What is the impact of disease-induced death in a Predator-Prey model experiencing an infectious disease ?

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RÉSUMÉ. Dans ce travail, nous discutons de l'incidence que peut avoir la surmortalité due à une maladie infectieuse sur la dynamique d'un modèle Proie-Prédateur de type Leslie-Gower avec maladie chez les Proies. La maladie infectieuse a le formalisme épidémiologique SIS (Susceptible-Infecté-Susceptible). Nous procédons à une analyze qualitative du modèle nous permettant de calculer des seuils écologiques qui résument les résultats de stabilité des différents équilibres. Nous mettons en exergue des conditions pour lesquelles la maladie disparaîtrait de la commuanauté ou deviendrait endémique. Finalement, nous présentons des simulations numériques qui illustrent nos résultats analytiques.

ABSTRACT. In this paper, we discuss the incidence of disease-induced death in a Leslie-Gower Prey-Predator model subjects to an infectious disease affecting only Preys. The infectious disease has the epidemiological SIS (Susceptible-Infectious-Susceptible) formalism. We carry out a qualitative analysis through which we compute ecological thresholds involving biological parameters of Preys, Predators and disease dynamic. We further investigate stability results of model steady states. We further highlight conditions, involving ecological thresholds, under which disease will disappear from the community or will become endemic. Finally, we show some numerical simulations in order to illustrate our analytical results.

MOTS-CLÉS : Modélisation, Maladie infectieuse, Surmortalité due à la maladie, Analyse qualitative

KEYWORDS : Modelling, Infectious disease, disease-induced death, Qualitative analysis

1. Introduction

A Leslie-Gower Predator-Prey model is a two species food chain with the particularity that the carrying capacity of Predator population is proportional to the number of Preys i.e. when there is a few quantity of Preys, predation is negligible so Predators find alternative foods ([10]). Since Predators and Preys that are involved in this model can be subjected to infectious disease, a major issue in mathematical modelling is to understand the effects of infectious diseases in regulating natural populations, decreasing their population sizes or reducing their natural fluctuations ([2], [9], [10], [8]). Many studies have been carried out in order to analyze the influence of infectious disease in Predator-Prey dynamics through mathematical modelling. Generally, there are more macroparasitic infections which can affect only preys, only predators or preys and predators. According to several epidemiological models and studies, infectious disease is able to leads a sur-mortality in the host population ([1], [3]).

Disease-induced death has been identify by a wide of authors as able to lead the so-called 'backward bifurcation' in epidemiological models ([1], [3] and references therein). Recall that in mathematical modelling theory, a backward bifurcation occurs when the disease-free equilibrium and the endemic equilibrium are simultaneously stable when a given threshold takes some values ([1] [3]). In other words, the infectious disease will not die out from the population. From public health policies, backward bifurcation is the worth think that can happen.

Based on that observations, a natural question that concerns the modelling of Predator-Prey dynamics experiencing infectious disease is : what is the incidence of disease-induced death in the outcomes of the model ? Despite the fact that there exist several study on Predator-Prey modelling in presence of infectious disease, this particular question has been scarcely addressed. Therefore, this paper aims to give an answer to that question at least for the particular case of the Leslie-Gower Predator-Prey model that has been widely study in the literature ([10] and references therein). For the authors knowledge, this paper is the first that addresses the question of taking into account or not disease-induced death in eco-epidemiological models.

2. The model formulation

Following ([6], [7]), the Leslie-Gower Predator-Prey model is given by

$$\begin{cases} \dot{H}(t) = (r_1 - a_1 P(t) - b_1 H(t))H(t), \qquad \dot{P}(t) = \left(r_2 - a_2 \dfrac{P(t)}{H(t)}\right) P(t), \\ H(0) > 0, \quad P(0) \geq 0 \end{cases} \tag{1}$$

where H denotes the Prey population, P the Predator population, r_1 the intrinsic growth rate of the Preys, r_2 is the intrinsic growth rate of the Predators, a_1 is the predation rate per unit of time, $K = \dfrac{r_1}{b_1}$ is the carrying capacity of the Prey's environment and $\dfrac{r_2}{a_2}H$ is the "carrying capacity" of the Predator's environment which is proportional to the number of Prey.

