EVOLUTIONARY CONSEQUENCES OF PREDATION FOR PATHOGENS IN PREY

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Abstract. This article investigates the impact of predation on the coexistence and competitive exclusion of pathogen strains in the prey. Two types of predator are considered — a generalist and a specialist. For each type of predator we assume that the predator can discriminate among susceptible and infected with each strain prey. The two strains will competitively exclude each other in the absence of predation with the strain with the larger reproduction number persisting. If a generalist predator preys discriminantly and the disease is fatal, then, depending on the predation level, a switch in the dominant pathogen may occur. Thus, for some predation levels the first strain may persist while for other predation levels the second strain may persist. Furthermore, a specialist predator preying discriminantly may mediate the coexistence of the two strains. Although in most cases increasing predation reduces the disease load in the prey, when predation leads to coexistence, it may also lead to *increase* in the the disease load.

Keywords: predator-prey, disease in prey, evolution, strains, competitive exclusion, predator mediated switch in dominant strain, predator mediated coexistence.

AMS Subject Classification: 92D30, 92D40

1. Introduction

The current threat of avian influenza has raised our awareness that we, the humans, are only one part in a complex web of interactions, where many disease-causing microorganisms are major players [23]. In terms of disease transmission the human population is linked to other, non-human species, through pathogens, such as influenza type A, that have the ability to cross the species borders and become adapted to new hosts. Many human diseases have emerged as a result of such adaptation. This suggests that understanding emerging pathogens in the human population requires the understanding of the infectious disease ecology in wildlife populations [5].

Diseases of animal and plant species impact their host species in various ways but often by decreasing their numbers [22], leading to a variety of implications for conservation [17]. The classical Kermack-McKendrick SIR epidemic model [14], which was developed for human populations [1], can also be applied to model disease spread in many non-human vertebrate species.

One assumption in the classical Kermack-McKendrick SIR epidemic model is that the population is closed, that is, the individuals in the population communicate only with other individuals in the same population, and the population is not subject to immigration or emigration. This assumption of complete isolation is rarely true in animal populations. Wildlife populations participate in ecological interactions with various

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other populations, occupying the same habitat, thus building an ecological community. Typically, the species in the community form complex web of trophic interactions called food web [20]. Because food webs may include hundreds of species, theoretical studies in community ecology often concentrate on subunits of a web, called "community modules" [10]. Community modules most frequently focus on two fundamental community interactions: competition for resources in the common habitat, and predation.

Integration of disease epidemiology with community ecology has recently begun to attract the attention of the scientists. Complex interdependence exists between the host-pathogen interactions and community interactions [5]. For instance, rabbit calicivirus disease (RCD) was introduced in Australia as a biological control agent of the wild rabbit population [7]. As a result, the rabbit population declined, but that may have had various consequences for the Australian flora and fauna. Investigation of these consequences through a mathematical model suggests that the reduction in the rabbit population may lead to reduction of foxes, a common predator for the rabbits, and an immediate small increase in the pasture biomass [19]. Conversely, changing community interactions may impact the disease prevalence in the affected species. Both experimental [15] and theoretical results [18] show that removal of predators leads to increase in disease prevalence in the prey.

Since the time of Lotka and Volterra [16] the most studied community module is the predator-prey relationship. It is hardly surprising that the study of the disease-ecology interdependence begins by the integration of simple epidemic SI or SIR models with predator-prey models. There are two main types of predators — generalist and specialist. Generalist predators feed on many types of species. Consequently, their dynamics is not coupled to the dynamics of a specific prey population. When a focal prey population is threatened by extinction, the predator is capable of changing his diet to another species and may continue to persist. Such a generalist predator acts on a specific prey population as an external added morality. The impact of such a predator on a prey, infected by a disease, is modeled through an SI or SIR model with additional mortality dependent on predator's density, assumed at equilibrium [18, 13]. Specialist predators feed almost exclusively one specific species of prey. As a consequence, the predator's numbers are strongly dependent on prey numbers, and prey extinction will almost surely lead to predator extinction. It is this type of predator-prey relationship that is modeled by the Lotka-Volterra model. Mathematical analysis focuses on the integration of Lotka-Volterra predator-prey models with SI or SIR disease models [4, 24, 25, 8] where sustained oscillations are found primarily as a result of presence of nonlinear functional response of the predator. Differential-delay models with constant delay in the equation of the predator [26], and eco-epidemiological models with age-structure in the prey have also been considered [6]. Saenz and Hethcote find that a disease common for two competing species may be able to change the competitive outcome [21].

It seems that little attention has been paid on the impact of predation on the evolution of the pathogens. Holt and Dobson [11] mention in a recent article that predation may be responsible for the coexistence of two competing pathogen strains. It is this scenario that we investigate in this article. The impact of predation or other community interactions on the disease evolution may be one of the mechanisms responsible for the emergence of new pathogen strains capable of crossing the species barriers.

In this article we investigate the impact of predation on competing pathogen strains in the prey population. Competitive exclusion is the only possible outcome for the two microorganisms in the absence of predation. We investigate the potential impact of both types of predators: generalist and specialist exerting discriminate (preying more intensely on some prey classes than others) predation. In Section 2 we consider the impact of a generalist predator. In Subsection 2.1 we examine the effect on the prey total population size when the predator attacks preferentially infected prey. In Subsection 2.2 we consider the various equilibria of the model. Subsection 2.3 is devoted to the impact of predation on the strain persistence and extinction. Section 3 is devoted to the impact of a specialist predator on the competing pathogen strains. In Subsection 3.1 we consider the boundary equilibria. In Subsection 3.2 we consider the coexistence equilibria. Section 2 and Section 3 focus of the case of a linear functional response of the predator. Section 4 considers extensions to non-linear functional response. Section 5 summarizes our observations and draws conclusions.

2. Generalist predator and the competition of pathogen variants in the prey population

We consider the spread of a disease in a non-human population subjected to predation. We model the disease spread with an SI model, thus effectively assuming that the prey does not recover from the disease. We assume that the disease is represented by two strains. Susceptible prey individuals, whose numbers at time t are given by $S(t)$, become infected with strain i if they get into contact with infected individuals with strain i. The number of infected individuals with strain i is given by $I_i(t)$. Transmission of strain i occurs at a rate β_i. Susceptible prey individuals die at a natural death rate μ_0, while infected individuals die at a rate μ_i for strain i. We assume that disease may add mortality, that is

$$\mu_1 \geq \mu_0 \qquad \mu_2 \geq \mu_0.$$

The total population size of the prey is $N(t) = S(t)+I_1(t)+I_2(t)$. The prey is subjected to predation by a generalist predator. We assume that the predator's dynamics has stabilized at equilibrial level and the predation on the focal prey acts as additional mortality for that prey. We assume the additional mortality is proportional to the predator equilibrial population size P, considered as a parameter. Therefore, the predation-added mortality to the prey is given by, aP, where a is the attack rate. In general, there are three different attack rates – an attack rate for susceptible prey, η, attack rate for prey individuals who are host to strain one, γ_1, and attack rate for prey individuals who are host to strain two, γ_2. With these notations the prey model with disease becomes:

$$\begin{aligned}
\frac{dS}{dt} &= rS\left(1-\frac{S+I_1+I_2}{K}\right) - \beta_1 SI_1 - \beta_2 SI_2 - (\mu_0+\eta P)S,\\
\frac{dI_1}{dt} &= \beta_1 SI_1 - (\mu_1+\gamma_1 P)I_1,\\
\frac{dI_2}{dt} &= \beta_2 SI_2 - (\mu_2+\gamma_2 P)I_2
\end{aligned} \tag{2.1}$$

Here we have denoted the intrinsic growth rate of the prey population by r. In addition we have assumed that the disease affects reproduction and only the susceptible population reproduces at the intrinsic reproduction rate r. Furthermore, we assume that the infected individuals do not reproduce. These assumptions appear to be common for

many predator-prey models with disease in prey. The parameter K denotes the baseline carrying capacity of the environment in the logistic equation.

It should be noted that we will iterpret the logistic term in the equation for the susceptible prey $r\left(1-\frac{S+I_1+I_2}{K}\right)$ as density-dependent per capita birth rate. To keep the birth rate non-negative, we will consider the system (2.1) (and later the system (3.1)) on the set

$$\Omega = \{(S, I_1, I_2) : 0 \leq S + I_1 + I_2 \leq K\}.$$

The set Ω is forward invariant for the system (2.1). Thus, solutions that start from that set, remain in the set for all time. In addition the set Ω contains the dynamics we are interested in.

Adding the three equations we obtain the differential equation of the total prey population

$$\frac{dN}{dt} = rS\left(1-\frac{N}{K}\right) - (\mu_0+\eta P)S - (\mu_1+\gamma_1 P)I_1 - (\mu_2+\gamma_2 P)I_2. \tag{2.2}$$

One observation that can be made from the equation for the total prey population size is that if $r < \mu_0$, then the total prey population goes to extinction, independent of the level of predation or disease. To see this, we recall that $\mu_1 \geq \mu_0$ and $\mu_2 \geq \mu_0$. These inequalities imply that $N'(t) \leq rN - \mu_0 N$. Therefore, if $r < \mu_0$, the total prey population $N(t) \to 0$ as $t \to \infty$. We summarize this observation in the following proposition.

