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Original Research Article

Revealing the role of predator interference in a predator–prey system with disease in prey population

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ABSTRACT

Predation on a species subjected to an infectious disease can affect both the infection level and the population dynamics. There is an ongoing debate about the act of managing disease in natural populations through predation. Recent theoretical and empirical evidence shows that predation on infected populations can have both positive and negative influences on disease in prey populations. Here, we present a predator–prey system where the prey population is subjected to an infectious disease to explore the impact of predator on disease dynamics. Specifically, we investigate how the interference among predators affects the dynamics and structure of the predator–prey community. We perform a detailed numerical bifurcation analysis and find an unusually large variety of complex dynamics, such as, bistability, torus and chaos, in the presence of predators. We show that, depending on the strength of interference among predators, predators enhance or control disease outbreaks and population persistence. Moreover, the presence of multistable regimes makes the system very sensitive to perturbations and facilitates a number of regime shifts. Since, the habitat structure and the choice of predators deeply influence the interference among predators, thus before applying predators to control disease in prey populations or applying predator control strategy for wildlife management, it is essential to carefully investigate how these predators interact with each other in that specific habitat; otherwise it may lead to ecological disaster.

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1. Introduction

The relationship between predation and infection in prey populations is complex. There is evidence showing both increase and decrease of infection in prey populations in response to predation (Packer et al., 2003; Holt and Roy, 2007; Cáceres et al., 2009). According to “healthy herds” hypothesis (Packer et al., 2003), selective predation by the predator on infected prey helps to eliminate infectious individuals from the healthy population and thereby prevents the spread of disease. Evidence from different other fields also support this hypothesis (Pulkinen and Dieter, 2006). Various programs for the management of disease in natural

populations also suggest the control of the diseased population through predation (Hudson et al., 1998; Choisy and Rohani, 2006; Greenman and Hoyle, 2010; Hawlena et al., 2010). On the other hand, there are also studies showing an increase in infection in prey populations due to the presence of predators (Holt and Roy, 2007; Bate and Hilker, 2013a). Recently, Cáceres et al. (2009) presented an example using field patterns, experiments and a model study to show that the release of infective spores of fungal parasite by the predator facilitates epidemics in *Daphnia* population. Predators can also affect the persistence of prey populations that are regulated by infectious diseases (Chattopadhyay and Arino, 1999; Roy and Chattopadhyay, 2005). Therefore, how predators affect the disease dynamics in prey populations is still not clear and, thus, an interesting topic of research.

In the presence of a predator, a system with disease in a prey population can show different complex dynamical behaviors, like bistability, quasi-periodicity and chaos. Previously, there are studies showing some of these complex dynamics. For example, Upadhyay et al. (2008) found the existence of chaos via a period-doubling route

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in a predator–prey system with disease in a prey population. After the addition of a free-living virus stage in a predator–prey model with disease in a prey population, Siekmann et al. (2010) found bistability where depending on the initial conditions, the system can be made disease-free. Hilker and Malchow (2006) found strange periodic attractors with complicated, long lasting transient dynamics in a predator–prey model with disease transmission in a prey population. Furthermore, Sieber and Hilker (2011) demonstrated the occurrence of chaos, bistability and attractor crisis. The existence of such complexity makes the disease dynamics more complicated and difficult to predict. Thus, to know how a predator population affects disease dynamics, first we need a more thorough study on how the predator population affects the complexity of the system and then analyzing those complex results we can get information regarding the disease dynamics.

In most of the studies, it is assumed that predators do not interfere with each other's activities; thus the competition among predators occurs only via depletion of prey abundance. In reality, there are several situations when predators have to encounter with other predators, especially when predators have to search for food (and therefore, have to share or compete for food). In fact, predator interference has been found to occur quite frequently in laboratory and natural systems (Kratina et al., 2009; Skalski and Gilliam, 2001; Salt, 1974). There is many significant evidence of predator interference in predator–prey systems involving herbivore–plant, snail–barnacle, parasite–host, mite–mite and beetle–cricket interactions (Arditi and Ginzburg, 1989; Salt, 1974). Analyzing published data on eight predator–prey and seven host–parasitoid systems, Arditi and Akakaya (1990) evidenced strong predator interference in twelve out of fifteen cases. Predator interference is also important at very low and high prey and predator densities (Kratina et al., 2009; Skalski and Gilliam, 2001). Moreover, previous studies have shown that interference among predators is a dominant driver of food-web stability (Chakraborty and Chattopadhyay, 2011; Rall et al., 2008; van Voorn et al., 2008; Huisman and De Boer, 1997) and also has the ability to generate patchiness in a homogeneous environment (Alonso et al., 2002). In spite of such huge importance, the effects of predator interference on the predator–prey–disease interactions have never been thoroughly investigated. This paper is aimed to bridge the existing gap.

There are different ways of incorporating predator interference in a mathematical model, e.g., by considering ratio dependent functional response (Arditi and Ginzburg, 1989), including predator interference in a Holling type I functional response (Seo and De Angelis, 2011), including predator interference in a Holling type II functional response (Beddington, 1975; DeAngelis et al., 1975), density dependent mortality of predators (Holt, 1977). However, several previous researchers have suggested in favor of using Beddington–DeAngelis functional response which is similar to Holling type II functional response, but contains an extra term describing mutual interference among predators (Kratina et al., 2009; Skalski and Gilliam, 2001; Huisman and De Boer, 1997).

In the present study, we consider a predator–prey system in which the prey population is subjected to an infectious disease. We assume that the disease is transmitted via both vertically and horizontally. For horizontal transmission, we consider the density dependent disease transmission among the prey population, whereas due to vertical transmission, an infected prey produces only infected individuals (Sieber et al., 2014). This kind of vertical transmission occurs in the case of lysogenic infection where viruses enter and integrate their genome into the host's genome and start reproducing as the host reproduces and duplicates its genome (Malchow et al., 2004). For example, plankton system is very prone to lysogenic infection (Fuhrman and Suttle, 1993). Previously, there are several mathematical modeling studies dealt

with lysogenic infection in prey populations (Sieber et al., 2014; Malchow et al., 2004, 2005; Hilker et al., 2006). We further assume that the growth rate of susceptible prey is higher than that of the infected one (Hilker and Malchow, 2006; Hilker et al., 2006). The predator can consume both infected and healthy preys; however, the attack rates on infected and healthy preys are different (Chattopadhyay and Arino, 1999; Malchow et al., 2004; Hilker and Malchow, 2006; Hilker et al., 2006). We consider that predators interfere with each other and we represent this interference by considering Beddington–DeAngelis functional response (Beddington, 1975; DeAngelis et al., 1975). In order to study the long-term dynamics of the model we use numerical analysis techniques and perform a detailed numerical bifurcation analysis using AUTO (Doedel and Oldeman, 2009).

Here, we focus on investigating the role of predator interference on the disease dynamics in a prey population. For this reason, first we observe how interference among predators affects the system dynamics, especially, the complexity of the system by varying the interference strength and carrying capacity. From there, we comment on how different predator populations with different interference strengths regulate disease outbreaks and the persistence of the prey population.

