

# Dynamics of SI epidemic with a demographic Allee effect

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## Abstract

In this paper, we present an extended SI model of Hilker et al. (2009). In the presented model the birth rate and the death rate are both modeled as quadratic polynomials. This approach provides ample opportunity for taking into account the major contributors to an Allee effect and effectively captures species' differential susceptibility to the Allee effects. It is shown that, the behaviors (persistence or extinction) of the model solutions are characterized by the two essential threshold parameters  $\lambda_0$  and  $\lambda_1$  of the transmissibility  $\lambda$  and a threshold quantity  $\mu^*$  of the disease pathogenicity  $\mu$ . If  $\lambda < \lambda_0$ , the model is bistable and a disease cannot invade from arbitrarily small introductions into the host population at the carrying capacity, while it persists when  $\lambda > \lambda_0$  and  $\mu < \mu^*$ . When  $\lambda > \lambda_1$  and  $\mu > \mu^*$ , the disease drives the host population to extinction with origin as the only global attractor. For the special cases of the model, verifiable conditions for host population persistence (with or without infected individuals) and host extinction are derived. Interestingly, we show that if the values of the parameters  $\alpha$  and  $\beta$  of the extended model are restricted, then the two models are similar. Numerical simulations show how the parameter  $\beta$  affects the dynamics of the model with respect to the host population persistence and extinction.

*Keywords:* Allee effect; threshold; persistence; extinction; carrying capacity.

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## 1. Introduction

The history of the term Allee effect can be traced back to the pioneering work of Allee (1931). It receives considerable attention recently in mathemati-

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cal models of ecology and epidemiology (for the recent works see (David et al., 2007; Friedman and Yakubu, 2012b; Hilker et al., 2007; Ling-ling and Cang, 2009; Kang and Castillo-Chavez, 2014; Sophia and Jang, 2011; Thieme et al., 2009) and the references therein). The Allee effect is a phenomenon in biology characterized by a positive relationship between population density or size and the *per capita* population growth rate in small populations Stephens et al. (1999). That is, it is manifested by an increase in *per capita* growth rate with increasing population density. When a population exhibits a critical size or density below which, the population declines to extinction and above which, it can increase, that population is said to exhibit a strong Allee effect. Various mechanisms cause Allee effects which are naturally related to reproduction and/or survival, for example, difficulties in finding suitable mate at low population densities and foraging efficiency (Courchamp and Mackdonald, 2001; David and Luděk, 2002; Stephens et al., 1999). When a strong Allee effect is present in the host demographic the impact of a disease can be detrimental to the host population since any further reduction could decrease host density below the Allee threshold and lead to the extinction of the host. Some species that experience both an Allee effect and a disease include the island fox *Urocyon littoralis* (Clifford et al., 2006; Angulo et al., 2007) and African wild dog *Lycaon pictus* (Burrows et al., 1995; Courchamp et al., 2000).

Single species models with demographic Allee effect are widely studied in the literature (see the review in (David and Luděk, 2002) and the references therein). An SI model is introduced by (Deredec and Courchamp, 2006) for a population whose dynamics already face strong Allee effects in the absence of disease infection, in order to compare the impact of the Allee effects on the disease dynamics. In addition, an alternative model in which the Allee effect only manifests on mortality is developed in (Deredec and Courchamp, 2006). That is, a growth rate is decomposed into constant birth rate and density-dependent death rate function. Deredec and Courchamp reported in (Deredec and Courchamp, 2006) that the impact of the Allee effects could be considered a tradeoff between disease and the Allee effects. Indeed, the Allee effects could safeguard native individuals by diminishing the population sizes that expedite parasitic spread. On the other hand, when the disease invades the population, the Allee effects reduce the population size and increase the range of parasitic species that could drive the population to extinction. Hilker and collaborators Hilker et al. (2009) analyzed a particular case of the model presented in (Deredec and Courchamp, 2006) with a quadratic fertility rate and a linear density-dependent death rate. It was proved that the model introduced in Hilker et al. (2009) exhibits rich dynamical behaviors multiple stable steady states and homoclinic bifurcations, which indicate disease invasion and host population extinction, respectively. Moreover, Hilker et al. (2009) noted that high transmissibility rates

could lead to disease-induced extinction.

Friedman and Yakubu (Friedman and Yakubu, 2012a) reconsidered the SI model of Hilker et al. (2009) to identify a parameter regime of the model that lead to host population persistence (with or without infected individuals) and host extinction. Furthermore, these authors proved that an Allee effect matters even at large population densities, as a small perturbation from the disease-free equilibrium can drive host's population to extinction. They also showed that additional deaths due to the disease infections increase the Allee threshold of the host population. Cai et al. (2013) used the same SI model of Hilker et al. (2009) to analytically study bifurcations and dynamical behaviors of the model. These researchers found that their qualitative conclusions support the numerical bifurcation analysis and conjunctures reported in Hilker et al. (2009). In addition, some new bifurcations phenomena are explored such as pitchfork bifurcation, Bogdanov-Takens (BT) bifurcation of codimension two, degenerate Hopf bifurcation and degenerate BT bifurcation of codimension three in elliptic case Cai et al. (2013). These bifurcations exhibit complicated dynamical behaviors of the model in Cai et al. (2013) such as multiple attractors, homoclinic loop, and limit cycles, whose respective biological consequences are disease persistence, host population extinction and disease cycles.

In a similar note, Thieme et al. (2009) developed an SI model with a strong Allee effect in the host demographics that incorporated distinct fertility and mortality functions compared to those used in the model of Hilker et al. (2009). Another distinguishing feature of their model with those in (Deredec and Courchamp, 2006; Hilker et al., 2009) is the assumption that the infected individuals do not reproduce. Thieme et al. (2009) proved that the transition from host's population decline to extinction is mediated by a Hopf bifurcation and is marked by the occurrence of a heteroclinic orbit. This means that there is an oscillation in the host population in form of limit cycles before the disappearance of the population, leading to an epidemic break out in a periodic manner in the host population.

This work is motivated by the fact that the presence of an Allee effect in host demographics affects qualitatively the dynamics of a population, see the review in (David and Luděk, 2002) and the references therein. In this study we chose the *per capita* growth rate function that is used in the model introduced by Hilker et al. (2009) and apply the demographic model given by the generalized logistic equation as presented in (Zhou and Hethcote, 1994). The decomposition of the growth rate into birth rate and death rate potentially can have a significant impacts on epidemiological dynamics of the model. The birth and death terms in our chosen structure are both modeled as quadratics polynomials. The model presented in this paper can be considered an extension of the one introduced by Hilker et al. (2009) where the death rate is assumed linear.

Using a quadratic death rate and one additional parameter provides a more realistic representation of the population dynamics particularly at low population density or size. As this is a mortality rate in populations where there is a positive correlation between individual survival probability and population density due to factors like joint defence, cooperative feeding and/or breeding and lower exposure to predators. More precisely, our chosen quadratic birth and death rate functions generalize the constant birth, linear death, and constant death rates considered in (Deredec and Courchamp, 2006; Hilker et al., 2009; Thieme et al., 2009), respectively. The advantage of these quadratic birth and death rate functions over those in the aforementioned studies is that they effectively capture species' differential susceptibility due to the Allee effect. That is, the Allee effect is more intense when the parameter  $\beta > 0$  than for  $\beta \leq 0$ . This follows from the fact that an Allee effect is more intense in some species than others (Courchamp and Mackdonald, 2001; Stephens and Sutherland, 1999). As for the relevant aforementioned models, our model exhibits complex dynamical behaviors such as stable limit cycles, leading to disease cycles. Moreover, it is shown that a disease-induced extinction is possible when the disease-induced death rate  $\mu$  and transmissibility  $\lambda$  are above some threshold values  $\mu^*$  and  $\lambda_1$ , respectively. Using a similar approach as in (Friedman and Yakubu, 2012a), we derive conditions for host population persistence (with or without infected individuals) and host extinction for the special cases of the model.

The paper is organized as follows: In Section 2, we introduce the basic assumptions and equations of the model and introduce the demographic functions. Section 3 presents the basic qualitative features of the model in a re-scaled form. The effects of disease-induced mortality rate on the model is also investigated in Section 3. In order to complete the investigation, the special cases of the model are analyzed in Section 4. In Section 5, conditions for host population persistence and extinction for each of these cases are derived. Regions of host population persistence and extinction for certain parameter ranges are illustrated *via* numerical simulations also in Section 5.

## 2. Model formulation

Let the host population density at time  $t$  be denoted by  $P(t)$ . The densities of susceptible and infected individuals when a disease divides the population into two parts are denoted by  $S(t)$  and  $I(t)$  respectively, see Figure 1.

There is no recovery from the disease and newborns of infected individuals move to the susceptible class (no vertical transmission). We assume a density-dependent transmission, which is described by mass action rate  $\sigma SI$ . An additional constant disease-related death rate  $\mu$  is assumed for infectious individuals. The *per capita* net growth rate function in which the strong Allee

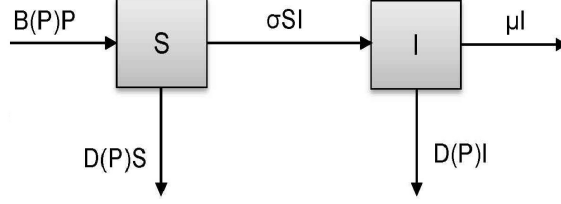


Figure 1: Flow diagram of model (1)

effect is manifested, denoted by  $G(P)$  and given in (2), is split into fertility function  $B(P) \geq 0$ , and mortality function  $D(P) \geq 0$ . Thus, the model equations are then described by the following system of differential equations:

$$\begin{aligned} \frac{dS}{dt} &= B(P)P - \sigma SI - D(P)S, \\ \frac{dI}{dt} &= \sigma SI - D(P)I - \mu I. \end{aligned} \quad (1)$$

The following *per capita* net growth rate is assumed to be in the form of the generalized logistic model introduced in (Gao and Hethcote, 1992).

$$G(P) = a(K_+ - P)(P - K_-), \quad 0 < K_- \ll K_+, \quad (2)$$

where the strong Allee effect is manifested through the minimum viable density  $K_-$ . As usual, the parameter  $K_+$  is the carrying capacity and the coefficient  $a > 0$  adjusts the maximum *per capita* growth rate.

