Role of horizontal incidence in the occurrence and control of chaos in an eco-epidemiological system

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A predator–prey model with disease in the prey population is proposed and analysed. The mode of disease transmission plays an important role in such dynamics. Keeping this factor in mind, we observe the dynamics of such a system for simple mass action incidence and standard incidence. Our observations indicate that the phenomenon of rarity or non-occurrence of chaos in our proposed model is well defined if the mode of disease transmission follows standard incidence. Moreover, using the method of Latin hypercube sampling, we show that the region of stability increases if the disease transmission follows the standard incidence law.

Keywords: eco-epidemiology; horizontal incidence; chaos; Lyapunov exponent; Latin hypercube sampling.

1. Introduction

Mathematical models have become important tools to analyse the spread and control of infectious diseases. Most models for the transmission of infectious diseases descend from the classical Susceptible-Infective-Recovered model of Kermack and McKendrick. Susceptible individuals become infectious by contact with infectious individuals. There are some biological differences between typical predator–prey interactions and infectious diseases, but these differences have identified some potentially fruitful avenues of research (Earn *et al.*, 1998). In the natural world, the species not only spreads the disease but also competes with other species for space or food or is predated by other species. Probably, Hadeler & Freedman (1989) were the first to describe a predator–prey model where the prey is infected by a parasite and the prey in turn infects the predator with the parasite. After that, a number of papers have already appeared in this direction (e.g. see Venturino, 1995, 2001; Hethcote, 2000; Chattopadhyay & Bairagi, 2001; Hethcote *et al.*, 2004, etc.) and these type of models are known as eco-epidemiological models (see Chattopadhyay & Arino, 1999).

Most of the earlier works on eco-epidemiology modelling are based on finding the stability and persistence of a system (e.g. Singh *et al.*, 2004). These models are analysed by considering a linear approximation to the non-linear equations that ecologists conventionally assume to be more complex situations (Hastings & Powell, 1991). But now the terms chaos, strange attractor and fractal are familiar to many, if not all, ecologists (Schaffer & Kot, 1986). In fact, Allen *et al.* (1993) showed that chaos can even prevent global population extinction if there are several distinct subpopulations that are weakly coupled by migration and subject to locally varying external noise. The key feature of chaotic dynamics is the sensitive dependence on initial conditions. Even a small change in initial conditions can lead

to different results. Chatterjee *et al.* (2006) proposed and analysed an eco-epidemiological model to observe the occurrence and control of chaos. They concluded that along with the rate of infection, the rate of predation also plays a pivotal role for monitoring the dynamics of the system.

Chatterjee *et al.* (2006) assumed that the disease transmission follows a simple mass action incidence law. It is seen that in the case of constant total population N, if the disease is not fatal and the model does not address vital dynamics (the normal birth and death dynamics), then the infection term bsi may be justified $\left(\text{since } \frac{b}{s+i} \text{ is now a constant}\right)$, where s is the susceptible population and i is the infective population. Here, the meaning of b becomes the encounter infection rate. But, for a large population, an individual's finite and often slow movements prevent it from making contact with a large number of individuals in a unit time. Such a mechanism is better described by $b\frac{si}{s+i}$ than bsi. For example, D'Amico *et al.* (1996) fitted a simple mass action model and found that the estimated transmission coefficient declined with both infected and susceptible host densities, showing that the simple mass action model was inadequate to describe the transmission process. Begon *et al.* (1998, 1999) concluded that standard incidence is a better descriptor of transmission dynamics than density-dependent transmission for cowpox. Many more small-scale experiments showed that simple mass action did not describe transmission adequately (e.g. see Reeson *et al.*, 2000; Barlow, 1991, 2000). The difference between the behaviour of different forms of the disease transmission are given in many papers (see Gao & Hethcote, 1992; Gao *et al.*, 1995, 1996; Hethcote & Van Ark, 1987; Mena-Lorca & Hethcote, 1992, to mention a few). In spite of the above observations, it is still an open question which functional form better describes the mode of disease transmission.

The main aim of this article is to compare the outcomes of two infection mechanisms, standard incidence or simple mass action incidence, with special emphasis on chaotic behaviour. To address this question, we have considered the model proposed by Chatterjee *et al.* (2006) and modified the model by assuming that the disease transmission follows standard incidence law. We compared the dynamical nature of the two systems numerically for a wider range of force of infection. Finally, we use Latin hypercube sampling (LHS), a stratified sampling technique that produces a more uniform distribution of sample points throughout the sample space (Smith *et al.*, 2005), to observe the dynamics of our considered model systems in the full range of parameter space. It helps us to indicate which form of the disease transmission will increase the stability region when the parameters are randomly generated. Our observations indicate that it is more easy to make the system stable around the positive interior equilibrium when disease transmission follows standard incidence instead of mass action incidence. It is also observed that the risk of getting chaos in the system becomes much less if the force of infection follows standard incidence.

The organization of the paper is as follows: We first discuss the basic formulation of the model in Section 2. In Section 3, we discuss some preliminary results that include the boundedness of the solutions and the conditions for the existence of different steady state. In Section 4, we find the conditions for the local stability of different equilibrium points and persistence of the coexistence equilibrium point. In Section 5, we perform the numerical simulations with special emphasis on the chaotic dynamic of the system. In Section 5.1, we have used LHS technique to see whether our result holds true when all the system parameters are varied simultaneously. The article ends with a discussion.

2. The basic mathematical model

Chatterjee *et al.* (2006) proposed a predator–prey model in which a transmissible disease is introduced into the prey population. They made the following assumptions.

They assumed that the disease spreads among the prey population and the disease is not genetically inherited. As a result, the total prey population is divided into two classes: one is the susceptible prey population 's' and the other is the infected prey population 'i'. Therefore, at any time t, the total prey population is $n(t) = s(t) + i(t)$. They assumed that the susceptible prey population grows in a logistic fashion with carrying capacity $k > 0$ and intrinsic growth rate constant $r > 0$. They further assumed that the infected prey population cannot grow, recover or reproduce. But the infected prey population is capable of contributing towards the carrying capacity of the susceptible prey population. The incidence is assumed to follow simple mass action incidence csi, where $c > 0$ is called the transmission coefficient. Finally, they assumed that the predator population p predates both the susceptible and the infected prey population and the predation of both the prey population has a positive effect on the growth rate of the predator population p

With these biological assumptions, they proposed the following mathematical model:

$$\frac{\mathrm{d}s}{\mathrm{d}T} = rs\left(1 - \frac{s+i}{k}\right) - pF(s,i) - csi,$$

$$\frac{\mathrm{d}i}{\mathrm{d}T} = csi - pG(s,i) - ei,$$

$$\frac{\mathrm{d}p}{\mathrm{d}T} = p(d_1F(s,i) + d_2G(s,i) - f),$$

where d_1 and d_2 are the conversion rates of the susceptible and the infected prey population, respectively, by the predator population. Moreover, d_1 and d_2 lie in the interval $(0, 1)$, e denotes the natural death rate of the infected prey populations and f is the natural death rate of the predator population. $F(s, i)$ and $G(s, i)$ denote the predator functional responses for the predator p, respectively.