The paper is organized as follows: in Section 2, we introduce the model for our investigation and mention about the possible equilibrium points. In Section 3, we examine how predator interference affects system dynamics by performing a rigorous bifurcation analysis on the model system. Finally, the paper ends with a discussion given in Section 4.

2. Basic model structure

We build an eco-epidemiological model that tracks population dynamics of susceptible prey $S(t)$, infected prey $I(t)$ and predator population $P(t)$ at time t . We construct the model based on the following assumptions:

- (A1) In the absence of infected (susceptible) prey and predation, the susceptible (infected) prey population follows logistic growth (Malchow et al., 2004; Hilker and Malchow, 2006).
- (A2) In the absence of predation, the susceptible and infected prey populations compete which is described by the classical Lotka–Volterra competition model. The interaction is weak–weak so that an interior stable equilibrium exists (Kot, 2001). Both susceptible and infected preys have a common carrying capacity K (Sieber et al., 2014). Moreover, the susceptible population becomes infected following the simple law of mass-action. We consider that the growth rate of infected prey is reduced due to infection (Hilker and Malchow, 2006; Hilker et al., 2006). We also assume that susceptible and infected individuals produce only susceptible and infected individuals, respectively.
- (A3) The infected prey does not recover or become immune but are removed by a constant death rate.
- (A4) The Beddington–DeAngelis functional response is chosen to represent predator's per capita feeding rate on susceptible and infected preys as $\beta_1 S / (1 + T_h(S + I) + T_i P)$ and $\beta_2 I / (1 + T_h(S + I) + T_i P)$ respectively, where β_1 / T_h and β_2 / T_h are the maximum uptake rates of susceptible and infected prey, respectively, and T_i is the constant positive parameter representing the interference among predators. It is to be mentioned here that the handling times for infected and susceptible prey are assumed to be same.
- (A5) Disease is spreading among the prey population only and the predator population is not directly affected by disease due to the predation of infected prey.

(A6) Conversion rate of susceptible prey into new predator is higher than that of the infected prey (Haque et al., 2009).

In view of the above assumptions the model takes the following form:

$$\frac{dS}{dt} = r_1 S \left(1 - \frac{S + a_{11}I}{K} \right) - \frac{\beta_1 SP}{1 + T_h(S + I) + T_i P} - \lambda SI, \quad (1)$$

$$\frac{dI}{dt} = r_2 I \left(1 - \frac{I + a_{22}S}{K} \right) - \frac{\beta_2 IP}{1 + T_h(S + I) + T_i P} + \lambda SI - \delta I, \quad (2)$$

$$\frac{dP}{dt} = \frac{\alpha_1 \beta_1 SP}{1 + T_h(S + I) + T_i P} + \frac{\alpha_2 \beta_2 IP}{1 + T_h(S + I) + T_i P} - \mu P. \quad (3)$$

Table 1 provides a list of the symbols for the variables and parameters. Here, r_1 and r_2 ($r_1 > r_2$) are the intrinsic growth rates of susceptible and infected prey, respectively (Hilker and Malchow, 2006; Hilker et al., 2006); a_{11} and a_{22} are the measures of the effect of infected prey on the growth of susceptible one and the measure of the effect of susceptible prey on the growth of infected one, respectively; λ is the disease transmission coefficient; δ is the death rate of infected population due to infection; α_1 and α_2 are the conversion factors of susceptible and infected prey, respectively, into new predators ($\alpha_1 > \alpha_2$); μ is the natural mortality rate of predator and T_h is the handling time times the searching rate needed for catching prey. In a similar way T_i is the interference time times the encounter rate between predator individuals.

We know that a model with explicit carrying capacity is suitable when susceptible and infected populations contribute equally to the density-dependence resulting from resource competition (in our case $a_{11} = a_{22} = 1$). However, in the case of different competitive abilities of susceptible and infected individuals, the concept of emergent carrying capacity is appropriate where the carrying capacities are not explicitly given, but can be seen as an upper limit of population growth that arises from reproduction and competition. A detailed description of emergent carrying capacity is provided in Sieber et al. (2014) and references therein. However, in the current paper, we consider a common carrying capacity for both susceptible and infected individuals with different competitive abilities, but the true carrying capacity would be emergent through the corresponding growth and the parameter K .

The model presented here is based on the structure of Sieber et al. (2014). The main difference with the previous model is that, in the present case, a density dependent disease transmission is considered instead of frequency dependent disease transmission

and predators interfere with each other. In the current form, the model itself is much more general; the model can also be used as a one predator–two prey competition model or as a one prey–intermediate predator–top predator system, with suitable choice of some of the parameter values (r_2 , a_{22} , λ and δ). However, we used this model as a SIP model to explore the impact of predator interference on the disease dynamics in the prey population.

System ((1)–(3)) has to be analyzed with the following set of initial conditions: $S(0) > 0$, $I(0) > 0$, $P(0) > 0$. This system possesses seven different equilibrium points: (i) prey and predator free equilibrium $E_0 = (0, 0, 0)$, (ii) infected prey and predator free equilibrium $E_S = (K, 0, 0)$, (iii) susceptible prey and predator free equilibrium $E_I = (0, (K/r_2)(r_2 - \delta), 0)$, (iv) predator free equilibrium $E_{SI}(S_{SI}, I_{SI}, 0)$, (v) susceptible prey free equilibrium $E_{IP}(0, I_{IP}, P_{IP})$, (vi) infected prey free equilibrium $E_{SP}(S_{SP}, 0, P_{SP})$, and (vii) the coexisting (interior) equilibrium $E_{SIP} = (S_{SIP}, I_{SIP}, P_{SIP})$.

However, we assume $r_2 < \delta$, which means that the infected prey can not survive in the absence of susceptible prey population, and therefore the $E_I = (0, (K/r_2)(r_2 - \delta), 0)$ equilibrium is not feasible and consequently also the $E_{IP}(0, I_{IP}, P_{IP})$ equilibrium is not feasible. The exact forms of the equilibria, their existence and stability conditions are given in the Appendix.

3. Results

In this section we focus on investigating the role of predator population on disease dynamics in the prey population. Specifically, we perform a rigorous bifurcation analysis to examine the impact of different predators with different interference strengths on disease dynamics by varying the interference strengths. Numerical simulation results are obtained by solving the set of ODE's with ode solvers available in MATLAB (MATLAB, 2012) and a numerical method based on bifurcation theory (Guckenheimer and Holmes, 1985; Kuznetsov, 2004 and references therein) and Kooi (2003) for applications in ecology. The numerical bifurcation analysis results are obtained using the numerical bifurcation package AUTO (Doedel and Oldeman, 2009). We calculate and continue equilibria and limit cycles and their stability as well as bifurcation points (critical parameter values) or curves of bifurcation points when two parameters, the interference strength among predators (T_i) and the carrying capacity (K), are varied simultaneously. For the numerical simulation, we have used the set of parameter values given in Table 1 unless it is specified otherwise. Since we want to investigate how predator population affects system dynamics, we choose the set of parameters in such a way that the absence of predator shows the stable coexistence of susceptible and infected prey. Table 2 gives a list of all attractors and bifurcation points and curves. For quasi-periodic and chaotic dynamic attractors, we calculated the Lyapunov exponents through the algorithm proposed in Wolf et al. (1985). In the following, we present full bifurcation analysis of our system.

