\begin{aligned}=& R_N \left(1 - \sigma_T R_T[S_T|I_T]\right)\partial[S_N|I'_N] + \sigma_T R_T \left(1 - R_N[S_N|I_N]\right)\partial[S_T|I'_T] \\ &+ \sigma_T R_N R_T [S_N|I_T]\partial[S_T|I'_N] + \sigma_T R_N R_T [S_T|I_N]\partial[S_N|I'_T]\end{aligned} \tag{S1.19}$$

$$\begin{aligned}=& R_N \left[\left(1 - \sigma_T R_T[S_T|I_T]\right)\partial[S_N|I'_N] + \sigma_T R_T[S_N|I_T]\partial[S_T|I'_N]\right] \\ &+ \sigma_T R_T \left[\left(1 - R_N[S_N|I_N]\right)\partial[S_T|I'_T] + R_N[S_T|I_N]\partial[S_N|I'_T]\right]\end{aligned} \tag{S1.20}$$

Furthermore, we have

$$R_T[S_N|I_T] = \frac{\delta_N p_{I_N}}{\delta_T p_{I_T}}(1 - R_N[S_N|I_N]) = \frac{h_N p_{S_N}}{\sigma_T h_T p_{S_T}}\tau_T \tag{S1.21}$$

$$\sigma_T R_N[S_T|I_N] = \frac{\delta_T p_{I_T}}{\delta_N p_{I_N}}(1 - \sigma_T R_T[S_T|I_T]) = \frac{\sigma_T h_T p_{S_T}}{h_N p_{S_N}}\tau_N \tag{S1.22}$$

This yields

$$\partial S = R_N \left[\tau_N \partial[S_N|I'_N] + \frac{h_N p_{S_N}}{h_T p_{S_T}}\tau_T \partial[S_T|I'_N]\right] + \sigma_T R_T \left[\tau_T \partial[S_T|I'_T] + \frac{h_T p_{S_T}}{h_N p_{S_N}}\tau_N \partial[S_N|I'_T]\right] \tag{S1.23}$$

or equivalently

$$\begin{aligned}\partial S =& \tau_N \left[R_N \partial[S_N|I'_N] + R_T \frac{\sigma_T h_T p_{S_T}}{h_N p_{S_N}}\partial[S_N|I'_T]\right] \\ &+ \tau_T \left[R_N \frac{h_N p_{S_N}}{\sigma_T h_T p_{S_T}}\sigma_T \partial[S_T|I'_N] + \sigma_T R_T \partial[S_T|I'_T]\right]\end{aligned} \tag{S1.24}$$

Link with reproductive values

The quantities τ_N and τ_T have a direct interpretation in terms of reproductive values. Indeed, we have

$$\tau_T = \frac{\beta_T[I_T|S_N]}{h_N} = \frac{\beta_T[S_N|I_T]p_{I_T}}{\delta_N p_{I_N}} = a_{TN}\frac{p_{I_T}}{p_{I_N}} = 1 - a_{NN} \tag{S1.25}$$

The last equation comes from equation (S1.2). Similarly, we have

$$\tau_N = a_{NT}\frac{p_{I_N}}{p_{I_T}} = 1 - a_{TT} \tag{S1.26}$$

Hence, it follows from equation (S1.4)

$$\tau_N + \tau_T = 1 + \sigma_T R_N R_T \mathcal{C} \tag{S1.27}$$

and

$$\frac{\tau_N}{\tau_N + \tau_T} = \frac{a_{NT} p_{I_N}^2}{a_{NT} p_{I_N}^2 + a_{TN} p_{I_T}^2} \tag{S1.28}$$

where the last expression can be identified as c_N in equation (S1.3).

