

## A predator–prey model with infected prey

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### Abstract

A predator–prey model with logistic growth in the prey is modified to include an SIS parasitic infection in the prey with infected prey being more vulnerable to predation. Thresholds are identified which determine when the predator population survives and when the disease remains endemic. For some parameter values the greater vulnerability of the infected prey allows the predator population to persist, when it would otherwise become extinct. Also the predation on the more vulnerable prey can cause the disease to die out, when it would remain endemic without the predators.

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### 1. Introduction

Infectious diseases can be a factor in regulating human and animal population sizes. For example, the Black Death in Europe in the 14th century killed up to one-fourth of the human population. European diseases such as smallpox brought by Cortez and others to Mexico decimated the native population there in the 16th century. Rinderpest caused high mortality in wild animals in Africa at the end of the 19th century. Myxomatosis caused enormous decreases in the rabbit population in Australia in the 1950s (McNeill, 1976).

In complex ecosystems predator–prey relationships can also be important in regulating the numbers of prey and predators. For example, when a bounty was placed on

natural predators such as cougars, wolves, and coyotes in the Kaibab Plateau in Arizona, the deer population increased beyond the food supply, and then over half of the deer died of starvation in 1923–25 (Curtis, 1972). However, predator control does not always cause the prey population to increase. Sih et al. (1985) found that predator removal decreased the prey population in 54 of the 135 systems examined. Packer et al. (2003) suggested that predator removal can lead to a decreased prey population size, if the prey also has an infectious disease.

Numerous studies have shown that predators take a disproportionate number of prey infected by parasites (Van Dobben, 1952; Vaughn and Coble, 1975; Temple, 1987). The review of Holmes and Bethel (1972) contains many examples in which the parasite changes the external features or behavior of the prey, so that infected prey are more vulnerable to predation. Infected prey may live in locations that are more accessible to predators; for example, fish or aquatic snails may live close to the water surface or snails may live on top of vegetation rather than under protective plant cover. Similarly, infected prey may be weaker or less active, so that they

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are caught more easily (Dobson, 1988; Moore, 2002). A review of studies showed that predators tend to forage for easily available prey (Krebs, 1978). For example, wolf attacks on moose on Isle Royale in Lake Superior are more successful if the moose are heavily infected with a lungworm (Peterson and Page, 1988). Predators such as wolves and lions save energy by selecting prey that may be weakened by a disease (Mech, 1970; Schaller, 1972).

Here we investigate the epidemiological and demographic effects in a predator–prey model in which the infected prey are more vulnerable to predation. The predator–prey model has logistic growth of the prey, so that the model without the infectious disease has an asymptotically stable positive equilibrium. It is assumed that the microparasitic infection does not induce immunity, so the epidemiology model is of SIS type. Threshold criteria are developed which determine when the predator population persists and when the disease remains endemic in the prey population. Of the six possible cases, case 3 is significant because the greater vulnerability of the infected prey allows the predator population to persist, when it would have become extinct without the disease. Case 5 is also significant because predation on the more vulnerable infected prey causes the disease to die out, when it would have remained endemic in the prey in the absence of predation. The importance of the analysis of the model here is not only the recognition that the unusual cases 3 and 5 can occur, but also the identification of the inequalities that must be satisfied by the parameter values in order for these cases to occur.

Previous modeling studies of infectious diseases in natural animal populations have considered the effects of disease-induced mortality or disease-reduced reproduction in regulating natural populations, decreasing their population sizes, reducing their natural fluctuations, or causing destabilizations of equilibria into oscillations of the population states (Anderson and May, 1978; May and Anderson, 1978; Gao and Hethcote, 1992; Mena-Lorca and Hethcote, 1992). The volume produced by the 1981 Dahlem Conference on the population biology of infectious diseases identified the major issues of infectious diseases in naturally fluctuating host populations (Anderson and May, 1982). A 1993 Conference at the Isaac Newton Institute in Cambridge, England resulted in a collection of papers focused on models for infectious diseases in natural animal populations (Grenfell and Dobson, 1995). The survey article by Begon and Bowers (1995) summarized previous work on models for host–pathogen dynamics.

In a prey–predator model with greater predation on infected prey and no reproduction in infected prey, Anderson and May (1986) found that the pathogen tends to destabilize the prey–predator interactions. In an SI modification of a Rosenzweig prey–predator model in

which predation is more likely on infected prey than on uninfected prey, predators get disease only from eating infected prey, and prey obtain the disease from parasites spread into the environment by predators, Haderler and Freedman (1989) found a threshold above which an infected equilibrium or an infected periodic solution appear. They also considered a second situation in which the predators could only survive on the prey if some of the prey were more easily caught due to being diseased. Venturino (1994) studied modifications of the classic Lotka–Volterra prey–predator model in which an SI or SIS disease spreads among either the prey or the predators. Venturino (1995) considered similar SI and SIS models with disease spread among the prey when there is logistic growth of the prey and the predators. In an SI model in which susceptible prey grow logistically and predators eat only infected prey, Chattopadhyay and Arino (1999) found persistence and extinction conditions for the population and also determined conditions for Hopf bifurcation to periodic solutions. Han et al. (2001) analyzed four predator–prey models for SIS and SIR diseases with the standard and mass action incidences. Venturino (2002) examined epidemic models in predator–prey models with disease in the predators. Hochberg (1989) considered models in which predators have more than one prey species, but the disease is specific to one prey species.

Diseases in animals caused by macroparasites are often more complicated, since the severity of the disease usually depends on the parasitic load in the animal. Hudson et al. (1992) formulated models for macroparasitic infections in red grouse and looked at situations in which parasitic infections of the prey made them more vulnerable to predation. Packer et al. (2003) examined the effects on prey population sizes of predator removal in models of microparasitic and macroparasitic infections with a constant predator population size.

The model considered here differs from previous models; it uses the standard incidence, it is based on a predator–prey model with an attractive equilibrium, and it focuses on the effects of the greater vulnerability to predation of the infected prey. Section 2 describes the basic predator–prey and SIS models. Section 3 contains the predator–prey SIS model with vulnerable infected prey, identifies the boundary equilibria and their stability thresholds, and presents conjectures about the behavior in the six possible cases. Proofs of many of the conjectures about the six cases are given in Section 4. Section 5 contains results on persistence of the predators and endemicity of the disease. The results are discussed in Section 6.