They took the predator functional responses (which play an important role in determining the long-term behaviour of a system; Hastings & Powell, 1991) for the predator p with respect to the susceptible prey population s (i.e. $F(s, i)$) and the infected prey population i (i.e. $G(s, i)$) as modified Holling type-II functional responses (see Gakkhar & Naji, 2003), i.e.

$$F(s,i) = \frac{a_1 s}{1 + b_1 s + b_2 i} \quad \text{and} \quad G(s,i) = \frac{a_2 i}{1 + b_1 s + b_2 i},$$

which satisfy the following two conditions:

H1. $F(0, i) = 0$ and $\frac{\mathrm{d}F}{\mathrm{d}s} > 0, \forall s \geqslant 0$,

H2. $G(s, 0) = 0$ and $\frac{\mathrm{d}G}{\mathrm{d}i} > 0, \forall i \geqslant 0$,

where a_1 and a_2 are the searching efficiency constants or equivalently the predation rates on the susceptible and the infected prey populations, respectively, and b_1 and b_2 are the positive parameters characterizing the modified Holling type-II functional response.

With these assumptions, they proposed the following set of differential equations:

$$\begin{aligned}
\frac{\mathrm{d}s}{\mathrm{d}T} &= rs\left(1 - \frac{s+i}{k}\right) - \frac{a_1 sp}{1 + b_1 s + b_2 i} - csi,\\
\frac{\mathrm{d}i}{\mathrm{d}T} &= csi - \frac{a_2 ip}{1 + b_1 s + b_2 i} - ei,\\
\frac{\mathrm{d}p}{\mathrm{d}T} &= \frac{(d_1 a_1 s + d_2 a_2 i)p}{1 + b_1 s + b_2 i} - fp.
\end{aligned} \tag{2.1}$$

All the rate parameters are positive and constant. They assumed the positive initial conditions as $s(0) \geqslant 0$, $i(0) \geqslant 0$ and $p(0) \geqslant 0$ for some initial time.

Here, we modify the model of Chatterjee *et al.* (2006) by taking standard incidence as the mode of disease transmission. Under this assumption, System (2.1) takes the following form:

$$\begin{aligned}
\frac{\mathrm{d}s}{\mathrm{d}T} &= rs\left(1-\frac{s+i}{k}\right)-\frac{a_1sp}{1+b_1s+b_2i}-\frac{csi}{s+i},\\
\frac{\mathrm{d}i}{\mathrm{d}T} &= \frac{csi}{s+i}-\frac{a_2ip}{1+b_1s+b_2i}-ei,\\
\frac{\mathrm{d}p}{\mathrm{d}T} &= \frac{(d_1a_1s+d_2a_2i)p}{1+b_1s+b_2i}-fp.
\end{aligned} \tag{2.2}$$

For simplicity, we non-dimensionalize the model system (2.2) with the following scaling: $S=\frac{s}{k}$, $I=\frac{i}{k}$, $P=\frac{a_1p}{r}$ and $t=rT$; with these quantities, System (2.2) is transformed into a dimensionless form as follows:

$$\begin{aligned}
\frac{\mathrm{d}S}{\mathrm{d}t} &= S(1-S-I)-\frac{SP}{1+\alpha S+\beta I}-\frac{\lambda SI}{S+I},\\
\frac{\mathrm{d}I}{\mathrm{d}t} &= \frac{\lambda SI}{S+I}-\frac{\gamma IP}{1+\alpha S+\beta I}-\delta I,\\
\frac{\mathrm{d}P}{\mathrm{d}t} &= \frac{(e_1S+e_2I)P}{1+\alpha S+\beta I}-\mu P,
\end{aligned} \tag{2.3}$$

where $\lambda=\frac{c}{r}$, $\delta=\frac{e}{r}$, $\gamma=\frac{a_2}{a_1}$, $\alpha=b_1k$, $\beta=b_2k$, $\mu=\frac{f}{r}$, $e_1=\frac{d_1a_1k}{r}$ and $e_2=\frac{d_2a_2k}{r}$.

3. Preliminaries

3.1 *Positive invariance*

Let us put (2.3) in a vector form by setting

$$X=\mathrm{col}(S,I,P)\in R^3, \tag{3.1.1}$$

$$F(X)=\begin{bmatrix}F_1(X)\\F_2(X)\\F_3(X)\end{bmatrix}=\begin{bmatrix}S(1-S-I)-\dfrac{SP}{1+\alpha S+\beta I}-\dfrac{\lambda SI}{S+I}\\[2ex] \dfrac{\lambda SI}{S+I}-\dfrac{\gamma IP}{1+\alpha S+\beta I}-\delta I\\[2ex] \dfrac{(e_1S+e_2I)P}{1+\alpha S+\beta I}-\mu P\end{bmatrix}, \tag{3.1.2}$$

where $F\colon C_+\to R^3$ and $F\in C^\infty(R^3)$. Then, (2.3) becomes

$$\dot{X}=F(X), \tag{3.1.3}$$

with $X(0)=X_0\in R^3_+$. It is easy to check in (3.1.2) that whenever choosing $X(0)\in R^3_+$ such that $X_i=0$, then $F_i(x)|_{X_i=0}\geqslant 0$ $(i=1,2,3)$. Due to the lemma of Nagumo (1942), any solution of (3.1.3) with $X_0\in R^3_+$, say $X(t)=X(t;X_0)$, is such that $X(t)\in R^3_+$ for all $t>0$.

3.2 *The equilibria and their existence conditions*

System (2.3) possesses the following biological feasible equilibria (other then the positive equilibrium point): $E_0 \equiv (0,0,0)$, $E_1 \equiv (1,0,0)$, $E_2 \equiv (S',0,P')$, where $S' = \frac{\mu}{e_1-\mu\alpha}$ and $P' = \frac{e_1(e_1-\mu\alpha-\mu)}{(e_1-\mu\alpha)^2}$, $E_3 \equiv (\bar{S},\bar{I},0)$, where $\bar{S} = \frac{\delta(1-\lambda+\delta)}{\lambda}$ and $\bar{I} = \frac{(\lambda-\delta)(1-\lambda+\delta)}{\lambda}$.