Full selection gradient

Plugging equation (S1.17) and (S1.24) into equation (S1.11), and noting that the denominator is $\tau_N + \tau_T$, we obtain the following expression for the selection gradient

$$\partial R = c_N \left[\frac{\partial R'_N}{R_N} + R_N \partial[S_N|I'_N] + R_T \frac{\sigma_T h_T p_{S_T}}{h_N p_{S_N}} \partial[S_N|I'_T] \right] \tag{S1.29a}$$

$$+ c_T \left[\frac{\partial R'_T}{R_T} + R_N \frac{h_N p_{S_N}}{h_T p_{S_T}} \partial[S_T|I'_N] + R_T \sigma_T \partial[S_T|I'_T] \right] \tag{S1.29b}$$

Although we have obtained this result by direct differentiation of the invasion fitness, we note that an alternative derivation can be obtained by noting that the selection gradient can be written as

$$\partial \mathcal{R} = \sum_{k,\ell} v_k u_\ell \, \partial(a_{\ell k})$$

By writing $a_{\ell k} = F_\ell m_{\ell k}$, we can write an equation similar to equation (5) in Rousset (1999), and further simplifications lead to equation (S1.29).

S1.4 Uncorrelated landscapes

If the landscape is uncorrelated, additional simplifications follow. First, the spatial correlation in treatment is always zero, hence $\mathcal{C} = \mathcal{C}' = 0$. It follows from equation (S1.5) that the invasion fitness of a rare mutant takes the following simple form:

$$\mathcal{R} = R'_N [S_N|I'_N] + R'_T \sigma_T [S_T|I'_T] \tag{S1.30}$$

Then the selection gradient can be written simply as

$$\partial \mathcal{R} = R_N [S_N|I'_N] \frac{\partial R'_N}{R_N} + \sigma_T R_T [S_T|I'_T] \frac{\partial R'_T}{R_T} + R_N \partial[S_N|I'_N] + R_T \sigma_T \partial[S_T|I'_T] \tag{S1.31}$$

For a neutral mutant, we have at equilibrium $[S_N|I'_N] = [S_N|I_N]$ and $[S_T|I'_T] = [S_T|I_T]$. Furthermore, we have at equilibrium

$$R_N [S_N|I_N] = c_N \tag{S1.32}$$

and

$$\sigma_T R_T [S_T|I_T] = c_T = 1 - c_N \tag{S1.33}$$

Combining equations (S1.30)-(S1.33), and noting that $\partial[S_x|I'_y] = (1 - g_P) q_{S_x/I'_y}$, we obtain equation (9) in the main text.

S1.5 Host reproduction

So far, our results depend neither on host reproduction nor on the specific mechanism generating heterogeneity. The only assumption we make is that the parasite can only transmit horizontally (i.e. there is no vertical transmission). For the specific example of vaccination, we consider density-dependent reproduction, following previous spatial models of host-parasite interactions (Boots & Sasaki, 2000; Lion & Gandon, 2015).

We assume that host reproduction occurs at rate b and can be either global (with probability g_H) or local (with probability $1 - g_H$). We also assume that only susceptible hosts can reproduce. Reproduction takes place into empty sites, which introduces density-dependence. Offspring are produced at rates $\lambda_N = b[o|S_N]$ and $\lambda_T = b[o|S_T]$ for naive and treated susceptible hosts, respectively, where $[o|S_i] = g_H p_o + (1 - g_H) q_{o/S_i}$.

For the vaccination example, we further consider that offspring have a probability ν of entering the treated class at birth, as depicted in figure 1a. Note that, for a fully imperfect vaccine ($r_i = 0$), all hosts are identical for the parasite and, as a result, $c = \nu$.

S1.6 Stochastic simulations

We performed stochastic individual-based simulations to analyse the effect of spatial structure and host quality on the evolution of host exploitation. The program was coded in C and implements the host-parasite life cycle (figure 1a in the main text) on a regular square lattice with 100× 100 sites. Each site can contain at most one individual. The lattice is updated asynchronously in continuous time using the Gillespie algorithm (Gillespie, 1977).