## 2. The predator–prey model and the SIS model

Let  $H(t)$  and  $P(t)$  be the sizes of prey population and predator population, respectively, where  $t$  is time. The

model that we use takes the form

$$\begin{aligned} \dot{H} &= r(1 - H/K)H - aHP = [r(1 - H/K) - aP]H, \\ \dot{P} &= kaHP - cP = (kaH - c)P, \end{aligned} \tag{1}$$

which is the modification of the classic Lotka–Volterra equations with density-dependent logistic growth of the prey. The initial per capita growth rate is  $r$  and the prey carrying capacity of the environment is  $K$ . The per capita death rate of predators is  $c$ . The predation rate is  $aHP$  and the feeding efficiency in turning predation into new predators is  $k$ , so that the birth rate of new predators is  $kaHP$ . This model has the desirable property that when the positive equilibrium  $(H_E, P_E)$  is feasible (i.e. the predator coordinate  $P_E$  is positive), it is globally asymptotically stable. Note that  $1/c$  is the average lifetime of predators, so that  $kaH/c$  is the average amount of prey  $H$  converted to predator biomass in a course of the predator’s life span. Thus the predator becomes extinct if it fails in its lifetime to replace itself even at maximum food level  $K$  ( $kaK/c \leq 1$ ). This model was formulated and explained by Pielou (1969). The lemma below follows from the Kolmogorov–Brauer theorem (Nisbet and Gurney, 1982).

**Lemma 1.** *For solution paths of system (1) in  $R_+^2$ , if  $H(0) > 0$  and  $kaK/c \leq 1$  or  $H(0) > 0$  and  $P(0) = 0$ , then  $H \rightarrow K$  and  $P \rightarrow 0$ , as  $t \rightarrow \infty$ . If  $H(0) > 0$ ,  $P(0) > 0$  and  $kaK/c > 1$ , then  $H \rightarrow H_E = c/ka$  and  $P \rightarrow P_E = r[1 - c/(kaK)]/a$ , as  $t \rightarrow \infty$ .*

Animals in the prey population are either susceptible (when they can become infected), or are infectious (when they are infected and can transmit the infection). Here we assume that the infection is either lifelong or that animals do not have immunity when they recover. These two cases correspond to an SI or an SIS disease. The total size of the prey population is  $H = X + Y$ , where  $X$  is the number of susceptible prey and  $Y$  is the number of infectious prey. Then  $S = X/H$  is the fraction of the prey that are susceptible and  $I = Y/H$  is the fraction that are infectious.

The incidence is the number of new cases per unit time due to the infection of susceptible prey through their contacts with infectious prey. If  $\beta$  is the average number of contacts (sufficient for transmission) of an animal per unit time, then  $\beta X/H = \beta S$  is the average number of contacts with susceptible animals per unit time of one infectious animal. Since there are  $Y = IH$  infectives, the number of new cases per unit time in the  $X$  susceptibles is equal to the standard incidence  $(\beta X/H)Y = \beta ISH$ . This standard incidence is the preferred formulation for animal populations (Hethcote, 1976; Anderson and May, 1982, p. 157; Hethcote and Van Ark, 1987; Gao and Hethcote, 1992; Mena-Lorca and Hethcote, 1992; De Jong et al., 1995; Hethcote, 2000). The movement out of the infectious class is given by  $\gamma Y$  in our ordinary

differential equation model. This corresponds to an exponentially distributed waiting time in the  $I$  class with  $e^{-\gamma t}$  as the fraction that is still in the infective class  $t$  units after entering this class and  $1/\gamma$  as the mean waiting time (Hethcote et al., 1981). Note that the SI model is a special case of the SIS model with  $\gamma = 0$ .

Bacterial infections tend to be of SIS type, while viral infections correspond to SIR diseases. The SIS and SIR models with logistic growth of the population and disease-related deaths were studied in Gao and Hethcote (1992). When there are no disease-related deaths, the SIS model in Section 7 of that paper is:

$$\begin{aligned} \dot{H} &= r(1 - H/K)H, \\ \dot{X} &= (b - \theta rH/K)H - [d + (1 - \theta)rH/K]X \\ &\quad - \beta XY/H + \gamma Y, \end{aligned} \tag{2}$$

$$\dot{Y} = \beta XY/H - \gamma Y - [d + (1 - \theta)rH/K]Y.$$

In the absence of disease, the prey population satisfies the logistic differential equation with birth rate coefficient  $b - \theta rH/K$  and death rate coefficient  $d + (1 - \theta)rH/K$ , where  $r = b - d > 0$  and  $0 \leq \theta \leq 1$ . The restricted growth in the logistic equation is due to a density-dependent death rate when  $\theta = 0$ , is due to a density-dependent birth rate when  $\theta = 1$ , and is due to a combination of these when  $0 < \theta < 1$ . Using  $H = X + Y$ ,  $I = Y/H$ , and  $S = 1 - I$ , the three differential equations above can be reduced to the following two-dimensional system:

$$\begin{aligned} \dot{H} &= [r(1 - H/K)]H, \\ \dot{I} &= [\beta(1 - I) - (\gamma + b - \theta rH/K)]I. \end{aligned} \tag{3}$$

This model is mathematically well posed and all solutions start or enter the positively invariant region  $D = \{(H, I) | 0 \leq I \leq 1, 0 \leq H \leq K\}$ . Because the model here does not include disease-related deaths, the two epidemiological threshold quantities are the basic reproduction number  $R_0$  and the modified basic reproduction number  $R_0^m$ . These are given by

$$R_0 = \frac{\beta}{\gamma + b - \theta r} \geq R_0^m = \frac{\beta}{\gamma + b}.$$

This system has the (extinction, disease-free) equilibrium  $E_0 = (0, 0)$  and the (carrying capacity, disease-free) equilibrium  $E_1 = (K, 0)$ . When  $R_0^m > 1$ , there is an (extinction, endemic) equilibrium  $E_3 = (0, 1 - 1/R_0^m)$ . This unusual equilibrium  $E_3$  with zero population size is a saddle with a repulsive direction into  $D$  for model (3), so it is biologically irrelevant here, but for the more general model in Gao and Hethcote (1992), disease-related deaths and persistence of the disease can drive the population to this extinction equilibrium. When  $R_0 > 1$ , there is an (carrying capacity, endemic) equilibrium  $E_4 = (K, 1 - 1/R_0)$ . All four equilibria are on the boundary of  $D$ . This model has the expected behavior

that the host population size approaches the carrying capacity  $K$ , the disease dies out if  $R_0 \leq 1$ , and the disease remains endemic if  $R_0 > 1$ . The lemma below on the global asymptotic behavior is a special case with no disease-related deaths of the result proved in Gao and Hethcote (1992).