The major objective here is to combine the preceding model (1) and an epidemiological SIS compartmental model, in order to analyze the influence of SIS infectious disease in a Predator-Prey community. The following hypothesis hold true in our model
(H1) The disease transmission follows the mass action law.

(H2) There is a disease-induced death for infectious populations.
(H3) The infected population do not become immune.
(H4) It is assumed that Predator cannot distinguish the infectious and healthy Preys.
(H5) We assume that only susceptible Preys are capable of reproducing.
Note that assumptions (H3)-(H5) was already described in [10]. Recall that irrespective to [10] our model acknowledges a major mechanism of infectious disease dynamic : the disease-induced death of infectious individuals.

3. Mathematical analysis

We start this study by recalling some meaningful results of model (1). The following results hold for system (1).

Theorem 3.1 *1) The nonnegative orthant $\mathbf{R}^2_+$ is positively invariant by system (1).*

2) Let $\varepsilon > 0$, the set $D = \left\{(H,P) : 0 < H \leq K+\varepsilon, 0 \leq P \leq \frac{r_2}{a_2}(K+\varepsilon)\right\}$ is a feasible region for system (1).

3) System (1) don't admit periodic solutions.

4) The predator-free equilibrium $E_1 = \left(\frac{r_1}{b_1}, 0\right) = (K,0)$ is a saddle point with stability for Prey population and instability for Predator population.

5) The coexistence equilibrium $E_2 = (H^*, P^*) = \left(\frac{r_1 a_2}{a_1 r_2 + a_2 b_1}, \frac{r_1 r_2}{a_1 r_2 + b_1 a_2}\right)$ is globally asymptotically stable (GAS).

Proof 3.1 *See Appendix A.*

Now we reach the step of the formulation and the study of the eco-epidemiological Predator-Prey model. For this purpose, let the variables S and I denote respectively the susceptible and infectious in Prey population. We further assume a density-dependent demographic mechanisms (birth and death) for Preys ([2]). Specifically, the parameter $0 \leq \theta \leq 1$ is such that $b - \frac{r_1 \theta H}{K}$ is the birth rate coefficient, $\mu + \frac{(1-\theta) r_1 H}{K}$ is the mortality rate, $r_1 = b - \mu$ is the intrinsic growth rate of Preys. The restricted growth in the logistic equation is due to a density-dependent death rate when $\theta = 0$, is due to a density-dependent birth rate when $\theta = 1$, and is due to a combination of these when $0 < \theta < 1$. σ denotes the recovery rate of infectious Preys. λ is the adequate contact rate between susceptibles and infectious in Prey that leads to disease transmission while d denotes the disease-induced death rate.
Based on these biological premise together with assumptions (H1)-(H6), the Leslie-Gower Predator-Prey model when the disease is present in Preys reads as

$$\left\{\begin{array}{rcl} \dot{H} &=& r_1\left(1-\frac{H}{K}\right)H - a_1 PH - dI, \\ \dot{S} &=& \left(b - r_1\theta\frac{H}{K}\right)H - \left[\mu + \frac{(1-\theta)r_1 H}{K}\right]S - \lambda SI + \sigma I - a_1 SP, \\ \dot{I} &=& \lambda SI - \sigma I - \left[\mu + \frac{(1-\theta)r_1 H}{K}\right]I - a_1 IP - dI, \\ \dot{P} &=& \left(r_2 - \frac{a_2 P}{H}\right)P, \end{array}\right. \quad [2]$$

Using the fact that $H = S + I$, (2) is reduced to

$$\left\{\begin{array}{lcl} \dot{H} & = & r_1\left(1-\dfrac{H}{K}\right)H - a_1PH - dI, \\ \dot{I} & = & \lambda(H-I)I - \sigma I - \left[\mu + \dfrac{(1-\theta)r_1H}{K}\right]I - a_1IP - dI, \\ \dot{P} & = & \left(r_2 - \dfrac{a_2P}{H}\right)P, \\ H(0) & > & 0, \quad I(0) \geq 0, \quad P(0) \geq 0. \end{array}\right. \qquad [3]$$

Using a similar reasoning as in Theorem 3.1, the following results hold for system (3).

