# Impact of a predator evolution on a predator-prey system

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## Motivation

- Environmental toxicity is becoming a global concern for the biological population.
- Toxicants, including pesticides, heavy metals, and industrial chemicals, disrupt the natural dynamics of ecosystems by interfering with species' survival, reproduction, and behavior.
- Predator-prey systems are highly vulnerable to such disruptions. This research focuses on developing and analyzing evolutionary mathematical models to understand the impact of toxicants on the dynamics of a discrete-time predator-prey system.
- In 2019, Azmy Ackleh et al. developed and studied the evolutionary responses of prey on a discrete-time predator-prey system.
- This project extends the case of predator evolution by considering three different toxicant effects.
  (i) Lethal effects, where the toxicant directly influences the predator's survival in a trait-dependent manner.
  (ii) Sublethal effects, where the toxicant impacts the fecundity of the predator.
  (iii) Mixed-effects, where the toxicant impacts both the predator's survival and fecundity.

## The Models

We formulate the models as follows:

Lethal Effects Model

$$\begin{aligned} n(t+1) &= \phi(n(t))n(t)(1-f(p(t),v)p(t))|_{v=u},\\ p(t+1) &= s(v)(1-\epsilon(v))p(t)+b(n(t))n(t)f(p(t),v)p(t)|_{v=u},\\ u(t+1) &= u(t)+\nu\partial_v \ln[R(n(t),p(t),v)]|_{v=u}. \end{aligned} \tag{1}$$

Sublethal Effects Model

$$\begin{aligned} n(t+1) &= \phi(n(t))n(t)(1-f(p(t),v)p(t))|_{v=u},\\ p(t+1) &= s_0p(t)+(1-\epsilon(v))b(v)\bar{b}(n(t))n(t)f(p(t),v)p(t)|_{v=u},\\ u(t+1) &= u(t)+\nu\partial_v \ln[R(n(t),p(t),v)]|_{v=u}. \end{aligned} \tag{2}$$

Mixed-Effects Model

$$\begin{aligned} n(t+1) &= \phi(n(t))n(t)(1-f(p(t),v)p(t))|_{v=u},\\ p(t+1) &= (1-\epsilon_1(v))s(v)p(t)+(1-\epsilon_2(v))b(v)\bar{b}(n(t))n(t)f(p(t),v)p(t)|_{v=u},\\ u(t+1) &= u(t)+\nu\partial_v \ln[R(n(t),p(t),v)]|_{v=u}. \end{aligned} \tag{3}$$

For all three models, we consider $n(t)$ and $p(t)$ to represent the number or density of prey and predator, respectively, and $u(t)$ is the mean phenotypic trait for the predator population representing toxicant resistance.

We assume the following Beverton-Holt nonlinearities $\phi(n)$ and $b(n)$ for the above-mentioned models:

$$\phi(n)=\frac{r_0}{1+mn} \quad \text{and} \quad b(n)=\frac{b_0}{1+\gamma n}.$$

In addition, we consider the following exponential trait-dependent nonlinearities for model (1):

$$s(v)=s_0e^{-w_sv^2}, \quad \epsilon(v)=\epsilon_0e^{-w_\epsilon v^2}, \quad \text{and} \quad f(p,v)=\frac{c(v)}{1+pc(v)},$$

where $c(v)=c_0e^{-w_cv^2}$.