Proposition 2.1. *Assume $r < \mu_0$. Then the total prey population declines to zero as time goes to infinity independently of the level of predation:*

$$N(t) \to 0 \qquad t \to \infty.$$

In view of Proposition 2.1, unless otherwise noted, we will assume in the rest of the paper that

$$r > \mu_0.$$

2.1. **The total prey population when $\gamma_1 \geq \eta$ and $\gamma_2 \geq \eta$.** In the case of more intense predation on infected individuals $\gamma_1 \geq \eta$ and $\gamma_2 \geq \eta$, or indiscriminate predation $\gamma_1 = \gamma_2 = \eta$ the equation of the total population size (2.2) can be rewritten as the following inequality

$$\frac{dN}{dt} \leq rN\left(1-\frac{N}{K}\right) - (\mu_0+\eta P)N \tag{2.3}$$

The solution of this inequality is dominated by the solution of the corresponding equality. The corresponding equality is the logistic equation with predation (harvesting). We observe that we can further simplify inequality (2.3) to obtain $N' \leq rN - (\mu_0+\eta P)N = (r-\mu_0-\eta P)N$. This implies that if the predation level is such that $r < \mu_0 + \eta P$, then the prey population goes to extinction $N(t) \to 0$ as $t \to \infty$. We summarize this result in the following proposition

Proposition 2.2. *Assume $\gamma_1 \geq \eta$ and $\gamma_2 \geq \eta$. Assume also $r < \mu_0 + \eta P$. Then the total prey population declines to zero as time goes to infinity:*

$$N(t) \to 0 \qquad t \to \infty.$$

Assuming that $r > \mu_0 + \eta P$, we denote by

$$r_P = r - \mu_0 - \eta P$$

the *predator-dependent intrinsic growth rate*, and by

$$K_P = \frac{K r_P}{r}$$

the *predator-dependent carrying capacity*. We note that both the predator-dependent intrinsic growth rate and the predator-dependent carrying capacity are linear decreasing functions of the predation level P. With this notation, we have that the solution of inequality (2.3) is dominated by the corresponding solution of the logistic equation

$$\frac{dN}{dt} = r_P N \left(1 - \frac{N}{K_P}\right) \tag{2.4}$$

and therefore satisfies [3]:

$$N(t) \leq \frac{N_0 K_P}{(K_P - N_0)e^{-r_P t} + N_0}. \tag{2.5}$$

The solutions of the logistic equation approach the carrying capacity K_P. This implies that if $r < \mu_0 + \eta P$ the limit of the total prey population size is zero, and if $r > \mu_0 + \eta P$ the limit may be zero, or may be non-zero but is smaller than K_P. Because of its threshold property, the inequality $r < \mu_0 + \eta P$ can be rewritten in the form $\mathcal{R} < 1$, where

$$\mathcal{R} = \frac{r}{\mu_0 + \eta P}$$

can be interpreted as the *prey reproduction number*. The prey reproduction number is a decreasing function of predation. Its largest value is the *predation-free prey reproduction number*, given by r/μ_0. Since $r > \mu_0$, there exists *threshold predation level*, given by

$$P^* = \frac{r - \mu_0}{\eta}. \tag{2.6}$$

The threshold predation level is proportional to the growth rate of the prey population outside predation and inversely proportional to the attack rate. The threshold predation level is such that for $P > P^*$, the prey reproduction number $\mathcal{R} < 1$ and the prey population dies out.

2.2. **Equilibria.** If the reproduction number of the prey population is above one, $\mathcal{R} > 1$, the system (2.1) has a disease-free equilibrium $\mathcal{E}_0 = (S^0, 0, 0)$, where S^0 satisfies the same equation as (2.4). Consequently, $S^0 = K_P$. There are two dominance equilibria corresponding to each strain. Strain one dominance equilibrium $\mathcal{E}_1 = (S_1^*, I_1^*, 0)$ satisfies the following system:

$$\begin{aligned} 0 &= rS\left(1 - \frac{S + I_1}{K}\right) - \beta_1 S I_1 - (\mu_0 + \eta P)S, \\ 0 &= \beta_1 S I_1 - (\mu_1 + \gamma_1 P) I_1. \end{aligned} \tag{2.7}$$

After canceling I_1 in the second expression we obtain

$$S_1^* = \frac{\mu_1 + \gamma_1 P}{\beta_1}.$$

Again, the number of susceptible prey at equilibrium is an increasing function of the predation level with slope equal to the ratio of the attack rate for individuals infected with strain one, γ_1, and the transmission coefficient, β_1. This means that when the attack rate is differential, and it is higher for individuals infected with strain one than for the susceptible individuals, $\gamma_1 > \eta$, there will be more susceptible individuals in the prey population compared to the case when the attack rate for all prey is indiscriminate and equal to the baseline attack rate for susceptible individuals. Canceling S in the first equation of the system (2.7), and replacing S with its value at equilibrium, we obtain the following expression for the disease load of strain one at equilibrium

$$I_1^* = \frac{\left(K_P - \frac{\mu_1+\gamma_1 P}{\beta_1}\right)}{1+\frac{\beta_1 K}{r}}.$$

We define the reproduction number of strain one for a generalist predator

$$\mathcal{R}_1 = \frac{K_P \beta_1}{\mu_1 + \gamma_1 P}.$$

We have a positive disease load of strain one if the reproduction number of strain one for discriminate predation is larger than one, that is, $\mathcal{R}_1 > 1$. The reproduction number of strain one for discriminate predation is a decreasing and concave up function of predation level, P. If strain one reproduction number in the absence of predation ($P = 0$) is larger than one, $\mathcal{R}_{0,1} > 1$, then there is a threshold predation level

$$P_1 = \frac{\mu_1(\mathcal{R}_{0,1} - 1)}{\beta_1 \frac{K}{r}\eta + \gamma_1}$$

such that for $P < P_1$ the reproduction number of strain one for discriminate predation $\mathcal{R}_1 > 1$ while for $P > P_1$ the reproduction number of strain one for discriminate predation $\mathcal{R}_1 < 1$. We call P_1 *strain one threshold predation level for a generalist predator*. The reproduction number of strain one gives the number of secondary cases one infected with strain one prey individual can produce in an entirely susceptible prey during its lifetime as infected. Indeed, K_P gives the number of susceptible prey in the disease-free prey population. The product $\beta_1 K_P$ gives the number of secondary infections one infected individual will produce in an entirely susceptible prey population per unit of time. One infected individual spends $1/(\mu_1 + \gamma_1 P)$ time units as infectious until that individual dies from natural causes or predation.

Symmetry gives a unique dominance equilibrium of strain two $\mathcal{E}_2 = (S_2^*, 0, I_2^*)$, where S_2^* and I_2^* are given as follows:

$$S_2^* = \frac{\mu_2 + \gamma_2 P}{\beta_2} \qquad I_2^* = \frac{\left(K_P - \frac{\mu_2+\gamma_2 P}{\beta_2}\right)}{1+\frac{\beta_2 K}{r}}.$$

The reproduction number of strain two and the strain two threshold predation level for a generalist predator are similarly given as:

$$\mathcal{R}_2 = \frac{K_P \beta_2}{\mu_2 + \gamma_2 P} \qquad P_2 = \frac{\mu_2(\mathcal{R}_{0,2} - 1)}{\beta_2 \frac{K}{r}\eta + \gamma_2}$$

The existence of boundary equilibria can be summarized in the following proposition

Proposition 2.3. *Assume $\mathcal{R} > 1$. Then, there always exists a disease-free equilibrium $\mathcal{E}_0 = (K_P, 0, 0)$. In addition, if the reproduction number of strain one is larger than one, $\mathcal{R}_1 > 1$, there exists a unique dominance equilibrium corresponding to strain one, $\mathcal{E}_1 = (S_1^*, I_1^*, 0)$. Similarly, if the reproduction number of strain two is larger than one, $\mathcal{R}_2 > 1$, there exists a unique dominance equilibrium corresponding to strain two, $\mathcal{E}_2 = (S_2^*, 0, I_2^*)$.*

Coexistence equilibria, if such exist, must satisfy the following two equations:

$$\begin{aligned} 0 &= \beta_1 S I_1 - (\mu_1 + \gamma_1 P) I_1, \\ 0 &= \beta_2 S I_2 - (\mu_2 + \gamma_2 P) I_2 \end{aligned} \tag{2.8}$$

From these two equations, after canceling I_1 and I_2, we obtain that

$$S^* = \frac{\mu_1 + \gamma_1 P}{\beta_1}, \qquad S^* = \frac{\mu_2 + \gamma_2 P}{\beta_2}.$$

These two expressions for S^* will be equal if the predation level P satisfies the following equality

$$\frac{\mu_1 + \gamma_1 P}{\beta_1} = \frac{\mu_2 + \gamma_2 P}{\beta_2}$$

The predation level that satisfies this equality is given by

$$P_c = \frac{\beta_2 \mu_1 - \beta_1 \mu_2}{\beta_1 \gamma_2 - \beta_2 \gamma_1}$$

provided the expression on the right-hand side is positive. We illustrate the area in the (β_1, β_2) plane where $P_c > 0$ in Figure 1. We summarize the result in the following proposition.

Proposition 2.4. *The predation level P_c for which coexistence may occur is positive if and only if*

$$\min\left\{\frac{\mu_2}{\mu_1}, \frac{\gamma_2}{\gamma_1}\right\} \leq \frac{\beta_2}{\beta_1} \leq \max\left\{\frac{\mu_2}{\mu_1}, \frac{\gamma_2}{\gamma_1}\right\}.$$

If $P_c > 0$ then both equations in (2.8) are simultaneously satisfied, and there are infinitely many coexistence equilibria as long as the following equation is also satisfied:

$$0 = r\left(1 - \frac{S + I_1 + I_2}{K}\right) - \beta_1 I_1 - \beta_2 I_2 - (\mu_0 + \eta P).$$

In fact, the coexistence that occurs in this case is the coexistence that occurs in the degenerate case $\mathcal{R}_1 = \mathcal{R}_2$. It is not hard to see that for $P = P_c$ we have exactly $\mathcal{R}_1 = \mathcal{R}_2$. We conclude that if the disease that affects the prey has differential mortality for the two strains and/or if the attack rate for prey individuals infected with the two strains is differential, then there is a unique predation level $P = P_c$ for which coexistence may occur. Thus coexistence may occur but it is rare. In general, coexistence does not occur in any other way, and the global dynamics of the system is given again by the following proposition

Proposition 2.5. *If $\mathcal{R}_1 < 1$ and $\mathcal{R}_2 < 1$ then the disease-free equilibrium is globally asymptotically stable. If $\mathcal{R}_1 > 1$ and/or $\mathcal{R}_2 > 1$, then the strain with the larger reproduction number persists and the other strain dies out. Coexistence does not occur outside of the degenerate case $\mathcal{R}_1 = \mathcal{R}_2$.*