REMARK 3.2.1 The equilibria E_0 and E_1 exist for any parametric value, while E_2 exists if $e_1 > \mu(\alpha+1)$ and E_3 exists if $\delta < \lambda < 1+\delta$.

We now seek the regions of parameter space for which the model system (2.3) admits a feasible interior equilibrium. Any feasible equilibria must correspond to a positive root S^* of the quadratic equation

$$w_1 S^2 + w_2 S + w_3, \tag{3.2.1}$$

where w_1, w_2, w_3 are given by

$$\begin{aligned}
\text{(i)}\quad w_1 &= 2\gamma\mu\alpha e_1 - \gamma\mu^2\alpha^2 + 2\gamma\mu^2\alpha\beta - 2\gamma e_1\mu\beta - \gamma\mu^2\beta^2 - \gamma e_1^2 \\
&\quad + 2\gamma e_1 e_2 - 2\gamma\mu\alpha e_2 - \gamma e_2^2 + 2\gamma e_2\mu\beta, \\
\text{(ii)}\quad w_2 &= \gamma e_2^2 + 2\gamma\mu e_1 - 2\gamma\mu^2\alpha - \gamma e_1 e_2 + \gamma\mu^2\beta^2 - \lambda e_2^2 + \delta e_2^2 \\
&\quad - \delta e_1 e_2 + \delta\mu^2\beta^2 - \lambda\gamma e_1\mu\beta + \lambda\gamma\mu^2\alpha\beta - \lambda\gamma\mu\alpha e_2 \\
&\quad - \gamma\mu^2\alpha\beta + \delta\mu\alpha e_2 - \delta\mu^2\alpha\beta + \delta e_1\mu\beta + 2\lambda e_2\mu\beta \\
&\quad - 2\delta e_2\mu\beta - 2\gamma\mu e_2 + 2\gamma\mu^2\beta + \lambda\gamma e_1 e_2 - 2\gamma e_2\mu\beta \\
&\quad + \gamma\mu\alpha e_2 + \gamma e_1\mu\beta - \lambda\mu^2\beta^2, \\
\text{(iii)}\quad w_3 &= \lambda\gamma\mu^2\beta - \gamma\mu^2 - \gamma\mu^2\beta - \delta\mu^2\beta + \gamma\mu e_2 - \lambda\gamma\mu e_2 + \delta\mu e_2,
\end{aligned} \tag{3.2.2}$$

for which additionally

$$I^* = \frac{\mu + (\alpha\mu - e_1)S^*}{e_2 - \mu\beta} > 0, \quad P^* = \frac{(e_1 S^* + e_2 I^*)((\lambda-\delta)S^* - \delta I^*)}{\mu\gamma(s^* + I^*)} > 0.$$

REMARK 3.2.2 The sufficient conditions for the existence of unique interior equilibrium point are as follows:

(i) $2(\mu(e_1\alpha + e_2\beta) + e_1 e_2 + \alpha\mu^2\beta) > \mu^2(\alpha^2+\beta^2) + 2\mu(e_1\beta + e_2\alpha) + e_1^2 + e_2^2$,
(ii) $\lambda\gamma > \delta$,
(iii) $e_2 + \lambda\mu\beta < \mu(1+\beta)$.

3.3 *Boundedness of the solutions*

Let us first recall (without proof) the following lemma due to Barbalat (1959).

LEMMA 3.3.1 Let g be a real-valued differential function defined on some half line $[a, +\infty)$, $a \in (-\infty, +\infty)$. If (i) $\lim_{t\to+\infty} g(t) = \alpha$, $|\alpha| < +\infty$, and (ii) $g'(t)$ is uniformly continuous for $t > a$, then $\lim_{t\to+\infty} g'(t) = 0$.

We shall prove the following key lemma.

LEMMA 3.3.2 Assume that the initial condition of (2.3) satisfies $S_0 + I_0 \geqslant 1$. Then, either (i) $S(t) + I(t) \geqslant 1$ for all $t \geqslant 0$ and therefore as $t \to +\infty$, $(S(t), I(t), P(t)) \to E_1 = (1, 0, 0)$ or (ii) there exists a $t_1 > 0$ such that $S(t) + I(t) < 1$ for all $t > t_1$. Finally, if $S_0 + I_0 < 1$, then $S(t) + I(t) < 1$ for all $t \geqslant 0$.

Proof. See Appendix A. □

LEMMA 3.3.3 Assuming $e_1 < 1$ and $e_2 < \gamma$, there is an $M > 0$ such that for any positive solution $(S(t), I(t), P(t))$ of System (2.3), $P(t) < M$ for all large t, where

$$M = \frac{1}{\xi}, \quad \xi = \min\{1, \delta, \mu\}.$$

Proof. See Appendix A. □

THEOREM 3.3.1 The set Ω is a global attractor in $R^3_{0,+}$ and, of course, it is positively invariant, where

$$\Omega = \{(S, I, P)\varepsilon R^3_{0,+} : S + I \leqslant 1, P \leqslant M\}.$$

Proof. See Appendix A. □

4. Local stability analysis and persistence

THEOREM 4.1 If the axial equilibrium is stable, then the disease-free equilibrium E_2 and the planar equilibrium E_3 do not exist, while the existence of E_2 or E_3 ensures the instability of E_1. The disease-free equilibrium E_2 is stable provided $\lambda - \gamma + \gamma\mu - \delta < 0$ and $e_1 + \mu\alpha < \alpha(e_1 - \mu\alpha)$. The planar equilibrium E_3 is stable if $\bar{\delta} > 0$, i.e. $(-\alpha + \beta)\delta^2 + (\lambda\alpha + \beta - \alpha - 2\beta\lambda)\delta - \lambda + \beta\lambda^2 - \beta\lambda > 0$.

