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Original Research Article

Complex dynamics of a predator–prey–parasite system: An interplay among infection rate, predator's reproductive gain and preference

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ABSTRACT

Parasites are considered as an important factor in regulating their host populations through trait-mediated effects. On the other hand, predation becomes particularly interesting in host–parasite systems because predation can significantly alter the abundance of parasites and their host population. The combined effects of parasites and predator on host population and community structure therefore may have larger effect. Different field experiments confirm that predators consume disproportionately large number of infected prey in comparison to their susceptible counterpart. There are also substantial evidences that predator has the ability to distinguish prey that have been infected by a parasite and avoid such prey to reduce fitness cost. In this paper we study the predator–prey dynamics, where the prey species is infected by some parasites and predators consume both the susceptible and infected prey with some preference. We demonstrate that complexity in such systems largely depends on the predator's selectivity, force of infection and predator's reproductive gain. If the force of infection and predator's reproductive gain are low, parasites and predators both go to extinction whatever be the predator's preference. The story may be totally different in the opposite case. Survival of species in stable, oscillatory or chaotic states, and their extinction largely depend on the predator's preference. The system may also show two coexistence equilibrium points for some parameter values. The equilibrium with lower susceptible prey density is always stable and the equilibrium with higher susceptible prey density is always unstable. These results suggest that understanding the consequences of predator's selectivity or preference may be crucial for community structure involving parasites.

1. Introduction

Study of predator–prey–parasite (PPP) systems starts with the pioneering work of Anderson and May in 1986 (Anderson and May, 1986). Since then literature in the field of predator–prey–parasite system has grown enormously in the last two and half decades and Chattopadhyay and Bairagi (2001), Xiao and Chen (2001), Venturino (2002), Packer et al. (2003), Hethcote et al. (2004), Hall et al. (2005), Fenton and Rands (2006), Bairagi et al. (2007, 2009), Hsieh and Hsiao (2008), Siekmann et al. (2010), Greenman and Hoyle (2010), Sieber and Hilker (2011) are just to name a few of such recent works. It is now well recognized that PPP models show more complicated and interesting dynamics than the traditional ecological or epidemiological models (Minchella and Scott, 1991). Parasites could be an important factor in regulating not only their host populations but also similarly important for the community structure (Cunningham and Daszak, 1998). It can greatly alter the quantitative dynamics of the community and can lead to high amplitude oscillations in abundance (Fenton and Rands, 2006) or extinction (Freeland, 1983; Richards et al., 1999; Muths et al., 2003). Therefore, the importance of parasites in ecology is being increasingly recognized to understand the role of parasites in biodiversity and ecosystem dynamics (Hudson et al., 1992a,b; McCallum et al., 2003, 2005).

Parasites also affect the predator–prey interaction significantly (Thomas et al., 1997) through trait-mediated effects (Hatcher et al., 2006; Raffel et al., 2010). There are many examples where prey becomes more vulnerable to predation due to parasitic infection (Lafferty and Morris, 1996; McCallum et al., 2005). Hudson et al. (1992a,b) performed series of field experiments on red grouse and fox populations, respectively prey and predators, in Scotland and north England. They observed that predators tend to capture a disproportionately high number of grouse that are infected by some parasites (nematode *Trichostrongylus*). Lafferty and Morris (1996)

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performed field experiments in many Southern California salt marshes. They observed some parasites (trematode *Euhaplorchis californiensis*) infect the brain of killifish (*Fundulus parvipinnis*). Parasitized fish frequently swam to surface and are eaten by piscivorous birds strikingly more than non-parasitized fish. They estimated that parasitized fish were, on average, 31 times more susceptible to predation than non-parasitized fish. In Salton Sea, USA, fishes are infected by a vibrio class of bacteria and causes fish mortality event frequently (Horvitz, in press; Kaiser, 1999). This bacteria is thought to weaken the fish and create anoxic conditions in their body. Since upper surface contains more oxygen, infected fish comes closer to water surface and becomes an easy catch for fish-eating birds like pelican. Vibrio is passed from one infected fish to another susceptible fish. Fish infected with vibrio dies within a few week and unable to reproduce (Horvitz, in press; Chattopadhyay and Bairagi, 2001). On the other hand, some studies show that predator can deliberately avoid the infected prey to reduce fitness cost (Sloan and Simmons, 1973; Schlichter, 1978; Jones et al., 2005; Meyling and Pell, 2006). In captive feeding trials, Jones et al. (2005) observed that birds prefer to forage upon non-parasitized armyworm (*Spodoptera frugiperda*) prey when birds were offered prey parasitized by the visible ectoparasitoid (*Euplectrus plathypenae*) larvae. Sloan and Simmons (1973) reported that parasitized jack pine budworm (*Choristoneura pinus*) larvae and pupae were unanimously rejected by foraging chipping sparrows (*Spizella passerina Bechstein*). In an another observation, Schlichter (1978) reported that black-capped chickadees (*Parus atricapillus Linnaeus*) largely avoided foraging upon galls on Canada goldenrod (*Solidago canadensis*) that had been parasitized by mordellid beetle (*Mordellistena unicolor*) larvae to extract gall fly (*Eurosta solidaginis Fitch*) larvae. Meyling and Pell (2006) observed that the generalist predator *Anthocoris nemorum* can detect and avoid the fungal pathogen *Beauveria bassiana* when it forages on host plants. Therefore, it may be interesting to observe the predator-prey dynamics in presence of parasitic infection and predator's preference or selectivity.