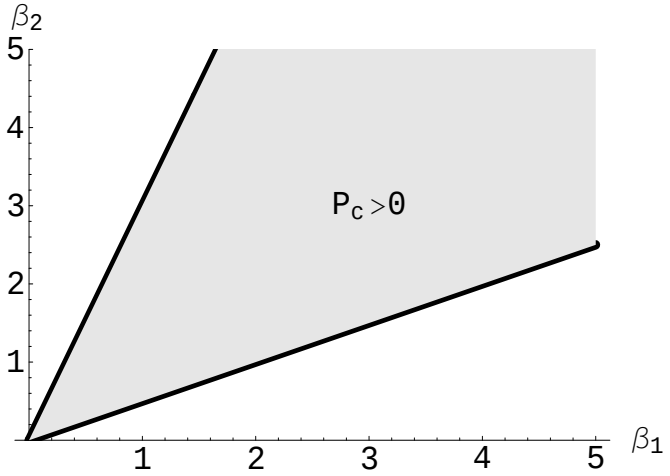

FIGURE 1. This figure shows the area in the (β_1, β_2) plane for which there is coexistence, that is, for which $P_c > 0$. This area in the figure is shaded in light grey. The lower boundary line of the shaded area is given by the line $\beta_2 = \min\left\{\frac{\mu_2}{\mu_1}, \frac{\gamma_2}{\gamma_1}\right\}\beta_1$. In the case of the figure above this line is given by the equation $\beta_2 = 0.5\beta_1$. The upper boundary line of the shaded area is given by the line $\beta_2 = \max\left\{\frac{\mu_2}{\mu_1}, \frac{\gamma_2}{\gamma_1}\right\}\beta_1$. In the case of the figure above this line is given by the equation $\beta_2 = 3\beta_1$. In the remaining part of the positive quadrant $P_c < 0$ and coexistence does not occur.

2.3. **Impact of predation on strain persistence.** However, it is important to note that the presence of a generalist predator can impact the outcome of the competition of strains in the prey. Predation level may determine which strain dominates. For instance, assume without loss of generality, that in the absence of predation $P = 0$ both reproduction numbers are above one, and also the reproduction number of the first strain is larger than the reproduction number of the second strain, $\mathcal{R}_{0,1} > \mathcal{R}_{0,2}$. In this case, according to Proposition 2.5, strain one will competitively exclude strain two and will dominate in the prey population. As predation level P is increased, there are two possibilities:

1. Strain one threshold predation level is smaller than the coexistence predation level: $P_1 < P_c$. In this case, as predation level P increases from zero, it first exceeds P_1. As soon as $P > P_1$, the reproduction number of strain one becomes smaller than one. The reproduction number of strain strain two is already smaller than one, as it is smaller than the reproduction number of strain one. Consequently, according to Proposition 2.5, the disease disappears from the prey population. Predation has eliminated the disease.
2. Strain one and strain two threshold predation levels are larger than the coexistence predation level: $P_1 > P_c$ and $P_2 > P_c$. In this case, as the predation level increases from zero it first becomes equal to the coexistence predation level, $P = P_c$. When $P < P_c$ strain one eliminates strain two and dominates in the prey population because we have $\mathcal{R}_1 > \mathcal{R}_2$ (see Proposition 2.5). When $P = P_c$, coexistence occurs. At this point the reproduction numbers of the two strains are

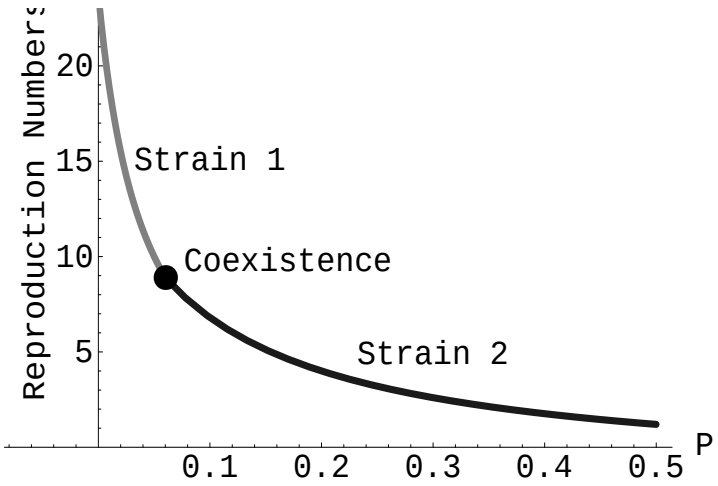

FIGURE 2. This figure shows a graph of the reproduction numbers $\mathcal{R}_1$ and $\mathcal{R}_2$ as functions of the predation level P. The predation level for which the two reproduction numbers are equal is $P_c = 0.06$. For $P < P_c$, the reproduction number $\mathcal{R}_1$ is larger and is the one graphed in grey. This means that strain one will dominate for those predation levels. For $P = P_c$ there is coexistence denoted with a large dot. For $P_2 > P > P_c$ the reproduction number $\mathcal{R}_2$ is larger and is graphed in black. This means that strain two will dominate for those predation levels. The remaining parameters of the figure are: $\beta_1 = 5$, $\beta_2 = 8$, $\mu_0 = 0.1$, $\mu_1 = 0.2$, $\mu_2 = 0.5$, $\gamma_1 = 5$, $\gamma_2 = 5$, $\eta = 2$, $K = 1$, $r = 2$.

equal. As predation becomes slightly larger than the coexistence predation level, $P > P_c$, the reproduction number of the second strain becomes larger than the reproduction number of the first strain but they are both still larger than one, $1 < \mathcal{R}_1 < \mathcal{R}_2$. Proposition 2.5 implies that in this case strain two will eliminate strain one, and persist in the population. Further increase in predation level will lead to the predation level increasing through P_1, when the reproduction number of strain one becomes smaller than one but there is no change in the dynamical outcome of the competition. If the predation level continues to increase, it will become larger than the strain two threshold predation level, $P > P_2$. At this point the reproduction number of strain two will also become smaller than one. Proposition 2.5 implies that both strains will be eliminated and the prey population will become disease-free. Predation has eliminated the disease, provided, it has not eliminated the entire prey population first. We illustrate this situation in Figure 2.

We conclude that for a fatal disease with differential disease-induced mortalities for the two strains and/or discriminate predation on individuals infected with the two strains, changing predation levels may lead to coexistence in a rare case but, more importantly, it may induce a switch in the dominant pathogen variant.

3. Specialist predator and the competition of pathogen variants in the prey population

In this section we consider again the spread of a disease in a prey population. We model the spread of the disease in the prey population the same way as with a generalist predator (see model (2.1)). However, we assume that the predator is a specialist predator, that is, it feeds exclusively on the focal prey population. The dynamics of a specialist predator is strongly coupled with the dynamics of the prey population. Thus, the number of predators is described as a dynamical variable $P(t)$ whose dynamics is given by differential equation. To the model (2.1) we add an equation for the dynamics of the predator. Its size increases in time by the biomass of the prey, that the predator has consumed, and decreases by the natural death rate of the predator. We denote by d the per capita death rate of the predator. The predator-prey model with disease in prey becomes:

$$\begin{aligned}
\frac{dS}{dt} &= rS\left(1-\frac{S+I_1+I_2}{K}\right)-\beta_1 SI_1-\beta_2 SI_2-(\mu_0+\eta P)S,\\
\frac{dI_1}{dt} &= \beta_1 SI_1-(\mu_1+\gamma_1 P)I_1,\\
\frac{dI_2}{dt} &= \beta_2 SI_2-(\mu_2+\gamma_2 P)I_2\\
\frac{dP}{dt} &= \epsilon(\eta S+\gamma_1 I_1+\gamma_2 I_2)P-dP,
\end{aligned} \tag{3.1}$$

where ϵ denotes predator's metabolic efficiency by which the biomass of consumed prey is converted to predator's biomass. The parameter ϵ is called *predator's conversion efficiency*. We assume in this section again that $\mu_1 \geq \mu_0$ and $\mu_2 \geq \mu_0$ as well as $r > \mu_0$. As before, we consider the full model (3.1) under the assumption that only susceptible prey give birth. We notice first that in the absence of disease the system above becomes

$$\begin{aligned}
\frac{dS}{dt} &= rS\left(1-\frac{S}{K}\right)-(\mu_0+\eta P)S,\\
\frac{dP}{dt} &= \epsilon\eta SP-dP.
\end{aligned} \tag{3.2}$$

This is the classical Lotka-Volterra predator-prey model. This model has been extensively studied. We introduce here some notation and results to be used later. If we denote by $K_P^\circ = \frac{K}{r}(r-\mu_0)$, we can call K_P° prey *carrying capacity in the absence of predation*. We introduce also the predator reproduction number:

$$\mathcal{R}_p = \frac{\epsilon\eta K_P^\circ}{d}. \tag{3.3}$$

The predator reproduction number gives the number of predators that will be produced in a population where the prey is at carrying capacity K_P°. To see this, notice that in a predator-free population, the prey is at carrying capacity K_P°. Consequently, ηK_P° gives the number of prey killed and eaten by one predator per unit of time, $\epsilon\eta K_P^\circ$ gives the number of prey killed and eaten by one predator, and converted into new predator biomass, per unit of time. Finally, $1/d$ is the lifespan of a predator. The predator-prey coexistence equilibrium $\hat{E} = (\hat{S}, \hat{P})$ is given by

$$\hat{S} = \frac{K_P^\circ}{\mathcal{R}_p} \qquad \hat{P} = \frac{r-\mu_0}{\eta}\left(1-\frac{1}{\mathcal{R}_p}\right). \tag{3.4}$$

We note that, as expected, the predator equilibrium size increases with the predator reproduction number $\mathcal{R}_p$ while the prey equilibrium size decreases with the predator reproduction number. Furthermore, both the predator equilibrium size and the prey equilibrium size increase with the prey intrinsic growth rate $r - \mu_0$. The increase of predator equilibrium size with the predator reproduction number $\mathcal{R}_p$ is saturating, where the saturation limit is directly proportional to the prey's growth rate and inversely proportional to predator's attack rate.