Proof. The variational matrix J of System (2.3) around any arbitrary point (S, I, P) is given by

$$J(S,I,P) = \begin{bmatrix} 1 - 2S - I - \frac{(1+\beta I)P}{(1+\alpha S+\beta I)^2} - \frac{\lambda I^2}{(S+I)^2} & -S + \frac{\beta SP}{(1+\alpha S+\beta I)^2} - \frac{\lambda S^2}{(S+I)^2} & \frac{-S}{1+\alpha S+\beta I} \\ \frac{\lambda I^2}{(S+I)^2} + \frac{\alpha\gamma IP}{(1+\alpha S+\beta I)^2} & \frac{\lambda S^2}{(S+I)^2} - \frac{\gamma P(1+\alpha S)}{(1+\alpha S+\beta I)^2} - \delta & -\frac{\gamma I}{1+\alpha S+\beta I} \\ \frac{P(e_1+e_1\beta I-\alpha e_2 I)}{(1+\alpha S+\beta I)^2} & \frac{P(-e_2-e_2\alpha S+\beta e_1 S)}{(1+\alpha S+\beta I)^2} & \frac{(e_1 S+e_2 I)}{1+\alpha S+\beta I} - \mu \end{bmatrix}. \tag{4.1}$$

At the axial equilibrium E_1, we have

$$J(1,0,0) = \begin{bmatrix} -1 & -(1+\lambda) & \frac{-1}{1+\alpha} \\ 0 & \lambda - \delta & 0 \\ 0 & 0 & \frac{e_1}{1+\alpha} - \mu \end{bmatrix}. \tag{4.2}$$

Since $J(1,0,0)$ is a upper triangular matrix, its eigenvalues are -1, $\lambda-\delta$ and $\frac{e_1}{1+\alpha}-\mu$. Accordingly, E_1 is stable if $\frac{e_1}{1+\alpha} < \mu$ and $\lambda < \delta$, and is saddle if $\frac{e_1}{1+\alpha} > \mu$ or $\lambda > \delta$. Thus, the existence of the disease-free equilibrium E_2 or the planar equilibrium E_3 ensures the instability of E_1 and vice versa.

Consider now the disease-free equilibrium E_2. We have

$$J(S',0,P') = \begin{bmatrix} \frac{(-e_1\alpha+e_1+\mu\alpha^2+\mu\alpha)\mu}{e_1(e_1-\mu\alpha)} & \frac{(\beta\alpha+\beta)\mu^2+(-\beta e_1+e_1-\lambda e_1\alpha)\mu+\lambda e_1^2}{e_1(e_1-\mu\alpha)} & \frac{-\mu}{e_1} \\ 0 & \frac{(\gamma\alpha+\gamma-\lambda\alpha+\delta\alpha)\mu+\lambda e_1-\delta e_1-\gamma e_1}{e_1-\mu\alpha} & 0 \\ e_1-\mu\alpha-\mu & \frac{-(e_2-\mu\beta)(e_1-\mu\alpha-\mu)}{e_1-\mu\alpha} & 0 \end{bmatrix}. \tag{4.3}$$

The characteristic equation of (4.3) is given by

$$[-(e_1-\mu\alpha)x+(e_1-\mu\alpha)(\lambda-\gamma+\gamma\mu-\delta)][-e_1(e_1-\mu\alpha)x^2 + \mu(\mu\alpha^2+\mu\alpha+e_1-e_1\alpha)x-\mu(e_1-\mu\alpha)(e_1-\mu-\mu\alpha)] = 0. \tag{4.4}$$

We know that E_2 exists if $e_1 > \mu(\alpha+1)$. Hence, (4.4) will have roots with negative real parts if $\lambda-\gamma+\gamma\mu-\delta < 0$ and $e_1+\mu\alpha < \alpha(e_1-\mu\alpha)$.

Finally, at the planar equilibrium E_3, we have

$$J(\bar{S},\bar{I},0) = \begin{bmatrix} \frac{(-1+2\lambda-2\delta)\delta}{\lambda} & \frac{(-1+\lambda-2\delta)\delta}{\lambda} & \delta\bar{\delta} \\ \frac{(\lambda-\delta)^2}{\lambda} & -\frac{(\lambda-\delta)\delta}{\lambda} & \gamma(\lambda-\delta)\bar{\delta} \\ 0 & 0 & -(\delta e_1+e_2\lambda-\delta e_2)\bar{\delta}-\mu \end{bmatrix}, \tag{4.5}$$

where

$$\bar{\delta} = \frac{(1-\lambda+\delta)}{(-\alpha+\beta)\delta^2+(\lambda\alpha+\beta-\alpha-2\beta\lambda)\delta-\lambda+\beta\lambda^2-\beta\lambda}.$$

The characteristic equation of (4.5) is given by

$$[x+\bar{\delta}\delta e_1+\bar{\delta}e_2(\lambda-\delta)+\mu][\lambda x^2+\delta(1+\delta-\lambda)x+\delta(\lambda-\delta)(1+\delta-\lambda)] = 0. \tag{4.6}$$

Assuming $\bar{\delta} > 0$, we see from (4.6) that whenever E_3 exists, it is locally, asymptotically stable. Hence the theorem.

For the positive steady state $E^* \equiv (S^*, I^*, P^*)$, it is not an easy task to find the explicit criteria for the local stability of the interior equilibrium points in terms of the system parameters. The application of the Routh–Hurwitz criteria gives rise to a complicated mathematical expression, and as such we do not find its biological meaning. Before proceeding to numerical experiments of the system around E^*, we would like to study the persistence of System (2.3). Biologically, persistence means the survival of all populations for all future time. To examine the persistence of the model systems under consideration, we shall use the method of 'average Lyapunov function' (see Gard & Hallam, 1979; Hofbauer, 1981). This method was first applied by Hutson & Vickers (1983) on ecological problems. □

THEOREM 4.2 System (2.3) is persistent if

(i)

$$(e_1 - \mu\alpha)(\lambda - \gamma e_1 - \delta) + e_1\gamma\mu > 0,$$

(ii)

$$(1+\delta-\lambda)(\delta e_1 + e_2(\lambda-\delta)) > \mu(-\beta\lambda^2 + ((2\beta-\alpha)\delta + 1 + \beta)\lambda + (\alpha-\beta)\delta^2 + (\alpha-\beta)\delta) > 0.$$

Proof. We consider the average Lyapunov function of the form

$$V(S, I, P) = S^{\alpha_1} I^{\alpha_2} P^{\alpha_3},$$

where each α_i $(i = 1, 2, 3)$ is assumed to be positive. In the interior of $R^3_{0,+}$, we have

$$\begin{aligned}\psi(S, I, P) &= \frac{\dot{V}}{V} \\ &= \alpha_1\left[(1 - S - I) - \frac{P}{1+\alpha S+\beta I} - \frac{\lambda I}{S+I}\right] \\ &\quad + \alpha_2\left[\frac{\lambda S}{S+I} - \frac{\gamma P}{1+\alpha S+\beta I} - \delta\right] + \alpha_3\left[\frac{e_1 S + e_2 I}{1+\alpha S+\beta I} - \mu\right]. \end{aligned} \tag{4.7}$$

We have already proved that the solutions are bounded in the region Ω (see Section 3.3) and the trivial equilibrium is a repeller under certain conditions (see Appendix B). To establish the persistence of the solution, we have to show that $\psi(S, I, P) > 0$ at the equilibria $E_1, E_2, E_3 \in R^3_{0,+}$, for any $\alpha_i > 0$ $(i = 1, 2, 3)$.