For the simulations, we used the following trade-off:

$$\beta(x) = 20 \ln(x + 1) \tag{S1.34}$$

$$\alpha(x) = x \tag{S1.35}$$

Upon infection, parasites can mutate at rate 0.05. Mutation effects were drawn from a normal distribution with 0 mean and standard deviation 0.05. All simulations were run with parameters values: $b = 8$, $d = 1$, starting from host exploitation $x = 1.25$. The mean equilibrium for each run was estimated as the average value of the trait between $t = 18000$ and the simulation end time $t = 20000$.

References

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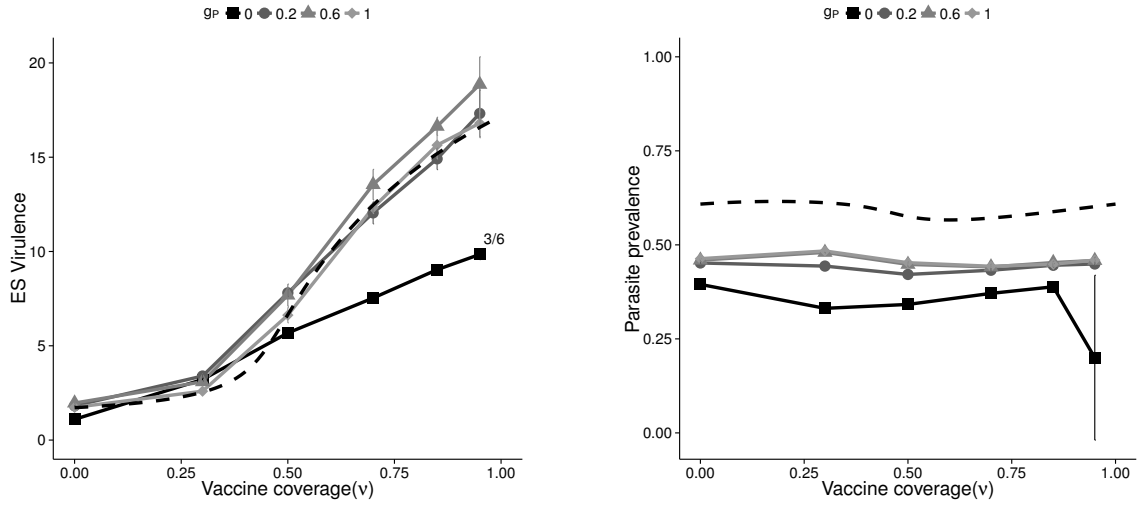
Appendix S2: Evolutionary consequences of an anti-growth vaccine: vaccine coverage (figure S2)

We show here the impact of vaccination coverage on parasite prevalence and virulence, for near-perfect vaccines ($r_2 = 0.9$). We broadly recover the predictions of Gandon et al. (2001, 2003): increasing vaccination coverage has little impact on parasite prevalence, but may select for higher virulence (figure S2a). Note that, as parasite dispersal becomes more local, parasite prevalence is minimised at lower vaccination coverage (figure S2b). Lower parasite dispersal leads to lower prevalence and more prudent exploitation over the whole range of vaccination coverage, but selection for increased virulence is stronger at intermediate parasite dispersal.



(a) **(b)**

Figure S2: The evolutionarily stable host exploitation (a) and prevalence (b) of the parasite as a function of vaccine coverage for an anti-growth vaccine r_2. The dashed lines indicate the predictions of non-spatial theory. The dots indicate the mean and standard deviation for six runs of the stochastic process. The fractions represent the number of runs that went extinct out of the six runs. The mean equilibrium for each run was estimated as the average value of the trait between $t = 18000$ and the simulation end time $t = 20000$. Mutations occured at rate 0.05. Mutation effects were drawn from a normal distribution with 0 mean and standard deviation 0.05. Simulations were performed on a regular lattice of four neighbours, with 10000 sites. Parameters: $b = 8$, $d = 1$, starting from host exploitation $x = 1.25$.