3.1. Endemic predator-free or SI-system

For the system where the predator is absent, in the reference parameter ranges the disease-free system consists of susceptible prey, S , only ($S > 0$ and $I = 0$) and is stable below a transcritical bifurcation TC_{SI} at $K = 4$ for all T_i . Above this TC_{SI} curve ($K > 4$) this disease-free system is unstable and the endemic system SI ($S > 0$ and $I > 0$), is stable. In the region corresponding to the stable SI -system, we are now interested to see how the infected population I together with the susceptible population S behave in the presence of the predator. Numerical bifurcation analysis shows that for the parameter values in the region studied in this paper given in Table 1, this SI -system is always unstable in the three dimensional

Table 1

List of symbols for variables and parameters and parameter values used in the text. With this parameter set we have inequality $r_2 < \delta$. Consequently, this condition holds for all numerical results presented in this manuscript.

Symbol	Value	Description
S, I, P	Variable	Populations: susceptible, infected prey and predator
a_{11}, a_{22}	0.1	Competition parameters
K	Variable	Carrying capacity
r_1	0.55	Intrinsic growth rate susceptible prey
r_2	0.1	Intrinsic growth rate infected rate prey
T_h	0.1	Handling time $\times$ searching rate
T_i	Variable	Interference time $\times$ encounter rate
t	Variable	Time
α_1	0.4	Efficiency coefficient susceptible prey–predator
α_2	0.1	Efficiency coefficient infected prey–predator
β_1	0.215	Maximum feeding rate susceptible prey–predator
β_2	0.25	Maximum feeding rate infected prey–predator
δ	0.25	Death rate infected prey due to infection
λ	0.04	Disease transmission coefficient
μ	0.2	Predator death rate

Table 2
List of the bifurcations curves and points.

	Description
Attractor	
E	Equilibrium
L	Limit cycle
C	Complex dynamics: quasi-periodic solution or chaos
Bifurcation	
TC_{SP}	Transcritical bifurcation invasion predator into susceptible prey E_S equilibrium
TC_{SI}	Transcritical bifurcation invasion infected-prey into susceptible prey E_S equilibrium
TC_{SPI}^c	Transcritical bifurcation invasion infected-prey into susceptible-prey-predator equilibrium E_{SP}
TC_{SPI}^c	Transcritical bifurcation invasion infected-prey into susceptible-prey-predator system limit cycle L_{SP}
T	Tangent or saddle-node bifurcation collision of two equilibria or limit cycles
H	Hopf bifurcation origin of (un)stable limit cycle
TR	Torus bifurcation onset of quasi-periodic solution or chaotic dynamics
S	Destruction of quasi-periodic solution on torus extinction of predator

state space of the SIP -system. Therefore, we do not discuss the results for the SI -system in this section.

3.2. Infected prey-predator or IP -system

Since we have considered vertical transmission of infection in our system, thus, there is a possibility that the predator–infected prey system exists ($I > 0$ and $P > 0$). However, for the parameter values given in Table 1, the infected prey population does not exist and consequently also the predator is not able to invade the infected prey population. Therefore, we do not analyze this IP -system in this section.

3.3. Disease-free predator–prey or SP -system

Now, we study the ecological subsystem where the disease is absent, that is $I = 0$ and the susceptible population is the whole prey population. Fig. 1 is a two-parameter bifurcation diagram

Fig. 1. Two-parameter diagram for T_i and K for the endemic system. Table 2 gives a list of all bifurcation points and curves.

where the bifurcation parameters T_i and K are varied simultaneously. The transcritical bifurcation TC_{SP} is a horizontal line at $K = 3.030303$. Below this curve only the prey population exists ($S > 0$ and $P = 0$) whereas the coexistence of the predator and the prey population occurs above this curve ($S > 0$ and $P > 0$). In the latter region a Hopf bifurcation H_{SP} separates the regions with stable and unstable equilibria. Below H_{SP} , the equilibrium E_{SP} is stable and above H_{SP} , the equilibrium is unstable and stable limit cycle exists (L_{SP}). This bifurcation diagram of the predator–prey system has been studied in detail in van Voorn et al. (2008) where analytical expressions for these curves are derived.

3.4. Dynamics for Holling type II functional response

In this section we give the results for the reference case without predator interference where $T_i = 0$. Then the Beddington–DeAngelis functional form reduces to the Holling type II functional response.

The results are presented in the bifurcation diagram given in Fig. 2 where K is the bifurcation parameter. Increasing K from zero, at first only the susceptible population exists and increases with K linearly. At the transcritical bifurcation point TC_{SP} where $K = 3.030303$, the predator can invade. Above this point, at the equilibrium E_{SP} , the value for S remains constant whereas the predator P increases. Above the Hopf bifurcation H_{SP} , this SP -system equilibrium E_{SP} becomes unstable and a stable limit cycle L_{SP} exists. It is observed that the minimum values during the cycles become very low when K is increased. During these episodes, extinction due to stochastic effects is likely. This phenomenon is called the paradox of enrichment.

At the bifurcation point TC_{SPI}^c , the oscillatory SP system is invaded by the infected population. Above this point the limit cycle L_{SP} exists and is stable. The stable limit cycle becomes unstable at a torus bifurcation TR_{SPI} where the dynamics becomes chaotic C_{SPI} . With this chaotic dynamics there are also episodes where the population size becomes very low again leading to the paradox of enrichment.

For the sake of completeness, we mention that at the transcritical bifurcation TC_{SI} ($K = 4$), the infected population can invade the susceptible population, but the resulting SI -system is unstable when the predator population exists.

Fig. 2. One-parameter bifurcation diagram for susceptible S , infected I and predator population P with free parameter K where $T_i = 0$. The solid (dashed) curves denote stable (unstable) equilibrium values and extreme values of stable (unstable) limit cycles, and dots denote local maximum values in the chaotic region. Table 2 gives a list of all attractors and bifurcation points and curves.

In the remaining part of this section we continue with the analysis of the SIP-system with predator interference described by system ((1)–(3)).

3.5. Endemic predator–prey or SIP-system

We now give the results for the Endemic predator–prey system with the Beddington–DeAngelis functional response, that is $T_i > 0$. This system is an extension of the SP-system. The susceptible prey population of the predator–prey SP-system can become infected in two regions, for low and high T_i ranges shown in Fig. 1. The SP-system can be invaded by the infected population either in an equilibrium or in a limit cycle state. In the following, we examine different dynamical behaviors of the system by varying T_i at different ranges of K .