3.1. **Boundary equilibria.** We investigate the equilibria of the system (3.1). The system has the extinction equilibrium $\mathcal{E}^0 = (0, 0, 0, 0)$ which is globally stable if $r < \mu_0$. Assuming that $r > \mu_0$ gives unstable extinction equilibrium and the system (3.1) has several disease-related equilibria. In the remainder of this section we will consider the case when $r > \mu_0$. The system has disease-free and predator-free equilibrium in which the prey population size is at carrying capacity in the absence of a predator: $\mathcal{E}_p^0 = (K_P^\circ, 0, 0, 0)$. Furthermore, the system has a disease-free, predator and susceptible prey equilibrium $\hat{\mathcal{E}}^0 = (\hat{S}, 0, 0, \hat{P})$, where $\hat{S}$ and $\hat{P}$ are as given in equations (3.4). The predator reproduction number is given by expression (3.3). This predator reproduction number is the predator reproduction number when the entire prey population consists of susceptible individuals. There are two dominance equilibria that correspond to strain one — one in the absence of predation, and another in the presence of predation. The strain one dominance equilibrium in the absence of predation $P = 0$ is given by $\mathcal{E}_{P,1} = \left(\frac{\mu_1}{\beta_1}, \frac{r}{\beta_1 K + r}\left(K_P^\circ - \frac{\mu_1}{\beta_1}\right), 0, 0\right)$. The strain one dominance equilibrium in the absence of predation exists if the reproduction number of strain one in the absence of predation $\mathcal{R}_1^\circ$ is larger than one: $\mathcal{R}_1^\circ > 1$. The reproduction number of strain one in the absence of predation is given by the following expression

$$\mathcal{R}_1^\circ = \frac{K_P^\circ \beta_1}{\mu_1}.$$

The strain one dominance equilibrium in the presence of predation $\hat{\mathcal{E}}_1 = (S_1^*, I_1^*, 0, P_1^*)$ satisfies the following system, obtained after canceling S in the first equation, I_1 in the second equation, and P in the third equation:

$$\begin{aligned} 0 &= r\left(1 - \frac{S + I_1}{K}\right) - \beta_1 I_1 - (\mu_0 + \eta P), \\ 0 &= \beta_1 S - (\mu_1 + \gamma_1 P), \\ 0 &= \epsilon(\eta S + \gamma_1 I_1) - d. \end{aligned} \tag{3.5}$$

From the second equation we can express S as a function of P, and from the first equation of system (3.5) we can express I_1 as a function if P:

$$S_1^* = \frac{\mu_1 + \gamma_1 P}{\beta_1} \qquad I_1^* = \frac{r}{\beta_1 K + r}\left(K_P^\circ - \frac{\mu_1}{\beta_1} - \left(\frac{K\eta}{r} + \frac{\gamma_1}{\beta_1}\right) P\right).$$

Substituting these in the last equation we obtain an equation for P only. Solving that we obtain:

$$P_1^* = \frac{d\beta_1}{\epsilon\gamma_1(\gamma_1 - \eta)}\left(\frac{K}{r}\beta_1 + 1\right)(\mathcal{R}_{p,1} - 1)$$

where

$$\mathcal{R}_{p,1} = \frac{\epsilon\eta}{d}\frac{\mu_1}{\beta_1} + \frac{\epsilon\gamma_1}{d}\frac{r}{\beta_1 K + r}\left(K_P^\circ - \frac{\mu_1}{\beta_1}\right)$$

We can interpret $\mathcal{R}_{p,1}$ as predator's reproduction number when the prey population consists of susceptible and infected with strain one individuals. The predator reproduction number gives the number of predators that will be produced in a population where there are $\frac{\mu_1}{\beta_1}$ susceptible prey and $\frac{r}{\beta_1 K+r}\left(K_P^\circ - \frac{\mu_1}{\beta_1}\right)$ infected with strain one prey. Consequently, $\eta\frac{\mu_1}{\beta_1}$ gives the number of susceptible prey killed and eaten by one predator per unit of time. In addition $\gamma_1\frac{r}{\beta_1 K+r}\left(K_P^\circ - \frac{\mu_1}{\beta_1}\right)$ gives the number of infected with strain one prey killed and eaten by one predator per unit of time. Furthermore, $\epsilon\left(\eta\frac{\mu_1}{\beta_1} + \gamma_1\frac{r}{\beta_1 K+r}\left(K_P^\circ - \frac{\mu_1}{\beta_1}\right)\right)$ gives the number of prey killed and eaten by one predator, and converted into new predator biomass, per unit of time. Finally, $1/d$ is the lifespan of the predator.

The predator numbers P_1^* in the dominance equilibrium of strain one $\hat{\mathcal{E}}_1$ is positive in the following cases:

- If the predator predates more intensively on individuals infected with strain one as compared to susceptible individuals, that is, if $\gamma_1 > \eta$, then the reproduction number of the predator in infected with strain one prey population must be larger than one: $\mathcal{R}_{p,1} > 1$.
- If the predator predates more intensively on susceptible individuals as compared to those infected with strain one, that is, if $\gamma_1 < \eta$, then the reproduction number of the predator in infected with strain one prey population must be smaller than one: $\mathcal{R}_{p,1} < 1$.

The number of infected with strain one individuals is positive ($I_1^* > 0$) if the reproduction number of strain one is larger than one: $\mathcal{R}_1 > 1$, where the reproduction number of strain one is defined as follows:

$$\mathcal{R}_1 = \frac{K_P^\circ\beta_1}{\mu_1 + \left(\frac{K\beta_1}{r}\eta + \gamma_1\right)P_1^*}.$$

By symmetry, there are two dominance equilibria that correspond to strain two — one in the absence of predation, and another in the presence of predation. The strain two dominance equilibrium in the absence of predation, $P = 0$, is given by $\mathcal{E}_{P,2} = \left(\frac{\mu_2}{\beta_2}, 0, \frac{r}{\beta_2 K+r}\left(K_P^\circ - \frac{\mu_2}{\beta_2}\right), 0\right)$. This equilibrium exists if the reproduction number of strain two in the absence of predation, given by

$$\mathcal{R}_2^\circ = \frac{K_P^\circ\beta_2}{\mu_2},$$

is larger than one: $\mathcal{R}_2^\circ > 1$. The strain two dominance equilibrium in the presence of predation is given by $\hat{\mathcal{E}}_2 = (S_2^*, 0, I_2^*, P_2^*)$ where

$$S_2^* = \frac{\mu_2 + \gamma_2 P_2^*}{\beta_2} \qquad I_2^* = \frac{r}{\beta_2 K + r}\left(K_P^\circ - \frac{\mu_2}{\beta_2} - \left(\frac{K\eta}{r} + \frac{\gamma_2}{\beta_2}\right)P_2^*\right).$$

The value of P_2^* is given by

$$P_2^* = \frac{d\beta_2}{\epsilon\gamma_2(\gamma_2 - \eta)}\left(\frac{K}{r}\beta_2 + 1\right)(\mathcal{R}_{p,2} - 1)$$

where

$$\mathcal{R}_{p,2} = \frac{\epsilon\eta}{d}\frac{\mu_2}{\beta_2} + \frac{\epsilon\gamma_2}{d}\frac{r}{\beta_2 K + r}\left(K_P^\circ - \frac{\mu_2}{\beta_2}\right)$$

As before, we can interpret $\mathcal{R}_{p,2}$ as predator's reproduction number when the prey population consists of susceptible and infected with strain two individuals.

The number of infected with strain two individuals is positive ($I_2^* > 0$) if the reproduction number of strain two is larger than one: $\mathcal{R}_2 > 1$, where the reproduction number of strain two is defined as follows:

$$\mathcal{R}_2 = \frac{K_P^\circ \beta_2}{\mu_2 + \left(\frac{K\beta_2}{r}\eta + \gamma_2\right) P_2^*}.$$

3.2. **Coexistence equilibria.** In this subsection, we investigate the coexistence equilibria. Those must satisfy the system which we obtain from (3.1) by setting the derivatives equal to zero and canceling S in the first equation, I_1 in the second equation, I_2 in the third equation, and P in the fourth equation:

$$\begin{aligned} 0 &= r\left(1 - \frac{S + I_1 + I_2}{K}\right) - \beta_1 I_1 - \beta_2 I_2 - (\mu_0 + \eta P), \\ 0 &= \beta_1 S - (\mu_1 + \gamma_1 P) \\ 0 &= \beta_2 S - (\mu_2 + \gamma_2 P) \\ 0 &= \epsilon(\eta S + \gamma_1 I_1 + \gamma_2 I_2) - d \end{aligned} \tag{3.6}$$

We note that the last equation with $\eta = 0$ implies that infected with strain one and infected with strain two prey individuals are in apparent competition mediated by the predator. Apparent competition means that if the equilibrial numbers of one of the two infected prey classes increases, then necessarily, the equilibrial numbers of the other infected prey class decrease. The term apparent competition was introduced by Holt [9]. The community context of the entire model (with $\eta \neq 0$) is equivalent to intra-guild predation [12]. Intra-guild predation (IGP) occurs when the top predator which predates on a prey species (in our case infected individuals) can also exploit the resource of its prey (in our case the resource of the infected individuals are susceptible individuals, and the predator predates on both infected and susceptible individuals).

