Mini-rotations - Trophic overyielding and infection dynamics

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Working through ideas for the mini-rotations project

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1 Background

Previous work has shown that the coexistence of a pair or mixture of species can reach higher abundance than the species with the highest carrying capacity (Loreau 2004; Poisot et al. 2013). This idea of "transgressive overyielding" is the basis for much work on biodiversity-ecosystem functioning relationships (BEF) (i.e., Schmid et al. 2008). Some research has suggested approaches to linking this concept to disease dynamics (Johnson et al. 2015), however, few studies have explicitly developed theory to this effect. Noteably Sieben et al. (2022) and others have provided elegant frameworks for considering competition and coexistence from the perspective of parasites themselves existing in both between- and within-host frameworks. However, what effects do infection dynamics have on transgressive overyielding? Is it possible that the inclusion of pathogen dynamics into classic models of competition would change the conditions under which transgressive overyielding is founded? If so, what is the relation of the virulence of the infection and the basic reproductive number \mathcal{R}_0 ?

2 Methods

We start from the assumption of two species competing, in a Lotka-Volterra model. We use the form of Abrams (2022, pg. 4), which includes terms for both inter- and intra-specific competition. We then have

$$\frac{dN_1}{dt} = \frac{r_1 N_1}{K_1} (K_1 - N_1 - \alpha_{12} N_2) \tag{1a}$$

$$\frac{dN_2}{dt} = \frac{r_2N_2}{K_2}(K_2 - N_2 - \alpha_{21}N_1) \tag{1b} \label{eq:1b}$$

As Case (1999) showed, there is a stable equilibrium IFF

$$\alpha_{21} < x < \frac{1}{\alpha_{12}} \tag{2}$$

where

$$x = \frac{K_2}{K_1} \tag{3}$$

and Loreau (2004) showed that transgressive overyielding is present when

$$(1 - \alpha_{21})(1 - x\alpha_{12}) > 0. \tag{4}$$

With this in mind, we aim to explore how this state can change under conditions of disease.

To start, we assume that one of the species, N_1 becomes infected with a generalized pathogen. A proportion of the population is infected, and this pathogen can have some impact on the ability of that population to grow. Our system Eqs.1 now can be represented as

$$\frac{\mathrm{d}N_1}{\mathrm{d}t} = (r_{1\phi}\phi N_1)(r_1(1-\phi)N_1)\left(1 - \frac{N_1 - N_2\alpha_{12}}{K_1}\right) \tag{5a}$$

$$\frac{\mathrm{d}N_2}{\mathrm{d}t} = r_2 N_2 \left(1 - \frac{N_2 - N_1 \alpha_{21}}{K_2} \right) \tag{5b}$$

Further and related, we can imagine one of these species becoming infected with a generalized pathogen. The closed population of that species now can be thought of as being described by a standard compartment model. That is, the number of individuals in N_1 can be thought of as divided into individuals that are susceptible, S_1 , infected I_1 , and recovered R_1 such that

$$N_1 = S_1 + I_1 + R_1 \tag{6}$$

Further, we can then think of these dynamics as being described by a classic SIR model with the dynamics given by:

$$\frac{dS}{dt} = -\beta SI + \delta R \tag{7}$$

$$\frac{dI}{dt} = \beta SI - \gamma I \tag{8}$$

$$\frac{dR}{dt} = \gamma I - \delta R \tag{9}$$

2.1 Transgressive Overyielding

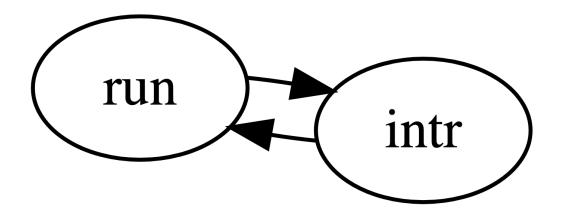
To understand how the incorporation of infection dynamics may affect the conditions under which transgressive overyielding happens, our goals are to:

- 1) Re-derive an expression for transgressive overyielding from the inter- and intra-specific version of the GLV (Eqns ?? ??)
- 2) Re-state this expression in terms of the infection parameters that will affect the interspecifi competition
- 3) Assess the assumption of non-linear growth on these expressions

4)

Model Parameters		
Term	Description	
N_i	Abundance of species i	
r_i	Growth rate of species i	
α_{ii}	Intraspecific competition within species i	
α_{ij}	Interspecific competition between species i and j	
$\begin{array}{c} \alpha_{ij} \\ S_i \\ I_i \\ R_i \end{array}$	Number of susceptible individuals in population i	
I_i	Number of infected individuals in population i	
R_i	Number of recovered individuals in population i	
β	Rate at which susceptible individuals become infected	
δ	Rate at which recovered individuals lose their immunity and	
0	become susceptible again	
γ	Rate at which the infected individuals recover from infection	

Table 1: Parameter values for our model systems



3 References

Source: Article Notebook

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