3.5.1. Dynamics for $K < 150$

At first we will discuss the dynamics for $K = 150$ shown in Fig. 3. There are two transcritical bifurcations, TC_{SPI}^e and TC_{SPI}^c , in addition to the one, TC_{SP} , in Fig. 1 discussed above. Above the transcritical bifurcation point TC_{SPI}^e , the disease-free E_{SP} system in equilibrium gets invaded by the disease. The resulting endemic system has a stable equilibrium E_{SIP} above the transcritical bifurcation TC_{SPI}^e . Between this bifurcation point and the Hopf bifurcation of the disease-free system H_{SP} , the disease-free system E_{SP} is stable. Between the Hopf bifurcation H_{SP} and the transcritical bifurcation TC_{SPI}^c , there is a stable SP-system limit cycle L_{SP} . For lower T_i values, this SP-system limit cycle L_{SP} becomes unstable and the positive SIP-system stable limit cycle L_{SIP} emerges. The endemic system limit cycles that originate from this transcritical bifurcation curve become unstable at a torus bifurcation curve TR_{SIP} shown in Fig. 1. Beyond this point the dynamics is complex: chaotic with possibly periodic windows.

Lowering K and for low T_i values, first of all bifurcation TR_{SIP} disappears, followed by the bifurcation TC_{SPI}^c and finally H_{SP} in which case the disease-free equilibrium E_{SP} is stable for a large interval of T_i before the predator goes extinct below TC_{SP} .

Next, we will discuss the dynamics for $150 < K < 225$ and later for $K > 225$. It is clear from Fig. 4, which is actually a detailed view of Fig. 1, that some interesting results occur for $K < 225$ with $T_i < 0.24$, and for $K > 225$ with $T_i > 0.24$, where the value $T_i = 0.24$ is in a region where the disease-free equilibrium E_{SP} is always

Fig. 3. One-parameter bifurcation diagram for susceptible S , infected I and predator population P with free parameter T_i where $K = 150$. The solid (dashed) curves denote stable (unstable) equilibrium values and extreme values of stable (unstable) limit cycles, and dots denote local maximum values in the chaotic region. Table 2 gives a list of all attractors and bifurcation points and curves.

Fig. 4. Two-parameter diagram for T_i and K . A detail of Fig. 1 for $0.14 \leq T_i \leq 0.32$ and $200 \leq K \leq 270$. Table 2 gives a list of all bifurcation points and curves.

stable for $3.0303 < K \leq 300$. To get a clearer view, in the following we will present one-parameter bifurcation diagrams for various fixed K -values and varying T_i -values.

3.5.2. Dynamics for $150 < K < 225$

Fig. 1 is the two-parameter bifurcation diagram for the SIP system. For higher T_i values, the endemic system has a stable equilibrium E_{SIP} between the transcritical bifurcation TC_{SPI}^e and the Hopf bifurcation H_{SIP} . Above the Hopf bifurcation H_{SIP} , a stable limit cycles L_{SIP} exists in the endemic region for higher K and T_i values. Furthermore, for smaller T_i values (above the Hopf bifurcation H_{SP}), the oscillating SP-system is invaded by the infected prey via the limit cycle above the transcritical bifurcation TC_{SPI}^c . Above the torus bifurcation curve TR_{SIP} , the dynamics is complex: chaotic with possibly periodic windows.

Now, we will discuss Fig. 4 to study the dynamics of the system. Interestingly, we find codimension-two bifurcation points where two codimension-one bifurcation curves intersect. For instance, as we increase K starting from 150, the bifurcation curve TR_{SIP} continues as a tangent bifurcation curve T_{SIP} at the codimension-two bifurcation point marked with a diamond.

In a similar way, from the transcritical bifurcation curve TC_{SPI}^c of the disease-free limit cycle, at the codimension-two bifurcation point marked by a bullet, a tangent bifurcation, now for endemic limit cycles T_{SIP} emerges. This tangent bifurcation curve undergoes two, often called, cusp bifurcations (not labeled in the figure) before it leaves the diagram at the top with $T_i \approx 0.29$. From this tangent curve for limit cycles, two torus bifurcation curves TR_{SIP} originate at different places. The first one occurs at $T_i \approx 0.16$ as we have shown above. The second one occurs at $T_i \approx 0.24$ and the emerging torus bifurcation curve leaves the diagram at the top at $T_i \approx 0.31$ as we increase T_i and K values. At this latter codimension-two bifurcation point which is also labeled with a diamond, a curve labeled S originates. This curve will be discussed later together with the quasi-periodic dynamics occurring between the curves TR_{SIP} and S .

It is clear from Fig. 1 that for moderate values of the interference strength, there exists a range of interference strengths where the system becomes disease-free. For a certain range around $T_i = 0.27$, the system remains even stable in equilibrium E_{SP} for sufficiently large values of the carrying capacity K . It is important to note that, in the absence of the predator, the disease was present in the

system in stable equilibrium E_{SI} in the same range of interference strength. Moreover, in the region corresponding to the stable interior equilibrium point (E_{SIP}), which covers a large region as shown in Fig. 1, it is found that the size of the infected population remains low compared to the case in the absence of predator (E_{SI}) (the figure is not shown). Therefore, in the regions of stable E_{SP} and E_{SIP} , the presence of predator actually helps to control or eradicate disease from the prey population.

Fig. 5 shows the stable attractor values for the state variables: susceptible prey S and infected prey I as a function of T_i for $K = 209$. The bifurcation pattern is similar to that as for $K = 150$ with the following exceptions. For high T_i values, above the TC_{SPI}^e bifurcation, a stable equilibrium E_{SIP} exists except in the region between the two Hopf bifurcations H_{SIP} where a limit cycle exists. In the lower T_i region, below this transcritical bifurcation TC_{SPI}^e , a disease-free equilibrium E_{SP} is stable and becomes unstable at a Hopf bifurcation H_{SP} where the disease-free system oscillates as a stable limit cycle L_{SP} . For lower T_i -values, the dynamical behavior is similar to that for the $K = 150$ case shown in Fig. 3.

Fig. 6 is a three-dimensional plot where the two-parameter diagram forms the bottom plane and along the vertical axis, the minimum values of the infected population are plotted. The chaotic region is plotted for $K = 209$ where the origin is formed by the tangent bifurcation T_{SIP} . The chaotic region is also plotted for $K = 200$ and $K = 220$ where the origin is formed by torus bifurcation curves TR_{SIP} such as in Fig. 3 and a transcritical bifurcation TC_{SPI}^e of the disease-free system, respectively. These changes are due to passing the codimension-two point where the torus bifurcation curve TR_{SIP} merges with the tangent bifurcation curve T_{SIP} , and the codimension-two point where this tangent bifurcation curve T_{SIP} merges with the transcritical bifurcation curve TC_{SPI}^e , following the arrows in Fig. 6. In addition to these chaotic attractors, the torus bifurcation TR_{SIP} , tangent bifurcation T_{SIP} , and transcritical bifurcation TC_{SPI}^e curves are plotted. These bifurcations are for limit cycles and only the minimum values of the infected populations are shown.