**Lemma 2.** For solution paths of system (3) in  $D$ ,

- (1) paths with  $I(0) > 0$  and  $H(0) = 0$  go to  $E_0 = (0, 0)$  if  $R_0^m \leq 1$ ; and go to  $E_3 = (1 - 1/R_0^m, 0)$  if  $R_0^m > 1$ ;
- (2) paths with  $I(0) = 0$  and  $H(0) > 0$  go to  $E_1 = (K, 0)$ ;
- (3)  $R_0 \leq 1 \Rightarrow$  paths with  $I(0) > 0$  and  $H(0) > 0$  go to  $E_1 = (K, 0)$ ;
- (4)  $R_0 > 1 \Rightarrow$  paths with  $I(0) > 0$  and  $H(0) > 0$  go to  $E_4 = (K, 1 - 1/R_0)$ .

The following results are used later in this paper. Consider the systems:

$$\dot{x} = f(t, x), \tag{4}$$

$$\dot{y} = g(y), \tag{5}$$

where  $f$  and  $g$  are continuous and locally Lipschitz in  $x$  in  $R^n$  and solutions exist for all positive time. Eq. (4) is called asymptotically autonomous with limit equation (5) if  $f(t, x) \rightarrow g(x)$  as  $t \rightarrow \infty$  uniformly for  $x$  in  $R^n$ .

**Lemma 3** (Thieme, 1992). Let  $e$  be a locally asymptotically stable equilibrium of (5) and  $\omega$  be the  $\omega$ -limit set of a forward bounded solution  $x(t)$  of (4). If  $\omega$  contains a point  $y_0$  such that the solution of (5), with  $y(0) = y_0$  converges to  $e$  as  $t \rightarrow \infty$ , then  $\omega = \{e\}$ , i.e.  $x(t) \rightarrow e$ , as  $t \rightarrow \infty$ .

**Corollary 4.** If solutions of system (4) are bounded and the equilibrium  $e$  of the limit system (5) is globally asymptotically stable, then any solution  $x(t)$  of system (4) satisfies  $x(t) \rightarrow e$  as  $t \rightarrow \infty$ .

### 3. The predator–prey SIS model with vulnerable infected prey

We combine the predator–prey and SIS models in the previous section to form a predator–prey model with an SIS disease in the prey population. It is assumed that infected prey are more vulnerable to predation by a factor  $q \geq 1$ . The combined model is

$$\begin{aligned} \dot{H} &= r(1 - H/K)H - a(X + qY)P, \\ \dot{X} &= (b - \theta rH/K)H - [d + (1 - \theta)rH/K]X \\ &\quad - \beta XY/H + \gamma Y - aXP, \end{aligned} \tag{6}$$

$$\dot{Y} = \beta XY/H - \gamma Y - [d + (1 - \theta)rH/K]Y - aqYP,$$

$$\dot{P} = ka(X + qY)P - cP.$$

Using  $H = X + Y$ ,  $I = Y/H$  and  $X/H = 1 - I$ , system (6) of four differential equations can be reduced to the

following three-dimensional system:

$$\begin{aligned} \dot{H} &= [r(1 - H/K) - a(1 + (q - 1)I)P]H, \\ \dot{I} &= [\beta(1 - I) - (\gamma + b - \theta rH/K) \\ &\quad - a(q - 1)(1 - I)P]I, \end{aligned} \tag{7}$$

$$\dot{P} = [kaH(1 + (q - 1)I) - c]P.$$

Model (7) can be shown to be mathematically well posed in the positively invariant region

$$G = \{(H, I, P) | 0 \leq H \leq K, 0 \leq I \leq 1, 0 \leq P\},$$

and solutions in  $G$  exist for all positive time. Solutions with  $H(0) > K$  approach or enter the region  $G$ , so it is sufficient to consider solutions in  $G$ .

#### 3.1. Boundary equilibria and their local stability

We start the mathematical analysis by looking at the equilibria on the boundary of region  $G$  and at their local stability. System (7) has up to five equilibria on the boundary of  $G$ . The first two are  $E_0 = (0, 0, 0)$  and  $E_1 = (K, 0, 0)$ . When  $kaK/c > 1$ , there is an equilibrium  $E_2 = (H_E, 0, P_E)$ , where  $H_E$  and  $P_E$  are given in Lemma 1. When  $R_0 > 1$ , there is an equilibrium  $E_4 = (K, 1 - 1/R_0, 0)$ . Note that paths in  $G$  on the  $H$  axis with  $H(0) > 0$  go to the equilibrium  $E_1 = (K, 0, 0)$ , since without any predators, the prey goes to its carrying capacity. Paths in  $G$  on the  $P$  axis go to the equilibrium  $E_0 = (0, 0, 0)$ , since without any prey, the predator population goes to extinction. When  $R_0^m > 1$ , there is an equilibrium  $E_3 = (0, 1 - 1/R_0^m, 0)$ . Paths in  $G$  on the  $I$  axis go to the equilibrium  $E_0 = (0, 0, 0)$  when  $R_0^m \leq 1$  and go to the equilibrium  $E_3 = (0, 1 - 1/R_0^m, 0)$  when  $R_0^m > 1$ .

Lemmas 1 and 2 give the behavior of paths in the  $I = 0$  and  $P = 0$  faces of region  $G$ . In the  $H = 0$  face of  $G$ ,  $P \rightarrow 0$ , so that paths in this face with  $I(0) > 0$  go to  $E_0 = (0, 0, 0)$  when  $R_0^m \leq 1$  and go to the equilibrium  $E_3 = (0, 1 - 1/R_0^m, 0)$  when  $R_0^m > 1$ . Paths (except the equilibrium  $E_1 = (K, 0, 0)$ ) starting on the  $H = K$  face of  $G$  enter the interior of  $G$ . All paths starting in the  $I = 1$  face of  $G$  enter the interior of  $G$ .

Next, we consider the local stability of each of the boundary equilibria. The Jacobian of system (7) is

$$J = \begin{bmatrix} j_{11} & -a(q - 1)HP & -a[1 + (q - 1)I]H \\ \theta rI/K & j_{22} & -a(q - 1)(1 - I)I \\ ka(1 + (q - 1)I)P & kaH(q - 1)P & kaH(1 + (q - 1)I) - c \end{bmatrix},$$

where  $j_{11} = r(1 - 2H/K) - a(1 + (q - 1)I)P$ , and  $j_{22} = [\beta - a(q - 1)P](1 - 2I) - (\gamma + b - \theta rH/K)$ .

At the equilibrium  $E_0 = (0, 0, 0)$ , the Jacobian is repulsive in the  $H$  direction with eigenvalue  $r > 0$ , since the prey goes to its carrying capacity, and is attractive in the  $P$  direction with eigenvalue  $-c$ , since the predators

go to extinction. In the  $I$  direction the eigenvalue is  $\beta - (\gamma + b)$ , so that  $E_0$  is attractive in the  $I$  direction if  $\mathbf{R}_0^m < 1$  and is repulsive if  $\mathbf{R}_0^m > 1$ .