We can solve the second and third equation for S and P:

$$\hat{P} = \frac{\beta_2\mu_1 - \beta_1\mu_2}{\beta_1\gamma_2 - \beta_2\gamma_1} \qquad \hat{S} = \frac{\mu_1 + \gamma_1\hat{P}}{\beta_1} = \frac{\mu_1\gamma_2 - \mu_2\gamma_1}{\beta_1\gamma_2 - \beta_2\gamma_1} \tag{3.7}$$

Proposition 2.4 gives the parameter values which determine the positivity of the coexistence value of the predator $\hat{P} > 0$. The corresponding value of the susceptibles is always positive, $\hat{S} > 0$, as long as $\hat{P}$ is positive. Solving the first and the last equation in the system (3.6) gives the values of the infected individuals with strain one and strain two.

$$\begin{aligned} \hat{I}_1 &= \frac{\left(\frac{r}{K} + \beta_2\right)\left(1 - \frac{\epsilon\eta S}{d}\right) - \frac{\epsilon\gamma_2}{d}\left[r\left(1 - \frac{S}{K}\right) - (\mu_0 + \eta P)\right]}{\frac{\epsilon\gamma_1}{d}\left(\frac{r}{K} + \beta_2\right) - \frac{\epsilon\gamma_2}{d}\left(\frac{r}{K} + \beta_1\right)} \\ \hat{I}_2 &= \frac{\frac{\epsilon\gamma_1}{d}\left[r\left(1 - \frac{S}{K}\right) - (\mu_0 + \eta P)\right] - \left(\frac{r}{K} + \beta_1\right)\left(1 - \frac{\epsilon\eta S}{d}\right)}{\frac{\epsilon\gamma_1}{d}\left(\frac{r}{K} + \beta_2\right) - \frac{\epsilon\gamma_2}{d}\left(\frac{r}{K} + \beta_1\right)} \end{aligned} \tag{3.8}$$

To investigate the positivity of the coexistence equilibrium, we introduce the invasion reproduction numbers of the two strains. The invasion reproduction number of strain

one when strain two is at equilibrium is defined as the number of secondary cases one strain-one infected individual can produce in a population where strain two is at equilibrium during its lifetime as infectious. The invasion reproduction number of strain one measures the invasion capabilities of strain one. This number is defined as follows:

$$\hat{\mathcal{R}}_1 = \frac{\beta_1 S_2^*}{\mu_1 + \gamma_1 P_2^*} = \frac{\beta_1(\mu_2 + \gamma_2 P_2^*)}{\beta_2(\mu_1 + \gamma_1 P_2^*)}.$$

The inequality $\hat{\mathcal{R}}_1 > 1$ says that strain one can invade the equilibrium of strain two, while the opposite inequality says that strain one cannot invade the equilibrium of strain two. It is easy to see from this expression that if $\gamma_2/\gamma_1 > \beta_2/\beta_1$ then the inequality $\hat{\mathcal{R}}_1 > 1$ is equivalent to the inequality $P_2^* > \hat{P}$ while if $\gamma_2/\gamma_1 < \beta_2/\beta_1$ then the inequality $\hat{\mathcal{R}}_1 > 1$ is equivalent to the inequality $P_2^* < \hat{P}$ (see equation (3.7)). In words, if the ratio of the predation rate of strain two-infected prey to strain one-infected prey is larger than the ratio of the transmission rates of strain two and strain one, then strain one can invade the equilibrium of strain two if and only if the predation level in the absence of strain one is higher than the predation level in presence of infectious individuals with both strains. If the relationship between the ratios is reversed then strain one can invade the equilibrium of strain two if and only if the predation level in the absence of strain one is lower than the predation level in presence of infectious individuals with both strains. The invasion capabilities of strain one increase with the predation level in an exclusive strain two equilibrium (P_2^*) if and only if $\gamma_2/\gamma_1 > \mu_2/\mu_1$. Similarly, we can define an invasion reproduction number of strain two:

$$\hat{\mathcal{R}}_2 = \frac{\beta_2 S_1^*}{\mu_2 + \gamma_2 P_1^*} = \frac{\beta_2(\mu_1 + \gamma_1 P_1^*)}{\beta_1(\mu_2 + \gamma_2 P_1^*)}.$$

As before, if $\beta_2/\beta_1 > \gamma_2/\gamma_1$, then the inequality $\hat{\mathcal{R}}_2 > 1$ is equivalent to the inequality $P_1^* > \hat{P}$ and if $\beta_2/\beta_1 < \gamma_2/\gamma_1$, then the inequality $\hat{\mathcal{R}}_2 > 1$ is equivalent to the inequality $P_1^* < \hat{P}$. The invasion capabilities of strain two increase with the predation level in an exclusive strain one equilibrium (P_1^*) if and only if $\gamma_2/\gamma_1 < \mu_2/\mu_1$. We conclude that all other things fixed predation levels act in opposing ways on the invasion capabilities of the two strains.

For a coexistence equilibrium to exist, we need that $\hat{I}_1 > 0$ and $\hat{I}_2 > 0$. There are two symmetric cases.

Case 1: Assume that the denominator of the expressions in (3.8) is positive:

$$\beta_2 > \frac{r}{K}\left(\frac{\gamma_2}{\gamma_1} - 1\right) + \frac{\gamma_2}{\gamma_1}\beta_1 \tag{3.9}$$

Case 2: Assume that the denominator of the expressions in (3.8) is negative:

$$\beta_2 < \frac{r}{K}\left(\frac{\gamma_2}{\gamma_1} - 1\right) + \frac{\gamma_2}{\gamma_1}\beta_1 \tag{3.10}$$

We consider more thoroughly Case 1. Case 2 is analogous. If the denominator of $\hat{I}_1$ and $\hat{I}_2$ is positive, then $\hat{I}_1$ and $\hat{I}_2$ will be both positive if their numerators are positive. Consider the numerator of $\hat{I}_1$. We express $\hat{S}$ as $(\mu_2 + \gamma_2\hat{P})/\beta_2$ and replace it in the numerator of $\hat{I}_1$. Separating the terms containing $\hat{P}$ from those that do not contain $\hat{P}$,

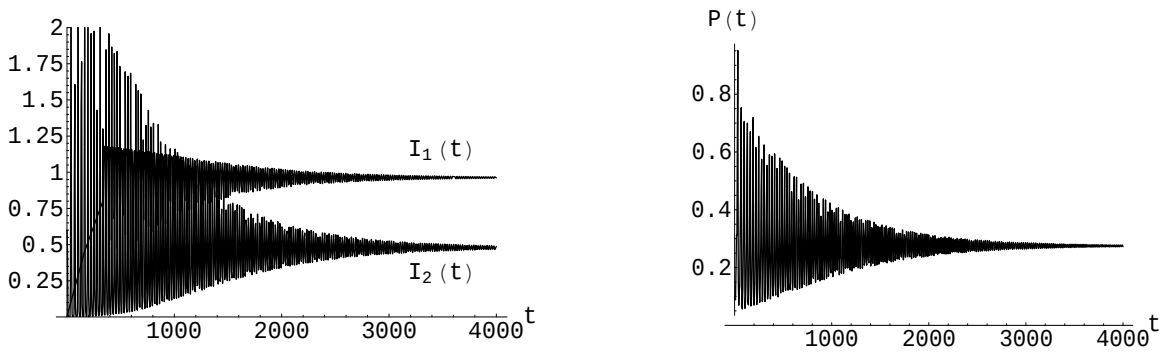

FIGURE 3. The left figure illustrates that the number infected prey with strain one $I_1(t)$ and the number infected prey with strain two $I_2(t)$ may tend toward a coexistence equilibrium. The right figure illustrates that the predator numbers of a specialist predator stabilize toward nonzero equilibrium. The parameter values for both figures are: $\beta_1 = 7$, $\beta_2 = 3$, $\eta = 2$, $\gamma_1 = 9$, $\gamma_2 = 1$, $\mu_0 = 0.1$, $\mu_1 = 0.5$, $\mu_2 = 1$, $d = 1$, $r = 9$, $K = 100$, $\epsilon = 0.1$. The predator's reproduction numbers for strain one and strain two are correspondingly $\mathcal{R}_{p,1} = 1.143$ and $\mathcal{R}_{p,2} = 0.3537$. The two invasion reproduction numbers of strain one and strain two are respectively $\hat{\mathcal{R}}_1 = 0.2596$, $\hat{\mathcal{R}}_2 = 3.588$. Since $\beta_2/\beta_1 = 0.429$, while $\gamma_2/\gamma_1 = 1/9$ and is smaller, then $\hat{\mathcal{R}}_1 < 1$ is equivalent to $\hat{P} < P_2^*$, and $\hat{\mathcal{R}}_2 > 1$ is equivalent to $\hat{P} < P_1^*$. We are in case (1) second scenario of Proposition 3.1.

we obtain the following expression for the numerator of $\hat{I}_1$:

$$\frac{r + \beta_2 K}{K}\left[1 - \mathcal{R}_{p,2} + \frac{r}{r + \beta_2 K}\frac{\epsilon\gamma_2}{d\beta_2}(\gamma_2 - \eta)\hat{P}\right].$$

Thus, if $\gamma_2 > \eta$, the inequality $\hat{I}_1 > 0$ is equivalent to the inequality $\hat{P} > P_2^*$. It also follows that, if $\gamma_2 < \eta$, the inequality $\hat{I}_1 > 0$ is equivalent to the inequality $\hat{P} < P_2^*$. Because the numerator of $\hat{I}_2$ is the same but multiplied by negative one, we will get that if $\gamma_1 > \eta$, the inequality $\hat{I}_2 > 0$ is equivalent to the inequality $\hat{P} < P_1^*$. We will also get that, if $\gamma_1 < \eta$, the inequality $\hat{I}_2 > 0$ is equivalent to the inequality $\hat{P} > P_1^*$. We summarize the coexistence result in the following proposition.