Appendix S3: Evolutionary consequences of an anti-transmission vaccine (figure S3)

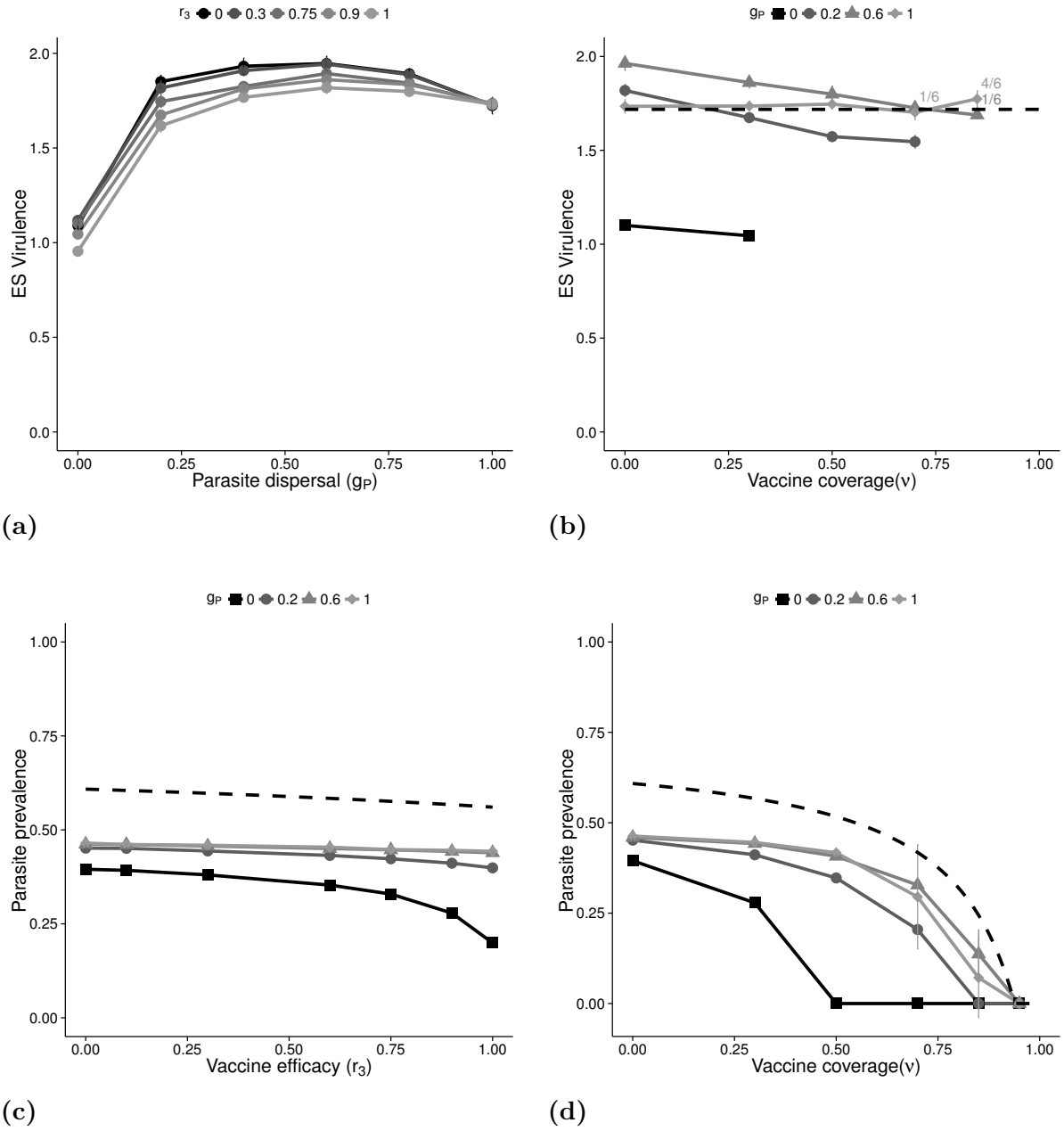


Figure S3: The evolutionarily stable host exploitation (a,b) and prevalence (c,d) of the parasite as a function of parasite dispersal, vaccine efficacy, and vaccine coverage for an anti-transmission vaccine r_3. The dashed lines indicate the predictions non-spatial theory. The dots indicate the mean and standard deviation for six runs of the stochastic process. The mean equilibrium for each run was estimated as the average value of the trait between $t = 18000$ and the simulation end time $t = 20000$. Mutations occured at rate 0.05. Mutation effects were drawn from a normal distribution with 0 mean and standard deviation 0.05. Simulations were performed on a regular lattice of four neighbours, with 10000 sites. Parameters: $b = 8$, $d = 1$, starting from host exploitation $x = 1.25$.

Appendix S4: Effect of parasite evolution on total host density (figure S4)