For E_1, we have

$$\psi_1(S, I, P) = \frac{\dot{V}}{V}\bigg|_{E_1} = \alpha_1(\lambda - \delta) + \alpha_3\left(\frac{e_1}{1+\alpha} - \mu\right). \tag{4.8}$$

Thus, $\psi_1(S, I, P) > 0$ whenever E_2 and E_3 exist.

For E_2, we have

$$\begin{aligned}\psi_2(S, I, P) = \frac{\dot{V}}{V}\bigg|_{E_2} &= \alpha_1\left(\frac{e_1 - \mu\alpha - \mu - e_1^2 + e_1\mu\alpha + e_1\mu}{e_1 - \mu\alpha}\right) \\ &\quad + \alpha_2\left(\frac{\lambda e_1 - \lambda\mu\alpha - \gamma e_1^2 + \gamma e_1\mu(\alpha+1) - \delta e_1 + \delta\mu\alpha}{e_1 - \mu\alpha}\right). \end{aligned} \tag{4.9}$$

Thus, $\psi_2(S, I, P) > 0$ if $e_1 - \mu\alpha - \mu - e_1^2 + e_1\mu\alpha + e_1\mu > 0$ and $\lambda e_1 - \lambda\mu\alpha - \gamma e_1^2 + \gamma e_1\mu(\alpha + 1) - \delta e_1 + \delta\mu\alpha > 0$. Since $e_1 < 1$ (condition for the boundedness of the solution of System (2.3), see Lemma 3.3.3), $e_1 - \mu\alpha - \mu - e_1^2 + e_1\mu\alpha + e_1\mu > 0$. Thus, if Condition (i) of Theorem 4.2 holds, then $\psi_2(S, I, P) > 0$.

For E_3, we have

$$\psi_3(S, I, P) = \frac{\dot{V}}{V}\bigg|_{E_3} = \alpha_3 \left(\frac{(1+\delta-\lambda)(\delta e_1 + e_2(\lambda-\delta))}{-\beta\lambda^2 + ((2\beta-\alpha)\delta + 1 + \beta)\lambda + (\alpha-\beta)\delta^2 + (\alpha-\beta)\delta} - \mu \right). \tag{4.10}$$

Thus, $\psi_3(S, I, P) > 0$ if Condition (ii) of Theorem 4.2 holds.
This completes the proof. □

5. Numerical results

Due to the complexity of the model system, the only choice for investigating the long-term behaviour of System (2.3) is numerical integration. In our numerical study, we shall confine our analysis to System (2.2). We have performed our numerical simulations with the help of MATLAB (version 6.5) software. First, we shall study System (2.2) and compare the results with that of System (2.1). Then, for the better understanding of disease transmission dynamics, we have used LHS techniques.

The main objective of the present analysis to follow is to investigate the role of the disease transmission to maintain the stability of an eco-epidemiological system. This is a very important problem from the current research point of view as the question 'how should disease transmission be modelled?' remains unsolved (McCallum *et al.*, 2001). Several laboratory studies have been performed to find an appropriate solution of this question, some of which have been addressed in the introduction. Our second concern is on the occurrence of chaotic behaviour in such a system and the role of disease transmission in such occurrences.

It should be noted here that the two models, Systems (2.1) and (2.2), differ only in the functional response associated with the parameter c. So, it is very reasonable to compare the stability of the two systems around their interior steady states by varying the key parameter c and keeping all the other parameters fixed at some desired feasible levels. The model system (2.1) was proposed and analysed by Chatterjee *et al.* (2006). Accordingly, we begin our numerical analysis with the set of hypothetical parameter values (see Table 1) previously used by Chatterjee *et al.* (2006) to represent an eco-epidemiological system where the disease factors influence the predator–prey system dynamics.

Chatterjee *et al.* (2006) observed in their paper that for $c = 0.845$, System (2.1) enters into a chaotic region. But here we observe that for the same parameter values, System (2.2) is stable around the positive steady state (see Fig. 1).

Now, if we decrease the value of c from 0.845 to 0.48, retaining the other parameter values same, we observe that the dynamical behaviour of System (2.2) changes from a stable focus to a strange attractor (see Fig. 2). Figure 2 is obtained by letting the system run for 20,000 time steps and examining for the last 16,000 time steps to eliminate transient behaviour (see Hastings & Powell, 1991).

Our next task is to observe whether the strange attractor is a chaotic attractor or not. We begin the study by examining plots of each species of System (2.2) against time for $c = 0.48$. Dynamics that have irregular behaviour, suggestive of chaos (see Hastings & Powell, 1991), are illustrated in Fig. 3. It is clear that the solutions are bounded but not periodic (see Fig. 3) and there is no observable regularity in the time evolution for individual species, e.g. a varying number of secondary maxima between primary maxima for species s and i. The solution plots reveal wandering solutions of an irregularly oscillating type without any uniform pattern. These type of solutions are said to display chaotic behaviour (Jordon & Smith, 1999).

Moreover, we know that there are quite a good number of available sophisticated mathematical tools to analyse the dynamical behaviour of autonomous systems, in order to conclude the actual nature of

TABLE 1 *Default parameter values taken from the paper of Chatterjee et al. (2006), which is treated as the fixed set of parameter values in our numerical section*

Parameters/variable	Definition	Default values	Unit
r	Intrinsic growth rate	1	day^{-1}
k	Carrying capacity	1	individuals ha^{-1}
a_1	Predation rate on s	1	ha per individual day^{-1}
a_2	Predation rate on i	0.344	ha per individual day^{-1}
b_1	Positive constant	4.055	ha per individual
b_2	Positive constant	8.0	ha per individual
f	Death rate of p	0.02	day^{-1}
e	Death rate of i	0.051	day^{-1}
d_1	Conversion rate	0.21	—
d_2	Conversion rate	0.67742	—
$s(0)$	Initial value of s	0.094	individuals ha^{-1}
$i(0)$	Initial value of i	0.091	individuals ha^{-1}
$p(0)$	Initial value of p	0.05	individuals ha^{-1}