We represent the demographic functions  $B(P)$  and  $D(P)$  in the following form

$$\begin{aligned} B(P) &= a\{-(1 - \alpha)P^2 + [K_+ + (1 - \beta)K_-]P + K_+\Gamma\}, \\ D(P) &= a(\alpha P^2 - \beta K_-P + K_+K_- + K_+\Gamma), \end{aligned} \quad (3)$$

where  $\alpha$ ,  $\beta$  and  $\Gamma$  are real parameters. The parameter  $\alpha \in [0, 1)$  determines the splitting of the quadratic term in (2) between the functions  $B(P)$  and  $D(P)$ . Moreover,  $\alpha$  and  $\beta$  determine the intensity of the Allee effects on both the demographic rate functions  $B(P)$  and  $D(P)$ . As in Hilker et al. (2009), the parameter  $\Gamma$  determines the effect of density-independence of the demographic rate functions. The basic requirement that both demographic functions need to be nonnegative places some constraints on the values of  $\beta$  and  $\Gamma$ . We assume that  $\beta \leq \min\{1, 2\sqrt{2\alpha}\}$  and that  $\Gamma \geq 0$ . It is easy to see that under these restrictions of the parameters both functions are positive on the interval  $[0, M]$  where  $M = \frac{(1-\beta)K_- + K_+}{1-\alpha}$ . It should be noted that, since  $B(P)$  is a quadratic polynomial

with negative leading coefficient it cannot be positive on an infinite interval. Therefore our aim is to have it positive on a practically relevant interval. In this regard, let us remark that the interval  $[0, M]$  contains all disease free equilibrium states of the population. The decomposition  $G(P) = B(P) - D(P)$  with  $B(P)$  and  $D(P)$  given by (3) is intended to model the factors causing the Allee effect. In Figure 2, for example, the steep gradient of the curve  $B(P)$  (i.e., the rapid population growth due to the decreasing mortality rate) is when  $P$  is small. This reflects factors like improved access (e.g. *via* cooperative strategies) to abundant resources. For small populations  $D(P)$  is either increasing at a slower rate ( $\beta \leq 0$ ) or is decreasing ( $\beta > 0$ ) while the values of all other parameters are fixed, representing what is referred to as safety in numbers (joint defence, lower individual exposure to predators, cooperation in raising the young). From biological point of view, the parameter  $\beta$  measures the intensity of an Allee effect and hence, our chosen fertility and mortality rate functions effectively capture species' differential susceptibility to the Allee effect since some species are more susceptible to the Allee effect than others. The two graphs intersect at the unstable equilibrium  $K_-$ . This critical value of  $P$ , a minimum viable population, is precisely the manifestation of the strong Allee effect. The graphs also intersect at the stable equilibrium  $K_+$ . When  $P$  is large, for example with values around  $K_+$ , the functions  $B(P)$  and  $D(P)$  exhibit the usual behavior of decreasing and increasing, respectively, due to adverse conditions caused by the larger population (food scarcity, stressful conditions owing to a strong competition, rise in predator numbers). As mentioned earlier, the demographic model described in terms of the functions  $B(P)$  and  $D(P)$  in (3) extends the model of Hilker et al. (2009) by using quadratic functions for modeling both the birth and the death rate with the aim of providing more realistic modeling tool as explained above. Note that the model in Hilker et al. (2009) is a particular case of (1) with (3) for  $\alpha = 0$  and some negative value of  $\beta$ .

To make the system (1) non-dimensional, we introduce the following dimensionless quantities as in Hilker et al. (2009):

$$p = \frac{P}{K_+}, \quad i = \frac{I}{K_+}, \quad s = \frac{S}{K_+}, \quad u = \frac{K_-}{K_+} \in (0, 1).$$

Substituting these quantities in system (1) the model equations are given by

$$\begin{aligned} \frac{dp}{dt} &= k(1-p)(p-u)p - \mu i, \\ \frac{di}{dt} &= [-\tau - k(\alpha p^2 - \beta u p) + \lambda p - \lambda i]i, \end{aligned} \tag{4}$$

where

$$k = aK_+^2, \quad \lambda = K_+\sigma, \quad \tau = \mu + k(u + \gamma), \quad \gamma = \Gamma/K_+. \tag{5}$$

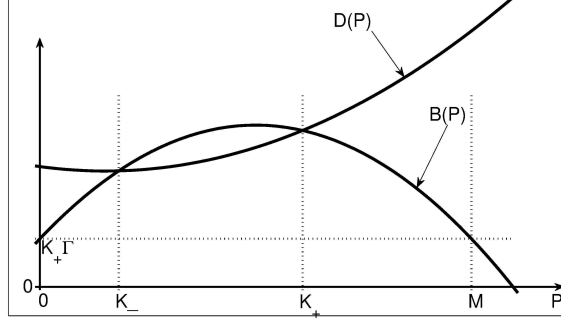


Figure 2: The demographic functions  $B(P)$  and  $D(P)$  for  $\beta > 0$

Furthermore, since  $P$  is considered in the practically relevant interval  $[0, M]$ , we have  $p \in [0, m]$  where

$$m = \frac{M}{K_+} = \frac{1 + (1 - \beta)u}{1 - \alpha}.$$

In what follows, we will base the model analysis on the ecologically interesting case in which the Allee threshold is far from the carrying capacity as in Hilker et al. (2009), i.e.  $0 < u < \frac{1}{2}$ .

### 3. Basic Qualitative Features of the Model

#### 3.1. Model (4) as a dynamical system.

**Theorem 1.** *The system of ordinary differential equations (4) defines a dynamical system on the domain*

$$\mathcal{F} = \{(p, i) : 0 \leq i \leq p \leq m\}.$$

*Proof.* We show first that all solutions of (4) initiated in  $\mathcal{F}$  remain in  $\mathcal{F}$  on the increasing interval of their existence. We consider the three line segments which make the boundary of  $\mathcal{F}$ , see Figure 3.

- (a) This line segment represents the disease free state of the population. It is positively invariant and contains the three disease free equilibria of the model.

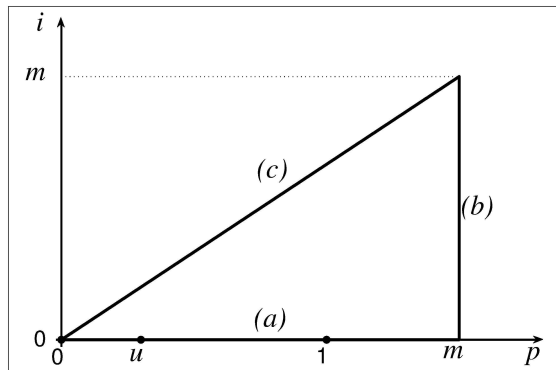


Figure 3: Domain  $\mathcal{F}$

- (b) If  $p = m$  one can see from the first equation of (4) that  $dp/dt < 0$ . Therefore the vector field defined by the system (4) is directed inwards on this line segment.
- (c) In order to prove that the vector field is directed inwards on this line segment we need to show that  $dp/dt - di/dt > 0$ . Let  $i = p \in [0, m]$ . Then

$$\frac{dp}{dt} - \frac{di}{dt} = pb(p) > 0,$$

which proves the required inequality.

Combining the results for the boundary segments (a), (b) and (c) we obtain that the solutions of (4) initiated in  $\mathcal{F}$  do not leave this domain. Then using the fact that  $\mathcal{F}$  is compact these solutions exist for  $t \in [0, \infty)$  (Stuart and Humphries, 1998, Theorem 2.1.5). Hence (4) defines a dynamical system on  $\mathcal{F}$ . ■

### 3.2. Threshold quantities

We introduce the critical host population density for disease establishment (the disease threshold) and the basic reproduction number as in Hilker et al. (2009). From the second equation of system (4) the replacement number of the disease is given by

$$\mathcal{R}(p) = \frac{\lambda p}{\tau + k(\alpha p^2 - \beta u p)}. \quad (6)$$

Notably, the replacement number is expressed as a function of  $p$  because the population size is variable. This threshold quantity  $\mathcal{R}(p)$  is defined to be the



average number of secondary infections produced by an infective individual during the entire infectious period Hethcote (2000). It should be noted that some authors use the term reproduction number/ratio instead of replacement number as in (Hilker et al., 2009; Hilker, 2010). Since in the absence of the disease the population being above the Allee threshold will settle at its carrying capacity then setting  $p = 1$  in Eq. (6) gives the basic reproduction number

$$\mathcal{R}_0 = \frac{\lambda}{\tau + k(\alpha - \beta u)}.$$

Notice that  $\tau + k(\alpha - \beta u) = \mu + ku(1 - \beta) + k(\alpha + \gamma) > 0$  since  $\beta \leq \min\{1, 2\sqrt{2\alpha}\}$ .

For the disease threshold (also known as critical community density or critical host density), set  $\mathcal{R}(p) = 1$  in (6) and solve for  $p$  in the equation

$$k\alpha p^2 - (k\beta u + \lambda)p + \tau = 0. \quad (7)$$

In the following analysis, we assume that  $\alpha \neq 0$  so that Eq. (7) is a quadratic equation. We shall see later that the results in Hilker et al. (2009) can be obtained when  $\alpha \rightarrow 0$ . Note from Eq. (7) that

- (i) there are two distinct real roots if  $(k\beta u + \lambda)^2 > 4k\alpha\tau$ ,
- (ii) there is one real root with multiplicity 2 if  $(k\beta u + \lambda)^2 = 4k\alpha\tau$ ,
- (iii) there is no real root if  $(k\beta u + \lambda)^2 < 4k\alpha\tau$ .

Denote by  $\lambda^*$  the threshold value of  $\lambda$  which discriminates between the three cases. Namely,  $\lambda^* = 2\sqrt{k\alpha\tau} - k\beta u$  is such that (7) has two, one or zero real roots according as  $\lambda > \lambda^*$ ,  $\lambda = \lambda^*$  and  $\lambda < \lambda^*$ , respectively.