Lemma 3.1 *1) The nonnegative orthant $\mathbf{R}^3_+$ is positively invariant by system (3).*

2) Let $\varepsilon > 0$, the set D defined as

$$D = \left\{(H, I, P) : 0 < H \leq K + \varepsilon, 0 \leq I \leq H, 0 \leq P \leq \frac{r_2}{a_2}(K+\varepsilon)\right\}$$

is a feasible region for system (3).

In order to analyze the impact of the disease-induced death rate on the outcomes of model (3), in the sequel, we will distinguish to cases. First, the case where $d = 0$ and second, $d > 0$.

3.1. The eco-epidemiological model without disease-induced death

Here we start, by assuming that the infectious disease does not lead supplement deaths. Therefore we should set $d = 0$ in model (3). Let

$$\mathcal{R}_1 = \frac{\lambda K}{\sigma + \mu + (1-\theta)r_1}, \quad \mathcal{Q}_1 = \frac{\lambda H^*}{\sigma + \mu + (1-\theta)b_1H^* + a_1P^*},$$

where H^* and P^* are given in Theorem 3.1. Setting the right hand side of model (3) equal to zero leads the following result.

Lemma 3.2 *Model (3) admits at most four equilibria :*

1) The point $E_1 = (K, 0, 0)$. That is, both Predators and disease die out.

2) When $\mathcal{R}_1$>1, the point $E_2 = \left(K, K\left(1 - \dfrac{1}{\mathcal{R}_1}\right), 0\right)$ is ecologically meaningful. In other words, Predators die out but disease persists in Preys.

3) The point $E_3 = (H^*, 0, P^*)$. There is a coexistence between Preys and Predators while disease dies out.

4) When $\mathcal{Q}_1 > 1$, the endemic point $E_4 = (H^*, I_e, P^*)$ with $I_e = H^*\left(1 - \dfrac{1}{\mathcal{Q}_1}\right)$ is ecologically meaningful.

Now we turn to investigate asymptotic stability results of equilibria of system (3). We first investigate local stability properties and further characterize their global asymptotic stability properties. To address local stability properties, we will compute jacobian matrix of system (3) at any of its equilibria. Recall that an equilibrium is locally asymptotically stable (LAS) whenever its jacobian matrix has eigenvalues with real part lying in negative real axis.

Theorem 3.2 *The following result holds for system (3).*

1) Both E_1 and E_2 are unstable.

2) If $\mathcal{Q}_1 < 1$ then E_3 is LAS.

3) Assume that the endemic equilibrium E_4 exists, that is, $\mathcal{Q}_1 > 1$ then it is LAS.

Proof 3.2 *See Appendix B.*

Remark 3.1 *At this step, it is not possible to conclude about what are the outcomes of model (3) when the threshold $\mathcal{Q}_1$ take the critical value 1. This issue will be addressed in the next result. Moreover, $\mathcal{Q}_1$ can be seen as the basic reproduction number of Preys when Predators are present while $\mathcal{R}_1$ can be seen as the basic reproduction number of Preys in absence of Predator. Recall that the basic reproduction number is the number of secondary infectious individuals that can be generated by an infectious individual, all over it infectious time, when he is in a population of susceptible individuals.*

We also derive the following result

Theorem 3.3 *1) If $\mathcal{Q}_1 \leq 1$ then E_3 is globally asymptotically stable (GAS).*

2) Assume that the endemic equilibrium E_4 exists, that is, $\mathcal{Q}_1 > 1$ then it is GAS.

Proof 3.3 *See Appendix C.*

At this step, we have characterized, from a qualitative point of view, the outcomes of model (3) when there is no disease-induced death. In the next section, we will carry out a similar study in order to obtain elements to characterize the impact of the disease-induced death in the Leslie-Gower Predator-Prey model with disease in Preys.

3.2. The eco-epidemiological model with disease-induced death

This section is devoted to the study of model (3) with $d > 0$. As the starting point, we computed its equilibria. To achieve that objective, we set the right hand side of system (3) equal to zero. Let $\mathcal{R}_2 = \dfrac{\lambda(db_1a_2 + r_1r_2a_1)}{b_1d(a_1r_2 + (1-\theta)a_2b_1)}$. The following result is valid.