Proposition 3.1. *Assume $r > \mu_0$ and that the inequality in Proposition 2.4 is satisfied. Consider the following two cases:*

(1) *Assume*

$$\beta_2 > \frac{r}{K}\left(\frac{\gamma_2}{\gamma_1} - 1\right) + \frac{\gamma_2}{\gamma_1}\beta_1.$$

Then we have the following scenarios:

- *$\gamma_1 > \eta$ and $\gamma_2 > \eta$. A unique coexistence equilibrium exists if and only if $\mathcal{R}_{p,1} > 1$, $\mathcal{R}_{p,2} > 1$ and $\hat{P} < P_1^*$ and $\hat{P} > P_2^*$.*
- *$\gamma_1 > \eta$ and $\gamma_2 < \eta$. A unique coexistence equilibrium exists if and only if $\mathcal{R}_{p,1} > 1$, $\mathcal{R}_{p,2} < 1$ and $\hat{P} < P_1^*$ and $\hat{P} < P_2^*$.*
- *$\gamma_1 < \eta$ and $\gamma_2 > \eta$. A unique coexistence equilibrium exists if and only if $\mathcal{R}_{p,1} < 1$, $\mathcal{R}_{p,2} > 1$ and $\hat{P} > P_1^*$ and $\hat{P} > P_2^*$.*

- $\gamma_1 < \eta$ *and* $\gamma_2 < \eta$. *A unique coexistence equilibrium exists if and only if* $\mathcal{R}_{p,1} < 1$, $\mathcal{R}_{p,2} < 1$ *and* $\hat{P} > P_1^*$ *and* $\hat{P} < P_2^*$.

(2) *Assume*

$$\beta_2 < \frac{r}{K}\left(\frac{\gamma_2}{\gamma_1} - 1\right) + \frac{\gamma_2}{\gamma_1}\beta_1.$$

Then we have the following scenarios:

- $\gamma_1 > \eta$ *and* $\gamma_2 > \eta$. *A unique coexistence equilibrium exists if and only if* $\mathcal{R}_{p,1} > 1$, $\mathcal{R}_{p,2} > 1$ *and* $\hat{P} > P_1^*$ *and* $\hat{P} < P_2^*$.
- $\gamma_1 > \eta$ *and* $\gamma_2 < \eta$. *A unique coexistence equilibrium exists if and only if* $\mathcal{R}_{p,1} > 1$, $\mathcal{R}_{p,2} < 1$ *and* $\hat{P} > P_1^*$ *and* $\hat{P} > P_2^*$.
- $\gamma_1 < \eta$ *and* $\gamma_2 > \eta$. *A unique coexistence equilibrium exists if and only if* $\mathcal{R}_{p,1} < 1$, $\mathcal{R}_{p,2} > 1$ *and* $\hat{P} < P_1^*$ *and* $\hat{P} < P_2^*$.
- $\gamma_1 < \eta$ *and* $\gamma_2 < \eta$. *A unique coexistence equilibrium exists if and only if* $\mathcal{R}_{p,1} < 1$, $\mathcal{R}_{p,2} < 1$ *and* $\hat{P} < P_1^*$ *and* $\hat{P} > P_2^*$.

Not all scenarios in Proposition 3.1 lead to stable coexistence. However, stable coexistence of the two strains and the predator occurs and we illustrate that in Figure 3. Consequently, in contrast to a generalist predator, specialist predators can lead to sustained coexistence of the two strains in the prey. This coexistence is robust under minor modifications of the parameters, and does not require special trivial values of the reproduction numbers. Both generalist and specialist predators impact the two competing pathogen strains in the prey population. Generalist predators, in particular, can change the amount of disease in the prey population, and even change the competitive advantage of the two competing strains. However, the generalist predator itself is not influenced by the prey and by the distribution of the disease strains in the prey. Consequently, there is no feedback mechanism to impact the predator. The case of a specialist predator is quite different. Specialist predator's numbers are strongly influenced by the prey's numbers and dynamically adapt to those. The distribution of the disease, and the strains in the prey, have full impact on the predator. Therefore, even though only one of the strains dominates in the absence of predation because it has a larger reproduction number, in the presence of a specialist predator, which prefers the prey individuals infected with that dominant strain, the reproductive capabilities of that dominant strain are reduced and a balance between the strain which has the competitive advantage and the strain that doesn't is created. This leads to stable coexistence.

A particularly interesting aspect is that, in the case when the predator brings about the coexistence of the two pathogens, the predator's equilibrial levels are completely independent of its intrinsic characteristics, such as predator's mortality d or conversion efficiency ϵ. In addition, the predator's equilibrial levels at the coexistence of the two pathogens do not explicitly depend on the predation rate of susceptible individuals η. This means that, if all parameters pertaining to the prey and the disease are fixed, predator's equilibrial levels can be changed only if the predator's predation rates on prey individuals infected with strain one (γ_1) or strain two (γ_2) change appropriately. We saw that increasing levels of a generalist predator decrease the equilibrial disease load of strain one or strain two, and thus the overall disease load. In the case of a specialist predator, we also see that increasing the predator's equilibrial levels decreases the number of infected prey with strain one (or strain two) when strain one (or strain two) is dominant. Simulations suggest that when the two strains coexist, increasing

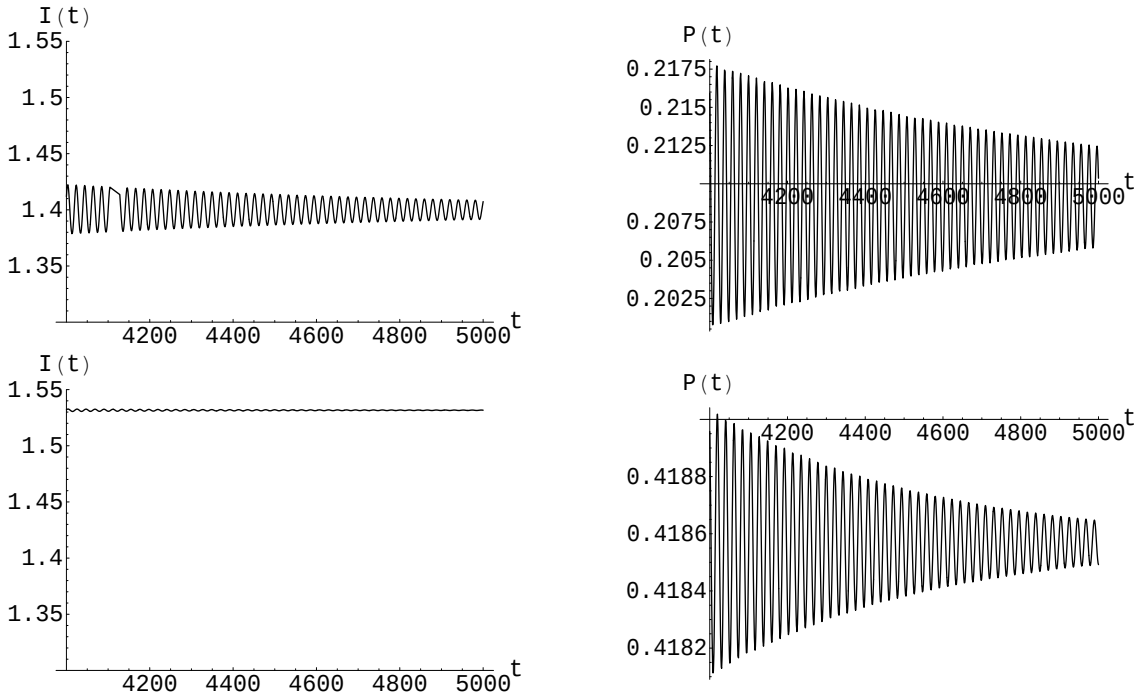

FIGURE 4. The left column of figures illustrates that the equilibrial total number of infected prey $I(t) = I_1(t) + I_2(t)$. The right column of figures illustrates the equilibrial predator numbers of a specialist predator. The upper row of figures gives the equilibrial total number of infected prey $I(t) = I_1(t) + I_2(t)$ and the equilibrial predator numbers for $\gamma_2 = 0.1$. The lower row of figures gives the equilibrial total number of infected prey $I(t) = I_1(t) + I_2(t)$ and the equilibrial predator numbers for $\gamma_2 = 1.98$. One can see that increasing γ_2 increases the equilibrial predator level from 0.21 to 0.41 and also increases the equilibrial total number of infected prey $I(t) = I_1(t) + I_2(t)$ from 1.4 to about 1.53. The remaining parameters of the figure are $\beta_1 = 7$, $\beta_2 = 3$, $\eta = 2$, $\gamma_1 = 9$, $\mu_0 = 0.1$, $\mu_1 = 0.5$, $\mu_2 = 1$, $d = 1$, $r = 9$, $K = 100$, $\epsilon = 0.1$.

predation level typically decreases the equilibrial disease load of one of the strains but increases the equilibrial disease load of the other strain. This reciprocal impact is not completely compensatory. Consequently, the impact of increasing specialist predator's levels on the total number of infected prey $I_1 + I_2$ may be negative as before leading to decrease in the total disease load, or positive, leading to *increase* in the total disease load. We illustrate this latter situation in Figure 4.

There is a simple but naive explanation of the increase in the total number of infected prey with increase of predation levels. Assume the equilibrial total prey number N is constant and does not depend of predation rate γ_2. Observe that the equilibrial number of susceptible prey, just like the predator equilibrial numbers, does not depend on the intrinsic parameters of the susceptible class, such as natural mortality of the prey μ_0 and predation rate on susceptible prey η. The equilibrial number of susceptible prey $\hat{S}$, however, depends on the predation rates of infected prey with strain one, γ_1, and strain two, γ_2. The total equilibrial number of infected prey $\hat{I}_1 + \hat{I}_2 = N - \hat{S}$. If the

equilibrial number of susceptible prey $\hat{S}$ decreases with γ_2 (while $\hat{P}$ increases), then the total equilibrial number of infected prey will increase. This happens if $\mu_2/\mu_1 > \beta_2/\beta_1$ and it is exactly the scenario illustrated in Figure 4. This explanation is naive because the total prey size N is not constant but possibly depends on γ_2 in a complex way.