When  $\mathbf{R}_0^m > 1$ , the equilibrium  $E_3 = (0, 1 - 1/\mathbf{R}_0^m, 0)$  is in  $G$  and the eigenvalue at  $E_3$  in the  $I$  direction is  $\gamma + b - \beta$  so that paths on the  $I$  axis go to  $E_3$ . The eigenvalue at  $E_3$  in the  $P$  direction is  $-c$ , so it is always attractive in this direction, but the eigenvalue in the  $H$  direction is  $r$ , so it is repulsive in this direction. Thus the existence of equilibrium  $E_3$  is biologically irrelevant, since solution paths with some initial prey never approach the equilibrium  $E_3$ .

At the equilibrium  $E_1 = (K, 0, 0)$ , the eigenvalues in the  $H$ ,  $I$ , and  $P$  directions are  $-r$ ,  $\beta - (\gamma + b - \theta r)$ , and  $kaK - c$ , respectively. So  $E_1$  is attractive in the  $H$  direction, is attractive in the  $I$  direction when  $\mathbf{R}_0 < 1$ , is repulsive in the  $I$  direction when  $\mathbf{R}_0 > 1$ , is attractive in the  $P$  direction when  $kaK/c < 1$ , and is repulsive in the  $P$  direction when  $kaK/c > 1$ . Thus the local stability behaviors at the three boundary equilibria  $E_0$ ,  $E_3$ , and  $E_1$  are consistent with the results in Lemmas 1 and 2.

By Lemma 1 we know that the equilibrium  $E_2 = (H_E, 0, P_E)$  exists in  $G$  and is attractive in the  $I = 0$  face of  $G$  when  $kaK/c > 1$ . The eigenvalue in the  $I$  direction is  $\beta - (\gamma + b - \theta rc/kaK) - a(q - 1)P_E$ , so that it is attractive if  $\mathbf{R}_1 < 1$  and is repulsive if  $\mathbf{R}_1 > 1$ , where

$$\mathbf{R}_1 = \frac{\beta}{(\gamma + b - \theta rc/kaK) + (q - 1)r(1 - c/kaK)}. \quad (8)$$

Note that  $\mathbf{R}_0 > \mathbf{R}_1$ .

By Lemma 2, we know that when  $\mathbf{R}_0 > 1$ , the equilibrium  $E_4 = (K, 1 - 1/\mathbf{R}_0, 0)$  exists in  $G$  and is attractive in the  $P = 0$  face of  $G$ . The eigenvalue in the  $P$  direction is  $kaK[1 + (q - 1)(1 - 1/\mathbf{R}_0)] - c$ , so that  $E_4$  is attractive in the  $P$  direction if  $\psi < 0$  and is repulsive if  $\psi > 0$  where

$$\psi = (q - 1)(1 - 1/\mathbf{R}_0) - (c/kaK - 1). \quad (9)$$

Note that the first factor is nonnegative and the second factor is positive when  $kaK/c > 1$ .

### 3.2. The six cases

The six possible cases for the behavior of solution paths starting in the interior of  $G$  depend on whether or not the boundary equilibria  $E_2$  and  $E_4$  are in the region  $G$ , and whether they are locally repulsive into the interior of  $G$  or are locally attractive for paths starting in the interior of  $G$ . First we consider the three cases with  $kaK/c \leq 1$ , so that without the disease, the feeding efficiency  $k$  of the predators is low enough that the predators become extinct. In these three cases the equilibrium point  $E_2 = (H_E, 0, P_E)$  with both predators and prey (but no disease) is not in region  $G$ .

*Case 1:* If  $\mathbf{R}_0 \leq 1$ , so that  $G$  has no equilibria  $E_2$ ,  $E_3$  or  $E_4$ , then all paths starting in the interior of  $G$  go to

$E_1 = (K, 0, 0)$ . Since the predators become extinct, we have the usual result that the prey goes to its carrying capacity  $K$ , and the disease in the prey dies out since  $\mathbf{R}_0 \leq 1$ .

*Case 2:* Suppose  $\mathbf{R}_0 > 1$ , so that  $G$  has a boundary equilibrium  $E_4 = (K, 1 - 1/\mathbf{R}_0, 0)$ , corresponding to endemicity of the disease. Here  $\psi < 0$ , so that  $E_4$  is attractive in the  $P$  direction, and all paths starting in the interior of  $G$  go to  $E_4 = (K, 1 - 1/\mathbf{R}_0, 0)$ . In this case the predators become extinct, the prey population goes to its carrying capacity  $K$ , and the disease remains endemic in the prey since  $\mathbf{R}_0 > 1$ .

*Case 3:* Suppose  $\mathbf{R}_0 > 1$ , so that  $G$  has a boundary equilibrium  $E_4 = (K, 1 - 1/\mathbf{R}_0, 0)$ . Here  $\psi > 0$ , so that  $E_4$  is repulsive in the  $P$  direction, which implies that no interior path can go to a boundary equilibrium. In this case an interior equilibrium exists, so we conjecture that all paths starting in the interior of  $G$  go to an interior equilibrium point. This interesting case illustrates how the greater vulnerability of the infected prey allows the predator population to persist, when it would have become extinct without the disease.

Next we consider the 3 cases with  $kaK/c > 1$ , so that the feeding efficiency  $k$  of the predators is high enough that without the disease, the prey and predators both persist. This means that the equilibrium  $E_2 = (H_E, 0, P_E)$  with both predators and prey (but no disease) is a boundary equilibrium in region  $G$ .

*Case 4:* Suppose  $\mathbf{R}_1 < \mathbf{R}_0 \leq 1$ , so that  $E_2 = (H_E, 0, P_E)$  is attractive in the  $I$  direction and the equilibrium  $E_4 = (K, 1 - 1/\mathbf{R}_0, 0)$  is not in  $G$ . Then all paths starting in the interior of  $G$  go to  $E_2 = (H_E, 0, P_E)$ . In this case the disease dies out since  $\mathbf{R}_0 \leq 1$ , and the prey and predators both persist.

*Case 5:* Suppose  $\mathbf{R}_1 \leq 1 < \mathbf{R}_0$ , so that  $E_2 = (H_E, 0, P_E)$  is attractive in the  $I$  direction. Here  $E_4 = (K, 1 - 1/\mathbf{R}_0, 0)$  is a boundary equilibrium in  $G$ , and it is repulsive in the  $P$  direction since  $\psi > 0$ . Because equilibrium  $E_2$  is attractive and there are no interior equilibria, we conjecture that all paths starting in the interior of  $G$  go to  $E_2 = (H_E, 0, P_E)$ . This interesting case illustrates how predation of the more vulnerable infected prey causes the disease to die out, when it would have remained endemic in the prey in the absence of predation.

