Appendix A from S. R. Hall et al., "Selective Predation and Productivity Jointly Drive Complex Behavior in Host-Parasite Systems"

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Details of *S-I*-Predation Models

No Predation

Without predators (i.e., $f_S[S, I, P] = f_I[S, I, P] = 0$), a boundary and an interior equilibrium emerge in the host-parasite system. The susceptible host-only, boundary equilibrium becomes

$$S_b^* = \frac{(b - d)}{b}\left(\frac{1}{c}\right),$$

$$I_b^* = 0, \tag{A1}$$

which requires only that birthrate (b) exceeds death rate (d) to be feasible (i.e., for $S_b^* > 0$). At this equilibrium, density of susceptible hosts increases linearly with the inverse of the strength of density dependence ($1/c$; eq. [A1]). The parasite can invade this boundary equilibrium (eq. [A1]) when per capita growth rate of the infected host class, dI/Idt, is positive when the parasite is rare, that is, when evaluated at the host-only equilibrium (S_b^*; eq. [A1]). Successful invasion requires sufficient productivity; that is, $1/c$ must exceed a critical level ($\hat{c}^{-1}$):

$$\hat{c}^{-1} = \left(\frac{d + \alpha}{\beta}\right)\frac{b}{b - d}. \tag{A2}$$

This condition captures the $R_0 > 1$ requirement typically cited in epidemiological literature (e.g., Holt et al. 2003). If this invasion criterion is met, the parasite can invade the host population, and an interior equilibrium emerges:

$$S_i^* = \frac{d + \alpha}{\beta},$$

$$I_i^* = \frac{b(1/c - S_i^*) - d/c}{b + \beta/c}. \tag{A3}$$

At this equilibrium, density of susceptible hosts becomes decoupled from $1/c$. Instead, S_i^* is determined solely by traits of the infected host class (the ratio of loss rates to transmission rate). This S_i^* reflects the minimal host population size that can sustain the parasite (loosely analogous to R^* in classical resource competition theory; Grover 1997). The equilibrial population size of infected hosts depends on the difference between host population size at the boundary and interior equilibria, $S_b^* - S_i^*$, and increases positively but nonlinearly with increases in 1/c (eq. [A3]).

It is straightforward to show that this no-predator coexistence equilibrium (eq. [A3]) is locally asymptotically stable using standard linear stability analysis (Kot 2001). If we define $F_1 \equiv dS/dt$ and $F_2 \equiv dI/dt$, the Jacobian (community) matrix (**J**) of the *S-I* system becomes

$$\mathbf{J} = \begin{bmatrix} \partial F_1/dS & \partial F_1/dI \\ \partial F_2/dS & \partial F_2/dI \end{bmatrix}. \tag{A4}$$

Matrix $\mathbf{J}$ has a characteristic equation $\lambda^2 + A_1\lambda + A_2 = 0$, and the Routh-Hurwitz stability criteria require that A_1 (-1 times the trace of the $\mathbf{J}$) >0 and A_2 (the determinant of $\mathbf{J}$) >0. Evaluated at the interior equilibrium without predators (eq. [A3]), $\mathbf{J}$ becomes

$$\begin{bmatrix} -\dfrac{b(d+\alpha)}{\beta/c} & -\dfrac{(b+\beta/c)(d+\alpha)}{\beta/c} \\ \dfrac{b(d+\alpha)-\beta/c(b-d)}{b+\beta/c} & 0 \end{bmatrix}. \tag{A5}$$

A feasible interior equilibrium yields negative elements J_{11} and J_{12} and positive element J_{21}. Given the signs of these elements, $A_1 = -J_{11} > 0$ always; also, $A_2 = -J_{21}J_{12} > 0$ always. Thus, when feasible, the coexistence equilibrium is always stable. This result means that predation must drive complex behavior in the *S-I* model with a predator with a type II functional response.

Predators with a Linear Functional Response

A predator with a linear functional response quantitatively alters invasion/feasibility criteria and equilibrial densities of susceptible (S) and infected hosts (I) but does not affect their qualitative dynamics. In a system with these predators, the boundary equilibrium becomes

$$S_b^* = \frac{(b-d-P)}{b}\left(\frac{1}{c}\right),$$

$$I_b^* = 0, \tag{A6}$$

which requires that maximal birthrate (b) exceeds both loss rates ($d+P$) and implies that S_b^* now increases less quickly with increases in productivity ($1/c$). The interior equilibrium becomes

$$S_i^* = \frac{d+\theta P+\alpha}{\beta},$$

$$I_i^* = \frac{b(1/c-S_i^*)-1/c(d+P)}{b+\beta/c}. \tag{A7}$$

This equilibrium requires (for both invasibility and feasibility) that the inverse of density dependence exceeds a minimum requirement ($\hat{c}^{-1} > c^{-1}$) but predation intensity not exceed a critical rate ($P < \hat{P}$), where

$$\hat{c}^{-1} = \frac{bS_i^*}{b-d-P},$$

$$\hat{P} = b-d-bcS_i^*. \tag{A8}$$

A predator with a linear functional response does not fundamentally change the behavior of the *S-I* system. At a feasible interior equilibrium (eq. [A8]), the elements of the associated Jacobian matrix become

$$J_{11} = -\frac{b(d + \alpha + \theta P)}{\beta/c} < 0, \tag{A9a}$$

$$J_{12} = -\frac{(b + \beta/c)(d + \alpha + \theta P)}{\beta/c} < 0, \tag{A9b}$$

$$J_{21} = \frac{b(d + \alpha + \theta P) - \beta/c(b - \alpha - P)}{b + \beta/c} > 0. \tag{A9c}$$

Since the signs of these elements equal those in the no-predation case and $J_{22} = 0$ still, this interior equilibrium is always stable when feasible. Thus, cycling behavior, Allee effects, and catastrophes require type II (saturating), density-dependent predation.