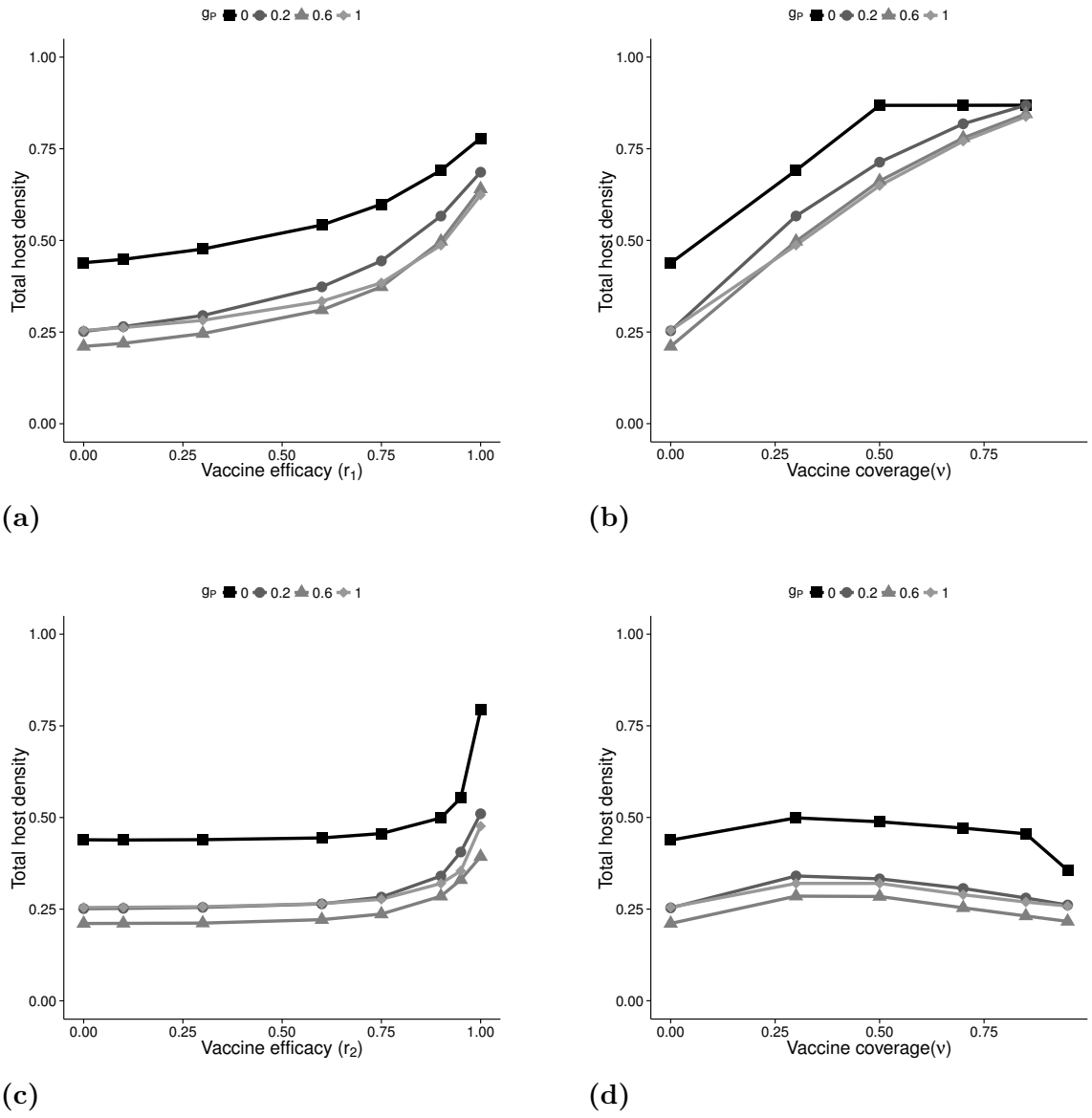


Figure S4: The total host density on the evolutionary attractor as a function of (a,c) vaccine efficacy and (b,d) vaccine coverage for (a,b) anti-infection (r_1) and (c,d) anti-growth (r_2) vaccines. The dashed lines indicate the predictions of non-spatial theory. The dots indicate the mean for six runs of the stochastic process. The mean equilibrium for each run was estimated as the average value of the trait between $t = 18000$ and the simulation end time $t = 20000$. Mutations occured at rate 0.05. Mutation effects were drawn from a normal distribution with 0 mean and standard deviation 0.05. Simulations were performed on a regular lattice of four neighbours, with 10000 sites. Parameters: $d = 1$, starting from host exploitation $x = 1.25$.

Appendix S5: Effect of host dispersal (figure S5)

In the main text, we investigate how changes in parasite dispersal affect the parasite evolution when host reproduce locally. Here, we show the robustness of our results when host dispersal is either partially ($g_H = 0.5$) or fully global ($g_H = 1$). For anti-growth (b) and anti-toxin (c) vaccines, global host dispersal weakens the effect of local parasite dispersal on the evolution of virulence. For anti-infection vaccines (a), the interplay between global host dispersal and local parasite dispersal gives rise to a non-linear relationship between vaccine efficacy and ES virulence, with a maximum for near-perfect vaccine. A complete study of the interplay between host and parasite dispersal kernels is beyond the scope of this paper, but this result suggests that the evolutionary outcome depends on both host and parasite dispersal patterns (see also Lion & Gandon, 2015 for a discussion in homogeneous spatially structured populations). Note that, as expected, global host dispersal always leads to higher prevalence (d,e,f).