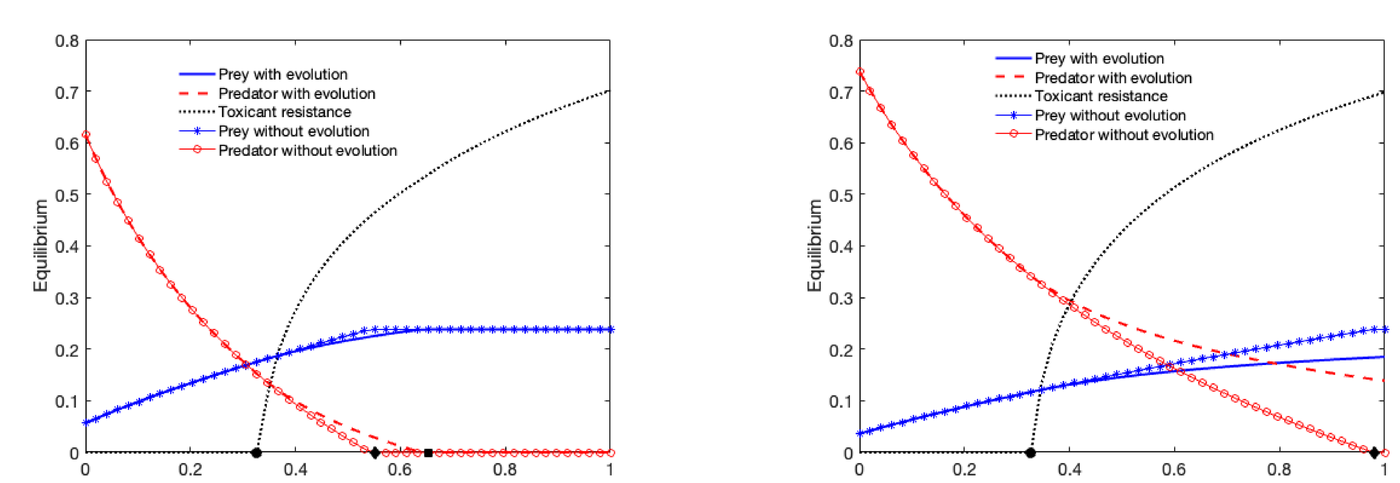
We also consider similar exponential nonlinearities for models (2) and (3).

## Stability Results for the Lethal Effects Model

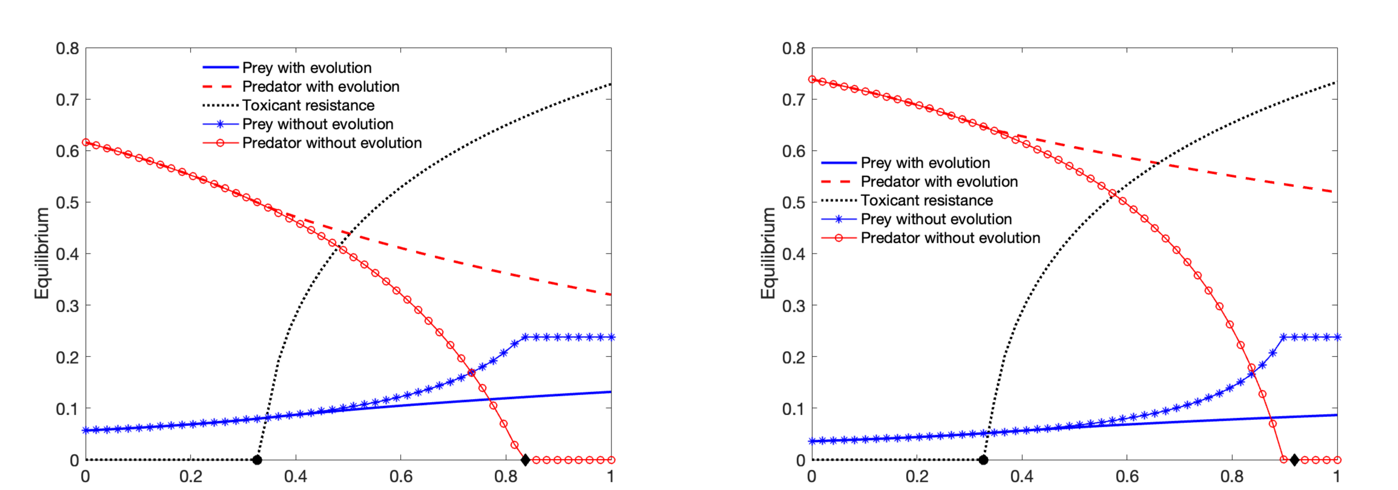
- The extinction equilibrium $(0,0,0)$ is globally asymptotically stable if $r_0<1$ and $\epsilon_0<\frac{w_s}{w_\epsilon+w_s}$.
- The predator-free equilibrium $(\bar{n},0,0)$, where $\bar{n}:=\phi^{-1}(1)$ exists if $r_0>1$ and is locally asymptotically stable if $\epsilon_0<\min\left\{\frac{\bar{n}b(\bar{n})c_0w_c+s_0w_s}{(w_s+w_\epsilon)s_0},1\right\}$, then the predator-free equilibrium $(\bar{n},0,0)$ is globally asymptotically stable for a sufficiently small $\nu$.
- The equilibrium $(n^*,p^*,0)$ with $n^*,p^*>0$ exists if $r_0>1$ and $s_0(1-\epsilon_0)+b(\bar{n})\bar{n}c_0>1$. Moreover, $(n^*,p^*,0)$ is locally asymptotically stable if $\epsilon_0<\min\left\{\frac{w_c\frac{n^*b(n^*)c_0}{s_0(1+p^*c_0)^2}+w_s}{w_s+w_\epsilon},1\right\}$ and $\nu$ is sufficiently small.