*Case 6:* Suppose  $1 < \mathbf{R}_1 < \mathbf{R}_0$ , so that  $E_2 = (H_E, 0, P_E)$  is repulsive in the  $I$  direction.  $E_4 = (K, 1 - 1/\mathbf{R}_0, 0)$  is a boundary equilibrium in  $G$ , and it is repulsive in the  $P$  direction since  $\psi > 0$ . Thus no interior path can go to a boundary equilibrium. In this case an interior equilibrium exists, so we conjecture that all paths starting in the interior of  $G$  go to an interior equilibrium. This case illustrates the situation in which the disease remains endemic even though the infected prey are more vulnerable to predation.

**4. Proofs**

*4.1. Proof of cases 1 and 4 with  $R_0 \leq 1$*

The differential equation for  $I$  in (7) satisfies

$$\begin{aligned} \dot{I} &= [\beta(1 - I) - (\gamma + b - \theta r) - \theta r(1 - H/K) \\ &\quad - a(q - 1)(1 - I)P]I \\ &\leq (\gamma + b - \theta r)[R_0(1 - I) - 1]I \\ &< 0 \text{ unless } I = 0. \end{aligned}$$

Thus  $I(t) \rightarrow 0$  as  $t \rightarrow \infty$ . Let system (7) be the system  $\dot{x} = f(t, x)$  in Corollary 4 in which the variable  $I(t)$  is considered to be a function of  $t$ . Then the limiting system as  $t \rightarrow \infty$  is system (1) with the added equation  $\dot{I} = 0$ . By Corollary 4 the solutions of the original system (7) approach the asymptotically stable equilibrium point of system (1), so that all solutions  $(H, I, P)$  go to  $E_1 = (K, 0, 0)$  if  $kaK/c \leq 1$ , and go to  $E_2 = (H_E, 0, P_E)$  if  $kaK/c > 1$ . This proves cases 1 and 4.

*4.2. Proof of case 2 with  $R_0 > 1$  and  $\psi < 0$*

The solutions  $H$  and  $I$  in system (7) satisfy the differential inequalities

$$\begin{aligned} \dot{H} &\leq r(1 - H/K)H, \\ \dot{I} &\leq [\beta(1 - I) - (\gamma + b) + \theta rH/K]I. \end{aligned}$$

These inequalities are similar to the SIS model equations (3) which have the globally asymptotically stable equilibrium  $E_4 = (K, 1 - 1/R_0)$  when  $R_0 > 1$ . It follows from the comparison theorem that solutions of the inequalities above satisfy

$$\limsup_{t \rightarrow \infty} H(t) \leq K, \quad \limsup_{t \rightarrow \infty} I(t) \leq 1 - 1/R_0. \tag{10}$$

Hence, if  $t$  is large enough, then  $P$  satisfies

$$\dot{P} \leq [ka(K + \eta)(1 + (q - 1)(1 - 1/R_0 + \eta) - c]P,$$

where  $\eta$  is small enough so that  $\psi < 0$  implies  $ka(K + \eta)(1 + (q - 1)(1 - 1/R_0 + \eta) - c) < 0$ . Thus  $\dot{P} < 0$  unless  $P = 0$ , so that  $P \rightarrow 0$  as  $t \rightarrow \infty$ . Corollary 4 implies that the solutions of system (7) go to the equilibrium  $E_4 = (K, 1 - 1/R_0, 0)$  as  $t \rightarrow \infty$ .

*4.3. Proofs when all prey have same vulnerability*

When  $q = 1$ , so that the infected prey have the same vulnerability as the susceptible prey, all conjectures regarding the cases in Section 3.2 hold globally. This model is related to a previously analyzed SIS model with disease in both the prey and the predators in Section 3 of Han et al. (2001). When  $q = 1$ ,  $\psi = 1 - c/kaK$ , so that case 3 is impossible. Moreover,  $q = 1$  implies that  $R_1 = \beta/(\gamma + b - \theta rc/kaK)$ . In case 6, it can be shown that (7)

has a unique interior equilibrium  $E_5 = (H_E, I_5, P_E)$  where  $I_5 = 1 - 1/R_1$ .

With  $q = 1$ , the equations for  $H$  and  $P$  in system (7) do not involve  $I$ , so that the asymptotic behaviors of  $H$  and  $P$  are given in Lemma 1. Thus when  $kaK/c \leq 1$ ,  $H \rightarrow K$  and  $P \rightarrow 0$  as  $t \rightarrow \infty$ , so that  $\dot{I} = \beta[1 - (\gamma + b - \theta r)/\beta - I + F(t)/\beta]I$ , where  $F(t) = \theta r(H/K - 1) \rightarrow 0$  as  $t \rightarrow \infty$ . Hence the differential equation has the limiting equation  $\dot{I} = \beta[1 - 1/R_0 - I]I$ . By Corollary 4, solutions must approach the globally asymptotically stable equilibrium of this logistic differential equation. This proves cases 1 and 2.

When  $kaK/c > 1$ ,  $H \rightarrow H_E$  and  $P \rightarrow P_E$  as  $t \rightarrow \infty$  by Lemma 1. Thus  $\dot{I} = \beta[1 - (\gamma + b - \theta rc/kaK)/\beta - I + F(t)/\beta]I$ , where  $F(t) = \theta r(H - H_E)/K \rightarrow 0$  as  $t \rightarrow \infty$ . Thus solutions approach the globally asymptotically stable equilibrium of the limiting equation  $\dot{I} = \beta[1 - 1/R_1 - I]I$ . This proves the global stability in cases 4–6.

*4.4. Existence of interior equilibria in cases 3 and 6*

When  $q > 1$ , we show that (7) has interior equilibria in  $G$  in cases 3 and 6. A positive equilibrium of (7) satisfies the equations

$$r \left( 1 - \frac{H}{K} \right) = a(1 + (q - 1)I)P, \tag{11}$$

$$\beta(1 - I) - \gamma - b + \frac{\theta rH}{K} = a(q - 1)P(1 - I), \tag{12}$$

$$kaH(1 + (q - 1)I) = c. \tag{13}$$

Combining (11) and (13), we obtain

$$P = (rk/c)H(1 - H/K) \triangleq f_1(H), \tag{14}$$

which is a parabola that is zero at  $H = 0$  and  $H = K$ . Eq. (13) implies that  $1 - I = (qH - H_E)/(q - 1)H$  so that  $H > H_E/q$ , where  $H_E = c/ka$  is the equilibrium value in Lemma 1. Substituting this into (12) yields

$$P = \frac{\beta}{a(q - 1)} - \frac{(\gamma + b - \theta rH/K)H}{a(qH - H_E)} \triangleq f_2(H). \tag{15}$$

It is easy to show that the function  $f_2(H)$  is increasing and concave downward on the interval  $[H_E/q, K]$  and approaches  $-\infty$  as  $H$  decreases to  $H_E/q$ .

