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Switching from simple to complex dynamics in a predator–prey–parasite model: An interplay between infection rate and incubation delay

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ABSTRACT

Parasites play a significant role in trophic interactions and can regulate both predator and prey populations. Mathematical models might be of great use in predicting different system dynamics because models have the potential to predict the system response due to different changes in system parameters. In this paper, we study a predator–prey–parasite (PPP) system where prey population is infected by some micro parasites and predator–prey interaction occurs following Leslie–Gower model with type II response function. Infection spreads following SI type epidemic model with standard incidence rate. The infection process is not instantaneous but mediated by a fixed incubation delay. We study the stability and instability of the endemic equilibrium point of the delay-induced PPP system with respect to two parameters, viz., the force of infection and the length of incubation delay under two cases: (i) the corresponding non-delayed system is stable and (ii) the corresponding non-delayed system is unstable. In the first case, the system populations coexist in stable state for all values of delay if the force of infection is low; or show oscillatory behavior when the force of infection is intermediate and the length of delay crosses some critical value. The system, however, exhibits very complicated dynamics if the force of infection is high, where the system is unstable in absence of delay. In this last case, the system shows oscillatory, stable or chaotic behavior depending on the length of delay.

1. Introduction

Theoreticians have used different mathematical models to understand, explain and predict complex dynamics of predator–prey interactions. Classical Lotka–Volterra or Rosenzweig–MacArthur predator–prey models and its variants assume that prey population only grows logistically to its carrying capacity but the predator has no such limitation. Leslie [1], for the first time, considered logistic growth of predator population with prey density as its upper limit. Thus, the predator's growth equation contains a negative term which has a reciprocal relationship with per capita availability of its preferred prey [2,3]. If $X(t)$ and $Y(t)$ are, respectively, the prey and predator densities at time t then Leslie–Gower model with type II predator's response function is represented by

$$\frac{dX}{dt} = r_1X - b_1X^2 - \frac{a_1XY}{k_1+X},$$
$$\frac{dY}{dt} = Y\left(r_2 - a_2\frac{Y}{X}\right). \quad (1)$$

It says, in absence of predator, the prey population grows exponentially with intrinsic per capita growth rate r_1 when prey is rare. However, prey population follows density-dependent birth rate, with b_1 as the strength of density dependency, when its size increases. Note that this model does not state a carrying capacity for the prey population in an explicit way, but models in an implicit way by means of intraspecific competition coefficient. This is known as emergent carrying capacity [4] since it is an emergent property based on actual life-history traits of prey, rather being a predetermined number (say K) as in popular logistic model. However, as an special case, one can easily obtain the explicit carrying capacity ($K = r_1/b_1$) from the emerging carrying capacity. Predator regulates prey population following Type II response function $\frac{a_1X}{k_1+X}$, where a_1 is the maximal per capita prey consumption rate and k_1 is the half saturation constant. Predators also grow logistically to its carrying capacity $\frac{r_2}{a_2}X$ with maximum per capita growth rate r_2 by consuming its prey. One can observe that predator's carrying capacity is not a constant, but a function of prey density, X. The proportionality constant a_2 represents the number of prey required to support one predator at equilibrium when the maximum per capita growth rate of predator is unity. All parameters are

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positive. Note that when $X \to \infty$ then $\frac{1}{Y}\frac{dY}{dt} \to r_2$; and $\frac{1}{Y}\frac{dY}{dt} \to -\infty$ when $X \to 0$. In other words, the per capita growth rate of predator reaches to its maximum when prey density is too high and becomes negative, leading predator extinction, when prey population is scarce. In recent past, many researchers have studied the Leslie–Gower predator–prey model and its variants with different modifications [5–10].

Parasites play a significant role in trophic interactions [11–14] and can regulate both predator and prey populations. There are many examples that show that prey population is infected by some parasites and predators consume disproportionately large number of infected prey [15–18]. Because of infection, the prey population is partitioned into two classes of susceptible population and infective population denoted by S and I, respectively. Recently, some authors have studied Leslie–Gower predator–prey model in presence of parasitic infection [19–23]. Haque and Venturino [19] studied a Leslie–Gower predator–prey model with prey infection and type I response function. It was shown that the behavior of the system largely depend on the disease incidence rate. Zhou et al. [23] studied a modified Leslie–Gower predator–prey model where prey population is infected by some parasites. They studied the local and global stability of the biologically feasible equilibria and permanence of the system. It was shown that the strictly positive interior equilibrium undergoes Hopf bifurcation when the rate of infection crosses a critical value. Shi et al. [22] studied the role of incubation delay in a Leslie–Gower predator–prey model with prey infection. They observed that the positive interior equilibrium undergoes a Hopf bifurcation if the delay crosses a critical value. In [20], authors show the influences of intraspecific competition and infection coefficient in the stability of the coexistence equilibrium point. In an another study [21], they modified Leslie–Gower predator–prey model where disease spreads among the predator species. They showed global stability and bifurcations for some of the equilibria and also determined the sufficient conditions for persistence of the ecosystem species. These studies show that the incidence rate plays key role in the dynamics of predator–prey–parasite system. All these models [19–23] consider simple mass action law or bilinear law to model incidence rate of disease. According to this law, the rate of new infection at any time t is proportional to the product of the densities of infected and susceptible individuals at that time and is represented by $\beta(t) = \lambda SI$, where λ is the disease transmission coefficient. This mass action law has some unrealistic features, viz., the number of newly infected individuals produced by a single infective individual depends on S and becomes very high when S is large [24]. Some authors [25–28] argued that standard incidence rate is more appropriate to express the disease transmission term. Following this law, the rate of new infection at any time t is defined as