From this figure we observe that increasing K starting from $K \approx 195$ and $T_i = 0.145$, the boundary of the chaotic regions is formed firstly by a torus bifurcation TR_{SIP} , then after passing a codimension-two point where the torus bifurcation terminates via a tangent bifurcation T_{SIP} , and finally above the codimension two

Fig. 6. Two-parameter plot with local minimum values for the infected I in the chaotic regions for $K = 200$, 209 and $K = 220$ projected. The bullets mark codimension-two points. Open circles mark the torus bifurcation TR_{SIP} at $K = 200$ and the tangent bifurcation T_{SIP} at $K = 209$ and transcritical bifurcation TC_{SPI}^e at $K = 220$. These points mark the end-points of the chaotic regions.

bifurcation point where the tangent bifurcation terminates via a transcritical bifurcation TC_{SPI}^e . The tangent bifurcation curve T_{SIP} forms the bridge (see Fig. 4) with the dynamics for higher predator interferences at high carrying capacities which is the subject of the next subsection. Specifically, Fig. 6 gives a more detailed view of Fig. 4 to observe three different routes to chaos in a clearer way in a single frame.

In conclusion, at high carrying capacities, there are two ranges of interference strength where the system shows fluctuation of all the populations: chaotic oscillation at the low range of interference strength and limit cycle oscillation at moderate to high interference strengths. One thing is common in both of the oscillating regions; minimum abundances of all the populations become very low so that environmental fluctuation can result stochastic extinction of any of the populations. Moreover, fluctuation of the infected population also represents outbreaks of disease. There is also a range of interference strength (for moderate values) where the system becomes disease-free.

3.5.3. Dynamics for $K > 225$

In order to study what happens in the region for $K > 225$ and $T_i > 0.24$ of the two parameter diagram given in Fig. 1, we also deal with one parameter bifurcation diagrams for $K = 247$ and $K = 250$.

Fig. 7 shows the stable attractor values for the state variable: susceptible prey S and infected prey I as a function of T_i for $K = 247$. For higher T_i -values, there are now two tangent bifurcations T_{SIP} between which the limit cycle is unstable. Furthermore, the transcritical bifurcation TC_{SPI}^e , where the chaotic region for lower T_i -values terminates, is now catastrophic and the originating endemic limit cycle is unstable. For increasing T_i , this unstable limit cycle becomes stable at a third tangent bifurcation T_{SIP} . This stable limit cycle loses its stability at a torus bifurcation TR_{SIP} . The dynamics on the torus appears to be quasi-periodic and it terminates by a collision with a saddle limit cycle.

Fig. 8, similar to Fig. 6, is a three-dimensional plot where the two parameter diagram forms the bottom plane and on the vertical axis, the minimum values of the infected population are plotted. At the back vertical plane, now the attractor values for the minimum abundances of the infected population I_{min} for $K = 250$ is shown together with the I_{min} values of the transcritical bifurcation TC_{SPI}^e of the disease-free system. Additionally, chaotic attractors, the torus

Fig. 5. One-parameter bifurcation diagram for susceptible S and infected I with free parameter T_i where $K = 209$. See Fig. 3 for a description of the shown symbols and curves.

Fig. 7. One-parameter bifurcation diagram for susceptible S and infected I with free parameter T_i where $K = 247$. See Fig. 3 for a description of the shown symbols and curves.

Fig. 9. One-parameter bifurcation diagram for susceptible S and infected I with free parameter T_i where $K = 250$. See Fig. 3 for a description of the shown symbols and curves.

bifurcation TR_{SIP} , tangent bifurcation T_{SIP} and transcritical bifurcation T_{SIP} curves are also drawn.

Fig. 9 gives the attractor values of susceptible prey S and infected prey I at $K = 250$. Comparing this bifurcation pattern with the one for $K = 247$ (Fig. 7) we find that, in the interval $247 < K < 250$, an unstable bifurcation occurs where the two stable and the two unstable limit cycles become connected as K is increased.

We now discuss the dynamics on the torus that originates from the torus bifurcation TR_{SIP} . This torus bifurcation occurs with $K = 250$ at $T_i = 0.26925342$. The dynamics on the torus emerging from this torus bifurcation is shown in Fig. 10 for the Poincaré plane for $dI/dt = 0$ where $T_i = 0.268$ and $T_i = 0.265$ (red dots). The dots in the diagram will form closed curves when simulations continue for longer times and hence show quasi-periodic dynamics on the torus. This was verified by the estimation the Lyapunov exponents through the algorithm proposed in Wolf et al. (1985). We found that two dominant Lyapunov exponents are zero.

Fig. 8. Two-parameter plot with local minimum values for the infected I in the region of the torus quasi-periodic solution and the chaotic region projected for $K = 250$. The bullet marks the codimension-two point where the torus bifurcation TR_{SIP} terminates at a tangent bifurcation T and the open circle marks the end-points of the chaotic region in the low T_i range at a transcritical bifurcation TC .

Here we describe the scenario, how the torus bifurcation is destroyed abruptly when the parameter T_i is decreased starting from the torus bifurcation TR_{SIP} . Between $T_i = 0.2619885$ and $T_i = 0.2619884$, the torus is destroyed. Fig. 10 shows the cross-sections with the Poincaré plane for the trajectories on the torus with $T_i = 0.2619885$ (red) and for $T_i = 0.2619884$ (blue); the dots leave the torus after two toroidal revolutions and converge to a stable equilibrium E_{SP} where $I = 0$.

Fig. 11 shows the phase-plot for the three state variables: susceptible prey S , infected prey I and predator P at $K = 250$ for $T_i = 0.2619885$ (red) and $T_i = 0.2619884$ (blue). Moreover, the unstable limit cycle for $T_i = 0.2698845$ (green) is also shown. At $T_i = 0.2619884$, the torus collided with the saddle limit cycle entirely along the surface of the torus the poloidal trajectories and the trajectory ends at the disease-free E_{SP} stable equilibrium. By showing only values for $S < 45$, Fig. 12 illustrates the dynamics on the Poincaré section for the plane $S = 45$ with the same color code as used in Fig. 11. When the dynamics on the torus is quasi-periodic

Fig. 10. Minimum map for I versus S for $K = 250$. The finite time dynamics on the torus for two intermediate values $T_i = 0.268$ and $T_i = 0.265$. Note that for longer times the dots form a closed curve in the Poincaré plane where the right-hand side of $dI/dt = 0$. Furthermore for two T_i just above and below the connection between the torus and the disease-free system equilibrium. Red $K = 250$ and $T_i = 0.2619885$ and blue $K = 250$ and $T_i = 0.2619884$. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 11. Phase plot susceptible S , infected I , and predator P close to the heteroclinic bifurcation for the torus quasi-periodic solution (red $T_i = 0.2619885$) and convergence to the disease-free system (blue $T_i = 0.2619884$) and the saddle limit cycle (green). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

such that the solution completely covers the torus surface this picture clearly shows the destruction. The dynamics on the invariant torus is broken at the T_i parameter where it hits the saddle limit cycle. So, the torus is destroyed by collision with a saddle limit cycle.