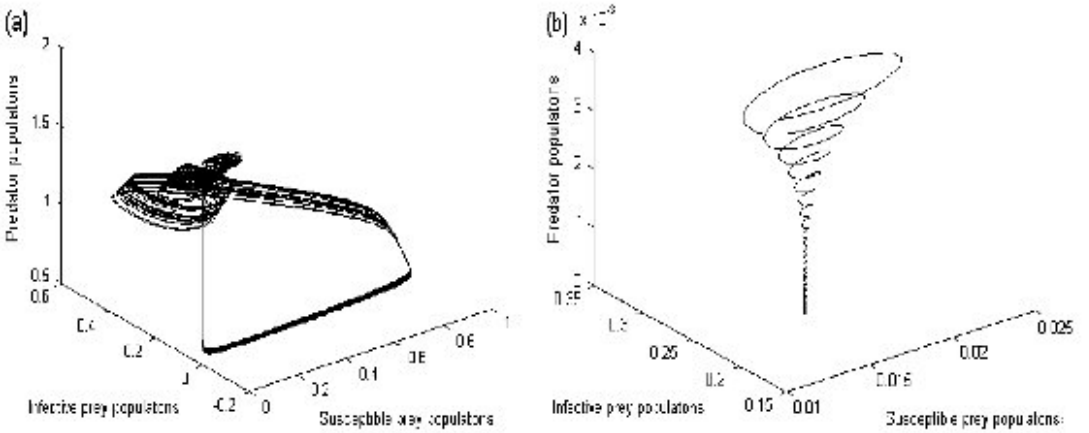

FIG. 1. The figure depicts the dynamics of (a) the system (2.1) with mass action incidence; and (b) the system (2.2) with standard incidence for $c = 0.845$.

it. Here, we find Lyapunov exponents (see Sprott, 2003) to show that the dynamics shown by System (2.2) for $c = 0.48$ is actually chaotic. A fundamental property of chaotic dynamics is sensitivity to small changes in initial conditions. Lyapunov exponents quantify this divergence by measuring the mean rate of exponential divergence of neighbouring trajectories. If the largest Lyapunov exponent of a trajectory is negative, then it is stable, while a trajectory with the largest Lyapunov exponent as zero is periodic, but if the largest Lyapunov exponent is positive then it is chaotic.

All the Lyapunov exponents corresponding to the strange attractor seen in Fig. 2 are depicted in Fig. 4, and it is clear that the largest Lyapunov exponent is positive ($\lambda_1 = 0.1221$, taking $\lambda_1 > \lambda_2 > \lambda_3$) and other two Lyapunov exponents are zero ($\lambda_2 \simeq 0.0$) and negative ($\lambda_3 = -0.195$), respectively. Consequently, the strange attractor is chaotic.

Now, we shall check for the sensitive dependence of the future dynamics on the current state, the final signature of chaos, where a small change in initial conditions may lead to different dynamical behaviour.

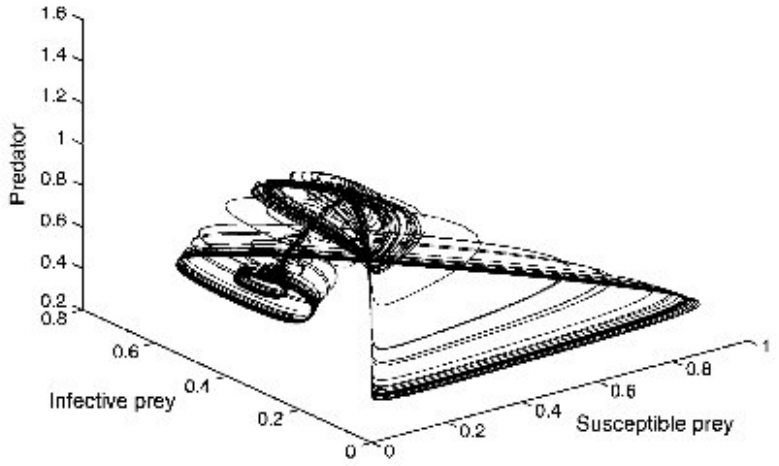

FIG. 2. The figure depicts the dynamics of System (2.2) (standard incidence) for $c = 0.48$.

We have illustrated this behaviour by comparing the trajectories generated by slightly different initial conditions. We have changed the initial value of the predator population by 0.01 (from 0.05 to 0.06), keeping the initial values of the susceptible and infected prey population fixed, and observed that the two initial conditions lead to dynamics that are essentially uncorrelated (although, of course, restricted to the attractor). It was seen that as time progresses, the system would be indeterminate (see Fig. 5). Thus, even a slight perturbation in species numbers, as would occur naturally, may lead to unpredictable results through time.

So, we observe that when $c = 0.48$, System (2.2) enters into a chaotic region. It is known that deterministic predator–prey models with strong periodic forcing have a complicated bifurcation diagram which includes limit cycles, the period-doubling route to chaos and the quasi-periodic route to chaos (Rinaldi *et al.*, 1993; Gragnani & Rinaldi, 1995). To understand the route to chaos, a systematic investigation of the dynamics was done by constructing a bifurcation diagram. Here, also we have run System (2.2) for 20,000 time steps and examined the last 16,000 time steps to eliminate transient behaviour. Then, we have plotted the successive maxima and minima of all the species with c as a function of the control parameter and other parameters are kept fixed at the level given in Table 1 (see Fig. 6).

One objective of studying chaos is to find the reasons behind the occurrence of such dynamics and hence to find a probable solution to control such dynamics. We observe from the bifurcation diagram (Fig. 6) that if the force of infection c is increased from 0.48 to 0.75, the dynamics of System (2.2) gradually changes from chaos to a stable focus. Thus, to keep System (2.2) stable around the positive steady state or to prevent the system from chaos, we shall have to keep the force of infection above certain threshold value $c_{I_1} = 0.75$.

We shall now try to find the role of simple mass incidence in the occurrence of chaos in an eco-epidemiological system and compare it with the standard incidence.

We observe from the bifurcation diagram (Fig. 7) that to keep System (2.1) stable around the positive steady state and to prevent the occurrence of chaos, we shall have to keep the force of infection above a certain threshold value $c_{I_2} = 2.8$ which is roughly four times that of c_{I_1}. So, we may say that it is more easy to keep System (2.2) stable around the positive steady state, than System (2.1).

The main result obtained by comparing the bifurcation diagrams (Figs 6 and 7) are presented in Table 2.