Note that  $\lambda^* > 0$ . Using the relation  $\tau = \mu + ku + k\gamma > ku$  and  $\beta < 2\sqrt{2\alpha}$  we obtain

$$\begin{aligned} \lambda^* &= 2\sqrt{k\alpha\tau} - k\beta u, \\ &> 2\sqrt{k^2\alpha u} - 2ku\sqrt{2\alpha}, \\ &= 2k\sqrt{2\alpha u} \left( \sqrt{\frac{1}{2}} - \sqrt{u} \right) > 0 \text{ since } u < 1/2. \end{aligned}$$

It follows that, for  $\lambda > \lambda^*$ , Eq. (7) has the following two real roots.

$$(p_T)_{1,2} = \frac{(k\beta u + \lambda) \pm \sqrt{(k\beta u + \lambda)^2 - 4k\alpha\tau}}{2k\alpha\tau}. \quad (8)$$

*Remark 1*

- (1) We note that, if  $(\lambda + k\beta u) \leq 0$ , then by the second equation of (4)

$$i(t) \rightarrow 0 \text{ as } t \rightarrow \infty,$$

so that the infected population goes extinct. Hereafter, we assume that  $(\lambda + k\beta u) > 0$ .

- (2)  $\lambda > \lambda^*$  implies that  $(\lambda + k\beta u) > 0$ . Therefore, the roots  $(p_T)_{1,2}$  are positive whenever they exist.

### 3.3. Existence and stability of equilibria

#### 3.3.1. Disease-free equilibria

In the absence of the disease, the steady states of system (4) are: the trivial extinction state  $E_0 = (0, 0)$ , the Allee threshold state  $E_1 = (u, 0)$  and the carrying capacity state  $E_2 = (1, 0)$ .

**Theorem 2.** *For the dynamical system (4),*

- (i) *the trivial extinction state  $E_0$  is always a stable node,*
- (ii) *if  $\mathcal{R}_0 < 1$ , the carrying capacity state  $E_2$  is a stable node and is a saddle point when  $\mathcal{R}_0 > 1$ ,*
- (iii) *the Allee threshold state  $E_1$  is a saddle point if  $\mathcal{R}_0 < 1$  and is an unstable node or a saddle point when  $\mathcal{R}_0 > 1$ .*

*Proof.* The Jacobian matrix of system (4) evaluated at a disease-free equilibrium  $(p, 0)$ , denoted by  $J(p, 0)$  is as follows:

$$J(p, 0) = \begin{pmatrix} -k[3p^2 - 2(1+u)p + u] & -\mu \\ 0 & A \end{pmatrix},$$

where  $A = \lambda p - \tau - kp(\alpha p - \beta u)$ .

(i) It follows that the trace and determinant of  $J(E_0)$  are  $\text{tr}(E_0) = -(ku + \tau) < 0$  and  $\det(E_0) = ku\tau > 0$ , respectively. The eigenvalues of  $J(E_0)$  have negative real parts and hence  $E_0$  is stable by the Routh Hurwitz criterion Sánchez (1979). Moreover,  $E_0$  is always a stable node since the eigenvalues  $-\tau$  and  $-ku$  are real and of negative sign irrespective of whether  $\mathcal{R}_0 \leq 1$ .

(ii) Similarly, the eigenvalues of the Jacobian matrix  $J(E_2)$  evaluated at  $E_2$  are, respectively, given by  $-k(1-u)$  and  $-\frac{\lambda}{\mathcal{R}_0}(1-\mathcal{R}_0)$ , which are real and of negative sign whenever  $\mathcal{R}_0 < 1$  (noting that  $u \in (0, 1)$ ). Hence  $E_2$  is a stable node. If  $\mathcal{R}_0 > 1$ , the first eigenvalue is still negative while the second eigenvalue is positive, which indicates that  $E_2$  is saddle point.

(iii) the first eigenvalue of  $J(E_1)$  is  $ku(1-u) > 0$  and the second eigenvalue is

$$-(\tau + ku^2\alpha) + u(\lambda + k\beta u),$$

which is negative if  $\mathcal{R}_0 < 1$  since  $(\lambda + k\beta u) \leq 0$  and positive or negative when  $\mathcal{R}_0 > 1$ . Therefore,  $E_1$  is either a saddle point or an unstable node.  $\blacksquare$

We infer from Theorem 2 that a disease cannot establish itself from arbitrarily small introductions into the host population at carrying capacity if  $\mathcal{R}_0 < 1$ . More precisely, if  $\mathcal{R}_0 < 1$  the host population either settles at the carrying capacity or undergoes extinction, depending on the initial condition being above or below the Allee threshold  $u$ , respectively. Hence, the dynamics of the system is only determined by the Allee effect. From biological point of view, this conclusion shows that the presence of an Allee effect in the host demographics could play a stabilizing and protective role in relation to invasion of a disease.

### 3.3.2. Endemic equilibria

Endemic equilibrium is the steady state solution of (4) when the infected compartment  $i$  is non empty. Hence, setting the right-hand side of (4) to zero, gives

$$\begin{aligned} i &= \frac{k}{\mu}(1-p)(p-u)p, \\ i &= \frac{1}{\lambda}[-\tau - k(\alpha p^2 - \beta up) + \lambda p]. \end{aligned} \tag{9}$$

Then, we obtain the following cubic equation by equating and rearranging the equations in (9).

$$Ap^3 + Bp^2 + Cp + D = 0, \tag{10}$$

where

$$A = -k, B = (k/\lambda)[\lambda(1+u) + \alpha\mu], C = -(1/\lambda)[ku(\beta\mu + \lambda) - \lambda\mu], D = \tau.$$

Notice that  $A$  is negative,  $B$  and  $D$  are positive while  $C$  is negative or positive or zero. Therefore, there is at least one sign change in the sequence of coefficients  $\{A, \dots, D\}$ . Hence, by Descartes rule of sign (Polyanin and Manzhirov, 2007), there is at least one positive real root of (10). Consequently, we obtain the following result.

**Theorem 3.** *If  $(p_T)_1 < 1$  and  $(p_T)_2 > 1$  then model (4) has:*

- (i) a unique endemic equilibrium if  $C \geq 0$ ;
- (ii) a unique endemic equilibrium or three endemic equilibria if  $C < 0$ .

We cannot exclude the existence of two endemic stationary states depending on the parameter values as in Figure 4, despite the fact that Theorem 3 does not state that. This is because it is algebraically impossible to find explicit necessary and sufficient conditions for the possible number of endemic equilibria of system

(4) depending on all the values of the model parameters (see the comment in Cai et al. (2013)). According to the first case of Theorem 3, it is possible for model (4) to have a unique endemic equilibrium. Thus, we obtain the following result.

**Theorem 4.** *Let  $Q(p) = \frac{d}{dp}[k(1-p)(p-u)p]$ . If  $0 < u < (p_T)_1 < 1 < (p_T)_2$ , then model (4) has a unique endemic equilibrium point  $E^* = (p^*, i^*)$  with*

$$(p_T)_1 < p^* < 1, i^* > 0,$$

and  $E^*$  is locally asymptotically stable if

$$Q(p^*) < \frac{\lambda p^*}{\mathcal{R}(p^*)} [\mathcal{R}(p^*) - 1]. \quad (11)$$

*Proof.* See Appendix A

#### 3.4. The effect of disease-induced mortality on the model

In order to explore the effect of disease related death on the stability results of model (4), we denote by  $\lambda_0$ , the denominator of the basic reproduction ratio  $\mathcal{R}_0$ . That is,  $\lambda_0 = \tau + k(\alpha - \beta u)$  so that  $\mathcal{R}_0 \leq 1$  if and only if  $\lambda \leq \lambda_0$ . Furthermore, the respective non-trivial nullclines of the model (4) are represented as follows:

$$\begin{aligned} \Lambda_p : i = \phi_1(p) &:= \frac{k}{\mu} p(p-u)(1-p), \\ \Lambda_i : i = \phi_2(\lambda, p) &:= p - \frac{kp(\alpha p - \beta u) + \tau}{\lambda}. \end{aligned}$$

It is to be noted that  $dp/dt = 0$  and  $di/dt = 0$  on the curves  $\Lambda_p$  and  $\Lambda_i$ , respectively, with only  $\Lambda_i$  depends on the coefficient of the force of infection  $\lambda$ . Also, one can easily see that  $\phi_2(\lambda, p) < p$  and  $\lim_{\lambda \rightarrow \infty} \phi_2(\lambda, p) = p$ . The dynamical system (4) behaves in two essentially different ways depending on whether or not  $\Lambda_p$  intersects the line  $i = p$ . These two cases are distinguished below *via* the threshold value

$$\mu^* = \frac{k(1-u)^2}{4}$$

of the disease induced mortality  $\mu$ , which is obtained by substituting  $i = p$  in the first equation of (9).

3.4.1. *The model with low disease-induced mortality:  $\mu < \mu^*$*

For the case when  $\lambda > \lambda_0$  system (4) always has an equilibrium  $\hat{E}_2$  on the decreasing portion of  $\Lambda_p$ . Moreover, it may or not have a second equilibrium  $\hat{E}_1$  on the increasing side of  $\Lambda_p$ .

**Proposition 1.** *Let  $\lambda > \lambda_0$ ,*

- (i) *then the endemic equilibrium  $\hat{E}_2$  is asymptotically stable,*
- (ii) *if the two endemic equilibria  $\hat{E}_1$  and  $\hat{E}_2$  exist, then  $\hat{E}_1$  is unstable (saddle point) and  $\hat{E}_2$  is asymptotically stable. Therefore, for every  $p \in (u, m]$  there exists  $\delta > 0$  such that any solution of system (4) initiated at a point  $(p, i)$  with  $i < \delta$  converges to  $\hat{E}_2$ .*

This proposition simply asserts that if the population is above the Allee threshold, the disease will establish itself in the population. This is illustrated in Figure 4A (one endemic equilibrium) and Figure 4B (two endemic equilibria).