If  $kaK/c \leq 1$ , then the curve  $f_2(H)$  intersects the parabola  $f_1(H)$  iff  $f_2(K) > f_1(K) = 0$ , which is equivalent to  $\psi > 0$ . Thus there is an interior equilibrium in  $G$  in case 3, but there is none in case 2. If  $kaK/c > 1$ , then we must have  $H \leq H_E < K$ , so that  $P$  in (14) is nonnegative. Here the curve  $f_2(H)$  intersects the parabola  $f_1(H)$  iff  $f_2(H_E) - f_1(H_E) > 0$ , which is equivalent to  $R_1 > 1$ . Thus there is an interior equilibrium in  $G$  in case 6 when  $R_1 > 1$ , but not in cases 4 or 5 when  $R_1 \leq 1$ . Although the increasing function  $f_2(H)$  intersected the parabola  $f_1(H)$  at only one point in numerical calculations for all parameter values that we

tried, we could not prove that the intersection is unique for all possible parameter sets. Setting  $f_1(H) = f_2(H)$  yields a cubic equation for  $H$ . Thus it is possible that there are three interior equilibria for some parameter values if the increasing, concave downward function  $f_2(H)$  intersects the parabola  $f_1(H)$  twice for  $H < K/2$  and then once for  $H > K/2$ . Since  $f_2(K)$  and  $f_2(H_E) \rightarrow \infty$  as  $\beta \rightarrow \infty$ , the value of the equilibrium prey population size  $H$  at an intersection of  $f_2(H)$  and the parabola  $f_1(H)$  should decrease from  $\min\{H_E, K\}$  towards its minimum of  $c/kaq$  as the contact rate  $\beta$  increases. As  $\beta$  increases, the equilibrium predator population size  $P$  goes up from  $P_E$  to its maximum of  $rkK^2/4c$  at the top of the parabola and then decreases towards a minimum of  $(1 - c/kaq)r/aq$ .

**5. Persistence and endemicity results**

For  $q > 1$  the interior equilibria could not be found explicitly and the stability of the interior equilibria for  $q > 1$  was intractable. Here we prove some results on the uniform persistence of the predator population  $P$  and the endemicity of the disease (i.e. uniform persistence of the infective fraction  $I$ ). A component  $x(t)$  of a positive solution of the model is defined to be uniformly persistent if its minimum (technically, its infimum for large time) always remains greater than some positive quantity (independent of the choice of the positive solution), so that it does not approach zero, i.e.  $\liminf_{t \rightarrow \infty} x(t) > \varepsilon > 0$  (Thieme, 1993).

**Theorem 5.** *If  $\mathbf{R}_0 < 1$  and  $kaK/c > 1$  are satisfied (case 4), or  $\mathbf{R}_0 > 1$  and  $\psi > 0$  are satisfied (cases 3, 5, 6), then there exists an  $\varepsilon > 0$ , independent of initial conditions, such that solutions starting in the interior of  $G$  satisfy  $\liminf_{t \rightarrow \infty} P(t) > \varepsilon$ .*

**Proof.** Define

$$X = \{(H, I, P) : 0 \leq H, 0 \leq I \leq 1, 0 \leq P\},$$

$$X_0 = \{(H, I, P) \in X : P > 0\},$$

$$\partial X_0 = X \setminus X_0.$$

It then suffices to show that (7) is uniformly persistent with respect to  $(X_0, \partial X_0)$ .

First, by the form of (7), it is easy to see that both  $X$  and  $X_0$  are positively invariant. Clearly,  $\partial X_0$  is relatively closed in  $X$ . We now show that (7) is point dissipative. By the first equation of (7), it is easy to see that any positive solution of (7) satisfies  $\limsup_{t \rightarrow \infty} H(t) \leq K$ . If  $V = kH + P$ , we have

$$\dot{V} = kr(1 - H/K)H - cP$$

$$= -cV + ckH + kr(1 - H/K)H.$$

Since  $\limsup_{t \rightarrow \infty} (ckH + kr(1 - H/K)H) \leq (c + r)kK$ , it follows that  $V(t)$  is ultimately bounded. Hence,  $P(t)$  is

ultimately bounded. Note that  $I(t)$  lies between 0 and 1. We conclude that (7) is point dissipative.

Set

$$M_\delta = \{(H(0), I(0), P(0)) : (H(t), I(t), P(t)) \text{ satisfies (7) and } (H(t), I(t), P(t)) \in \partial X_0, \forall t \geq 0\}.$$

Then it is easy to see that  $M_\delta = \{(0, 0, 0), (K, 0, 0)\}$  if  $\mathbf{R}_0 < 1$ , and

$$M_\delta = \{(0, 0, 0), (K, 0, 0), (K, 1 - 1/\mathbf{R}_0, 0)\}$$

if  $\mathbf{R}_0 > 1$ .

Take  $\varepsilon_1 > 0$  and  $\varepsilon_2 > 0$  small enough such that

$$ka(K - \varepsilon_1) > c \tag{16}$$

if  $\mathbf{R}_0 < 1$  and  $kaK/c > 1$  are satisfied, and such that

$$ka(K - \varepsilon_1)[1 + (q - 1)(1 - \varepsilon_2 - 1/\mathbf{R}_0)] > c \tag{17}$$

if  $\mathbf{R}_0 > 1$  and  $\psi > 0$  are satisfied. We now claim that  $\limsup_{t \rightarrow \infty} P(t) > 0$ . Suppose that  $\limsup_{t \rightarrow \infty} P(t) = 0$ . Then for any  $\xi > 0$ , there is a  $T > 0$  such that  $P(t) \leq \xi$  for all  $t \geq T$ . Let us choose  $\eta > 0$  small enough such that any positive solution of the following equation:

$$\dot{H} = [r(1 - H/K) - \eta]H$$

satisfies  $H(t) \geq K - \varepsilon_1$  for all large  $t$ . Now, we further restrict  $\eta > 0$  and  $\varepsilon_1$  small enough such that any positive solution of the following equation:

$$\dot{I} = I[\beta(1 - I) - (\gamma + b - \theta r(K - \varepsilon_1)/K - \eta)]$$

satisfies  $I(t) \geq 1 - \varepsilon_2 - 1/\mathbf{R}_0$  for all large  $t$ . Clearly, we can restrict  $\xi$  small enough such that any positive solution of (7) satisfies