Also, when $K = 250$, just as for $K = 220$ shown in Fig. 6, there is chaotic dynamics in the low T_i -range below the transcritical bifurcation TC_{SP}^c . Fig. 13 is the phase-plot for the three state variables: susceptible prey S , infected prey I and predator P where $K = 250$ and $T_i = 0.1785$ (red). Also the limit cycle at the transcritical bifurcation TC_{SP}^c at $T_i = 0.1785$ (green) is shown. With T_i at the transcritical bifurcation the chaotic attractor hits the invariant plane $I = 0$ and the solution converges to the stable SP limit cycle.

In the following, we will discuss the bistabilities and the regime shifts that have been observed during bifurcation analysis for $K > 225$.

3.5.4. Bistabilities

We observe the existence of several bistabilities when both, the carrying capacity and the strength of predator interference, remain high. We find that the tangent bifurcation gives birth to bistability between two coexistent oscillations. Specifically, the coexistent stable periodic orbit becomes unstable via a tangent bifurcation and further becomes stable via another tangent bifurcation (Fig. 7). Within these two tangent bifurcations, there is a range of T_i for which both coexistent oscillations coexist. We also find bistability between coexistent oscillations and coexistent equilibria. There are also scenarios when there is bistability between a coexistent

Fig. 12. Phase plot susceptible S , infected I , and predator P same as in Fig. 11 but now only for $S < 45$. Torus quasi-periodic solution (red $T_i = 0.2619885$) and convergence to the disease-free system (blue $T_i = 0.2619884$) and the saddle limit cycle (green). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Fig. 13. Phase plot susceptible S , infected I close to the transition from the cyclic endemic system and convergence to the cyclic disease-free system (red $K = 250$, $T_i = 0.17851$) and the unstable limit cycle (green) at the transcritical bifurcation. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

oscillation and disease-free equilibria. Furthermore, a bistability between a disease-free equilibria and a coexisting torus has also been observed. In the last two scenarios, it is the initial condition that determines whether the disease will be endemic or not.

3.5.5. Regime shifts and hystereses

At high carrying capacities and high strengths of predator interference, we find the possibility of regime shifts and hystereses (Figs. 7 and 9). Regime shifts can be defined as large, unexpected, persistent changes in the structure and function of a system (Biggs et al., 2009). According to Scheffer (2009), a regime shift is a “critical transition” which occurs when there is a drastic change toward another state caused by minor perturbations and/or a gradual change in the system parameters. This definition does not include drastic changes due to the large and sudden changes to the system. According to this definition, a regime shift occurs when there is a discontinuity in stable attractors due to the variation of a particular parameter. In the present study, we find several regime shifts because of the existence of tangent bifurcation, bistability, and the destruction of a stable torus. According to Bate and Hilker (2013b), we can classify these regime shifts into two different classes; reversible and irreversible.

In the case of a reversible regime shift, due to a sequence of small changes in the bifurcation parameter, it is possible to return to the starting point, via a hysteresis loop Bate and Hilker (2013b). An example of a reversible regime shift can be found in Fig. 7. There are three tangent bifurcations in Fig. 7. If we start just right to the second tangent bifurcation, we will be in a coexistent oscillatory state. Now, slowly diminishing the strength of predator interference below the tangent bifurcation point will mean that the system will eventually approach the other coexistent oscillatory state (say, second) after some oscillatory transient. Now, we are in a second coexistent oscillatory state and if we start increasing the strength of predator interference slowly, we will still be on the second oscillatory state until the first tangent bifurcation (from the right) is passed. If we pass this first tangent bifurcation point, we will be in the first oscillatory state, however the current interference strength will be much higher than the original interference strength. Once there, slowly decreasing the interference strength will move the system to the original state near the tangent point on the initial coexistent oscillations.

On the other hand, in the case of an irreversible regime shift, there is no such sequence of small changes to come back to the starting point, i.e., there is no hysteresis loop (Bate and Hilker, 2013b). Therefore, in this case, once the system goes away from the starting point and leaves one stable state, only a huge perturbation away from another stable state can make it possible to return to the

starting point. For example, in Fig. 7, it is really difficult to reach to the torus or adjacent endemic limit cycle via equilibria. This means when starting on the stable torus or adjacent coexistent limit cycle, slowly decreasing interference strength below the destruction of the torus or increasing interference strength beyond the third transcritical bifurcation point (from right) of coexistent oscillation would lead to the end of coexistent limit cycle/torus forever.

From the above analysis, it is clear that the dynamics are more complex for $K > 225$. Similar to the case of $K < 225$, here also we find two ranges of interference strength where all the populations fluctuate (chaotic oscillation, quasi-periodic oscillation and limit cycle oscillation). On the one hand, this fluctuations increase the possibility of population extinction due to stochastic fluctuation, on the other hand, oscillations of the infected population represent outbreaks of disease. Moreover, the range of interference strength for limit cycle oscillation is larger in the current case compared to $K < 225$. Here also, there is a range of interference strength for which disease-free system exists. However, due to bistability, the disease-free equilibrium exists with the coexisting equilibrium for a small range of interference strength, which means that, initial conditions will play a big role in determining whether the system will be disease-free or not. In this case, if the system is not disease-free, due to the irreversible regime shift, a perturbation can make the system disease-free and the system stays in this state unless there is a huge change in the system.

4. Discussion

Various programs for the management of disease in natural populations suggest the control of infection through predation (Hudson et al., 1998; Choisy and Rohani, 2006; Hawlena et al., 2010; Greenman and Hoyle, 2010). However, several theoretical and empirical evidence shows both increase and decrease of the infection prevalence of the disease due to the predation on infected population (Packer et al., 2003; Holt and Roy, 2007; Cáceres et al., 2009). In the present study, we also explored the role of a predator in influencing disease dynamics in a prey population but from a different perspective. Moreover, we also investigated how different predators with different interference strengths ($T_i > 0$) lead to different dynamical behaviors and influence disease dynamics.

4.1. Disease dynamics at different interference strengths

We have found that the infected prey disappears from the system for moderate values of interference strengths, however they are successfully invaded in the system at very high and low interference strengths (Fig. 1). We can explain the existence and disappearance of disease from the system due to the variation in interference strengths as follows. Due to higher growth rate, susceptible prey grows faster and gets an advantage in competition compared to the infected prey. Now when interference strength is low, predation remains important and decreases the competition among susceptible and infected preys which helps infected prey to survive. Now, as interference among predators increases, predation decreases, and susceptible prey starts growing faster compared to the infected one. In the situation when interference strength becomes very high, disease transmission plays a big role for the infected prey, i.e., gain due to the conversion of susceptible to infected prey becomes high which helps infected population to overcome their competitive disadvantage and as a result infection exists in the system. However, for moderate values of the interference strength, predation is relatively less important and also the abundance of susceptible prey is not high enough to provide sufficient input to the infected class so that infected prey can overcome their competitive disadvantage. As a result, infected prey cannot survive in this range of interference strength and the

system remains disease-free which is important from an ecological point of view.