Predators with a Type II, Multiple-Prey Functional Response

In the model with saturating predation, there may be zero, one, or two susceptible host-only (boundary) equilibria (May 1977; fig. 1A). These boundary equilibria are

$$S_b^* = \frac{b(1/c - h_S) - d/c + \sqrt{[d/c - b(1/c + h_S)]^2 - 4bP/c}}{2b}, \tag{A10a}$$

$$S_b^* = \frac{b(1/c - h_S) - d/c - \sqrt{[d/c - b(1/c + h_S)]^2 - 4bP/c}}{2b}. \tag{A10b}$$

Assuming that the host could persist without the predator ($b > d$), only the upper, stable equilibrium (eq. [A10a]) is feasible at low predation intensity (P). As P increases further, the system crosses a transcritical bifurcation at

$$P = h_S(b - d). \tag{A11}$$

At this point, the lower boundary equilibrium (eq. [A10b]) emerges, but it is a saddle. Therefore, the susceptible host experiences alternative stable states because the saddle separates a stable boundary equilibrium from a stable trivial ($S^* = 0$, $I^* = 0$) equilibrium. As P increases further yet, the stable attractor and saddle meet at a fold bifurcation:

$$P = \frac{b^2(h_S^2 + 1/c^2) + 2bh_S(b - d)/c - d/c^2(2b - d)}{4b/c}. \tag{A12}$$

Beyond this critical P, the susceptible host cannot persist with the predator (fig. 1A).

The whole S-I system with a predator feeding according to a type II, multiple-prey functional response becomes more complex to analyze than the version with a linear functional response, but several aspects of the model are readily understood analytically. The nullcline for susceptible hosts ($dS/dt = 0$) is

$$S = \frac{-(d + BI)/c - b[h_S - 1/c + I(1 + \gamma\theta)] + \sqrt{X}}{2b}, \tag{A13}$$

where

$$X = 1/c^2(d + \beta I)^2 + b^2[h_S + I(\gamma\theta - 1) + 1/c]^2 - 2b/c[2P + (d + \beta I)][h_S + 1/c + I(\gamma\theta - 1)], \tag{A14}$$

and the nullcline for infected hosts ($dI/dt = 0$) is

$$I = \frac{P}{\beta S - (d + \alpha)} - \frac{h_S + S}{\gamma\theta}. \tag{A15}$$

The nullcline for I always decreases with increasing S. The nullcline for S, however, can take a variety of

shapes, and it can disappear and reappear in the positive quadrant (e.g., figs. 3, 5). As a result, these nullclines can cross zero, one, or two times in the positive quadrant. However, the complex expressions for the interior equilibria prevent meaningful presentation here.

The bifurcation diagrams reveal three types of bifurcations for which one can solve analytically. The second interior equilibrium emerges at an interior transcritical bifurcation:

$$P = -\frac{[h_S(\beta/c + b\gamma\theta) + \gamma\theta(b - d)/c + (d + \alpha)/c][b(d + \alpha) - \beta(d + \alpha)/c]}{(\beta/c + b\gamma\theta)^2}. \tag{A16}$$

This second, high S–low I equilibrium is always a saddle (determined numerically using our parameter ranges; table 1). Once P exceeds this level and this interior saddle emerges, the multiple equilibria arise. We determined the domains of attraction of these two equilibria in figure 3C using an adaptive-step, numerical integrator (MathWorks 1999).

Both of these interior equilibria meet at even higher P at an interior fold bifurcation. This collision occurs when

$$P = \frac{\{(\beta/c + b)(d + \alpha + \beta h_S) + \gamma\theta[\beta/c(b - d) - b(d + \alpha)]\}^2}{4b\beta\gamma\theta(b + \beta/c)(\gamma\theta - 1)}. \tag{A17}$$

If predation intensity exceeds this level, the parasite can no longer coexist because the interior equilibrium becomes unfeasible (see fig. 1A and eq. [A12] for an analogous boundary fold).

The S-I-predator system can begin to oscillate due to a Hopf bifurcation. This bifurcation results from a degeneracy of its associated Jacobian matrix (**J**). With predators feeding with a multiple-prey, type II functional response, **J** becomes

$$\mathbf{J} = \left\{ \begin{array}{cc} b[1 - c(I + 2S)] - \dfrac{P(h_S + \gamma\theta I)}{(h_S + \gamma\theta I + S)^2} - \beta I - d & -S\left[\beta + bc - \dfrac{\gamma\theta P}{(h_S + \gamma\theta I + S)^2}\right] \\[2em] I\left[\beta + \dfrac{\gamma\theta P}{(h_S + \gamma\theta I + S)^2}\right] & \beta S - (d + \alpha) - \dfrac{\gamma\theta P(h_S + S)}{(h_S + \gamma\theta I + S)^2} \end{array} \right\}. \tag{A18}$$

(This Jacobian matrix was used to numerically characterize the behavior of the five biologically feasible equilibria of this system.) Hopf bifurcations occur when the trace ($J_{11} + J_{22}$) of **J** is 0, meaning that negative intraspecific feedback no longer stabilizes the system (Kot 2001). This bifurcation occurs when

$$P = -\frac{(h_S + S^* + \gamma\theta I^*)^2[bS^* + (d + \alpha)/c]}{h_S\gamma\theta/c}. \tag{A19}$$

The global homoclinic bifurcations are difficult to solve analytically (Kot 2001). Therefore, we used a standard numerical ODE integrator to find them (MathWorks 1999). Combined, these four bifurcations together with the two boundary bifurcations yield a qualitative understanding of the behavioral regimes of the S-I-predator system.