Lemma 3.3 *Model (3) admits at most four equilibria :*

1) The point $e_1 = (K, 0, 0)$. Both Predators and disease die out.

2) Assume $\mathcal{R}_1 > 1$ and let $0 < \overline{H} \leq K$ the positive solution of

$$-b_1\lambda H^2 + H(\lambda(r_1 - d) + db_1(1-\theta)) + d(\sigma + \mu + d) = 0. \quad [4]$$

Let also $\mathcal{Q}_2 = \dfrac{\lambda\overline{H}}{\sigma + \mu + (1-\theta)b_1\overline{H} + d}$. Therefore, if $\mathcal{Q}_2 > 1$ then the point $e_2 = \left(\overline{H}, \overline{H}\left(1 - \dfrac{1}{\mathcal{Q}_2}\right), 0\right)$ is a meaningful equilibrium. In other words, Predators die out but disease persists in Preys.

3) The point $e_3 = (H^*, 0, P^*)$. There is a coexistence between Preys and Predators while disease dies out.

4) Suppose that $\mathcal{R}_2 > 1$ and let $0 < H^\dagger \leq K$ the positive solution of

$$-\lambda\left(b_1 + \frac{a_1r_2}{a_2}\right)H^2 + H\left(\lambda(r_1 - d) + db_1(1-\theta) + d\frac{a_1r_2}{a_2}\right) + d(\sigma + \mu + d) = 0. \quad [5]$$

Let also $P^\dagger = \dfrac{r_2}{a_2}H^\dagger$ *and* $\mathcal{Q}_3 = \dfrac{\lambda H^\dagger}{\sigma+\mu+(1-\theta)b_1H^\dagger+d+a_1P^\dagger}$. *Therefore, if* $\mathcal{Q}_3 > 1$ *then the point* $e_4 = \left(H^\dagger, H^\dagger\left(1-\dfrac{1}{\mathcal{Q}_3}\right), P^\dagger\right)$ *is a meaningful equilibrium. It denotes the endemicity of the disease in Preys coexisting with Predators.*

Remark 3.2 *We stress the fact that in Lemma 3.3, assumptions* $\mathcal{R}_1 > 1$ *and* $\mathcal{R}_2 > 1$ *are necessary and sufficient to have the positive solution of (4) and (5), respectively, in the feasible domain. That is, lower than* K.
At this step, a first observation that can be made while comparing model (3) without and with disease-induced death is the complexity of computations of equilibria in the latter case.

Now we reach the step of characterizing the stability property of various equilibria. As previously (see Theorem 3.2), we will achieve that goal by characterizing the real parts of eigenvalues of the jacobian matrices computed at any of these equilibria. The following results address that issue. Theorem 3.4 is obtained similarly as Theorem 3.2, so we omit the proof.

Theorem 3.4 *The following result holds for system (3).*

1) Both e_1 *and* e_2 *are unstable.*

2) Let $\mathcal{Q}_3^* = \dfrac{\lambda H^*}{\sigma+\mu+(1-\theta)b_1H^*+d+a_1P^*}$. *If* $\mathcal{Q}_3^* < 1$ *then* e_3 *is LAS.*

The next result addresses the asymptotic stability of the endemic equilibrium.

Theorem 3.5 *Assume that the endemic equilibrium* $e_4 = \left(H^\dagger, H^\dagger\left(1-\dfrac{1}{\mathcal{Q}_3}\right), P^\dagger\right)$ *exists, that is* $\mathcal{R}_2 > 1$ *and* $\mathcal{Q}_3 > 1$. *Then is LAS.*

Proof 3.4 *See Appendix D.*

Remark 3.3 *From a qualitative point of view, one can conclude that irrespective of epidemiological models ([1], [3]), the Leslie-Gower Predator-Prey model experiencing infectious disease in Preys present similar results without and with disease-induced death. We observe in this study that the disease-induced death only leads more complexity in terms of analytical treatments of the model.*

