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Appendix A. Analysis of the "predator–spreader" model and its variants.

In this appendix, we provide further analysis of the model presented in the text (Eq. A.1). We compare the structural form of the invasion/persistence threshold (Eq. A.2) with that derived from several variants of the model. Comparisons with these variants help to explain behavior of these key thresholds.

Equilibria and infection prevalence from Eq. 1

The model presented in the text (Eq. A.1) produces only two equilibria – either all species are extinct (a trivial case) or host and parasite coexist (and internal equilibrium). Otherwise if it is not controlled by the parasite, the host population increases towards infinity. The interior (coexistence) equilibrium is quite complex (and local stability analysis is even more analytically inscrutable), but we can understand some key constraints placed on it from the simple expression produced for spores in the water column (Z_w^*):

$$Z_w^* = \frac{(b-d-f_C C)(d+v+\theta f_C C)}{uf(d+v-\rho b+\theta f_C C)} = -\frac{r_{S,C} d_{I,C}}{u f_{S,C}(r_{I,C})} \quad \text{(A.1)}$$

where the compound parameters follow those in the text (Eq. A.3). To ensure a positive value of Z_W^*, this expression demands that:

$$b-d-f_C C = r_{I,C} > 0 \text{ but} \quad \text{(A.2a)}$$

$$d+v-\rho b+\theta f_C C = -r_{I,C} > 0 \quad \text{(A.2b)}$$

which implies that the host must be sufficiently productive to support the predator (Eq. A.2a) but the parasite must be sufficiently virulent (i.e., large effect on mortality [high v] and/or large reduction in fecundity [low ρ]) to control the host in the face of selective predation (since predation itself might undermine parasite control of the host; Eq. A.2b). Infection prevalence, p^*, also follows a simple expression; it equals:

$$p^* = \frac{I^*}{I^* + S^*} = \frac{r_{S,C}}{r_{S,C} - r_{I,C}}; \quad \text{(A.3)}$$

this term decreases with predator density, C, and predator selectivity, θ, since:

$$\frac{\partial p^*}{\partial C} = \frac{f_C(-\theta(b-d) - r_{I,C})}{(r_{S,C} - r_{I,C})^2} \quad \text{(A.4a)}$$

$$\frac{\partial p^*}{\partial \theta} = -\frac{f_C C(r_{S,C})}{(r_{S,C} - r_{I,C})^2}; \quad \text{(A.4b)}$$

a little bit of algebra indicates that prevalence always decreases with predator density, C (unless $\theta < 0$ which is impossible; Eq. A.4a) and with selectivity of predation, θ (Eq. A.4b). Thus, infection prevalence decreases with predator density and increasing selectivity, even if highly selective predation enhances persistence of the parasite (Fig. 4). Interestingly, infection prevalence depends only on the vital rates of the uninfected and infected host classes, not on other key features of this disease system (transmission; mixing, sinking; and loss rates of spores from the bottom pool). Instead, those other factors help to determine equilibrial density of the classes of hosts and pools of spores.

The model without predation ($C = 0$) but with the benthic and water column pools of spores produces a more manageable equilibrium, which we present here for interested readers:

$$Z_w^* = \frac{(b-d)(d+v)}{uf(d+v-\rho b)} = -\frac{r_S d_I}{u f_S(r_I)} \quad \text{(A.5a)}$$

$$S^* = \frac{\lambda d_Z r_I}{f\left[(r_S - r_I)(d_Z + m) + m r_I(u\sigma)\right]} \quad \text{(A.5b)}$$

$$I^* = \frac{\lambda d_Z r_S}{f\left[(r_S - r_I)(d_Z + m) + m r_I(u\sigma)\right]} \quad \text{(A.5c)}$$

$$Z_b^* = \frac{\lambda Z_W^*(r_I(1-u\sigma) - r_S)}{\left[(r_S - r_I)(d_Z + m) + m r_I(u\sigma)\right]} \quad \text{(A.5d)}$$

$$p^* = r_S/(r_S - r_I) \quad \text{(A.5e)}$$

where the compound parameters closely follow those in Eq. A.3, except that now $C = 0$ (i.e., $d_I = d_s + v$, $r_s = b - d_s$, and $r_I = pb - d_I$). This equilibrium requires that the host is sufficiently productive to persist itself (i.e., $b > d_s$) but the parasite is virulent enough (high v, low ρ) to control the host ($d_I > pb$, or $r_I < 0$). These conditions are less stringent than those described above.