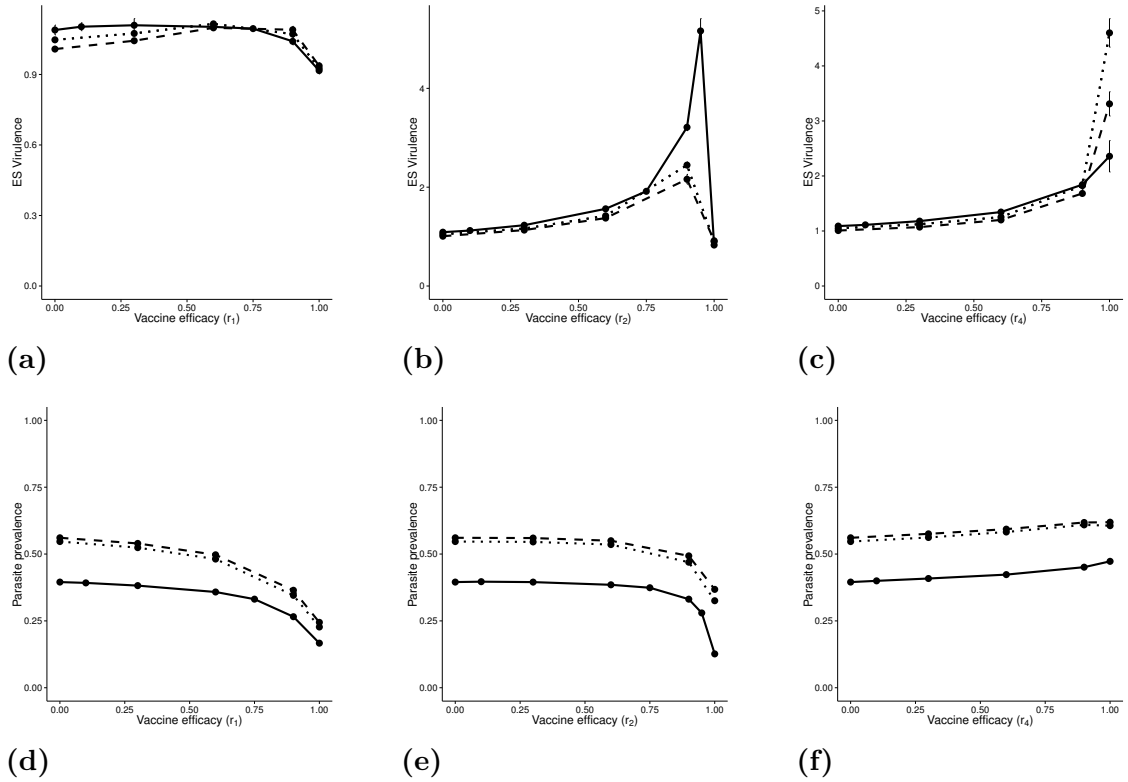


Figure S5: The evolutionarily stable host exploitation (a,b,c) and prevalence (d,e,f) for (a,d) anti-infection (r_1), (b,e) anti-growth (r_2) and (c,f) anti-toxin (r_4) vaccines. For each figure, the results for fully local parasite dispersal ($g_P = 0$) and either fully local ($g_H = 0$, plain lines), partially global ($g_H = 0.5$, dotted lines), or fully global ($g_H = 1$, dashed lines) host dispersal are shown. The dots indicate the mean and standard deviation for six runs of the stochastic process. The mean equilibrium for each run was estimated as the average value of the trait between $t = 18000$ and the simulation end time $t = 20000$. Mutations occured at rate 0.05. Mutation effects were drawn from a normal distribution with 0 mean and standard deviation 0.05. Simulations were performed on a regular lattice of four neighbours, with 10000 sites. Parameters: $d = 1$, starting from host exploitation $x = 1.25$.

Appendix S6: Effect of host fecundity (figure S6)

Previous studies have shown that, in the absence of vaccination, the kin competition effect is predicted to vanish when habitat saturation increases: as host fecundity increases, the differences between spatial and non-spatial models flatten out (Lion & Boots, 2010). Indeed, when host fecundity is infinite, the model converge towards a simple SIS model without demography, for which parasite dispersal only affects the speed of evolution, but not the endpoint. Stochastic simulations lead to the same result for anti-infection and anti-transmission vaccines, although for an anti-growth vaccine, the effect of host fecundity appears to be more complex (figure S6).