4. Numerical simulations

In this section, we provide numerical simulations using an implicit nonstandard algorithm (see [10]) to illustrate and validate analytical results obtained in the previous sections. Indeed, as mentioned in [10], standard numerical methods (Euler, Runge Kutta methods, etc.) included in software package such as Scilab and Matlab sometimes present spurious behaviors which are not in adequacy with the continuous system properties that they aim to approximate i.e., lead to negative solutions, exhibit numerical instabilities, or even converge to the wrong equilibrium for certain values of the time discretization or the model parameters ([10]). Moreover, parameter values have been chosen in such a way

that they obey the conditions for stability or bifurcation. For our numerical treatments, we consider parameter values summarized in Table 1.

Tableau 1. *Parameter values for the Leslie-Gower predator-prey models*

Parameter	Value	Reference
r_1	1	Sharma et al. (2015) [8]
r_2	0.2	Sharma et al. (2015) [8]
a_1	0.1	Tewa et al. (2012) [9]
a_2	0.4	Sharma et al. (2015) [8]
b_1	0.01	Assumed
σ	0.1	Assumed
μ	0.2	Assumed
θ	0.8	Tewa et al. (2012) [9]

Figure 1 illustrates the coexistence of Preys and Predators in the disease-free case.

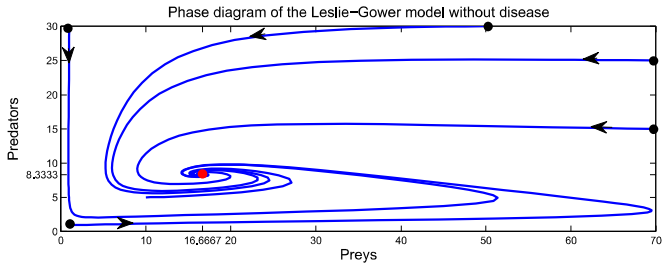

Figure 1. *Predators and Preys coexist in the disease-free case.*

When there is no disease-induced death and as we saw in Theorem 3.3, page 5, the threshold $\mathcal{Q}_1$ captures the whole dynamic of model 3. We illustrate it in figure 2.

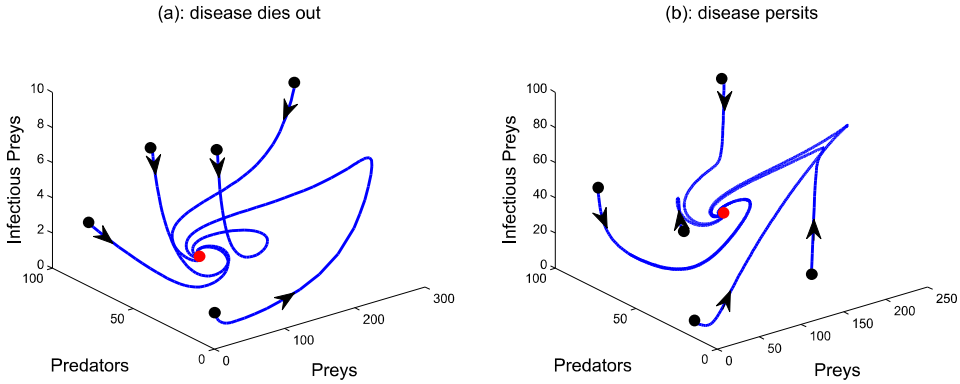

Figure 2. *Disease dies out ($\mathcal{Q}_1$<1) or persists ($\mathcal{Q}_1 > 1$). In panel (a), $\lambda = 0.006$, $d = 0$, in panel (b), $\lambda = 0.2$, $d = 0$. The rest of parameter values in Table 1.*

5. Conclusion

In this paper we carry out the study of a Leslie-Gower Predator-prey model experiencing an infectious disease only in Preys. We distinguished the cases where the model acknowledges or not a disease-induced death. Our qualitative analysis have highlighted several thresholds that summarize the whole dynamics of the model. We further compute conditions, involving afore-mentioned thresholds, under which the infectious disease will disappear or will become endemic in the community. Moreover, we can also conclude that, from a qualitative point of view, disease-induced death has not incidence in the outcomes of the model, irrespective of epidemiological finding ([1], [3]). However, this finding should be improved by the study of several other eco-epidemiological models. At this step, we just have a first indication, a first study and it remains to be validated by several others works. This paper just gives an insight concerning the question of taking into account or not disease-induced death in eco-epidemiological models. We finally illustrate our theoretical results with relevant numerical simulations.