Invasion/persistence thresholds: incremental build-up for two-spore-compartments

The key threshold in the text (Eq. A.2) yielded some surprising results. Namely, predators can enhance invasion and persistence of parasites through two key mechanisms: they release spores directly into the water column and they cull some infected hosts that otherwise remove spores from the water column. We can understand these results more clearly by considering similar thresholds produced from variations on this model. In several instances below, we imagine that infected hosts do not remove spores from the water column, an unrealistic scenario. In other words, we would modify the equation for change in water column spores from Eq. A.1d to:

$$dZ_w/dt = \sigma_C \theta f_C C I + m Z_b - f_s S Z_w - \lambda Z_w \quad \text{(A.6)}$$

This assumption seems unrealistic because *Daphnia* graze generally and would contact and eat spores whether healthy or infected (Ebert 2005, Hall et al. 2007). Nonetheless, it proves informative when compared to the more realistic case (i.e., when dZ_w/dt follows Eq. A.1d).

Let us start with the simplest case where there is no predation (C=0) and infected hosts do not consume spores (i.e., Eq. A.6 applies). The invasion threshold is:

$$\hat{m} = d_Z\left(\frac{d_S + v}{(\quad)(\quad)}\right) = d_Z\left(\frac{d_I}{(\quad)(\quad)}\right) = d_Z\left(\frac{1}{(\quad)}\right) \quad \text{(A.7)}$$

which is smaller than the similar threshold produced from Eq. A.1 but assuming no predation:

$$\hat{m} = d_Z\left(\frac{r_S - r_I}{-\phi r_I - (r_S - r_I)}\right). \quad \text{(A.8)}$$

(Since $r_S - r_I > d_I$, the numerator in Eq. A.8 is larger than that in Eq. A.7; furthermore, given this result and that $-r_I < d_I$, the denominator of Eq. A.8 is smaller than that in Eq. A.7). This result is illustrated in comparisons of Figure 4.A and A.1a (looking along the Y-axis; C = 0). These two models only differ in the assumption about removal of spores in the water column by infected hosts. Therefore, we can conclude that this spore-sink mechanism creates an additional hurdle for the parasite. Returning to the "unrealistic scenario" (i.e., Eq. A.6 applies) with predation ($C > 0$), we see that the invasion/persistence threshold becomes:

$$\hat{m} = d_Z\left(\frac{d_{I,C} - \phi_C}{\phi + \phi_C - d_{I,C}}\right) \quad \text{(A.9)}$$

which now incorporates infectivity of spores produced by release from the predator (Φ_C)and those entering the bottom pool following death of infected hosts from infection (Φ)This threshold (Eq. A.9) can either increase or decrease with predator density (C), depending on the number of spores released per host that is predated, σ_C. Since the slope of the threshold is:

$$\frac{\partial \hat{m}}{\partial C} = -\frac{d_z \phi(\theta f_C(\sigma_C u - 1))}{(\phi + \phi_C - d_{I,C})^2} \qquad \text{(A.10)}$$

we readily find that this threshold always increases with C if predators do not release sufficient numbers of spores ($\sigma_C u < 1$) but always decreases otherwise (see Fig. A.1). One can show that the slope is steeper (either negative or positive) when predator selectivity (θ) elevates. This effect appears in Fig. A.1. Furthermore, we can solve for the predator density required to sustain epidemics with no mixing from the bottom pool of spores (i.e., when $m = 0$):

$$\tilde{C} = \frac{d_I}{\theta f_C(\sigma_C u - 1)} \qquad \text{(A.11)}$$

which of course requires that $\sigma_C > 1$. As seen in Fig. A.1 and intuited from equation A.11, this level of predation ($\tilde{C}$) decreases as predators become more selective (higher θ) and as more spores are released from predators (higher σ_C).

Now we again imagine that infected hosts remove spores (i.e., the more realistic scenario where Eq. A.1d applies) but predators do not release spores ($\sigma_C = 0$). We find yet another threshold (the $\sigma_C = 0$ lines of Fig. 4):

$$\hat{m} = d_Z \left(\frac{d_{I,C}(r_{S,C} - r_{I,C})}{-\phi r_{I,C} - d_{I,C}(r_{S,C} - r_{I,C})} \right) \quad . \qquad \text{(A.12)}$$

The slope of this curve with predator density (C) is very complex (i.e., a quadratic polynomial of C divided by a quartic polynomial); in the example illustrated, we see that the slope it can first decrease, then increase with C (Fig. 4). Once predators release spores ($\sigma_C > 0$) we arrive at the curve already presented in the text (Eq. A.3):

$$\left(d_{\cdot,\cdot}(r_{\cdot,\cdot} - r_{\cdot,\cdot}) + r_{\cdot,\cdot}\phi_{\cdot} \right) \qquad \text{(A.13)}$$

which a tiny bit of algebra shows is always lower than the threshold without spore release from predators (Eq. A.12; Fig. 4). Unfortunately, the X-intercept of this curve in Fig. 4 is also difficult to express simply (i.e., an expression analogous to Eq. A.11 is difficult to represent compactly here). However, we see that like in the more unrealistic scenario (as governed by Eq. A.11), predators can support epidemics without mixing from the bottom pool when selectivity (θ) and spore release (σ_C) are sufficiently high.

