Competing for resources in the presence of infection: Mathematical models of foraging interference and disease transmission

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14 February 2018


Work in progress: I. M. Bulai, F. Hilker. Competing for resources in the presence of infection: Mathematical models of foraging interference and disease transmission.

Work in progress: I. M. Bulai, F. Hilker. The "effect" of the interference on the predator population in a predator prey model.
Introduction

The predator-prey model
- Disease and interference in the predator population

Sensitivity analysis: comparison between the models

Disease and Beddington-deAngelis functional response

Conclusions
A predator-prey model is introduced with a disease in the predator population.
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One can consider no vertical transmission (NoVT) model or the vertical transmission (VT) one: we will study the most general one, partial vertical transmission (PVT) (includes both NoVT and VT).
Introduction to the problem (1)

- A predator-prey model is introduced with a disease in the predator population.
- One can consider no vertical transmission (NoVT) model or the vertical transmission (VT) one: we will study the most general one, partial vertical transmission (PVT) (includes both NoVT and VT).
- The transmission of the disease could be density dependent (DDT) or not, thus frequency dependent (FDT). Which are the differences?
On one side the interference in the predator population is considered and on the other side both interference and handling term are considered in the functional response (Beddington-DeAngelis); There are differences?
On one side the **interference in the predator population** is considered and on the other side both **interference and handling term** are considered in the functional response (Beddington-DeAngelis); There are differences?

Which direct effect have the disease and the interference on the predator population, and indirectly on the prey population?
Hypothesis for the first two models

- There is a **disease in the predator population**, could be 
density-dependent disease transmission (DDT) or 
frequency-dependent disease transmission (FDT)

- There is the **interference** between the predators
Hypothesis for the first two models

- There is a disease in the predator population, could be density-dependent disease transmission (DDT) or frequency-dependent disease transmission (FDT).
- There is the interference between the predators.
- The predator population is divided only in two classes, the susceptible ones, $S$, and the infected, $I$, and not treatment is given to the infected predators.
- The prey population $\tilde{N}$ is the only resource of food for the predators.
The mathematical model

The model reads:

\[
\begin{align*}
\frac{d\tilde{N}}{d\tau} &= r \left(1 - \frac{\tilde{N}}{K}\right) \tilde{N} - f(S, I)S - g(S, I)I \\
\frac{dS}{d\tau} &= -\tilde{m}S - \beta(S, I) + e_S f(S, I)S + (1 - \nu)e_I g(S, I)I \\
\frac{dI}{d\tau} &= -\tilde{m}I - \tilde{\mu}I + \beta(S, I) + \nu e_I g(S, I)I.
\end{align*}
\]

With

\[
\begin{align*}
f(S, I) &= \frac{a_S \tilde{N}}{1 + a_S w_{SS}S + a_S w_{SI}I} \\
g(S, I) &= \frac{a_I \tilde{N}}{1 + a_I w_{IS}S + a_I w_{II}I}.
\end{align*}
\]
The mathematical model (2)

Where

\[ \beta(S, I) = \tilde{\beta}SI \quad \text{DDT} \]

or

\[ \beta(S, I) = \frac{\tilde{\beta}SI}{S + I} \quad \text{FDT}. \]

In FDT, contact rate is assumed to be independent of host density. In DDT transmission, contact rate is assumed to linearly increase with host density.
We will assume

\[ a_S = a_I := \tilde{a}, \quad e_S = e_I := \tilde{e} \quad \text{and} \]

\[ w_{SS} = w_{SI} = w_{IS} = w_{II} := \tilde{w}. \]
Simplification of the models

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- We apply the coordinate transformations

\[ \tilde{P} = S + I \quad \text{and} \quad \tilde{i} = \frac{I}{S + I}. \]
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- We nondimensionalize it by applying the next substitutions

\[ N(t) = \frac{1}{K} \tilde{N}(\tau), \quad P(t) = \frac{\tilde{a}}{r} \tilde{P}(\tau) \quad \text{and} \quad t = r\tau \]
Simplified models

We get

\[
\frac{dN}{dt} = (1 - N) N - \frac{NP}{1 + wP} \\
\frac{dP}{dt} = a \left( -mP - \mu Pi + \frac{NP}{1 + wP} \right) \\
\frac{di}{dt} = ia \left( (\beta - \mu)(1 - i) - \frac{N(1 - \nu)}{1 + wP} \right) \quad \text{FDT}
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\end{align*}
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System’s equilibria for FDT model and the stability analysis (1)

Proposition 1.

(i) The trivial equilibrium point \( E_0 = (0, 0, 0) \) and the prey-and-predator-free point \( E_3 = (0, 0, 1) \) are always feasible, furthermore they are both unstable.
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(ii) The predator-and-disease-free point \( E_1 = (1, 0, 0) \) is always feasible, and it’s stable if \( m - 1 > 0 \) and \( \beta - \mu < 1 - \nu \) hold.
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(iii) The disease-free point

\[
E_2 = (m(1 + wP_2), P_2, 0)
\]

is feasible if the predator population is non-negative \( m - 1 < 0 \). Furthermore it’s stable if \( \beta - \mu < (1 - \nu)m \) hold.
System’s equilibria for FDT model and the stability analysis (3)

(iv) The predator-disease-induced extinction point

\[ E_4 = \left( 1, 0, \frac{\beta - \mu - (1 - \nu)}{\beta - \mu} \right) \]

is feasible if \( \beta - \mu > 0 \), and it’s stable if \( m - 1 + \mu i_4 > 0 \) hold.
System’s equilibria for FDT model and the stability analysis (3)