6. Bibliographie

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A. Proof of Theorem 3.1

From system (1), one has for all $t \geq 0$,

$$\begin{array}{rcl} H(t) & = & H(0)\exp\left(\displaystyle\int_0^t (r_1 - a_1P(s) - b_1H(s))\,ds\right) > 0 \\ P(t) & = & P(0)\exp\left(\displaystyle\int_0^t \left(r_2 - \frac{a_2P(s)}{H(s)}\right)ds\right) \geq 0. \end{array} \quad [6]$$

Therefore, part 1 holds.

To prove part 2 we need to establish that the set D is a positively invariant and absorbing set. Let $([0,T), X = (H,P))$ be the maximal solution of the Cauchy problem (1) with $0 < T \leq +\infty$. Let $t_1 \in [0,T)$. It suffices to show that

– if $H(t_1) \leq K$ then for all $t \in [t_1, T)$, $H(t) \leq K$

– if $P(t_1) \leq \dfrac{r_2}{a_2}K$ then for all $t \in [t_1, T)$, $P(t) \leq \dfrac{r_2}{a_2}K$

since we have already shown that solutions are nonnegative. Assume that $\varepsilon_1 > 0$ exists such that $H(t_1 + \varepsilon_1) > K$. Let $t_1^* = \inf\{t \geq t_1 | H(t) > K\}$. Since $H(t_1^*) = K$, then $H(t) = K + H'(t_1^*)(t - t_1^*) + o(t - t_1)_{t \to t_1^*}$. Moreover, from the first equation of (1), $H'(t_1^*) = -a_1P(t_1^*)K \leq 0$. Then there exists $\xi > 0$ such that $\forall t_1^* \leq t < t_1^* + \xi$, $H(t) < K$ which is a contradiction. As a result, $\forall t \in [0,T), H(t) \leq K$. Similarly one can prove that if $P(t_1) \leq \dfrac{r_2}{a_2}K$ then for all $t \in [t_1, T)$, $P(t) \leq \dfrac{r_2}{a_2}K$.

Now we reach the step that aims to show that the set D is an absorbing set. From the first equation of system (1) one has $\dot{H}(t) \leq r_1\left(1 - \dfrac{H}{K}\right)H$ which implies that

$$H(t) \leq u(t) \to K \quad \text{as} \quad t \to +\infty,$$

where u is the unique solution of $\dot{u} = r_1\left(1 - \dfrac{u}{K}\right)u$ with $u(0) = H(0)$. Hence for all $\varepsilon > 0$, $\exists T_1 > 0 / H(t) \leq K + \varepsilon$, $\forall t > T_1$. Similarly, from the second equation of system (1) one has $\forall t > T_1$, $\dot{P}(t) \leq r_2\left(1 - \dfrac{a_2P}{r_2(K+\varepsilon)}\right)P$ which also implies that $P(t) \leq v(t) \to \dfrac{r_2}{a_2}(K + \varepsilon)$ as $t \to +\infty$, where v is the unique solution of $\dot{v} = r_2\left(1 - \dfrac{a_2v}{r_2(K+\varepsilon)}\right)v$ with $v(0) = P(0)$. Thus there exists $\exists T_2 > 0 / P(t) \leq \dfrac{r_2}{a_2}(K + \varepsilon)$. These end the proof of part 2.

To prove part 3, one uses the Dulac function $B(H,P) = \dfrac{1}{HP}$. Since $-r_1 < 0$ and $r_2 > 0$ are the eigenvalues of the jacobian matrix of system (1) at E_1, it follows that E_1 is a saddle point. Finally, to prove part 5 one can use the Lyapunov function proposed by Korobeinikov (see [5]).

B. Proof of Theorem 3.2

Since $r_2 > 0$ is an eigenvalue of the jacobian matrices of system (3) at E_1 and E_2, it therefore follows that both E_1 and E_2 are unstable.