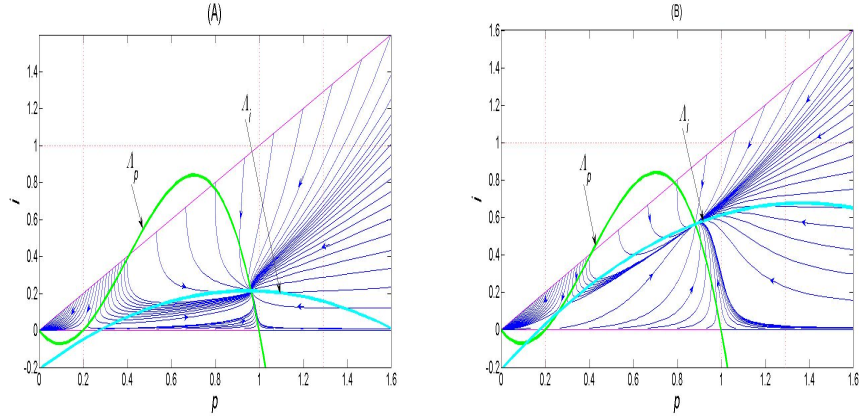


Figure 4: (A) Low disease-induced mortality and unique endemic equilibrium; (B) Low disease-induced mortality and two endemic equilibria. The green curve  $\Lambda_p$  is the  $p$ -nullcline and the cyan curve  $\Lambda_i$  is the  $i$ -nullcline. The diagonal magenta line is the line  $p = i$ .

3.4.2. *The model with high disease-induced mortality:  $\mu > \mu^*$*

In this case, for sufficiently large  $\lambda$  the graph of  $\Lambda_i$  is very close to the line  $i = p$  so that  $\Lambda_i$  and  $\Lambda_p$  do not intersect. Denoting  $\lambda_1 = \min\{\lambda : \phi_1(p) \leq \phi_2(\lambda, p)\}$  and  $\mathcal{F}_0 = \{(p, i) : i = 0\}$ , we obtain the following result.

**Proposition 2.** *If  $\lambda > \lambda_1$  the only stable equilibrium of the dynamical system (4) is the origin and its basin of attraction is  $\mathcal{F} \setminus \mathcal{F}_0$ .*

In simple terms, for  $\lambda > \lambda_1$  the disease drive the host population to extinction (see Figure 5A). On the other hand, when  $\lambda \in (\lambda_0, \lambda_1)$  the disease can either persist endemically or drive the host population to extinction. That is, the eventual outcome (endemic state or extinction) depends on the host population size and the size of the initial number of infection. As  $\lambda$  decreases from  $\lambda_1$  to  $\lambda_0$  the dynamics presented in Figure 5B-D can be observed.

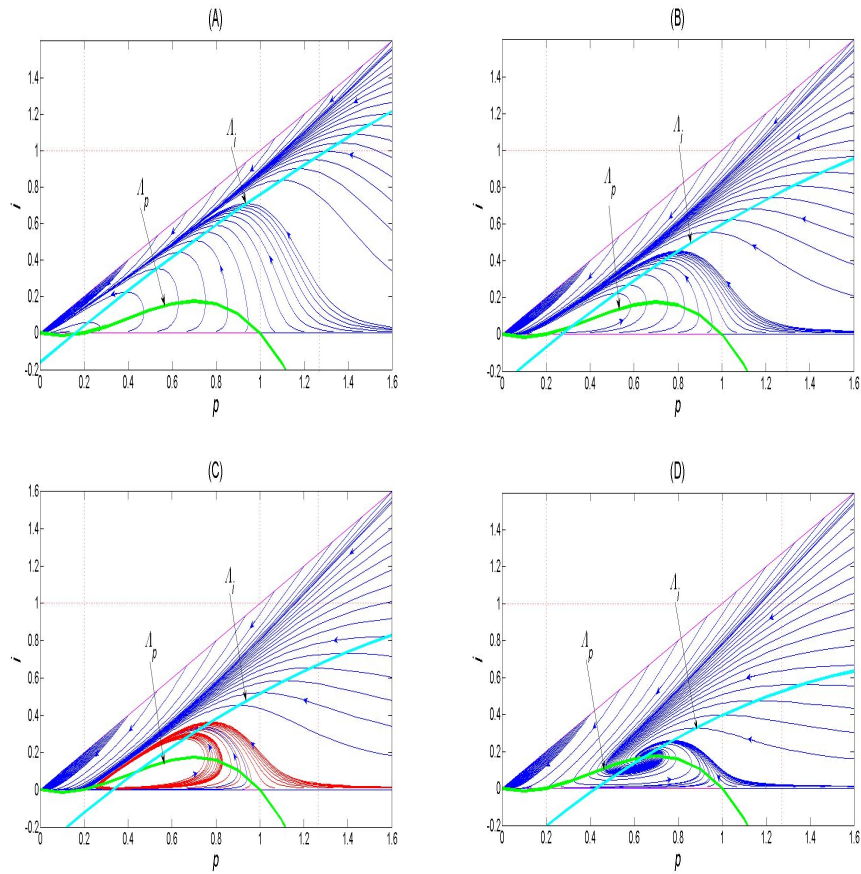


Figure 5: (A) Extinction:  $\lambda > \lambda_1$ ; (B) Unique unstable endemic equilibrium; (C) Stable endemic limit cycle and (D) Stable Spiral point. The green curve  $\Lambda_p$  is the  $p$ -nullcline and the cyan curve  $\Lambda_i$  is the  $i$ -nullcline. The diagonal magenta line is the line  $p = i$ .

Biologically, the results of propositions 1 and 2 follow from the fact that the maximum degree of depression of the host population equilibrium, here

leading to extinction, is achieved by a disease with intermediate pathogenicity (Anderson, 1979; Anderson and May, 1979). If a disease pathogenicity is low ( $\mu < \mu^*$ ), the disease has a little detrimental effect on the host and so, the host persists at endemic state with large population density. On the other hand, if the disease pathogenicity is high, i.e.  $\mu > \mu^*$  such that  $\lambda \in (\lambda_0, \lambda_1)$ , the infection can either be stably maintained in the population or drive the host to extinction depending on the initial sizes of the host and infected sub-populations owing to the strong Allee effect. Furthermore, if the disease pathogenicity is high ( $\mu > \mu^*$ ) and  $\lambda > \lambda_1$  then the disease drive the host population to extinction.

#### 4. Special cases of the model

In this section, we consider the dynamics of system (4) for the case when  $\alpha = 0$  and  $\beta \neq -\frac{1}{ku}$ , which implies that the mortality rate function of system (4) becomes linear (i.e. different from that of Hilker et al. (2009)).

*Case I:  $\alpha = 0$  and  $\beta \neq 0$*

When  $\alpha = 0$  and  $\beta \neq 0$  such that  $\beta \neq -\frac{1}{ku}$ , system (4) reduces to

$$\begin{aligned}\frac{dp}{dt} &= k(1-p)(p-u)p - \mu i, \\ \frac{di}{dt} &= [-\tau + (\lambda + k\beta u)p - \lambda i]i.\end{aligned}\tag{12}$$

Setting  $\alpha$  to zero in equations (6) and (7), we obtain the following pertinent threshold quantities for model (12). The replacement number,

$$\mathcal{R}(p) = \frac{\lambda p}{\tau - k\beta u p}\tag{13}$$

and the basic reproduction number,

$$\mathcal{R}_0 = \frac{\lambda}{\tau - k\beta u}.\tag{14}$$

The disease threshold,

$$p_T = \frac{\tau}{\lambda + k\beta u},\tag{15}$$

is the point at which the linear  $i$ -nullcline crosses the horizontal axis ( $p$ -axis). Moreover,  $0 < p_T < 1$  is equivalent to  $\mathcal{R}_0 > 1$ , and  $p_T > 1$  is equivalent to  $\mathcal{R}_0 < 1$ .

The results of Theorem 1, Theorem 3 and the existence and stability results of the disease free equilibria follow for  $\alpha = 0$ . Moreover, for  $\alpha = 0$  and  $p_T$  as defined in (15), Theorem 4 is restated as follows.

**Theorem 5.** Let  $Q(p) = \frac{d}{dp}[k(1-p)(p-u)p]$ . If  $0 < u < p_T < 1$ , then model (12) has a unique endemic equilibrium point  $E^* = (p^*, i^*)$  with

$$p_T < p^* < 1, i^* > 0,$$

and  $E^*$  is locally asymptotically stable if

$$Q(p^*) < (\lambda + k\beta u)(p^* - p_T).$$

It is worth mentioning here that, if  $\alpha = 0$  and  $\beta = -\frac{1}{ku}$ , then model (4) reduces to that of Hilker et al. (2009). Detailed analysis of this case can be found in (Cai et al., 2013; Friedman and Yakubu, 2012a; Hilker et al., 2009).

*Case II:  $\alpha = \beta = 0$*

For the case when  $\alpha = \beta = 0$ , the model (4) reduces to

$$\begin{aligned} \frac{dp}{dt} &= k(1-p)(p-u)p - \mu i, \\ \frac{di}{dt} &= [-\tau + \lambda(p-i)]i. \end{aligned} \tag{16}$$

The feasible region (Theorem 1) and the disease free equilibria with their associated stability results are as that of system (12). For the endemic equilibria Eq. (10) becomes

$$Ap^3 + Bp^2 + Cp + D = 0, \tag{17}$$

where

$$A = -k, B = k(1+u), C = -(ku + \mu), D = \mu p_T,$$

so that  $A, C$  are negatives and  $B, D$  are positives. Then there are three sign changes in the sequence of coefficients of Eq. (17). Therefore, Theorem 3 becomes

**Theorem 6.** If  $0 < u < p_T < 1$  then model (16) has either a unique endemic equilibrium or three endemic equilibria.

It follows that Theorem 4 holds if a unique endemic equilibrium exists. Moreover, it is observed that the results of Theorem 6 and similar results for model (12) do not exclude the existence of two endemic stationary states, depending on the parameter values as in Figure 7B. The threshold quantities: replacement



number, basic reproduction number and disease threshold are respectively, given by

$$\mathcal{R}(p) = \lambda p / \tau, \quad (18)$$

$$\mathcal{R}_0 = \lambda / \tau, \quad (19)$$

and

$$p_T = \tau / \lambda. \quad (20)$$

## 5. Persistence and Extinction

Following (Friedman and Yakubu, 2012a), here we state the conditions for persistence/extinction of the infected/host population of model (12) and model (16). All the theorems, lemmas and their proofs we present here are based on the approach in (Friedman and Yakubu, 2012a).

