

SUPPORTING INFORMATION: ANALYTIC TECHNIQUES

Here we provide a derivation of the results given in the main text. For simplicity, we will derive the stability conditions for the baseline model, where increased tolerance of parasite-induced mortality incurs a cost to resistance through recovery, with density-dependent birth and no infected reproduction. For clarity, we give the model again here,

$$\frac{dX}{dt} = (a - qN)X - bX - \beta XY + \gamma Y, \quad (\text{A1a})$$

$$\frac{dY}{dt} = \beta XY - (\alpha + b + \gamma)Y. \quad (\text{A1b})$$

The invasion fitness of the host is defined as the growth rate of a mutant invading a resident equilibrium population. By rewriting the above dynamics for a mutant host with parameters $(\bar{\alpha}, \bar{\gamma})$ and taking the Jacobian, we find the invasion fitness,

$$s = (a - qN^* - b - \beta Y^*)(\bar{\alpha} + b + \bar{\gamma}) + \beta \bar{\gamma} Y^*. \quad (\text{A2})$$

where *s indicate the resident population densities. (In fact this expression is a proxy for the true fitness as it is not derived directly from the mutant's growth rate. However, it is sign equivalent to the true fitness and does not affect our analysis.) If $s > 0$ the mutant may invade the resident population (provided it overcomes demographic stochasticity whilst rare), but if $s < 0$ the mutant will always die out.

Our aim has been to identify those evolutionary scenarios where variation can arise through evolutionary branching. The outcomes of evolution in theoretical models are known to be highly dependent on the shape of the trade-off that is assumed. So, to keep our conclusions general, we will focus on identifying those defence mechanisms and associated costs where evolutionary branching can occur *for some trade-off* and those where it can never occur. As such our analysis follows the geometric methods of reference (25) in the main text.

Since the fitness depends on both mutant and resident parameters we write $s = s(\bar{\gamma}, \bar{\alpha}, \gamma, \alpha)$. An evolutionary singularity occurs when the local fitness gradient is zero. Using subscripts to

denote derivatives (i.e. $s_{14} = \partial^2 s / \partial \bar{\gamma} \partial \alpha$), this can be written as $s_1 + s_2 g'(\gamma) = 0$. We thus obtain a condition for the singularity on the gradient of the host's trade-off,

$$g'(\gamma) = -\frac{s_1}{s_2} = \frac{\alpha + b}{\gamma}. \quad (\text{A3})$$

Evolutionary stability determines whether the singularity is a local fitness maximum and is given by,

$$E = \frac{\partial^2 s}{\partial \bar{\alpha}^2} = s_{11} + 2s_{12}g'(\gamma) + s_{22}g'(\gamma)^2 + s_2 g''(\gamma). \quad (\text{A4})$$

which when evaluated at the singular point gives,

$$E = \frac{\gamma(q(\alpha + b + \gamma) - \beta(a - b))}{q(\alpha + b + \gamma) + \beta(\alpha + b)} g''(\gamma). \quad (\text{A5})$$

Convergence stability depends upon the combination of evolutionary stability and mutual invasibility. As such we calculate the mutual invasibility condition,

$$M = \frac{\partial^2 s}{\partial \bar{\alpha} \partial \alpha} = s_{13} + (s_{14} + s_{23})g'(\gamma) + s_{24}g'(\gamma)^2. \quad (\text{A6})$$

Notice that M does not depend upon the curvature of the trade-off, $g''(\gamma)$, and so is independent of the trade-off shape. As stated in the main text, evaluating this condition for our baseline model gives $M = 0$ necessarily.

As we explain in the main text, for evolutionary branching to occur, a singular point must be evolutionarily unstable ($E > 0$) but convergence stable ($E + M < 0$). By changing the trade-off shape, $g''(\gamma)$, we can ensure that $E > 0$. However, for both conditions to be satisfied, we must have $M < 0$. Since we have found that this is never the case, we know that in this model, evolutionary branching can never occur.

This model can easily be developed to add reproduction from infecteds (an additional birth term afY in (A7a)) or move density-dependence on to death or on to parasite-induced death (an additional density term qN in (A7b)). In these cases the analytic expressions for E and M become rather lengthy, but numerical investigation shows that M is always positive in these cases, and so branching still cannot occur.

These methods can also easily be adapted to alternate resistance mechanisms. For example, if resistance is through avoidance (lowered transmission), then we simply take the various derivatives with respect to β rather than γ . However, we found that the general conclusions of the model stay the same when this is done.

When considering sterility tolerance we simply modify our system of equations to,

$$\frac{dX}{dt} = (a - qN)(X + fY) - bX - \beta XY + \gamma Y, \quad (\text{A7a})$$

$$\frac{dY}{dt} = \beta XY - (\alpha + b + \gamma)Y. \quad (\text{A7b})$$

We now assume that infected hosts (Y) can give birth to susceptible offspring but that this is reduced by some factor f . We assume that a host can reduce the sterility effects of the parasite (increase f) at a cost to its death rate (increase b). By implementing the methods described above we find that parameter values exist where $M < 0$ and thus the host can branch for certain trade-offs.