4.2. Regime shifts

We found different bistable regions at moderate interference strengths with various types of bistabilities. Previously, only a few studies found the existence of bistability in eco-epidemiological systems (e.g., Siekmann et al., 2010; Kooi et al., 2011; Sieber and Hilker, 2011; Bate and Hilker, 2013b). In our present study, in the case of a bistability between two different coexistent oscillations, fluctuations of infected population are different. Although, both of the situations show outbreak of disease, but depending on the initial conditions, disease outbreak can be large or small. In the other case, when bistability exists between a coexistent oscillations and coexistent equilibria, depending on the initial condition, there can be widespread outbreaks of disease or disease can persist in the system at a low level. However, the bistability between disease-free equilibria and coexistent oscillations, and disease-free equilibria and coexisting torus show the possibility of widespread outbreaks of disease or complete removal of disease from the system depending on the initial population abundances.

All these bistabilities results in several hystereses and regime shifts. Some of them are reversible via long and complex sequences of small changes in parameter values, and others are irreversible. We found that the stable coexistent torus and some of the stable oscillations in Figs. 7 and 9 are not recoverable when once lost, without huge perturbations. Previously, Bate and Hilker (2013b) observed similar type of reversible and irreversible regime shifts in a predator–prey system with disease in predator population. In our case, due to the regime shift, a proper small change in the interference strength can prevent widespread outbreaks of disease and make the system disease-free, and due to the irreversibility, the system remains disease-free for a certain range of interference strength unless there is a huge change in the system.

4.3. Existence of torus and chaos

We found the existence of quasi-periodic dynamics on a torus in our system. There are many ways how this dynamics on the torus can bifurcate or disappear. For example, via generation of chaos or there could be phase locking into a periodic orbit or can be destruction by a saddle-cycle, called a homoclinic bifurcation in (Bate and Hilker, 2013b). We also found that the torus was destructed by a saddle limit cycle, the saddle limit cycle itself acts as a separatrix. It is tempting by seeing the results in Fig. 12 to call this a homoclinic bifurcation as in Bate and Hilker (2013b), but it can be problematic to denote it as “homoclinic connection”. When we do not know whether the dynamics on the torus remains quasi-periodic, a candidate is this dynamics at the parameter values where the collision occurs. However, in Scheffer et al. (1997), it is mentioned that the torus can disappear when it collides with the saddle cycle, but they continue with: “Actually the torus deconstruction occurs after a sequence of bifurcations, through which the torus loses its smoothness before finally disappearing. Just before this happens, the torus is very close to the saddle cycle, so that from time to time the trajectories in the torus remain very similar to the saddle cycle for a long period”. We denote this global bifurcation phenomenon by “torus destruction by a saddle limit cycle” because calling it a homoclinic bifurcation would require the description of the homoclinic connection and this is beyond the scope of this paper.

Another interesting result of this study is the existence of different ways of origination of chaos at low predator interference strengths. Although chaotic behavior itself is interesting, routes to chaos are also important. The most observed route is via a cascade

of period doubling (Thompson and Stewart, 1986). Also other routes, like “Intermittency” (Pomeau and Manneville, 1980) and “Ruelle-Takens-Newhouse” (Newhouse et al., 1978) exist. In a series of papers, see the references in Deng and Loladse (2007), a simple Rosenzweig–MacArthur tri-trophic food chain model satisfying the trophic time diversification hypothesis which translates the model into a singular perturbed system of three time scales, shows at least four different types of chaos generation mechanisms have been classified. In Boer et al. (1999, 2001), Kooi et al. (2004) and van Voorn et al. (2010), numerical algorithms and software are developed for detecting and continuing bifurcations related to homoclinic (cycle-to-cycle) or heteroclinic (point-to-cycle) orbits and used to systematically calculate the boundaries of the chaotic regions in the parameter space. In Kooi et al. (2011) this was done with the analysis of the eco-epidemiological predator–prey model with predator suffering from an infectious disease. Here, we found three known different ways in which chaos can originate under parameter variation: via a torus bifurcation (Newhouse et al., 1978), a tangent bifurcation (Pomeau and Manneville, 1980) and a transcritical bifurcation (Deng and Hines, 2003).

The existence of the torus and chaos has a deep impact on the disease dynamics. One hand, chaos brings huge fluctuations in all the populations representing huge outbreaks of disease. On the other hand, the existence of oscillations, torus and chaos lead to a concern regarding dangerously small population sizes which is common with many other models exhibiting oscillation and complex dynamics (Bate and Hilker, 2013b; Thomas et al., 1980). Some of the interesting dynamics occur in scenarios of major boom and bust, cases that are likely to cause extinctions due to the stochastic fluctuation. In particular, looking at the bifurcation diagrams in Figs. 3, 5, 7 and 9, we see that the amplitude of the fluctuating populations is very large and the minimum values of all the populations become very small in the cases of limit cycles, torus and chaotic oscillations which increases the possibility of population extinction.

4.4. Comparison of results with Sieber et al. (2014)

Our model system is similar in structure with the model considered by Sieber et al. (2014) except in the disease transmission term where we considered a density dependent transmission instead of frequency dependent transmission used by Sieber et al. (2014) and also the incorporation of interference among predators in our model. Considering emergent carrying for the prey population, Sieber et al. (2014) observed the existence of stationary coexistence of all the populations which was not possible with explicit carrying capacity. Moreover, they found the existence of bistability in their model which indicates the possibility of disease-induced extinction due to the overcritical inflow of additional infected individuals. Our system also follows emergent carrying capacity formulation and shows stationary coexistence and bistability in the presence of interference among predators. Moreover, we observed the existence of more complex dynamics, such as torus and chaos. However, with the parametric setup considered for the numerical simulation, we did not observe the stationary stable coexistence of all the populations in the absence of interference among predators.

4.5. Some real world applications

Here we uncovered a new mechanism: depending on the strength of interference among predators, introduction of predator to control disease in prey populations may have both positive and negative influences on disease spread and persistence. This can have a practical application in the field of pest control. If we consider pests as prey and natural predators as a predator, then our study provides an insight into the management strategies in a pest

control program. When a pest population becomes large, crop will be affected heavily, resulting in economic losses. Thus, our target always remains to increase crop by decreasing pest abundance. According to our study, if the pest population is affected by disease, then depending on the strength of predator interference, one can set strategy to control pest population. Specifically, the introduction of a predator with very low interference strength would be beneficial because the incorporation of a predator with proper amount will induce oscillation in the system and makes the pest abundance very low. Now, putting a little bit effort at this stage would easily eradicate pest population from the system. Another application of our study can be in the field of predator control as a wildlife management tool. Predator control is considered as one of the oldest and most widespread wildlife management tools in the world (Murie, 1940). Although several predator-management programs exist (Hone, 1994; Cote and Sutherland, 1997), but only a few works have been devoted to analyze the effectiveness of control strategies (Hone, 1994; Cote and Sutherland, 1997; Conner et al., 1998). The success of predator control is most often judged by an increase in prey abundance (Conner et al., 1998; Boggess et al., 1990). Now, if the prey population is affected by a disease, then it would be beneficial to introduce a predator with moderate to high interference strength (depending on carrying capacity) which will decrease the infected prey and ultimately there will be an increase in the susceptible prey abundance. However, the introduction of predator with low interference strength can completely eliminate the prey population from the system. Thus, our study suggests that, before the introduction of predator to control prey, it is essential to carefully investigate the interference among predators otherwise predator control can have unexpected consequences on the abundance of the target prey population (Cote and Sutherland, 1997; Sih et al., 1985).