$$\beta(t) = \frac{\lambda SI}{S+I}. \tag{2}$$

One can see that the per capita infection rate is independent of infective and tends to a constant when I is large. In this study, we therefore assume that the mode of disease transmission follows the standard incidence rate. Castration [29], conspicuousness [15], behavior modification [30,31], increased mortality, increased predation [17,32,33], competitive abilities of hosts [34], feeding rate [35] etc. are supposed to be parasitic effects on hosts. We thus assume that infectious prey cannot give birth due to castration caused by parasite [36]. It is also assumed that an infected individual does not recover or become immune, and the disease spreads horizontally from an infectious individual to a susceptible individual. Infectious preys are either removed by predation or removed by disease related death [37]. Since the prey species are weakened due to infection, predators can easily catch infected individuals. Susceptible preys however maintain their density dependent growth at the same rate as it was in absence of infection. Following other similar studies [12,38–41], we assume that infectious preys contribute negatively to the growth rate of host as they are still in the environment and share resources with susceptible preys. Lafferty and Moris [17] observed through field experiment that parasitized fishes increase their risk of avian predation by swimming close to the water surface and predation rates of piscivorous birds on infected fish is, on an average, 31 times higher than the predation rates on susceptible fish. Therefore, it will not be unrealistic to assume that predators consume only infected preys. Assuming μ as the disease related death rate of infectious prey, we study the following PPP model:

$$\begin{aligned}
\frac{dS}{dt} &= r_1 S - b_1 S\Big(S+I\Big) - \frac{\lambda SI}{S+I},\\
\frac{dI}{dt} &= \frac{\lambda SI}{S+I} - \frac{a_1 IY}{k_1+I} - \mu I,\\
\frac{dY}{dt} &= Y\Big(r_2 - \frac{a_2 Y}{I}\Big), \quad \text{if} \quad (S,I) \neq (0,0),\\
\frac{dY}{dt} &= 0, \quad \text{if} \quad (S,I) = (0,0),
\end{aligned} \tag{3}$$

subjected to positive initial conditions $S(0) > 0$, $I(0) > 0$, $Y(0) > 0$. The system is thus defined in the set Ω, where

$$\Omega = \{(S,I,Y) \in R^3 : S \geq 0, I \geq 0, Y \geq 0\}.$$

All parameters are assumed to be positive.

Different kind of delays are used in mathematical models to represent the biological events more accurately. For example, a negative feedback delay is considered in the logistic prey growth rate to represent density dependent feedback mechanism [42] and a positive feedback delay is considered to represent the gestation time of the predator [43]. Model system (3) assumes that the infection process is instantaneous. That means, as soon as an infected prey contacts a susceptible prey, the latter becomes infectious. However, in reality, there is a time-delay between the two events, viz., the first effective contact between susceptible and infectious preys and the newly infected prey becomes productively infectious. To incorporate this incubation delay, we rewrite the system (3) as

$$\begin{aligned}
\frac{dS}{dt} &= r_1 S - b_1 S(S+I) - \frac{\lambda SI}{S+I},\\
\frac{dI}{dt} &= \lambda \int_{-\infty}^{t} \frac{S(\tilde{u})I(\tilde{u})F(t-\tilde{u})d\tilde{u}}{S(\tilde{u})+I(\tilde{u})} - \frac{a_1 IY}{k_1+I} - \mu I,\\
\frac{dY}{dt} &= Y\Big(r_2 - \frac{a_2 Y}{I}\Big), \quad \text{if} \quad (S,I) \neq (0,0),\\
\frac{dY}{dt} &= 0, \quad \text{if} \quad (S,I) = (0,0).
\end{aligned} \tag{4}$$

Here we have assumed that the number of infectious preys at time t is arising from the contacts of actual population of susceptible and infectious preys at time $(t-\tilde{u})$, where $\tilde{u}$ is distributed according to a probability distribution function $F(\tilde{u})$, known as delay kernel (or memory function), defined by

$$F(\tilde{u}) = \frac{a^{p+1}\tilde{u}^p}{p!}\exp(-a\tilde{u}),$$

where a (>0) is a constant and p, known as order of the delay, is a non-negative integer. The average delay is defined as [44]

$$\bar{T} = \int_0^\infty \tilde{u}F(\tilde{u})d\tilde{u} = \frac{p+1}{a}.$$

If the kernel takes the form of a delta function, namely

$$F(\tilde{u}) = \delta(\tilde{u}-\tau),$$