4. Non-linear functional response

In Section 2 and Section 3 we assume that the predator's functional response is linear, that is we assume functional response of type I. A natural question to be addressed is: Would the results hold if the predator's functional response is of type II or type III? With this question in mind we consider the following generalization of model (2.1) modeling the predation of a generalist predator:

$$\begin{aligned}
\frac{dS}{dt} &= rS\left(1-\frac{S+I_1+I_2}{K}\right)-\beta_1 SI_1-\beta_2 SI_2-\mu_0 S-\frac{\eta P S^\alpha}{aS^\alpha+1},\\
\frac{dI_1}{dt} &= \beta_1 SI_1-\mu_1 I_1-\frac{\gamma_1 P I_1^\alpha}{\xi_1 I_1^\alpha+1},\\
\frac{dI_2}{dt} &= \beta_2 SI_2-\mu_2 I_2-\frac{\gamma_2 P I_2^\alpha}{\xi_2 I_2^\alpha+1}
\end{aligned} \tag{4.1}$$

where a, ξ_1, and ξ_2 are parameters associated with the functional response. The parameter $\alpha \geq 1$ determines the type of the predator's functional response. If $\alpha = 1$, and $\xi_1 \neq 0$, $\xi_2 \neq 0$ then the functional response is Holling's type II. If $\alpha = 1$, and $\xi_1 = 0$, $\xi_2 = 0$ then the functional response is Holling's type I. We obtain model (2.1) in this case. If $\alpha > 1$, then the functional response is Holling's type III, and has sigmoidal shape.

We established that in contrast to model (2.1), model (4.1) has coexistence equilibria for nontrivial values of the reproduction numbers, that is even if the reproduction numbers of the two strains are different. The analysis of the general case is somewhat technical. To support our claim, we will consider a simpler, although perhaps not very realistic, version of the model above. We assume $a = 0$ and, say, $\xi_1 = 0$. In addition, we consider the case $\alpha = 1$. This simplified version allows for explicit computation of the coexistence equilibrium. In this case the model (4.1) has the same disease-free equilibrium as model (2.1), namely $\mathcal{E}_0 = (S^0, 0, 0)$ with $S^0 = K_P$. The reproduction numbers of the two strains, as before, are given by

$$\mathcal{R}_i = \frac{K_P \beta_i}{\mu_i + \gamma_i P} \qquad\qquad i = 1, 2.$$

Strain one equilibrium is $\mathcal{E}_1 = (S_1^*, I_1^*, 0)$ where the components are given explicitly by the same expressions as in model (2.1). Strain two equilibrium, however, is different. It is given by the ordered triple $\mathcal{E}_2 = (S_2^*, 0, I_2^*)$ where the components S_2^* and I_2^* are solutions of the following system:

$$\begin{aligned}
0 &= r\left(1-\frac{S+I_2}{K}\right)-\beta_2 I_2-\mu_0 S-\eta P,\\
0 &= \beta_2 S-\mu_2-\frac{\gamma_2 P}{\xi_2 I_2+1}
\end{aligned} \tag{4.2}$$

From this system I_2 is given by the following expression that depends on S:

$$I_2 = \frac{\gamma_2 P + \mu_2 - \beta_2 S}{\xi_2(\beta_2 S - \mu_2)}.$$

Replacing I_2 in the first equation in (4.2) we obtain that S_2^* is a solution of the equation:

$$r\left(1 - \frac{S}{K}\right) - \mu_0 S - \eta P = \left(\beta_2 + \frac{r}{K}\right)\frac{\gamma_2 P + \mu_2 - \beta_2 S}{\xi_2(\beta_2 S - \mu_2)}.$$

Let $f(S)$ denote the left-hand side of the above equation, considered as a function of S, while $g(S)$ denote the right-hand side. Both functions are decreasing when positive. We have that $f(S)$ is linear decreasing with $f(S^0) = 0$. On the other hand $g(S) \to \infty$ as $S \to \frac{\mu_2}{\beta_2}^-$, and $f((\gamma_2 P + \mu_2)/\beta_2) = 0$. Thus, if $\mathcal{R}_2 > 1$, the equation above has at least one solution, which gives S_2^*.

Coexistence equilibria, if they exist, satisfy the system:

$$\begin{aligned} 0 &= r\left(1 - \frac{S + I_2}{K}\right) - \beta_2 I_2 - \mu_0 S - \eta P, \\ 0 &= \beta_1 S - \mu_1 - \gamma_1 P \\ 0 &= \beta_2 S - \mu_2 - \frac{\gamma_2 P}{\xi_2 I_2 + 1} \end{aligned} \tag{4.3}$$

The presence or absence of coexistence equilibria depends on the invasion reproduction numbers. As in Section 3, these measure the ability of each strain to invade the equilibrium of the other strain. The invasion reproduction number of strain one at the equilibrium of strain two depends on the predation level, P, and is given by:

$$\hat{\mathcal{R}}_1 = \frac{\beta_1 S_2^*}{\gamma_1 P + \mu_1}.$$

The invasion reproduction number of strain two at the equilibrium of strain one also depends on the predation level, P, and is given by:

$$\hat{\mathcal{R}}_2 = \frac{\beta_2 S_1^*}{\gamma_2 P + \mu_2} = \frac{\beta_2(\gamma_1 P + \mu_1)}{(\gamma_2 P + \mu_2)\beta_1}.$$

Solving the system (4.3) we obtain a unique coexistence equilibrium $\mathcal{E}^* = (S^{**}, I_1^{**}, I_2^{**})$ whose components are given by:

$$\begin{aligned} S^{**} &= \frac{\gamma_1 P + \mu_1}{\beta_1} \\ I_2^{**} &= \frac{(\gamma_2 P + \mu_2)(1 - \hat{\mathcal{R}}_2)}{\xi_2(\beta_2 S^{**} - \mu_2)} \\ I_1^{**} &= \frac{r\left(1 - \frac{S^{**}}{K}\right) - \mu_0 - \eta P - \left(\beta_2 + \frac{r}{K}\right) I_2^{**}}{\left(\beta_1 + \frac{r}{K}\right)} \end{aligned} \tag{4.4}$$

The expression for I_2^{**} is positive if and only if $\hat{\mathcal{R}}_2 < 1$. It is easy to see that if ξ_2 is taken large enough, the expression for I_1^{**} is also positive. Thus, the region in parameter space where the coexistence equilibrium exists is nontrivial.

We established rigorously that in the general case, as in the case above, coexistence equilibrium exists if and only if at least one of the invasion reproduction numbers is smaller than one. From simulations it seems that both invasion reproduction numbers are smaller than one when a viable coexistence equilibrium exists. Simulations also

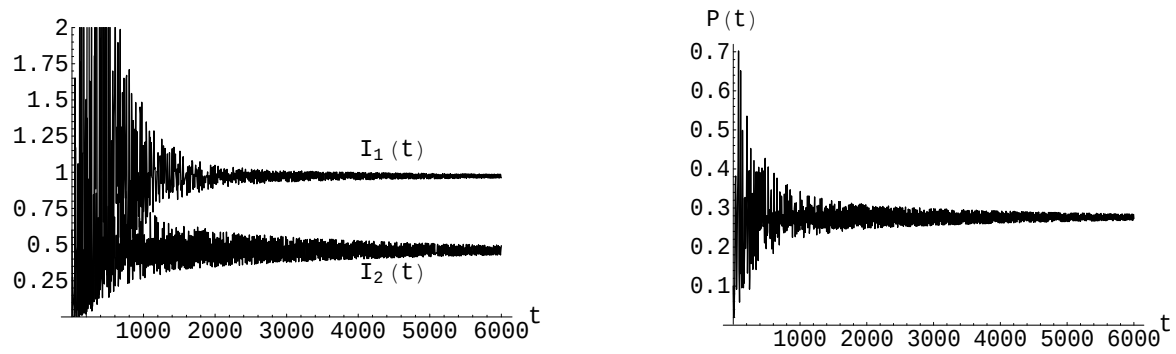

FIGURE 5. The left figure illustrates that the number infected prey with strain one $I_1(t)$ and the number infected prey with strain two $I_2(t)$ may tend toward a coexistence equilibrium. The right figure illustrates that the predator numbers of a specialist predator stabilize toward nonzero equilibrium. The parameter values for both figures are: $\beta_1 = 7$, $\beta_2 = 3$, $\eta = 2$, $\gamma_1 = 9$, $\gamma_2 = 1$, $\mu_0 = 0.1$, $\mu_1 = 0.5$, $\mu_2 = 1$, $d = 1$, $r = 9$, $K = 100$, $\epsilon = 0.1$. In addition, the parameters for the functional response are $a = 0.09$, $\xi_1 = 0.005$, and $\xi_2 = 0.01$, $\alpha = 1$.

suggest that the resulting coexistence equilibrium in this case is not stable. Thus, dynamical outcome of the competition is competitive exclusion. Further investigations on this model should focus on the question whether a coexistence equilibrium exists when one of the invasion reproduction numbers is greater than one, and whether this equilibrium is stable. Extensive simulations so far, however, seem to suggest that stable coexistence is at best difficult to attain.

Generalizing the model (3.1) to include nonlinear functional response, we obtain the system:

$$
\begin{aligned}
\frac{dS}{dt} &= rS\left(1 - \frac{S + I_1 + I_2}{K}\right) - \beta_1 S I_1 - \beta_2 S I_2 - \mu_0 S + \frac{\eta P S^\alpha}{aS^\alpha + 1} \\
\frac{dI_1}{dt} &= \beta_1 S I_1 - \mu_1 I_1 + \frac{\gamma_1 P I_1^\alpha}{\xi_1 I_1^\alpha + 1}, \\
\frac{dI_2}{dt} &= \beta_2 S I_2 - \mu_2 I_2 + \frac{\gamma_2 P I_2^\alpha}{\xi_2 I_2^\alpha + 1}, \\
\frac{dP}{dt} &= \epsilon\left(\frac{\eta S^\alpha}{aS^\alpha + 1} + \frac{\gamma_1 I_1^\alpha}{\xi_1 I_1^\alpha + 1} + \frac{\gamma_2 I_2^\alpha}{\xi_2 I_2^\alpha + 1}\right) P - dP,
\end{aligned}
\tag{4.5}
$$

Simulations confirm that this model, just as its counterpart with linear response (3.1), supports coexistence of the two strains. Results from the simulations are presented in Figure 5. We want to note that simulations with this model also suggested that coexistence of the two strains in the form of sustained oscillations, as well as chaos, are some of the complex behaviors that this model seems to exhibit. Further investigations of the model is necessary to understand more completely its complexity, but it is beyond the scope of this article.