Before we state the conditions for persistence/extinction, we claim the following auxiliary results.

**Lemma 1.** *Let  $0 < p_T < 1$ . Then for any  $0 < \rho_0 < 1 - u$ , there exists a sufficiently small  $\rho > 0$  and a function  $t^0 = t^0[\rho, \rho_0, i(0)]$  such that if*

$$0 < i(0) < \rho \text{ and } u + \rho_0 < p(0) \leq 1,$$

*then*

$$i(t_1) = \rho \text{ for some } t_1 < t^0[\rho, \rho_0, i(0)].$$

*Proof.* Suppose

$$i(t) < \rho \text{ for all } t < t^{0*}. \quad (21)$$

Then we need to show that  $t^{0*}$  has a bound in terms of  $\rho, \rho_0$ , and  $i(0)$ . We claim that

$$p(t) > u + \rho_0 \text{ for all } t < t^{0*}. \quad (22)$$

Proof of the claim: If the assertion in (22) is not true, then there is a smallest  $t = t_1$  such that  $p(t_1) = u + \rho_0$ , so that  $\frac{dp(t_1)}{dt} \leq 0$ . But by the first equation of (12) and inequalities (21) and (22)

$$\frac{dp}{dt} > k(1-p)(p-u)p - \mu\rho = k[1 - (u + \rho_0)]\rho_0(u + \rho_0) > 0$$

at  $t = t_1$  for

$$\rho < \frac{k[1 - (u + \rho_0)]\rho_0(u + \rho_0)}{\mu},$$

which is a contradiction. Thus, inequality (22) holds.

We further claim that

$$\frac{dp}{dt} > 0 \text{ whenever } p(t) < 1 - \rho_1,$$

where  $\rho_1 = v\rho$  and  $v$  is a positive constant such that  $u + \rho_0 \leq 1 - \rho_1 \leq 1$ . In fact, by the first equation of (12) and inequalities (21) and (22), at any time  $t_2$  where  $p(t_2) < 1 - \rho_1$ , we have

$$\frac{dp}{dt} > k\rho_1\rho_0(u + \rho_0) - \mu\rho = \mu\rho$$

for

$$\rho_1 = \frac{2\mu\rho}{k\rho_0(u + \rho_0)} = v\rho.$$

It follows that,

$$p(t) > 1 - v\rho \text{ if } t > t_1^0(\rho, \rho_0) \text{ for some } t_1^0 = t_1^0(\rho, \rho_0). \quad (23)$$

Also, from the second equation of (12) and inequalities (21) and (23), we have

$$\begin{aligned} \frac{di(t)}{dt} &= -\tau + (\lambda + k\beta u)p - \lambda i, \\ &> -\tau + (\lambda + k\beta u)(1 - v\rho) - \lambda\rho, \\ &= \frac{1}{p_T}(1 - p_T)\tau - v_1\rho = \xi\tau - v_1\rho, \end{aligned}$$

where  $t > t_1^0(\rho, \rho_0)$ ,  $\xi = \frac{1-p_T}{p_T} > 0$  and  $v_1 > 0$  is a constant. Therefore,

$$i(t) > i[t_1^0(\rho, \rho_0)]e^{(1/2)\xi\tau[t-t_1^0(\rho, \rho_0)]} \text{ for } \rho \leq \frac{1}{2v_1}\xi\tau.$$

Hence,  $i(t_1) > \rho$  for some  $t_1$ , where

$$t_1 \leq t^{0*} \equiv t_1^0(\rho, \rho_0) + t_2^0(\rho, \rho_0, i[t_1^0(\rho, \rho_0)]). \quad (24)$$

It is to be noted that,  $i[t_1^0(\rho, \rho_0)]$  depends on the initial condition  $i(0)$ , as such if  $i(0) \rightarrow 0$ , then  $i[t_1^0(\rho, \rho_0)] \rightarrow 0$ . Hence, the right-hand side of Eq. (24) is indeed a function  $t^0$  of  $\rho, \rho_0$  and  $i(0)$  which approaches infinity as  $i(0)$  approaches 0. ■

**Lemma 2.** Let  $0 < p_T < 1$  and

$$i(t_a) = \rho, p(t_a) > u + \rho_0 \text{ for some } \rho_0 \in (0, 1 - u),$$

where,  $\rho$  is a sufficiently small positive number depending on  $\rho_0$ . If  $i(t) \leq \rho$  for  $t_a < t < t_b$ , then

$$t_b - t_a < t^0(\rho, \rho_0).$$

**Theorem 7.** If

$$0 < u < p_T < 1 \tag{25}$$

and

$$\max_{u \leq x \leq p_T} \{k(1-x)(x-u)x\} > \frac{\mu(\lambda + k\beta u)}{\lambda}(1 - p_T) \tag{26}$$

then for any solution  $(p(t), i(t))$  of (12) with  $p(0) > u + \rho_0, 0 < i(0) < \rho$  for some positive numbers  $\rho_0$  and  $\rho$ , there exists an  $\eta > 0$  depending on  $\rho_0$  and  $\rho$  and a time  $t^0 = t^0[\rho, \rho_0, i(0)]$  such that

$$i(t) \geq \eta \text{ for all } t \geq t^0[\rho, \rho_0, i(0)]. \tag{27}$$

*Proof.* See Appendix B

As stated earlier, in model (12), all the trajectories  $(p(t), i(t))$  with  $p(0) < u$  lead to the extinction of the host population. But for  $0 < p_T < 1$ , Theorem 7 asserts that in the presence of disease infection, the host population size need to be larger than the Allee threshold  $u$  in order to guarantee persistence of the infected population. That is, the Allee threshold is increased to  $u + \rho_0$ .

*Remark 2*

(i) If  $g(x) = \frac{k}{\mu}(1-x)(x-u)x$  and  $x_c = \frac{1+u+\sqrt{(1+u)^2-3u}}{3}$ , then (26) becomes

$$\begin{aligned} \frac{(\lambda + k\beta u)}{\lambda}(1 - p_T) &< \max_{u \leq x \leq p_T} g(x) \\ &= \begin{cases} g(p_T) & \text{if } u \leq p_T \leq x_c, \\ g(x_c) & \text{if } u < x_c < p_T. \end{cases} \end{aligned} \tag{28}$$

(ii) If  $p_T \leq x_c$  as in the first case of (i), the inequality (26) becomes

$$k(p_T - u)p_T > \frac{\mu(\lambda + k\beta u)}{\lambda}. \quad (29)$$

We illustrate in Figure 6 that inequality (26) is satisfied whenever the maximum value of  $g$  in the second case of (28),  $g(x_c)$  is greater than  $\frac{(\lambda + k\beta u)}{\lambda}(1 - p_T)$ , where  $u < x_c < p_T$ .

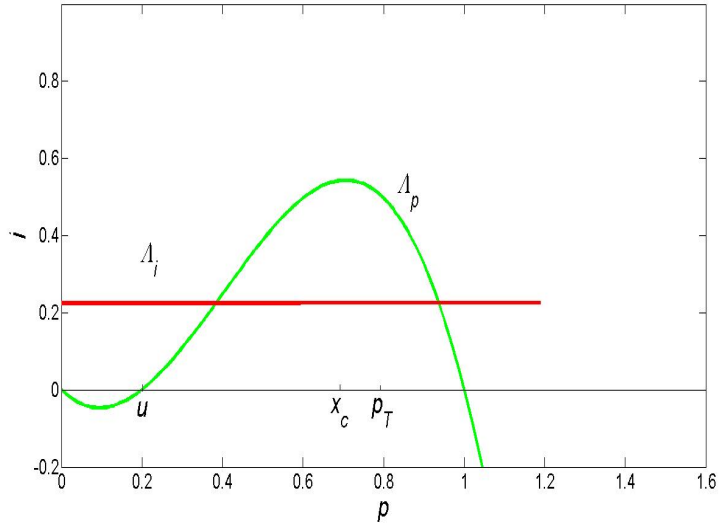


Figure 6: Inequality (26) holds where  $u < x_c < p_T$  and the  $i$ -nullcline,  $\Lambda_i = \frac{(\lambda + k\beta u)}{\lambda}(1 - p_T)$ , is below the maximum value of the  $p$ -nullcline,  $\Lambda_p = \frac{k}{\mu}(1 - p)(p - u)p$ .

We observe that in model (12), it is possible for the host population to go extinct with  $0 < p_T < 1$  and  $p(0) \geq u + \rho_0$ . This can be seen when we consider  $p_T < u$  instead of  $p_T > u$  in (26). Hence, we obtain the following result.

**Theorem 8.** *If  $0 < p_T < u$  and*

$$\max_{u \leq x \leq 1} \left\{ k(1 - x)(x - u)x - \frac{\mu(\lambda + k\beta u)}{\lambda}(x - p_T) \right\} \leq \rho \quad (30)$$

*for some small enough  $\rho > 0$ , then any solution of model (12) with  $1 - \eta < p(0) \leq 1$  for any sufficiently small  $\eta > 0$  and  $i(0) > 0$  satisfies*

$$p(t) \rightarrow 0 \text{ and } i(t) \rightarrow 0 \text{ as } t \rightarrow \infty.$$

*Proof.* See Appendix C

Inequality (30) holds whenever the  $p$ -nullcline,  $\Lambda_p$ , is below the  $i$ -nullcline,  $\Lambda_i$ , of system (12) (see Figure 7A). It is to be noted that, the assumption that  $\rho$  in condition (30) is small is necessary. As shown in Figure 7B, for example, if the two nullclines intersect at two points  $\omega_1$  and  $\omega_2$  which are not sufficiently close, then any solution  $(p(t), i(t))$  of model (12) with initial condition that  $p(0) \in (1 - \eta, 1]$  and a very small number of infectives  $i(0)$  converges to  $\omega_2$  as  $t \rightarrow \infty$ .