Invasion/persistence thresholds: incremental build-up for fully mixed spores

In order to offer some completeness, we should consider the case in which spores are fully mixed. This assumption obviates the bottom spore equation; thus, the model retains the dS/dt and dI/dt equations (Eq. A.1a, b) but replaces the two spore equations (Eq. A.1c, d) with:

$$dZ_w/dt = \sigma(d_s + v)I + \sigma_C \theta f_C C I - f_s(S+I)Z_w - d_z Z_w \qquad \text{(A.14)}$$

Here, spores in the water column (Z_w) increase as infected animals die from infection (first term), as infected animals are eaten by the predator (second term); they decrease as both host classes remove them (third term) and are lost at a constant background rate (d_Z).

In principle, some of the same concepts developed for the model with two spore compartments apply to this model with one fully mixed spore compartment. For instance, if we assume no predation ($C = 0$), we see that spore clearance by infected hosts makes it more difficult for the parasite to invade. We now solve for invasion-persistence thresholds for infectivity of spores released from infected hosts (Φ from hosts dying from infection, or $\Phi + \Phi_C$ for those spores plus those released from consumed infected hosts, assuming $\sigma_C > 0$). We see two thresholds arise:

(A.15a)

(A.15b)

for the case in which infected hosts do not remove spores (Eq. A.15a) or where they do remove spores (Eq. A.15b). Clearly, removal of spores by infected hosts demands higher Φ. Then, if we add selective predation ($C > 0$), we can derive new thresholds:

(A.16a)

(A.16b)

where the top threshold applies to the case in which infected hosts do not remove spores, while the bottom one includes removal of spores by infected hosts. If predators do not release spores ($\Phi_C = 0$), then predators raise the level of Φ required for invasion-persistence, regardless of whether infected hosts remove spores (i.e., Eq. A.16a is more stringent than is A.15a; Eq. A.16b is more stringent than Eq. A.15b). Thus, the phenomenon seen in Fig. 4 in which predators enhance invasion-persistence of parasites by removing infected hosts depends on having two pools of spores. Finally, if predators do release spores ($\Phi_C > 0$), predators can enhance invasion of the parasite as long as they release a sufficient amount of spores per infected host consumed (σ_C):

(A.17)

In principle, it nice to see some of these parallel results produced by this simpler model with one fully mixed spore compartment. In practice, parasites are limited by spores produced per host in this variation of the model in only very extreme conditions. When the system is fully mixed, the more applicable restrictive condition for parasite invasion and persistence requires that predator density (C) should not exceed:

(A.18)

Unless loss rate of spores (d_Z) is quite large or spore production (σ, σ_C) per host is very small, this condition (Eq. A.18) mainly applies. This condition largely ignores all of the host-parasite biology captured in Eqs. A.15–A.17. Thus, in fully mixed systems, assuming that infected hosts produce at least a modest number of spores, many of the results discussed above (Eqs. A.15–A.17) do not apply (again, because Eq. A.18 is more restrictive). This conclusion emphasizes an important caveat about the role of predators: predators enhance disease when spore losses are very high and/or mixing from a bottom pool otherwise limits disease significantly.

FIG. A1. Analogue to the more realistic scenario in Fig. 4 in the text, except here infected hosts do not clear spores from the water column (Z_b; see Eq.A.6). This case is purely theoretical (since infected *Daphnia* remove spores as do uninfected ones), but it helps us to understand results from the model presented in the text. All other parameters and situations parallel the other case in the text (Fig. 4). We can note several key differences. First, without predators, the parasite can invade at lower mixing rate (m) than the parasite in the more realistic scenario (see Eq. A.7 vs. A.8). Thus, clearance of spores by live infected hosts makes it harder for the parasite to invade. Second, if predators do not release spores ($\sigma_C = 0$), we find that increasing predator density makes it much more difficult for parasites to invade (i.e., higher m is required with increasing C). Therefore, part of the predator effect in the more realistic effect stems from culling of infected hosts that would otherwise remove spores that could infect susceptible hosts: again, live infected hosts are sinks for spores. Third, if predators do release many spores (high σ_C), lower densities of predators can sustain epidemics without any mixing than required in the more realistic scenario. Again, this result indicates that spore removal by infected hosts makes it more difficult for the parasite to invade and persist, since they remove spores that predators release into the water column.

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