$$\dot{H} \geq [r(1 - H/K) - \eta]H$$

for all large  $t$ . As a consequence,  $H(t) \geq K - \varepsilon_1$  for all large  $t$ . Then, we further restrict  $\xi$  small enough such that any positive solution of (7) satisfies

$$\dot{I} \geq I[\beta(1 - I) - (\gamma + b - \theta r(K - \varepsilon_1)/K - \eta)]$$

for all large  $t$ . Thus,  $I(t) \geq 1 - \varepsilon_2 - 1/\mathbf{R}_0$  for all large  $t$ . Consequently, if  $\mathbf{R}_0 > 1$  and  $\psi > 0$  are satisfied, we have

$$\dot{P} \geq P[ka(K - \varepsilon_1)(1 + (q - 1)(1 - \varepsilon_2 - 1/\mathbf{R}_0)) - c]$$

for all large  $t$ . It follows that  $\limsup_{t \rightarrow \infty} P(t) = +\infty$ , so that we have reached a contradiction to the boundedness of  $P$ . The case where  $\mathbf{R}_0 < 1$  and  $kaK/c > 1$  can be treated similarly.

If  $\mathbf{R}_0 < 1$ , it is easy to see that any solution  $(H(t), I(t), 0)$  of (7) with  $H(0) > 0$  approaches  $E_1 = (K, 0, 0)$  as  $t$  approaches infinity. If  $\mathbf{R}_0 > 1$ , any solution  $(H(t), I(t), 0)$  of (7) with  $H(0) > 0, I(0) > 0$  approaches  $E_4 = (K, 1 - 1/\mathbf{R}_0, 0)$  as  $t$  approaches infinity and the solution with  $H(0) > 0, I(0) = 0$  approaches  $E_1 = (K, 0, 0)$  as  $t$  approaches infinity. Then by the aforementioned claim, it follows that  $E_0 = (0, 0, 0), E_1 = (K, 0, 0)$  and  $E_4 = (K, 1 - 1/\mathbf{R}_0, 0)$  are isolated invariant sets in  $X$ , so any stable manifolds of these points cannot

occur in  $X_0$ . Clearly, these equilibria are acyclic in  $M_\partial$ . By Theorem 4.6 in Thieme (1993) (see also Hirsch et al. (2001) for a stronger repelling property of  $\partial X_0$ ), we conclude that system (7) is uniformly persistent with respect to  $(X_0, \partial X_0)$ .  $\square$

**Theorem 6.** *If  $kaK/c > 1$  and  $\mathbf{R}_1 > 1$  (case 6), then there exists an  $\varepsilon > 0$  such that each positive solution of (7) satisfies  $\liminf_{t \rightarrow \infty} I(t) > \varepsilon$ .*

**Proof.** Define

$$Y = \{(H, I, P) : 0 \leq H, 0 \leq I \leq 1, 0 \leq P\},$$

$$Y_0 = \{(H, I, P) \in Y : I > 0\},$$

$$\partial Y_0 = Y \setminus Y_0.$$

It then suffices to show that (7) is uniformly persistent with respect to  $(Y_0, \partial Y_0)$ . By the discussions above, we know that (7) is point dissipative.

Set

$$L_\partial = \{(H(0), I(0), P(0)) : (H(t), I(t), P(t)) \text{ satisfies (7) and } (H(t), I(t), P(t)) \in \partial Y_0, \forall t \geq 0\}.$$

Then it is easy to see that  $L_\partial = \{(0, 0, 0), (H_E, 0, P_E)\}$ .

Take  $\varepsilon_3 > 0$  and  $\varepsilon_4 > 0$  small enough such that

$$\begin{aligned} \beta - (\gamma + b - \theta r(H_E - \varepsilon_3)/K) \\ - a(q - 1)(P_E + \varepsilon_4) > 0. \end{aligned} \tag{18}$$

By (7), we have

$$\begin{aligned} \frac{\ln\left(\frac{H(t)}{H(0)}\right)}{t} &= r \left( 1 - \frac{\int_0^t H(s) ds}{tK} \right) \\ &\quad - a \frac{\int_0^t [1 + (q - 1)I(s)]P(s) ds}{t}, \end{aligned} \tag{19}$$

$$\frac{\ln\left(\frac{P(t)}{P(0)}\right)}{t} = -c + ka \frac{\int_0^t [1 + (q - 1)I(s)]H(s) ds}{t}.$$

For a continuous function  $f(t)$  with  $t \in [0, \infty)$ , we define

$$\langle f \rangle_t = \int_0^t f(s) ds / t.$$

Then (19) can be rewritten as

$$r(1 - \langle H \rangle_t / K) - a \langle P \rangle_t = r_1(t), \quad -c + ka \langle H \rangle_t = r_2(t). \tag{20}$$

We now claim that  $\limsup_{t \rightarrow \infty} I(t) > 0$ . Suppose that  $\limsup_{t \rightarrow \infty} I(t) = 0$ . Then for any  $\xi_1 > 0$ , there is a  $T_1 > 0$  such that  $I(t) \leq \xi_1$  for all  $t \geq T_1$ . By Theorem 5, we have  $\liminf_{t \rightarrow \infty} P(t) > \varepsilon$ . By similar arguments as those in the proof of Theorem 5, we can deduce that  $\liminf_{t \rightarrow \infty} H(t) > \varepsilon_5 > 0$  where  $\varepsilon_5$  is a constant. As a consequence of these facts, by taking  $\xi_1$  small enough and let  $t$  be sufficiently large, we can obtain from (20) that

$$\langle H \rangle_t > H_E - \varepsilon_3, \quad \langle P \rangle_t < P_E - \varepsilon_4. \tag{21}$$

Then by the second equation of (7), we have

$$\begin{aligned} I(t) \geq I(0) \exp\{t[\beta - (\gamma + b - \theta r(H_E - \varepsilon_3)/K) \\ - a(q - 1)(P_E + \varepsilon_4) - \beta \langle I \rangle_t + a(q - 1) \langle IP \rangle_t]\} \end{aligned} \tag{22}$$

By restricting  $\xi_1$  small enough, it follows from (18) and (22) that  $I(t) \rightarrow \infty$  as  $t \rightarrow \infty$ , which is a contradiction to the boundedness of  $I$ . This proves the claim. Then, by similar discussions as those in the proof of Theorem 5, we conclude that the disease is uniformly persistent.  $\square$

**Corollary 7.** *If  $kaK/c > 1$  and  $\mathbf{R}_1 > 1$  (case 6), then the disease and the populations are uniformly persistent.*

When  $kaK/c > 1$  and  $q$  is large enough, then  $\mathbf{R}_1 < 1$ . Then we can prove that the disease dies out for any initial position.