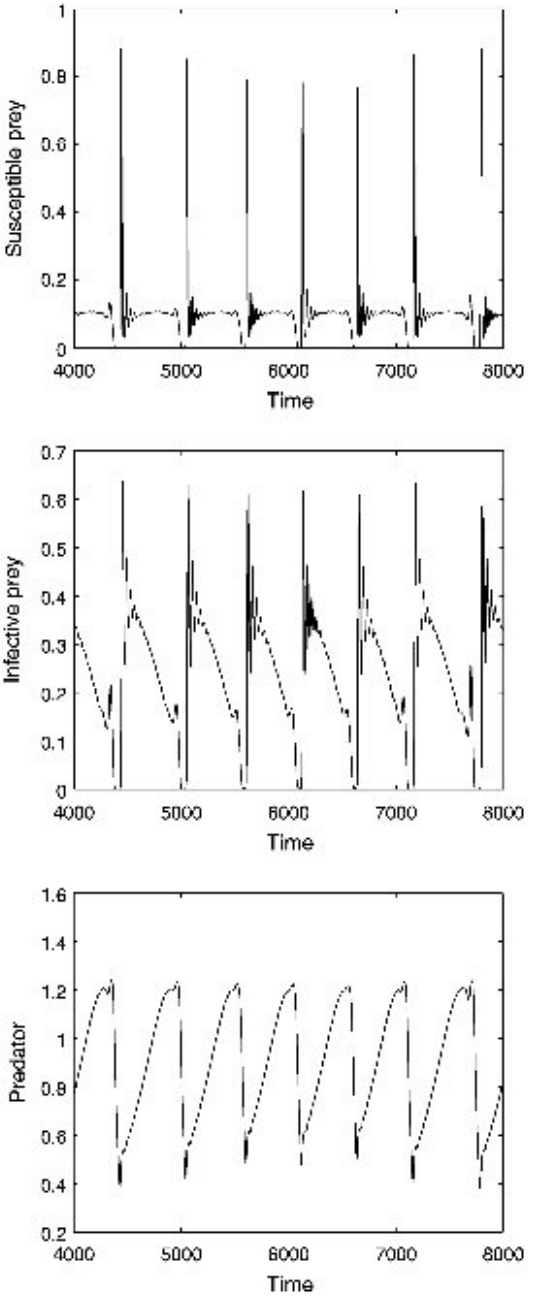

FIG. 3. Time evolution of different population components for the parametric value $c = 0.48$.

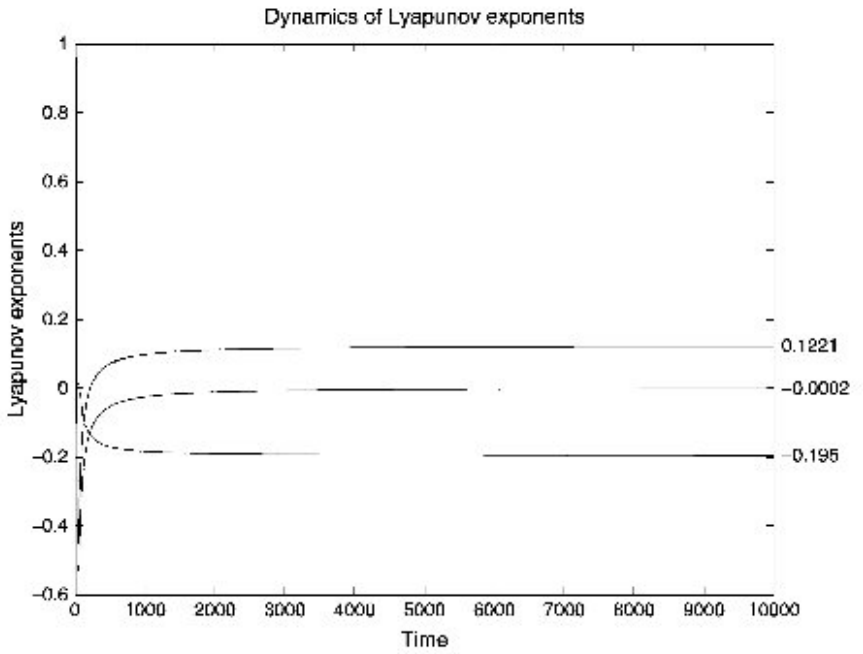

FIG. 4. The spectrum of Lyapunov exponent for System (2.2) around the strange attractor obtained for $c = 0.48$, see Fig. 2.

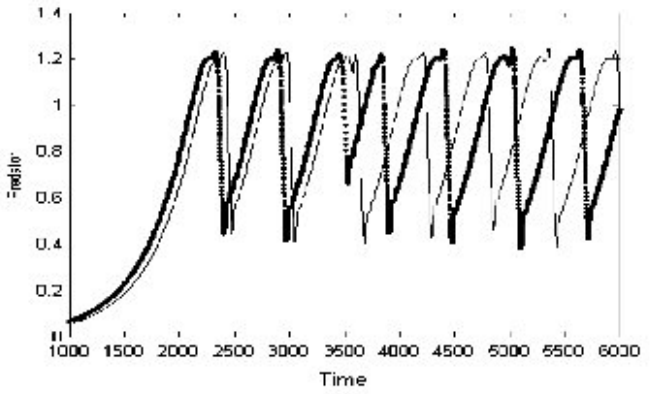

FIG. 5. Divergence of trajectories for the predator population for two different initial conditions '—' and '...' differing only by 0.01 with $s(0)$ and $i(0)$ unchanged.

From Table 2, it is clear that the range of c for which chaos occurs is much smaller for System (2.2) in comparison to System (2.1). Thus, we may conclude that the occurrence of chaos in the case of the population following standard incidence rate as the mode of disease transmission is much less than the population following simple mass action. Hence, we see that the phenomenon of non-occurrence or rarity of chaos in nature is well defined by the model where the mode of disease transmission