- The extinction equilibrium $(0,0,\tilde{u})$ exists if $\epsilon_0>\frac{w_s}{w_s+w_\epsilon}$, and is globally asymptotically stable if $r_0<1$ and $\nu$ is sufficiently small.
- The predator-free equilibrium $(\bar{n},0,\tilde{u})$ exists if $r_0>1$ and $\epsilon_0>\frac{\bar{n}b(\bar{n})c_0w_c+s_0w_s}{(w_s+w_\epsilon)s_0}$, and is locally asymptotically stable if $s(\tilde{u})\left(1-\epsilon(\tilde{u})\right)+b(\bar{n})\bar{n}c(\tilde{u})<1$ and $\nu$ is sufficiently small.
- If $\epsilon_0>\frac{\bar{n}b(\bar{n})c_0w_c+s_0w_s}{(w_s+w_\epsilon)s_0}$ and $s(\tilde{u})\left(1-\epsilon(\tilde{u})\right)+b(\bar{n})\bar{n}c(\tilde{u})>1$, then model (1) is uniformly persistent, *i.e.*, there exists an $\epsilon>0$ such that
$$\min\{\liminf_{t\to\infty} n(t), \liminf_{t\to\infty} p(t), \liminf_{t\to\infty} u(t)\}>\epsilon$$
for any initial condition with $n(0),p(0),u(0)>0$.