**Theorem 8.** *If  $kaK/c > 1$  and  $1 < \mathbf{R}_0$ , then the disease dies out when  $q$  is large enough (case 5).*

**Proof.** By Theorem 5, we have  $\liminf_{t \rightarrow \infty} P(t) > \varepsilon$ . By similar discussions as those in the proof of Theorem 5, we see that there is an  $0 < \varepsilon_6 < 1$  such that  $\limsup_{t \rightarrow \infty} I(t) < 1 - \varepsilon_6$ . It follows from the second equation of (7) that  $\dot{I} < -I$  when  $q$  is large enough. Consequently,  $I(t) \rightarrow 0$  as  $t \rightarrow \infty$ . By the proof of Theorem 6, we have  $\liminf_{t \rightarrow \infty} H(t) > \varepsilon_5 > 0$ . Hence, the number of infectious prey approaches zero as time evolves to infinity.  $\square$

We use numerical calculations to illustrate the asymptotic behavior of the model. Let us fix  $\beta = 1.55, \gamma = 0.15, r = 1, c = 1, k = 1, a = 1, K = 2.6, b = 0.9, \theta = 0.8$ , so that  $kaK/c = 1.625 > 1$ . Then, for  $q$  in the interval  $[1, 2.312]$ , we have  $\mathbf{R}_1 > 1$ . For these parameter values we are in case 6 and there is an attractive interior equilibrium  $E_5$ . Moreover, the endemic infective fraction  $I_5$  is decreasing as  $q$  increases. When  $q$  passes 2.312, the endemic equilibrium infective fraction disappears. Then we are in case 5, so  $I(t)$  approaches 0 as  $t \rightarrow \infty$ , and  $H(t)$  and  $P(t)$  tend to their equilibrium values  $H_E$  and  $P_E$ . This means that the disease dies out in the prey, and the uninfected prey and the predator coexist in a global steady state  $E_2 = (H_E, 0, P_E)$  (see Fig. 1).

### 6. Discussion

Both pathogens and predation can be important in regulating host populations. Here we have investigated the simultaneous effects of microparasites and predators not only on the prey population size, but also on each other. In some of the six possible cases in Section 3.2, the two regulatory mechanisms of disease and predation seem to operate independently. For example, in cases 1 and 2, the feeding efficiency  $k$  of the predator is low ( $kaK/c \leq 1$ ), so that the predator population becomes extinct and the prey population size goes to its carrying

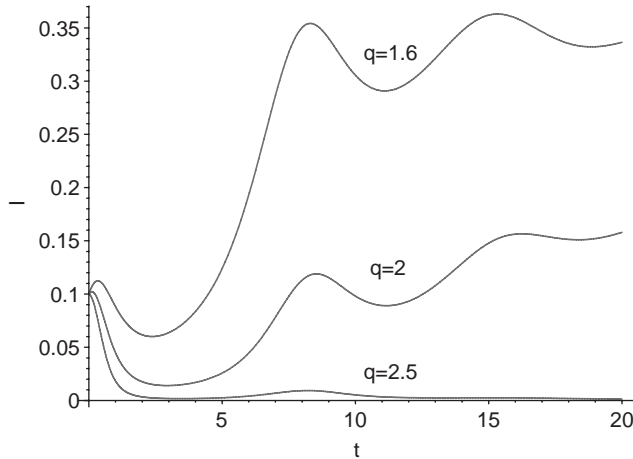


Fig. 1. Graphs of  $I(t)$  when  $\beta = 1.55$ ,  $\gamma = 0.15$ ,  $r = 1$ ,  $c = 1$ ,  $k = 1$ ,  $a = 1$ ,  $K = 2.6$ ,  $b = 0.9$ , and  $\theta = 0.8$  where initial values are  $H(0) = 0.8$ ,  $I(0) = 0.1$ , and  $P(0) = 1$ . The curves represent  $I(t)$  when  $q = 1.6, 2$ , and  $2.5$ .

capacity  $K$ . In these cases the basic reproduction number  $R_0$  determines whether the disease dies out ( $R_0 \leq 1$ ) or remains endemic ( $R_0 > 1$ ) in the prey population.

But in case 3, the endemicity of the disease and the greater vulnerability of the infected prey ( $q > 1$ ) allows the predator population to persist, when it would have become extinct without the disease. In this case, the presence of the disease is beneficial to the predator population, because it provides some prey who are more easily caught by the predators. We determined that for this to occur, the parameter values must satisfy  $(q - 1)(1 - 1/R_0) > (c/kaK - 1)$ . Note that for case 3 with  $kaK/c \leq 1$  and  $R_0 > 1$ , this condition is never satisfied when the vulnerabilities are the same for the susceptible and infected prey ( $q = 1$ ), but it is satisfied when the vulnerability  $q$  of the infected prey is large enough.

In the last three cases in Section 3.2, the feeding efficiency  $k$  of the predators is large enough ( $kaK/c > 1$ ), so that the predator population always persists. Case 4 corresponds to the situation where the basic reproduction number  $R_0$  satisfies  $R_0 \leq 1$ , so that the disease dies out and the prey and predator populations approach their usual equilibrium values  $H_E$  and  $P_E$ . When  $R_0 > 1$ , there are two cases depending on the value of another threshold quantity  $R_1$  given by Eq. (8).

In case 5 with  $R_1 \leq 1 < R_0$ , the disease dies out and the prey and predator populations approach their usual equilibrium values  $H_E$  and  $P_E$ . But case 5 is unusual, since the disease dies out even though the basic reproduction number  $R_0$  is greater than 1, so that the disease would have remained endemic without the predators. From the expressions for  $R_1$  and  $R_0$ , we

see that case 5 occurs if the parameter values satisfy

$$\gamma + b - \theta r < \beta \leq (\gamma + b - \theta rc/kaK) + (q - 1)r(1 - c/kaK).$$

The right inequality above shows that case 5 can occur even if the susceptible and infected prey have the same vulnerability ( $q = 1$ ). In this situation the predation lowers the prey population size  $H$  below its carrying capacity  $K$ , which decreases the mean time in the infectious class given by  $1/(\gamma + b - \theta rH/K)$ , so that the disease can die out. The right inequality above is even more likely to occur when the infected prey are more vulnerable to predation ( $q > 1$ ) and is certain to occur for large enough  $q$ . Hence, the more vulnerable the infected prey are to predation, the more likely the disease can be eliminated by the predators.

In case 6 with  $1 < R_1 < R_0$ , the disease remains endemic and solutions approach an interior equilibrium with an equilibrium infective fraction that is less than  $1 - 1/R_0$ , and an equilibrium prey population size that is less than the disease free equilibrium  $H_E$ . Thus predation on weaker, diseased animals helps keep the prey population healthy by reducing the fraction of the population that is infected.

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