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(v) The coexistence equilibrium is

\[ E_* = \left( \frac{1 + (w - 1)P_*}{1 + wP_*}, P_*, \frac{\beta - \mu - m(1 - \nu)}{\beta - \mu \nu} \right), \]

\( P_* \) is the root of the second degree polynomial

\[ AP^2 + BP + C = 0. \]
System’s equilibria for FDT model and the stability analysis (3)

For the feasibility of the coexistence equilibrium we need to have

\[ 1 + (w - 1)P_* > 0, \quad \frac{\beta - \mu - m(1 - \nu)}{\beta - \mu \nu} > 0 \]

and one between

(a) \( A < 0 \) and one between \( B \) or \( C \) less than 0 or

(b) \( A > 0 \) and one between \( B \) or \( C \) greater than 0

must hold, \( A, B \) and \( C \) are the coefficient of the 2\textit{nd} degree polynomial. While for it’s stability the Routh-Hurwitz conditions for a 3\textit{rd} degree polynomial must hold.
### Density-dependent disease transmission

<table>
<thead>
<tr>
<th>( E = (N, P, i) )</th>
<th>Feasibility conditions</th>
<th>Stability conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>( E_0 = (0, 0, 0) )</td>
<td>always feasible</td>
<td>unstable</td>
</tr>
<tr>
<td>( E_1 = (\ast, 0, 0) )</td>
<td>always feasible</td>
<td>( m - 1 &gt; 0 )</td>
</tr>
<tr>
<td>( E_2 = (\ast, \ast, 0) )</td>
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<tr>
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</tr>
<tr>
<td>( E_* = (\ast, \ast, \ast) )</td>
<td>( N_* &gt; 0, i_* &gt; 0 ),</td>
<td>Routh-Hurwitz cond.</td>
</tr>
</tbody>
</table>

**Table:** Density-dependent disease transmission

- The predator-prey model
- Disease and interference in the predator population
- Sensitivity analysis: comparison between the models
- Disease and Beddington-deAngelis functional response
- Conclusions
In both system’s were found five equilibrium points plus the trivial equilibrium point.

$E_4$, in the DDT case is feasible if we consider the particular case $\nu = 1$ and unstable while in FDT case the non negativity of the prevalence must be required and under proper conditions could be stable.

The conditions for the stability of $E_1$, $E_2$ and $E_*$ changes from a model to another.

The coexistence equilibrium, in FDT case an analytical expression of it was found while for DDT it was showed that exist only numerically.
Transcritical bifurcation diagram for FDT and DDT varying the transmissibility

Figure: Left: the bifurcation diagram for FDT varying $\beta$, the transmissibility; Right: the bifurcation diagram for DDT varying $\beta$, the transmissibility.
Transcritical bifurcation diagram for FDT and DDT varying the disease-induced death rate

**Figure:** Left: the bifurcation diagram for FDT varying $\mu$, the disease-induced death rate; Right: the bifurcation diagram for DDT varying $\mu$, the disease-induced death rate.
Transcritical bifurcation diagram for FDT and DDT varying the waste time

Figure: Left: the bifurcation diagram for FDT varying $w$, the waste time; Right: the bifurcation diagram for DDT varying $w$, the waste time.
We would like to predict how changes in the parameters will affect the solutions. We will make a sensitivity analysis of the parameters: $\beta$ and $w$. 
Sensitivity analysis: FDT

Introduction

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Sensitivity analysis: DDT

- Prey population (N)
- Predator population (P)
- Prevalence (i)
Sensitivity analysis: FDT vs DDT (predator pop.)

Figure: Predator population varying both $\beta$ and $w$. Left: FDT; Right: DDT.
For simplicity we directly write the nondimensionalized models again for FDT and DDT, respectively.

\[
\begin{align*}
\frac{dN}{dt} &= (1 - N)N - \frac{NP}{1 + wP + hN} \\
\frac{dP}{dt} &= a \left( -mP - \mu Pi + \frac{NP}{1 + wP + hN} \right) \\
\frac{di}{dt} &= ia \left( (\beta - \mu)(1 - i) - \frac{N(1 - \nu)}{1 + wP + hN} \right) \\
\end{align*}
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\end{align*}
\]
Sensitivity solutions for BdA-FDT
Sensitivity solutions for BdA-DDT

Figure: From top to bottom: the prey population, predator population and prevalence varying both $\beta$ and $w$. Left: FDT; Right: DDT.
Conclusions (1)

- We investigated the impact of predator interference on predator-prey systems with infectious diseases circulating in the predator population.
- We have shown that interference influences predator population size and can thus impact disease emergence and infection levels. Similarly, the strength of the disease may impact predator population size and cascade to the prey level.
- We have considered two types of disease transmission, namely frequency-dependent and density-dependent transmission. Contact rates in the former are independent of host population size and proportional to host population size in the latter. These are two extremes in a continuum of possibilities, and the truth for many species may be somewhere in the middle.
We introduced predator interference using the approach of “wasting time“ similar to Beddington (1975) and carefully distinguish between different wasting times of susceptible and infected predators.

We included handling time as well, giving a functional response analogous to the Beddington-DeAngelis functional response (Beddington 1975, DeAngelis et al 1975).
Andrew M. Bate, Frank M. Hilker, Disease in group-defending prey can benefit predators, Theor Ecol (2014) 7:87–100.


Thanks for your attention!