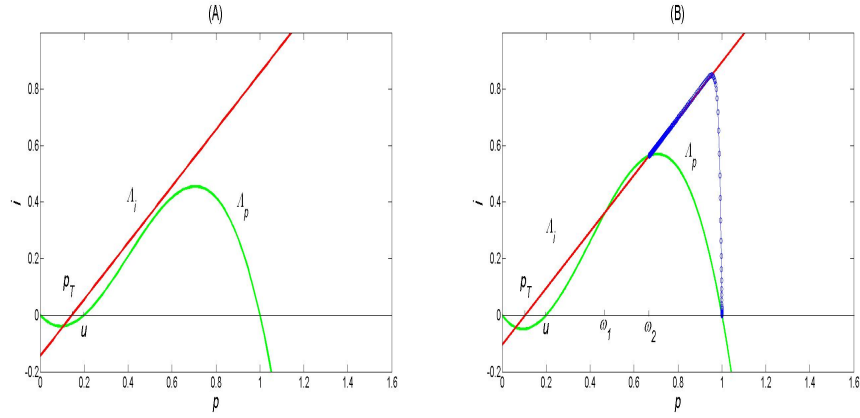


Figure 7: (A) Inequality (30) holds where  $p_T < u < 1$  and the  $p$ -nullcline  $\Lambda_p$  is below the  $i$ -nullcline  $\Lambda_i$ ; (B) Two interior positive equilibrium points of model (12) and a solution (circles) with initial condition  $(p(0), i(0)) = (1, 0.0001)$  converges to an equilibrium point  $\omega_2$  as  $t \rightarrow \infty$ . Parameter values used are:  $k = 1.3; \gamma = 1.8, u = 0.2, \beta = 0.07, \lambda = 19.797$  and (A)  $\mu = 0.3$ ; (B)  $\mu = 0.25$ .

On the issue of persistence and extinction for model (16), Lemma 1 and Lemma 2 hold. But Theorem 7 and Theorem 8, respectively, become

**Theorem 9.** *If*

$$0 < u < p_T < 1 \quad (31)$$

and

$$\max_{u \leq x \leq p_T} \{k(1-x)(x-u)x\} > \mu(1-p_T) \quad (32)$$

then for any solution  $(p(t), i(t))$  of (16) with  $p(0) > u + \rho_0, 0 < i(0) < \rho$  for some positive numbers  $\rho_0$  and  $\rho$ , there exists an  $\eta > 0$  depending on  $\rho_0$  and  $\rho$

and a time  $t^0 = t^0[\rho, \rho_0, i(0)]$  such that

$$i(t) \geq \eta \text{ for all } t \geq t^0[\rho, \rho_0, i(0)]. \quad (33)$$

*Remark 3*

(i) If  $g(x) = \frac{k}{\mu}(1-x)(x-u)x$  and  $x_c = \frac{1+u+\sqrt{(1+u)^2-3u}}{3}$ , then (32) becomes

$$\begin{aligned} (1-p_T) &< \max_{u \leq x \leq p_T} g(x) \\ &= \begin{cases} g(p_T) & \text{if } u \leq p_T \leq x_c, \\ g(x_c) & \text{if } u < x_c < p_T. \end{cases} \end{aligned} \quad (34)$$

(ii) If  $p_T \leq x_c$  as in the first case of (i), the inequality (32) becomes

$$k(p_T - u)p_T > \mu. \quad (35)$$

**Theorem 10.** *If  $0 < p_T < u$  and*

$$\max_{u \leq x \leq 1} \{k(1-x)(x-u)x - \mu(1-p_T)\} \leq \rho \quad (36)$$

for some small enough  $\rho > 0$ , then any solution of model (16) with  $1 - \eta < p(0) \leq 1$  for any sufficiently small  $\eta > 0$  and  $i(0) > 0$  satisfies

$$p(t) \rightarrow 0 \text{ and } i(t) \rightarrow 0 \text{ as } t \rightarrow \infty.$$

We can biologically conclude that the results of Theorems 8 and 10 indicate that the synergistic interplay between Allee effects and infectious diseases is death blow for the host population if the disease threshold  $p_T$  is low and the transmissibility  $\lambda$  is large. That is, the eventual outcome in such a situation is the extinction of the whole population.

### 5.1. Numerical simulations

Here, we focus on the model parameters where a small number of individuals infected with a fatal disease cause the host population subject to the strong Allee effect in the vital dynamics with  $p(0) = 1$  to persist or to go extinct. If extinction occurs for no matter how small the initial number of the infected individuals,  $i(0)$  is, then the model parameters are in the *host extinction phase*; otherwise they are said to be in the *host persistence phase* (Friedman and Yakubu, 2012a).

According to Theorem 7, when  $0 < p_T < 1$ , under conditions (25) and (26), if  $p(0) > u + \rho_0$  for some  $\rho_0 > 0$  ( $p(0) = 1$ , in particular), the inequality (27)

holds for any small number of infected individuals  $i(0)$ . Hence,  $(u, \lambda)$  is a point of persistence of the infected population and also of the host population by Theorem 1. If  $0 < p_T < \min\{u, 1\}$  and inequality (30) holds, then by Theorem 8,  $(u, \lambda)$  is a point of host extinction.

For illustration, in Figure 8, we vary the Allee threshold,  $u$  and the transmissibility,  $\lambda$ , keeping all other parameters fixed, see Table 1 for the parameter values, which we adopt from (Friedman and Yakubu, 2012a; Hilker et al., 2009).

Table 1: Parameter values

Parameter	Nominal value
$k$	0.2
$\gamma$	1.25 (scaled by $k$ to 0.25)
$\mu$	0.1
$u$	(0, 0.5)
$\beta$	-0.6 (Assumed)

Define

- (1) The curve  $\Lambda_1 : \lambda = \lambda_1(u)$  by setting  $p_T$  to 1 in (15).
- (2) The curve  $\Lambda_2 : \lambda = \lambda_2(u)$  by setting  $p_T$  to  $u$  in (15).
- (3) The curve  $\Lambda_3 : \lambda = \lambda_3(u)$  such that with  $\lambda = \lambda_3(u)$ , equality holds in inequality (26). Then inequality (26) holds if  $\lambda > \lambda_3(u)$ .
- (4) The curve  $\Lambda_4 : \lambda = \lambda_4(u)$ , such that with  $\lambda = \lambda_4(u)$ , equality holds in inequality (30). Then inequality (30) holds if  $\lambda > \lambda_4(u)$ .

By the assertion of Theorem 7, the region between  $\Lambda_3$  and  $\Lambda_2$  is a region of persistence of the infected population. The region  $\lambda > \lambda_4(u) + \epsilon$  for some  $\epsilon > 0$  is a region of host population extinction as asserted by Theorem 8. Points of disease population persistence or extinction may either be points in the regions between  $\Lambda_1$  and  $\Lambda_3$  and between  $\Lambda_2$  and  $\Lambda_4$ . In Figure 8, simulations depict points of host population persistence with no infected individuals (tildes), points of disease persistence (stars), and points of host population extinction (open circles).

For system (16), we define the curves  $\Lambda_1$  and  $\Lambda_2$  by replacing Eq. (15) with Eq. (20). The curves  $\Lambda_3$  and  $\Lambda_4$  are defined respectively, by replacing inequalities (26) and (30) with inequalities (32) and (36). Then we have in Figure 8B, numerical simulations similar to that of system (12).

It should be noted that, if  $\alpha = 0$  and  $\beta = -\frac{1}{ku}$ , then model (4) reduces to that of Hilker *et al.* Hilker et al. (2009) with  $\lambda > 1$ , see simulations in Figure 8D and compare with Figure 5 in Friedman and Yakubu (2012a).

One can observe from Figure 8 that it is possible for the model parameters to shift from host population persistence phase to host population extinction phase if we increase the transmissibility  $\lambda$  and the Allee threshold  $u$ , respectively, while the values of all the other parameters are fixed. Moreover, it can also be seen that the regions for persistence and extinction vary in size with altering value of  $\beta$ , noting that Figure 8A-D are drawn with  $\beta = 0.6, 0, -0.6$ , and  $-\frac{1}{ku}$ , respectively, and the same set of values of all the other parameters.

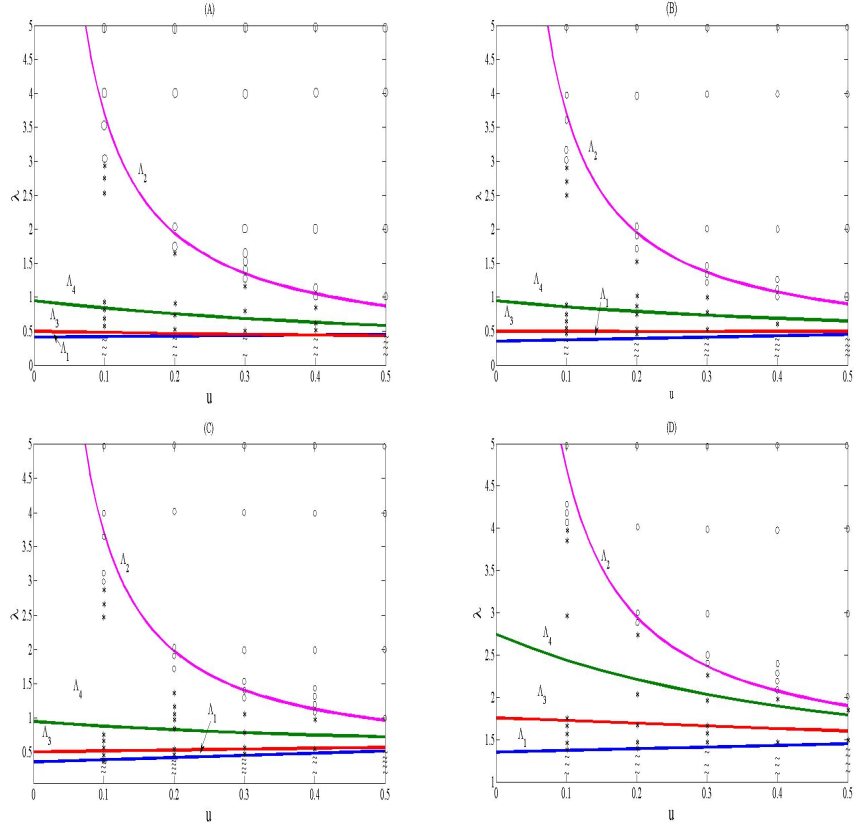


Figure 8: Region of disease extinction (host persistence) is denoted by 'tildes', region of disease persistence is denoted by 'stars', and region of host extinction is denoted by 'open circles' in  $(u, \lambda)$ -plane with initial condition  $(p, i) = (1, 0.0001)$ . (A)  $\alpha = 0, \beta = 0.6, \lambda > 0$ ; (B)  $\alpha = \beta = 0, \lambda > 0$ ; (C)  $\alpha = 0, \lambda > 0$ ; (D)  $\beta = -\frac{1}{ku}$  and the values of other parameters are as stated in Table 1.