Here, $r_0$ is the inherent growth rate of prey, $s_0(1-\epsilon_0)+b(\bar{n})\bar{n}c_0$ is the invasion growth rate of the predator, and $\epsilon_0$ is the initial toxicant level.
We have similar theoretical results when the toxicant effects are sublethal. However, we analyze the mixed effects case only numerically.

## Comparison Between Lethal and Sublethal Effects Model

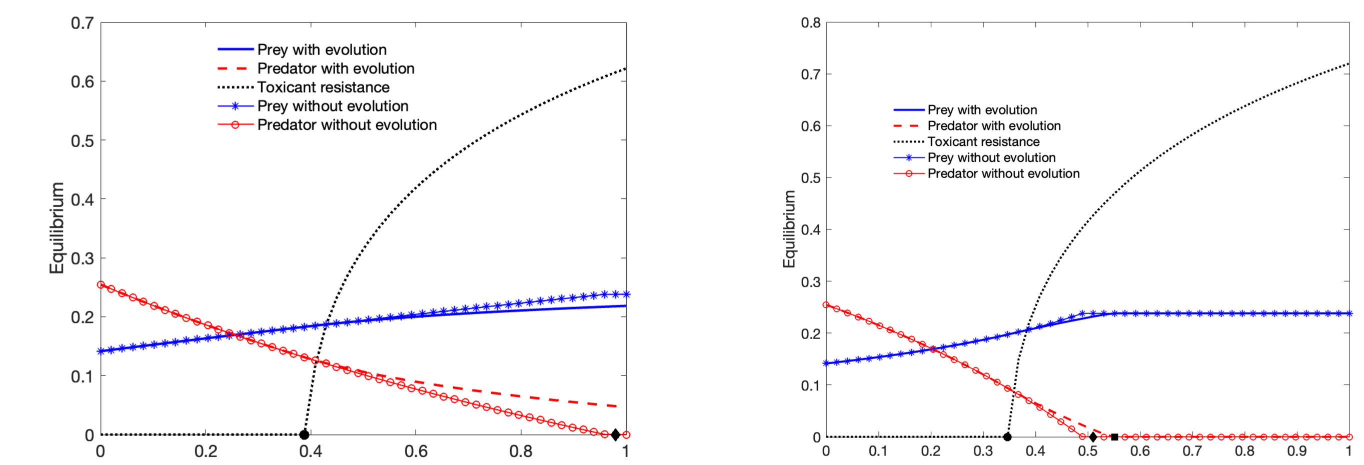



Bifurcation diagrams for the lethal model with a higher predator survival.



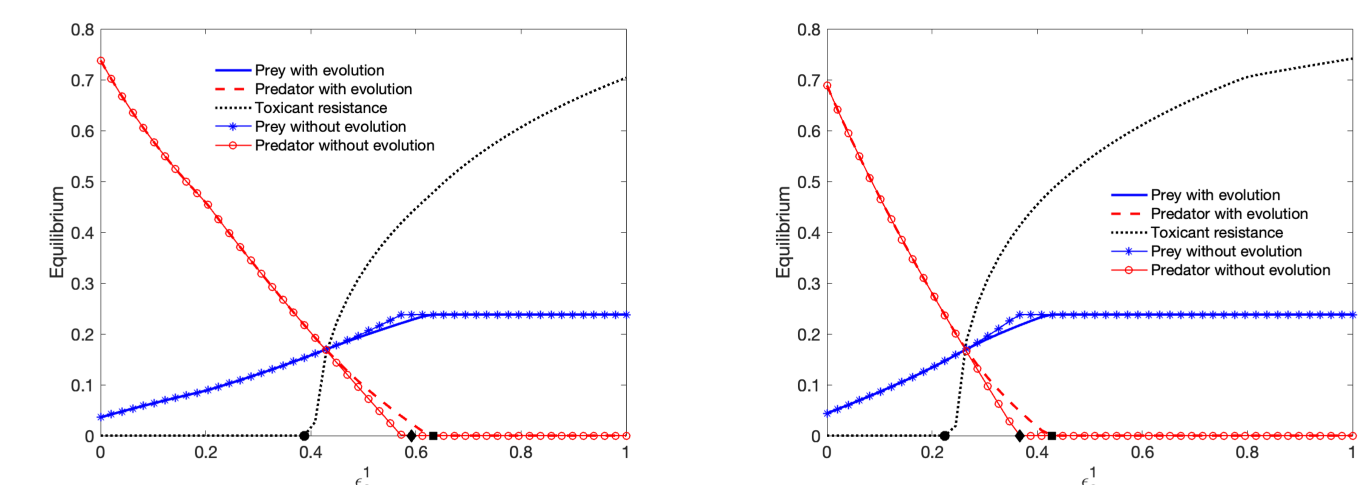

Bifurcation diagrams for the sublethal model with a higher predator survival.

## Results for the Low Predator Survival




Bifurcation diagrams for the lethal and sublethal models.

## Results for the Mixed-Effects Model




Bifurcation diagrams for the mixed effect model.

## Concluding Remarks

- In the non-evolutionary scenario, when fecundity is lower, the predator goes extinct at a lower toxicant level in the lethal case compared to the sublethal case. In contrast, when fecundity is higher, this scenario is reversed. Thus, lethal effects are greater when fecundity is low, but sublethal effects become greater when fecundity is high.
- While comparing the lethal and sublethal cases, the toxicant threshold level is the same for all scenarios from where the predator starts evolving in response to toxicants.
- In all scenarios, evolution produces higher predator densities in the toxic environment.
- When the predator survival is higher, the sublethal effects produce higher predator densities. However, the scenario is reversed when the predator survival is low.
- The mixed effects case produces qualitatively similar dynamics to the low predator survival scenario.

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## References

[1] Azmy S Ackleh, Neerob Basak, and Amy Veprauskas. The impact of predator evolution on the dynamics of a discrete-time predator-prey system. (About to be submitted)