In the last three decades a good number of theoretical works have been published on multi-trophic ecological systems that exhibit chaotic behavior (Gragnani et al., 1998; Hastings and Powell, 1999; Vayenas and Pavlou, 1999; Cushing et al., 2003; Vandermeer, 2006; Stone and He, 2007). However, there is still a lack of evidence that chaos exists in multi-species systems in real world scenario. There are few experimental demonstrations in laboratory setting which support existence of chaos in multi-species food web (Jost et al., 1973; Costantino et al., 1997; Dennis et al., 1997; Fussmann et al., 2000; Becks et al., 2005). Some theoretical works in predator–prey–parasite systems also show the existence of complex dynamics like chaos (Lenbury et al., 1999; Hilker and Malchow, 2006; Upadhyay et al., 2008; Stiefs et al., 2009; Kooi et al., 2011). Extensive numerical simulations of the model of Chattopadhyay and Bairagi (2001) show that the system exhibits chaotic dynamics when some key parameters attain their critical values. It was commented that chaotic behavior may not be observed in natural system because it occurs intermittently with stable limit cycles in narrow parameter regimes (Upadhyay et al., 2008). Hilker and Malchow (2006) studied the interaction of phytoplankton and zooplankton where the first species is infected by some disease. They observed some strange attractor when the virulence parameter was varied. Stiefs et al. (2009) demonstrated that to obtain complex dynamics like quasi-periodicity and chaos a SIRS type model is essential instead of a SIS type when predator is infected by parasite. Kooi et al. (2011) have shown that an infectious disease in the predator population can lead to complex dynamics, such as chaos, in a PPP system. Though this complexity was shown to occur under biologically very feeble assumptions like infected predator does not feed. Lenbury et al. (1999) studied a three-dimensional PPP model where predator population is invaded by some parasite. Using singular perturbation argument, they showed that the system exhibits limit cycle behavior as well as other stable dynamical characteristics. Simulation results of their extended four-dimensional system, where both prey and predator are infected by parasite, exhibit chaotic dynamics with respect to the parameter γ, which measures the maximum susceptible prey attack rate by the infected predator. None of these models however considers predator's preference to infected prey over the susceptible one or vice-versa. In this paper, we study a PPP model where prey is infected by some microparasite and predators consume both the susceptible and infected preys with some preference. Our study reveals that complexity in the dynamics of a predator–prey–parasite model largely depends on the predator's preference or selectivity.

We organize the rest of this paper in the following manner. Section 2 deals with the development of the model. Section 3 provides some preliminary results concerning the boundedness of the solutions as well as the existence of equilibria of the model system with their stability properties. The paper ends with a discussion in Section 4.

2. Model formulation

Recent studies reveal that outcomes of trophic interactions and community processes are greatly determined by the parasites and pathogens (MacNeil et al., 2003; Malmstrom et al., 2006; Collinge and Ray, 2006; Wilmers et al., 2006). Since any realistic community system is composed of many subsystems (Bascompte and Melian, 2005), it has always been suggested to decompose the bigger system into smaller subsystems to understand interactions within each subsystem and then to combine the smaller subsystems to better understand the interactions among the subsystems and its consequences on the community system (Hatcher et al., 2006). Keeping this in mind, we consider a predator–prey subsystem of a community where a prey population (X) grows logistically to the environmental carrying capacity K in absence of predator with intrinsic growth rate r, and a predator population (P) feeds upon this prey population with response function $f(.)$. If predator follows type II response function then $f(X) = nX/(a + X)$, where $f(0) = 0$, $f'(X) > 0$, n is the predator's per capita prey capture rate when prey density is very high and a is the half-saturation constant. Thus, we have a predator–prey model which is represented by the following coupled equations:

$$\begin{aligned} \dot{X} &= rX\left(1-\frac{X}{K}\right) - f(X)P, \\ \dot{P} &= b_1 f(X)P - dP. \end{aligned} \tag{1}$$

Parameters b_1 and d are positive and represent, respectively, the biomass conversion factor and food-independent death rate of predator.

Suppose our prey population is infected by some microparasites. The prey population will then be divided into two subpopulations, viz., susceptible prey (S) and infected prey (I), where the total population is given by $X = S + I$. Assuming that the disease is of SIS type and infection spreads following mass action law then the interaction between two subpopulations of this epidemic model can be represented by

$$\begin{aligned} \dot{S} &= rS\left(1-\frac{S+I}{K}\right) - \lambda SI + \gamma I, \\ \dot{I} &= \lambda SI - cI - \gamma I, \end{aligned} \tag{2}$$

where λ is the force of infection and γ is the recovery rate. Parameter $c(= c_1 + \mu)$ represents the death rate of infected prey by natural mortality (c_1) and virulence (μ) of the disease. It is