5. Summary of results

This paper considers two types of models — one with a generalist predator predating discriminantly, and one with a specialist predator, also predating discriminantly. The population numbers of a generalist predator, which feeds on many types of prey, are not strongly affected by the dynamics of one specific type of prey. We model this type of predation on a focal prey species as a parameter, which potentially increases the mortality of the prey. The functional response of the predator for the baseline model is linear. We are interested how predation of a generalist predator impacts the competition of two strains in the prey population infected with a microparasite. To study this effect, we compute the relevant equilibria and reproduction numbers of the strains. We made the following observations:

(1) Increasing levels of predation decrease the total prey population size. More interestingly, increasing predation levels decrease the reproduction number of the disease and overall disease load, thus facilitating eradication of the disease without necessarily leading to extinction of the prey.
(2) Competitive exclusion of the two strains is the predominant outcome. Selection again favors the strain with the higher reproduction number. However, predation levels impact the reproduction numbers of the two strains differently and can *switch* the competitive advantage between the two strains. In particular, if at low predation levels the first strain has a higher reproduction number and will exclude the second strain, it is possible that at higher predation level the second strain has higher reproduction number and will dominate in the prey population. Thus which strain is prevalent depends on the amount of predation pressure exerted by the predator on the prey. Why is this important and why it can occur in nature? Many generalist predators have preferred prey as a food source but feed on a number of other types of prey. If our focal species is one of the side food sources for the predator, it does not affect the dynamics of the predator. However, the preferred food source for the predator may impact predators numbers by increasing or decreasing them. As we show this increase or decrease can cause a switch in the pathogen causing the disease in our focal species. In this case, coexistence of the two strains is possible only in the non-generic case of equality of the reproduction numbers of the two strains.

In contrast a specialist predator feeds exclusively on a particular species of prey. The population dynamics of the prey impacts tremendously the population dynamics of the predator. It is this type of predation that is traditionally modeled with Lotka-Volterra type predator-prey models. We model the impact of a specialist predator on the competition of disease strains in the prey by structuring the total prey population in a Lotka-Volterra predator-prey model with linear functional response into susceptible, infected with strain one and infected with strain two individuals. We computed the relevant equilibria. Equilibria depend on a number of threshold parameters: the reproduction numbers of the two strains, the reproduction number of the predator, the reproduction number of the predator at the equilibrium of strain one or at the equilibrium of strain two, as well as the invasion reproduction numbers of strain one and strain two. We made the following observations:

(1) If the disease imparts differential virulence on the prey population, and the specialist predator predates discriminantly on the various prey classes generated by the disease, a variety of dynamical outcomes are possible. If one of the strains dominates in the population in the presence of the predator (a scenario that occurs when both the reproduction number of the strain and the reproduction number of the predator in the presence of the strain are above one), then increasing predation levels decrease the reproduction number of the corresponding strain as well as the disease load.
(2) In contrast of a generalist predator, however, specialist predator may lead to coexistence of the two strains which occurs in non-generic cases (that is, even when the reproduction numbers of the two strains are not the same). This happens because the prey's numbers, and particularly the number of susceptible prey, exert a feedback control on predator's equilibrial numbers, adapting those to appropriate level that mediates the coexistence of the strains. This result applies both for linear and non-linear response of the predator.
(3) Discriminate predation mediates coexistence of pathogen strains in a prey population because appropriate attack rates and predation levels may counteract the intrinsic vital differences in the strains. In particular, in the example of stable coexistence in this paper, we see that strain one has higher transmission rate, and lower virulence but also much higher attack rate than strain two.
(4) Conventional wisdom in models suggests that stable coexistence of strains occurs when both invasion numbers are larger than one (that is, each strain has a positive growth rate when the other strain is at equilibrium). Surprisingly enough, that is not necessarily the case with stable coexistence mediated by predation. In the example presented in this article stable coexistence occurs when the invasion reproduction number of strain one is smaller than one while the invasion reproduction number of strain two is larger than one. This, perhaps, happens because predation also impacts the invasion reproduction numbers of the strains.
(5) Another surprising observation is that the attack rates for both types of infected prey are the only predation-related characteristics that impact the equilibrial level of the predator (predator's mortality rate and conversion efficiency, for instance, do not impact the equilibrial level of the predator) in the case of coexistence of the pathogen strains.
(6) In general predation reduces disease load and prevalence. This is the impact we observe with generalist predator or in any case when one of the strains eliminates the other. A specialist predator that differentiates among the various disease-related classes and mediates coexistence of the two pathogens can also lead to *increase* of the total disease load (that is the number of cases generated by both strains). Although predation impacts differently the two strains (increases the number of cases with one of the strains, and decreases the number of cases with the other) when they coexist, the combined effect may be increase in the total disease load. Predation leading to increase in the disease has only recently been found in an SIR model with predation where presumably the predator predates exclusively on the recovered class (and the total population size is constant). Preferential predation of the predator on the recovered individuals leads to increase in the number of susceptible individuals, which in turn, increases the

disease incidence and prevalence [13]. In the models we discuss here there is no recovered class and the pathways leading to increase in disease load appear to be more subtle.

It is important to note that the two types of predation – generalist and specialist – can be modeled with one model which contains the two very disparate models (2.1) and (3.1) as special cases. The idea of such model is to model a predator who has a choice of a number of species to feed on. We assume the total weighted population size of all prey remains constant. The predator may feed on all species of prey without discriminating among them. In this case we will consider such predator to be a complete generalist. However, the predator may feed on several, or even just one, species of prey. We will introduce a parameter that measures the amount of specialization of the predator. If the predator feeds on only one species of prey, then the predator is a complete specialist.

To introduce the general model, consider a predator of size $P(t)$ who potentially may feed on $(n+1)$ species. The population size of susceptible, and infected with each strain individuals in the ith species is given respectively by $S^i(t)$, $I_1^i(t)$ and $I_2^i(t)$. The parameters have the same meanings as in model (3.1) but are specific for the ith species. The model takes the form:

$$\begin{aligned}
\frac{dS}{dt} &= r^i S^i \left(1 - \frac{S^i + I_1^i + I_2^i}{K^i}\right) - \beta_1^i S^i I_1^i - \beta_2^i S^i I_2^i - (\mu_0^i + \eta^i P) S^i, \\
\frac{dI_1^i}{dt} &= \beta_1^i S^i I_1^i - (\mu_1^i + \gamma_1^i P) I_1^i, \\
\frac{dI_2^i}{dt} &= \beta_2^i S^i I_2^i - (\mu_2^i + \gamma_2^i P) I_2^i, \qquad i = 0, \ldots, n \\
\frac{dP}{dt} &= \epsilon P \textstyle\sum_{i=0}^{n} (\eta^i S^i + \gamma_1^i I_1^i + \gamma_2^i I_2^i) - dP.
\end{aligned} \tag{5.1}$$

Let:

$$\begin{aligned}
\eta &= \max\{\eta^0, \ldots, \eta^n\} \\
\gamma_1 &= \max\{\gamma_1^0, \ldots, \gamma_1^n\} \\
\gamma_2 &= \max\{\gamma_2^0, \ldots, \gamma_2^n\}
\end{aligned} \tag{5.2}$$

We assume that the maximum in the attack rates for susceptibles, and infected individuals with each strain is attained for the same species. That means that the predator prefers certain species, whether they are healthy or sick, as opposed to preferring healthy individuals from one species but sick individuals from another species. We may assume without loss of generality that this maximum is attained for the species numbered as zero (that is, $\eta = \eta^0$, $\gamma_1 = \gamma_1^0$, $\gamma_2 = \gamma_2^0$). Thus, the predator prefers best species zero. The predator may prefer to equal or lesser degree the remaining species, which determines its specialization level. To measure this, let Δ^i be predator's specialization constant for species i. The predator's specialization constant for species i measures the reduction of the predator's attack rates to species i compared to the attack rates of species zero:

$$\eta^i = \eta(1-\Delta^i) \qquad \gamma_1^i = \gamma_1(1-\Delta^i) \qquad \gamma_2^i = \gamma_2(1-\Delta^i) \qquad i = 1, \ldots, n$$

where it is assumed that $0 \le \Delta^i \le 1$. The amount of predator specialization is measured by the *predator specialization constant* Δ defined as follows:

$$\Delta = \frac{1}{n} \sum_{i=1}^{n} \Delta^i.$$

Since the number of species is large, we assume that the total weighted population size of all species of prey remains constant:

$$W(t) = \eta \sum_{i=0}^{n} S^i + \gamma_1 \sum_{i=0}^{n} I_1^i + \gamma_2 \sum_{i=0}^{n} I_2^i = W = \text{const.}$$

Moreover, we assume that $\epsilon W = d$. These assumptions imply that if $\Delta = 0$, the predator feeds on all species with the same attack rates, and its total population size is constant, say P. Then each of the systems for the $(n+1)$ species is the same, and it is equivalent to system (2.1). If, on the other hand, $\Delta = 1$, the predator feeds only on species zero, and the presence of the other species has no impact on the dynamics of the predator. Thus, the model for species zero in this case is equivalent to model (3.1).

The main observation in this article is that predation may increase genetic diversity of pathogens circulating in a prey population. Differential predation by a specialist predator ($\Delta \approx 1$) causes coexistence between two strains infecting the prey which in the absence of predation would exclude each other. On the other hand, as amount of specialization of the predator $\Delta \to 0$, the region in parameter space of coexistence of the strains shrinks to the trivial case when the two reproduction numbers are equal. One question remains open: Would a specialist predator cause the coexistence of more than two pathogens? We surmise that will not be possible unless some other trade-off mechanism is in place.

Predation also has evolutionary consequences on the pathogens in prey populations as it exercises selection on their hosts, based on the phenotypic (behavioral) differences that the pathogen creates in the different classes of hosts. If in the absence of predation, a pathogen of higher transmissibility and lower virulence persists in the prey population and excludes all other strains, predation may lead to the persistence of a pathogen of lower transmissibility and higher virulence, provided that the predator attack rate of prey infected with a strain of higher virulence is lower. Further studies are needed to elucidate the impact of predation on the evolution of virulence.

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