## Discussion and conclusion

The Allee effects and parasitism are some of the extinction drivers that recently received considerable attention in extinction research. Their joint interplay have long been recognized to drive host population to extinction. An SI model with a strong Allee effect in which the vital dynamics (birth and death) are both modeled as quadratic polynomials is designed and rigorously analyzed. This approach provides ample opportunity for taking into account the major contributors to the Allee effect and makes the presented model more general. The specific choice of the mortality rate in Eq. (3) arises from the Allee mechanisms that affect survival or both survival and reproduction at all densities Berec et al. (2006) and intraspecific competition. This allow us to explore a range of values of the parameter  $\alpha$  and that of  $\beta$ , which determine the intensity of the Allee effects on both fertility and mortality rate functions. More specifically, the mortality rate decreases when  $\beta > 0$  and it increases if  $\beta \leq 0$  while the values of all the other parameters are fixed, showing that some species are more susceptible to the Allee effects than others. This follows from the fact that species whose individuals benefit from the presence of conspecifics are more susceptible to the Allee effects than others (Courchamp and Mackdonald, 2001; Stephens and Sutherland, 1999). This study generalizes some of the previous studies in the sense that if  $\alpha = \beta = 0$ , we have a nonlinear fertility function and a constant mortality rate as in Thieme et al. (2009). For  $\alpha = 0, \beta = -\frac{1}{ku}$ , the demographic functions of the proposed model are quadratic and linear similar to those in Hilker et al. (2009). In this case the presented model can be considered as an extension of the model of Hilker et al. (2009).

If  $\mathcal{R}_0 < 1$ , the host population will either undergo extinction or settles at the carrying capacity depending on the initial population size (Theorem 2). In such a case the disease cannot invade from arbitrarily small introductions into the host population at carrying capacity. From biological point of view, in the absence of disease the presence of an Allee effect in the host demographic plays a protective and a stabilizing role in relations to the disease invasion. On the other hand, in the presence of infection an additional disease-related mortality increases the likelihood of population extinction. More precisely, the disease establishes an effective host eradication threshold above the Allee threshold  $u$  (Theorem 7).

It is well known that the maximum degree of depression of a host population equilibrium is achieved by intermediate disease pathogenicity, i.e. low to moderate pathogenicity (Anderson, 1979; Anderson and May, 1979). In view of that, we obtain two important threshold quantities  $\lambda_0$  and  $\lambda_1$  of the transmissibility  $\lambda$ . If the disease pathogenicity is low ( $\mu < \mu^*$ ) such that  $\lambda > \lambda_0$ , the disease could establish itself in the host population (Figure 4). In contrast, if the disease pathogenicity is high, i.e.  $\mu > \mu^*$  such that  $\lambda \in (\lambda_0, \lambda_1)$ , the disease

can either invade the population or drive the host population to extinction depending on the initial sizes of the host and infected sub-populations due to the strong Allee effect. For some high value of  $\mu$ , the dynamics of system (4) could undergo some substantial changes as  $\lambda$  decreases from  $\lambda_1$  to  $\lambda_0$ . First, the stable endemic equilibrium becomes unstable and so, both the total population and the infected sub-population start oscillating in form of stable limit cycles (illustrated in Figure 5C). This mathematically corresponds to a Hopf bifurcation scenario. Then, the oscillations disappear as a result of a collision between the increasing limit cycles and the Allee threshold state  $E_1$ , which is known as a Homoclinic bifurcation. Despite the disappearance of the limit cycles, the unstable endemic equilibrium still persists and so, there is no endemic attractor any more. This leads to the extinction of the whole population. Moreover, the unstable endemic equilibrium also disappears when the total population falls below the Allee threshold due to disease related mortality. As expected, the model presented in Hilker et al. (2009) exhibits all these dynamical behaviors being a special case of the extended model in this paper. In addition, if the disease pathogenicity is high ( $\mu > \mu^*$ ) and  $\lambda > \lambda_1$  the disease drives the host population to extinction. Thus, the system is rendered monostable with the trivial extinction state  $E_0$  being the only global attractor, see for example Figure 5A. In particular, when  $\lambda > \lambda_1$  there is a disease-induced extinction for any initial state.

In a recent study, (Friedman and Yakubu, 2012a) used the model of Hilker et al. (2009) and focused on the role of the Allee effect at large population densities. Following the approach in (Friedman and Yakubu, 2012a), verifiable conditions for the special cases of the proposed model that guarantee host persistence (with or without infected individuals) and host extinction are derived *via* the relative position of a disease threshold to the Allee threshold and host population carrying capacity. This extinction scenario shows how a small perturbation to the disease-free equilibrium can lead to the catastrophic extinction of the host population (Theorems 8 and 10). In addition, we prove that there is an effective increase in the Allee threshold when a fatal disease invades the host population whose demographics are manifested with a strong Allee effect (Theorems 7 and 9).

In summary, the results of the proposed model indicate that if a strong Allee effect would be present in the host demographic both the host and disease dynamics might be very sensitive to parameter perturbations. As a consequence, intensified host population depression, increased prevalence, cycling, or host extinction could be the possible outcomes. These results are obtained before in (Friedman and Yakubu, 2012a; Hilker et al., 2009), which shows the robustness of the presented model. In addition, the quadratic birth and death rates functions introduced here effectively capture species' susceptibility variations due to

the Allee effects since the range of values of the parameters  $\alpha$  and  $\beta$  determine the intensity of the Allee effects on both the fertility and mortality rate functions  $B(P)$  and  $D(P)$ . This support the results in (Courchamp and Mackdonald, 2001; Stephens and Sutherland, 1999) that an Allee effect is more intense in some species than others and the fact that animal species that aggregate due to the Allee effects would be more prone to negative effects of parasite Christe et al. (2006).

Mathematically, we provide a new approach to investigate species' differential susceptibility to the Allee effects. An approach which makes the presented model more general than some of the previous studies (Hilker et al., 2009; Thieme et al., 2009). Indeed, determining the range of values of the parameters  $\alpha$  and  $\beta$  which determine the intensity of the Allee effects on both the fertility and mortality rate functions  $B(P)$  and  $D(P)$  would be of crucial importance in ecology and conservation for identifying potential extinction risks and guiding management actions.

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### Appendix A. Proof of Theorem 4

*Proof.* It should be noted that  $E^*$  is an endemic equilibrium point with  $i^* > 0$  if and only if  $x = p^*$  is a zero of the function

$$\begin{aligned} q(x) &= kx(1-x)(x-u) - \frac{\mu}{\lambda}[\tau + k(\alpha x^2 - \beta ux)][\mathcal{R}(x) - 1] \\ &= kx(1-x)(x-u) - \mu x \frac{[\mathcal{R}(x) - 1]}{\mathcal{R}(x)}. \end{aligned}$$

This may occur only in the interval  $(p_t)_1 < x < 1$ . It can be verified that  $q(1) < 0$ ,  $q[(p_t)_1] > 0$  and  $q''(x) < 0$  if  $u < x < 1$ . Thus, by Intermediate Value

Theorem Stewart (2008),  $q(x)$  has precisely one zero  $x = p^*$  in the interval  $(p_t)_1 < x < 1$ . But the slope of the  $i$ -nullcline is greater than that of the  $p$ -nullcline at  $E^*$ , that is,

$$Q(p^*) < \frac{\mu}{\mathcal{R}(p)} \left\{ [\mathcal{R}(p) - 1] + \frac{p}{\mathcal{R}(p)} \frac{d}{dp}[\mathcal{R}(p)] \right\}. \quad (\text{A.1})$$

The Jacobian matrix of system (4) at  $E^*$  is

$$J(E^*) = \begin{pmatrix} Q(p^*) & -\mu \\ (\lambda - k(2\alpha p^* - \beta u))i^* & H_{i^*} \end{pmatrix},$$

where

$$H_{i^*} = \frac{\lambda p^*}{\mathcal{R}(p^*)}[\mathcal{R}(p^*) - 1] - 2\lambda i^*, \quad i^* = \frac{p^*}{\mathcal{R}(p^*)}[\mathcal{R}(p^*) - 1].$$

The eigenvalues of  $J(E^*)$  have negative real parts if the trace,  $tr[J(E^*)] < 0$  and the determinant,  $det[J(E^*)] > 0$ . That is

$$tr(E^*) = Q(p^*) + H_{i^*} = Q(p^*) - \frac{\lambda p^*}{\mathcal{R}(p^*)}[\mathcal{R}(p^*) - 1] < 0,$$

which is the inequality (11) and

$$\begin{aligned} det[J(E^*)] &= Q(p^*) \left\{ \frac{\lambda p^*}{\mathcal{R}(p^*)}[\mathcal{R}(p^*) - 1] - 2\lambda i^* \right\} \\ &\quad + \frac{\mu}{\mathcal{R}(p^*)} \left\{ [\mathcal{R}(p^*) - 1] + \frac{p^*}{\mathcal{R}(p^*)} \frac{d}{dp}[\mathcal{R}(p^*)] \right\} i^*, \\ &= \frac{\lambda p^*}{\mathcal{R}(p^*)}[\mathcal{R}(p^*) - 1] \\ &\quad \times \left\{ \frac{\mu}{\mathcal{R}(p^*)} \left( [\mathcal{R}(p^*) - 1] + \frac{p}{\mathcal{R}(p)} \frac{d}{dp}[\mathcal{R}(p^*)] \right) - Q(p^*) \right\} > 0 \end{aligned}$$

only with inequality (A.1). ■

## Appendix B. Proof of Theorem 7

*Proof.* Considering Lemma 2, it suffices to show that  $i(t)$  is bounded from below in the interval  $t_a < t < t_b$ , where  $i(t) \leq \rho$  and  $t_a > 0$ . Let  $(t_a, t_b)$  be a maximal interval for which  $i(t) \leq \rho$  so that  $i(t_a) = \rho$  and then,  $i(t) > \rho$  for some  $t < t_a$ . Suppose  $t_1$  is the largest value of  $t, t < t_1$  such that  $i(t)$  is monotonically decreasing from  $t_1$  to  $t_a$ . Then,

$$i(t) < i(t_1) \text{ for } t_1 < t < t_a \tag{B.1}$$

and

$$\frac{di(t_1)}{dt} = 0,$$

or by the second equation of (12),

$$-\tau + (\lambda + k\beta u)p(t_1) - \lambda i(t_1) = 0. \tag{B.2}$$

Hence,

$$\lambda p(t_1) = p_T + \frac{\lambda}{\lambda + k\beta u} i(t_1) > p_T = u + \rho_1, \quad (\text{B.3})$$

where  $\rho_1 \equiv p_T - u > 0$ .