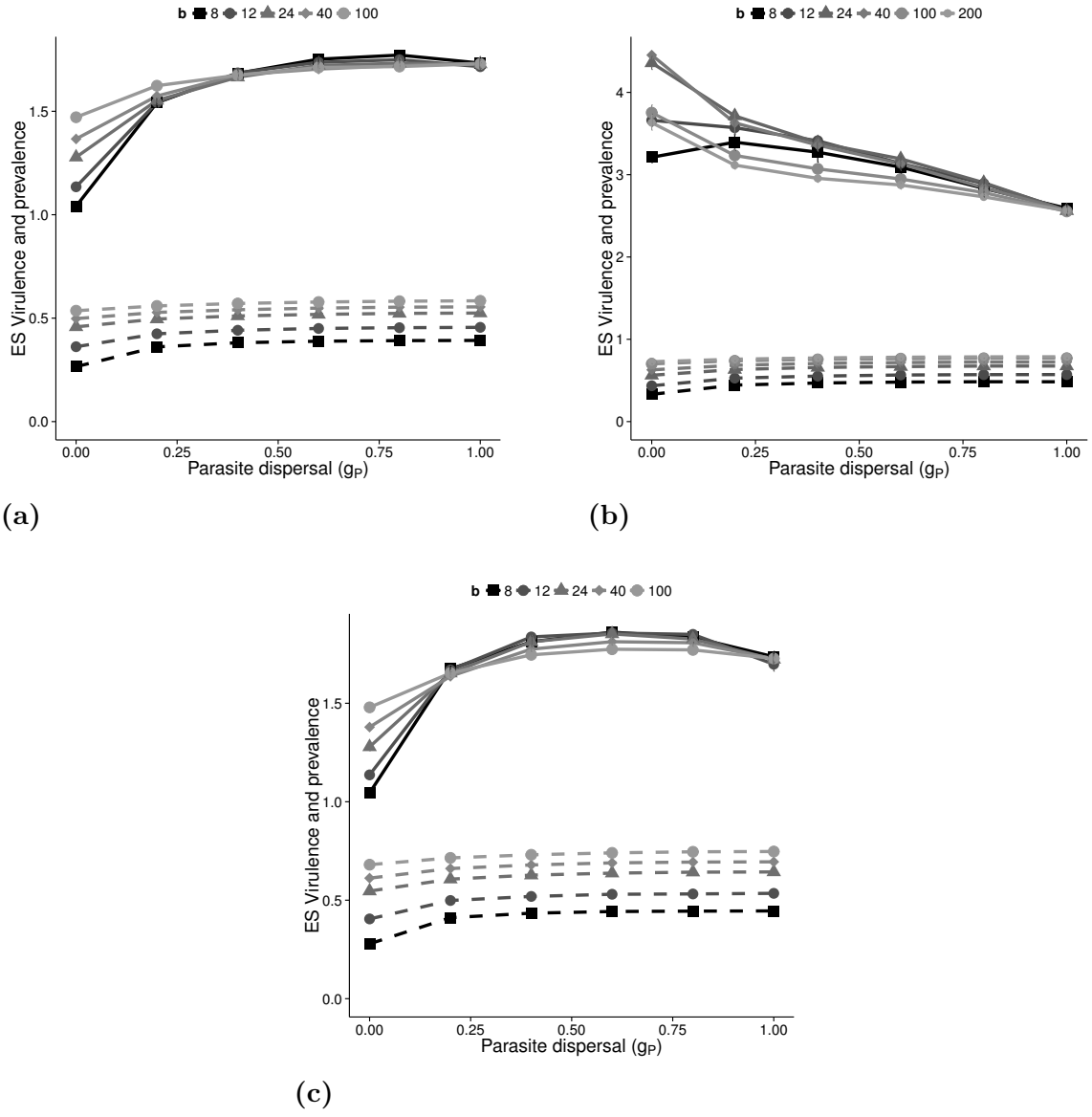


Figure S6: The evolutionarily stable host exploitation (plain lines) and prevalence (dashed lines) of the parasite as a function of parasite dispersal for (a) an anti-infection vaccine (r_1), (b) an anti-infection vaccine (r_2) and (c) an anti-transmission vaccine (r_3) for a near-perfect vaccine ($\nu = 0.9$ and $r_i = 0.9$) and increasing values of host fecundity ($b = 8$, 12, 24, 40, 100). The dots indicate the mean and standard deviation for six runs of the stochastic process. The mean equilibrium for each run was estimated as the average value of the trait between $t = 18000$ and the simulation end time $t = 20000$. Mutations occured at rate 0.05. Mutation effects were drawn from a normal distribution with 0 mean and standard deviation 0.05. Simulations were performed on a regular lattice of four neighbours, with 10000 sites. Parameters: $d = 1$, starting from host exploitation $x = 1.25$.