Since the variable I does not appear in the first and the third equation of system (3) and together we Lemma 3.1 it suffices to compute the eigenvalue of the jacobian matrices

of both E_3 and E_4 in the I-direction. A direct computation leads that the eigenvalue of the jacobian matrix at E_3 is $\eta_{E_3,I} = \lambda H^* \left(1 - \frac{1}{\mathcal{Q}_1}\right)$ while at E_4 it is $\eta_{E_4,I} = -\lambda I_e$. Therefore, E_3 is LAS whenever $\mathcal{Q}_1 < 1$ while E_4 when it exists, i.e. $I_e > 0$, it is LAS. This ends the proof.

C. Proof of Theorem 3.3

Since system (3) is dissipative, that is, its solutions are bounded (see the feasible region D) then one can apply results on triangular systems (see Corollary 4 in [4]). Following Theorem 3.1, we deduce that $\lim_{t\to+\infty}(H,P)(t) = (H^*, P^*)$. Therefore, the limiting equation of variable I is $\dot{I} = \left(\lambda H^* \left(1 - \frac{1}{\mathcal{Q}_1}\right) - \lambda I\right) I$. Finally, it follows that if $\mathcal{Q}_1 \leq 1$ then $I \to 0$ and E_3 is GAS. Similarly, if $\mathcal{Q}_1 > 1$ then $I \to I_e$ and E_4 is GAS. This completes the proof.

D. Proof of Theorem 3.5

Since the endemic equilibrium $e_4 = \left(H^\dagger, H^\dagger \left(1 - \frac{1}{\mathcal{Q}_3}\right), P^\dagger\right)$ exists, that is $\mathcal{R}_2 > 1$ and $\mathcal{Q}_3 > 1$, one has

$$d - \lambda H^\dagger < 0. \qquad [7]$$

For simplicity, in the sequel we note H (resp. I, P) instead of $H^\dagger$ (resp. $I^\dagger$, $P^\dagger$). Moreover, let $A_1 = -b_1 H + d\left(1 - \frac{1}{\mathcal{Q}_3}\right)$; $A_2 = -d$; $A_3 = -a_1 H$;$A_4 = (\lambda - (1-\theta)b_1)I$; $A_5 = -\lambda I$; $A_6 = -a_1 I$; $A_7 = \frac{r_2^2}{a_2}$; $A_8 = -r_2$. $C_0 = A_1A_5A_8 + A_7A_2A_6 - A_7A_3A_5 - A_4A_2A_8$; $C_1 = -A_1A_5 + A_4A_2 - A_1A_8 - A_5A_8 + A_7A_3$; $C_2 = A_1 + A_5 + A_8$. Following Routh-Hurvitz theorem, the endemic equilibrium e_4 is LAS whenever $C_0 < 0$ and $C_2 < 0$ and $C_1C_2 + C_0 > 0$. Straightforward computations lead $C_2 = -b_H + (d - \lambda H)\left(1 - \frac{1}{\mathcal{Q}_3}\right) < 0$; $C_0 = -r_2\left(\lambda b_1 IH + \frac{dI(\sigma+\mu+d+a_1P)}{H}\right) + \frac{r_2^2}{a_2}a_1 I(d - \lambda H) < 0$;

$$\begin{aligned} C_1C_2 + C_0 = \quad & \left(-b_1H + (d-\lambda H)\left(1 - \tfrac{1}{\mathcal{Q}_3}\right)\right)\left(-d(1-\theta)b_1I - \lambda b_1IH - \lambda d\tfrac{I}{\mathcal{Q}_3}\right. \\ & -r_2b_1H + r_2(d-\lambda H)\left(1-\tfrac{1}{\mathcal{Q}_3}\right)\Big) + \tfrac{r_2^2}{a_2}(a_1b_1H^2) \\ & -r_2\left(-a_1H\tfrac{r_2^2}{a_2} + r_2\left(-b_1H + (d-\lambda H)\left(1-\tfrac{1}{\mathcal{Q}_3}\right)\right)\right) \\ & > 0. \end{aligned} \qquad [8]$$

Thus, when the endemic equilibrium, e_4, exists, it is LAS.