Now, we claim that there exists  $\rho_0^* \in (0, \rho_1)$  such that

$$p(t) > u + \rho_0^* \text{ for all } t_1 < t \leq t_a. \quad (\text{B.4})$$

In fact, since  $p(t_1) > u + \rho_1 > u + \rho_0^*$ , if the assertion (B.4) is not true, then there exists  $t_1^* \in (t_1, t_a)$  such that

$$p(t) > u + \rho_0^* \text{ if } t_1 < t < t_1^*, p(t_1^*) = u + \rho_0^*.$$

Thus,

$$\frac{dp(t_1^*)}{dt} \leq 0,$$

or by the first equation of (12), we have

$$k[1 - (u + \rho_0^*)]\rho_0^*(u + \rho_0^*) - \mu i(t_1^*) \leq 0$$

so that

$$i(t_1^*) \geq \frac{k}{\mu} [1 - (u + \rho_0^*)]\rho_0^*(u + \rho_0^*).$$

But, by inequality (B.1) and Eq. (B.2),

$$i(t_1^*) \leq i(t_1) = \frac{1}{\lambda} [(\lambda + k\beta u)p(t_1^*) - \tau] < \frac{1}{\lambda} [(\lambda + k\beta u) - \tau],$$

so that,

$$k[1 - (u + \rho_0^*)]\rho_0^*(u + \rho_0^*) < \frac{\mu}{\lambda} (\lambda + k\beta u)(1 - p_T).$$

Indeed, this is a contradiction to inequality (26) when  $\rho_0^*$  is chosen for  $x = u + \rho_0^*$  to be the value at which the left-hand side of (26) attains the maximum. We infer that with this chosen value of  $\rho_0^*$ , (B.4) holds and specifically,

$$p(t_a) > u + \rho_0^*.$$

Therefore, by applying Lemma 2, we can now deduce that

$$t_b - t_a < t^0(\rho, \rho_0^*).$$

Considering the way  $\rho_0^*$  is determined,  $t^0(\rho, \rho_0^*)$  may be taken as a function depending on  $\rho$  only (*i.e.*  $t^0 = t^0(\rho)$ ). We also observe from the second equation of (12) that

$$\frac{di}{dt} \geq -ci \text{ for all } t > 0,$$

where  $c > 0$  is a constant. Therefore,

$$i(t) \geq \rho e^{-c(t_b-t_a)} \geq \rho e^{-ct^0(\rho)} \equiv \eta \text{ if } t_a < t < t_b.$$

Indeed, this estimate is true for any such interval  $t_a < t < t_b$ , where  $i(t) \leq \rho$  and  $i(t_a) = \rho$ . It follows that,  $i(t) > \eta$  if  $t > t^0[\rho, \rho_0, i(0)]$  by combining this estimate and Lemma 1.  $\blacksquare$

### Appendix C. Proof of Theorem 8

*Proof.* Let  $\Lambda_i$  and  $\Lambda_p$  be the  $i$ - and  $p$ -nullclines of model (12) respectively. Define

$$\Pi_1 = \{(p, i) \in [0, \infty) \times [0, \infty) : i > 0, p_T < p < 1\},$$

$$\Pi_2 = \{(p, i) \in [0, \infty) \times [0, \infty) : i = 0, 0 < p \leq p_T\},$$

$$\Pi_3 = \{(p, i) \in [0, \infty) \times [0, \infty) : i > 0, u < p < 1\},$$

$$\Pi_4 = \{(p, i) \in [0, \infty) \times [0, \infty) : i = 0, 0 < p \leq u\}.$$

We denote by  $\Lambda_i^+$  the union of  $\Lambda_i \cap \Pi_1$  and the interval,  $\Pi_2$ , that is

$$\Lambda_i^+ = \Lambda_i \cap \Pi_1 \cup \Pi_2$$

and  $\Lambda_p^+$  the union of  $\Lambda_p \cap \Pi_3$  and the interval,  $\Pi_4$ , so that

$$\Lambda_p^+ = \Lambda_p \cap \Pi_3 \cup \Pi_4.$$

Therefore,

$$\frac{dp}{dt} > 0 \text{ below } \Lambda_p^+ \text{ and } \frac{dp}{dt} < 0 \text{ above } \Lambda_p^+,$$

$$\frac{di}{dt} > 0 \text{ below } \Lambda_i^+ \text{ and } \frac{di}{dt} < 0 \text{ above } \Lambda_i^+.$$

Considering condition (30), where

$$\max_{u \leq x \leq 1} \left\{ k(1-x)(x-u)x - \frac{\mu(\lambda + k\beta u)}{\lambda}(x-p_T) \right\} < 0$$

we have

$$\Lambda_i \cap \{(p, i) \in [0, \infty) \times [0, \infty) : i > 0\}$$

to be strictly above

$$\Lambda_p \cap \{(p, i) \in [0, \infty) \times [0, \infty) : i > 0\}.$$

Thus,

$$\frac{di}{dt} \geq \rho_1 \text{ below } \Lambda_p^+ \text{ and } \frac{dp}{dt} \leq -\rho_1 \text{ above } \Lambda_i^+, \text{ for some } \rho_1 > 0.$$

Hence, every trajectory of model (12) must cross  $\Lambda_i^+$  at some time to enter the region above  $\Lambda_i^+$  unless it is initially above it. Then it remains there so that  $p(t) \rightarrow 0$  and  $i(t) \rightarrow 0$  as  $t \rightarrow \infty$ .

Consider now the case when equality holds in inequality (30) with  $\rho = 0$ . Then,  $\Lambda_p^+$  and  $\Lambda_i^+$  are tangent to each other with point of intersection  $\omega = (p^*, i^*)$ . The Jacobian matrix of (12) evaluated at  $\omega$

$$J(\omega) = \begin{pmatrix} Q(p) & -\mu \\ (\lambda + k\beta u)i^* & -\lambda i^* \end{pmatrix}$$

has eigenvalues 0 and  $\frac{\mu(\lambda+k\beta u)}{\lambda} - \lambda i^*$ . Therefore,  $\omega$  is a single degenerate point, so that no trajectory  $(p(t), i(t))$  which is above  $\Lambda_i^+$  can converge to  $\omega$  for some time  $t = t_1$  as  $t$  approaches infinity.

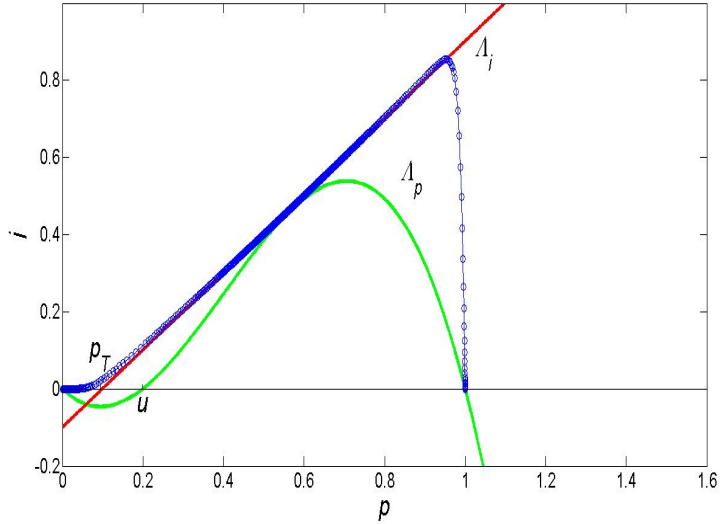


Figure 9: Phase plane of model (27) with the  $i$ -nullcline  $\Lambda_i$  tangent to the  $p$ -nullcline  $\Lambda_p$  and a solution (circles) with initial condition  $(1, 0.0001)$  converges to  $(0, 0)$  as  $t \rightarrow \infty$ . Here,  $k = 1.2, \mu = 0.232, \gamma = 1.27, u = 0.2, \beta = 0.07$  and  $\lambda = 19.997$ .

It follows, as illustrated in Figure 9, that any trajectory  $(p(t), i(t))$  with  $(p(0), i(0)) = (1 - \eta_1, \eta_2)$  must cross  $\Lambda_p^+$  and  $\Lambda_i^+$ , where  $\eta_1 \geq 0$  is sufficiently small and  $\eta_2 > 0$ . Hence,  $(p(t), i(t)) \rightarrow (0, 0)$  as  $t \rightarrow \infty$  as in the first case considered above. Precisely,  $p(t_\eta) < \frac{1}{2}u$  for some finite time  $t_\eta$ .

Furthermore, when  $\rho$  in condition (30) is small enough, the  $i$ -nullcline  $\Lambda_i$  intersects the  $p$ -nullcline  $\Lambda_p$  at two points  $\omega_1$  and  $\omega_2$  such that  $|\omega_1 - \omega|$  and  $|\omega_2 - \omega|$  are sufficiently small. Then, by continuity, the corresponding trajectory  $(p^*(t), i^*(t))$  with  $(p^*(0), i^*(0)) = (1 - \eta_1, \eta_2)$  satisfies  $p^*(t_\eta) < u$ , where  $\eta_1 \geq 0$  is small enough and  $\eta_2 > 0$ . Hence,  $p^*(t) \rightarrow 0$  as  $t \rightarrow \infty$ .

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