We conclude that the presence of a predator can both increase and suppress infection in a prey population depending on the interference strength among predators. However, the relationship between the interference strength and its effects on systems is very complex and needs further investigation. The strength of interference among predators is habitat specific. Habitat structures can change encounters between predators (Norton et al., 2001) and may therefore alter interference among predators (Grabowski and Powers, 2004). As a result, changes in habitat may lead to different outcomes in response to introduction of predator to control disease. Moreover, different predators with different interference strengths can have different impacts on system dynamics. In conclusion, our study prompts a very important, general warning: before applying predator to control disease in prey populations or applying predator control strategy for wildlife management, it is essential to carefully investigate the role of that predator in controlling disease and how the predators interact with each other in that specific habitat; otherwise it may lead to ecological disaster.

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Appendix A. Existence and stability of equilibrium points: linear stability analysis

In this Appendix we study the equilibria of the system analytically using the symbolic program Maple (2008) to obtain expressions for the equilibrium values of the state variables and the eigenvalues of

the Jacobian matrix evaluated at the equilibria. This gives a complete overview in all possible equilibria which can be used to get starting points for continuation of equilibria and limit cycles using AUTO (Doedel and Oldeman, 2009). For such a numerical bifurcation analysis parameter values are needed. In parameter regions with chaotic dynamics brute-force simulation are performed using MATLAB (2012) code.

All numerical results presented in this article were obtained for the parameter values given in Table 1. We restrict the analysis here to cases where one relationship between the two parameters r_2 and δ , namely $r_2 < \delta$, holds true in addition to the property that all parameter values are positive. This restriction represents that the infected population cannot survive in the absence of susceptible population.

We start with an analysis of the one and two-dimensional subsystems.

A.1. S-System

The S-system Eq. (1) with $I = P = 0$ is the logistic growth equation and there is convergence to the carrying capacity $S^* = K$.

A.2. I-System

The I-system Eq. (2) with $S = P = 0$ is the logistic growth equation but the effective intrinsic growth rate is negative because $r_2 < \delta$. Therefore the zero equilibrium $I_j^* = 0$ is stable and consequently this equilibrium is uninvadable by the predator population. Therefore the two-dimensional IP-system is not further discussed.

A.3. SI-System

The SI-system Eqs. (1) and (2) with $P = 0$ in the Lotka–Volterra competition model together with the classical epidemiological SI-model with an additional death rate of the infected prey. The Jacobian matrix evaluated at the $(0, 0)$ zero equilibrium is a diagonal matrix with elements $(r_1, r_2 - \delta)$ which are the eigenvalues. Because $r_1 > 0$ equilibrium E_0 is always unstable for the two dimensional SI-system.

The interior two-dimensional equilibrium (S_{SI}, I_{SI}) is given by $S_{SI}^* = (r_1 K - (r_1 a_{11} + \lambda K) I_{SI}) / r_1$ and $I_{SI}^* = \frac{r_1 K (r_2 a_{22} - r_2 - \lambda K + \delta)}{(r_1 a_{11} + \lambda K)(r_2 a_{22} - \lambda K) - r_1 r_2}$. The predator free state exists if $r_1 K - (r_1 a_{11} + \lambda K) I_{SI} > 0$ together with either both $r_2 a_{22} - r_2 - \lambda K + \delta > 0$ and $(r_1 a_{11} + \lambda K)(r_2 a_{22} - \lambda K) - r_1 r_2 > 0$ holds or both $r_2 a_{22} - r_2 - \lambda K + \delta < 0$ and $(r_1 a_{11} + \lambda K)(r_2 a_{22} - \lambda K) - r_1 r_2 < 0$ holds. One of the eigenvalues is $\frac{\alpha_1 \beta_1 S_{SI} + \alpha_2 \beta_2 I_{SI}}{1 + T_h I_{SI} + T_h S_{SI}} - \mu$ and the other two eigenvalues are the roots of the quadratic equation

$$\xi^2 + D_1 \xi + D_2 = 0,$$

where

$$D_1 = \frac{(r_1 S_2 + r_2 I_2)}{K},$$

$$D_2 = \left[\frac{r_1 r_2}{K^2} + \left(\lambda + \frac{r_1 a_{11}}{K} \right) \left(\lambda - \frac{r_2 a_{22}}{K} \right) \right] S_{SI} I_{SI}.$$

Therefore, E_{SI} is LAS if $\frac{\alpha_1 \beta_1 S_{SI} + \alpha_2 \beta_2 I_{SI}}{1 + T_h I_{SI} + T_h S_{SI}} < \mu$ and $(r_1 r_2 / K^2) + (\lambda + (r_1 a_{11} / K))(\lambda - (r_2 a_{22} / K)) > 0$.

A.4. SIP-System

System (1)–(3) possesses the following seven equilibria, $E_0, E_S, E_I, E_{SI}, E_{SP}, E_{IP}, E_{SIP}$. As mentioned above because $r_2 < \delta$ we do not discuss the E_I and E_{IP} equilibria.

E_0 : The prey and predator free equilibrium point $E_0 = (0, 0, 0)$. The Jacobian matrix is a diagonal matrix with elements $(r_1, r_2 - \delta, -\mu)$ which are the eigenvalues. Because $r_1 > 0, r_2 - \delta$, and $\mu > 0$, equilibrium E_0 is always a saddle point for the three dimensional SIP-system.

E_S : The infected prey and predator free equilibrium $E_S = (K, 0, 0)$. The eigenvalues are $-r_1 < 0, r_2 + (\lambda - (r_2 a_{22} / K))K - \delta$, and $(\alpha_1 \beta_1 K / (1 + T_h K)) - \mu$. Therefore E_S is locally asymptotically stable (LAS) if $r_2 + (\lambda - (r_2 a_{22} / K))K < \delta$ and $\alpha_1 \beta_1 K / (1 + T_h K) < \mu$. E_{SI} : Predator free equilibrium values $E_{SI}(S_{SI}^*, I_{SI}^*, 0)$ were already derived above as solutions for the two-dimensional system. Numerical bifurcation analysis shows that for the parameter values given in Table 1 that this boundary equilibrium of the three dimensional system is always unstable and that the predator is able to invade leading to an interior E_{SIP} equilibrium.

The analysis results for the remaining equilibria E_{SP} and E_{SIP} are difficult to interpret (the equilibrium values are solutions of quadratic equations) and therefore not reported here. However, we recall that explicit expressions for the equilibrium values as well as the eigenvalues of the Jacobian matrix evaluated in that equilibrium, can be obtained with the program Maple (2008).

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