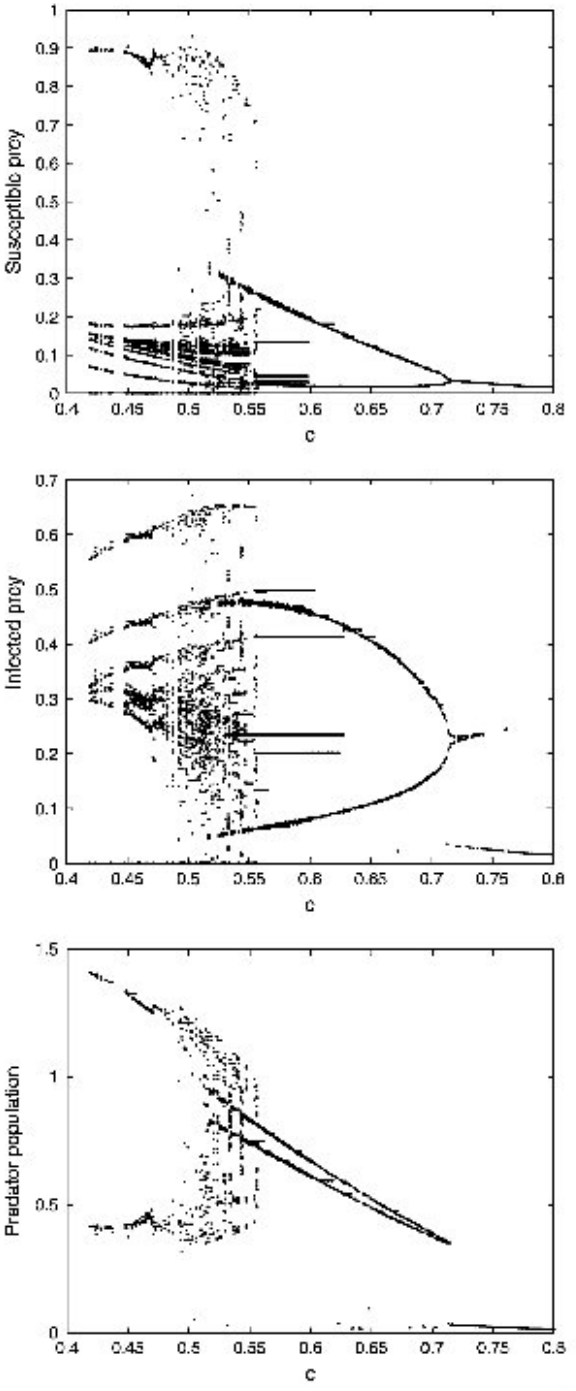

FIG. 6. Bifurcation diagram depicting the dynamical nature of different populations of System (2.2) (where the mode of disease transmission follows standard incidence) by varying the parameter c, holding the other parameter values fixed at the level given in Table 1.

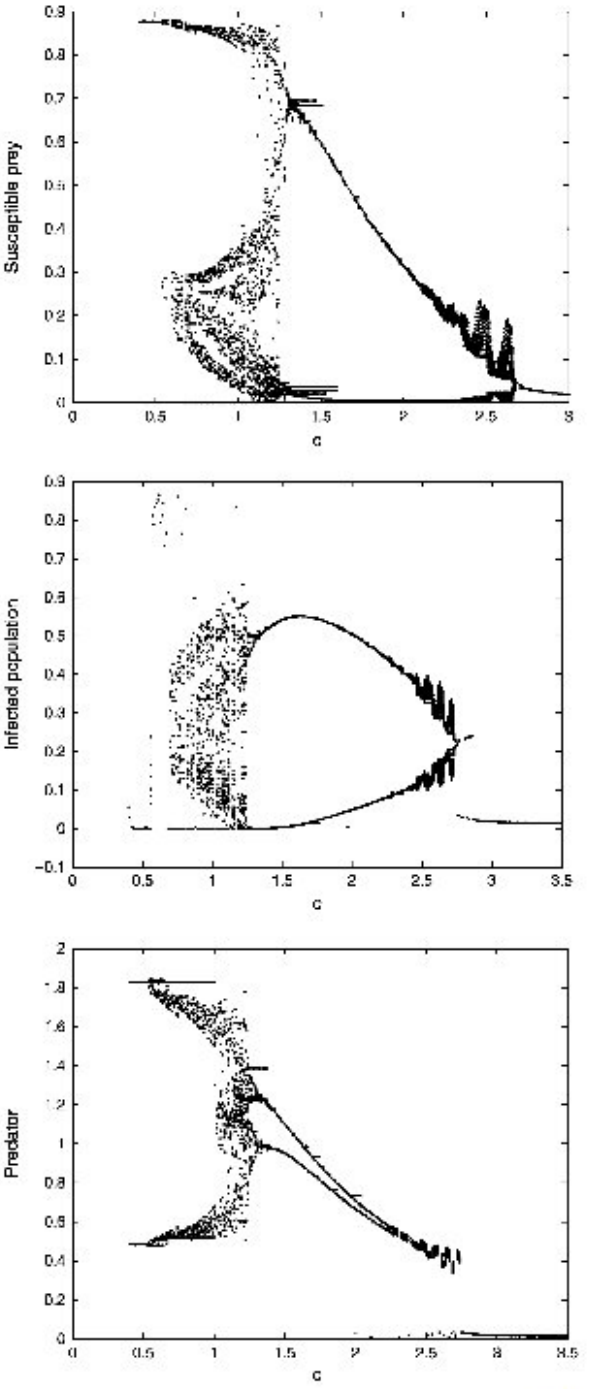

FIG. 7. Bifurcation diagram depicting the dynamical nature of different populations of System (2.1) (where the mode the of disease transmission follows simple mass action incidence) by varying the parameter c, holding the other parameter values fixed at the level given in Table 1.

TABLE 2 *Simulation experiments of model systems* (2.1) *and* (2.2) *with other parameter values fixed at the level given in Table* 1

Range in which the parameter c is varied	Dynamical behaviour of System (2.1) (where the mode of disease transmission follows simple mass action)	Dynamical behaviour of System (2.2) (where the mode of disease transmission follows standard incidence)
$0.42 \leqslant c \leqslant 0.47$	Limit cycle	Limit cycle
$0.48 \leqslant c \leqslant 0.56$	Limit cycle	Chaos
$0.57 \leqslant c \leqslant 0.75$	Limit cycle	Limit cycle
$0.75 \leqslant c \leqslant 1.25$	Chaos	Stable focus
$1.25 \leqslant c \leqslant 2.7$	Limit cycle	Stable focus
$c > 2.7$	Stable focus	Stable focus

follows standard incidence rate. Moreover, the system with mass action incidence as the mode of disease transmission is more stable around the interior steady state than the system with standard incidence.

We have reached such a conclusion by varying the parameter c only, holding the other parameter values fixed at the level given in Table 1. Naturally, a question arises whether the same result holds when the parameters are not fixed but are chosen randomly from a joint probability distribution. So a possible extension, as a more sophisticated and powerful support of the above results, is obtained by assuming that a set of parameter values is a random sample from the joint probability distribution of the whole parameter space. We assume a Gaussian distribution of the parameters. The logic behind this assumption is very simple and realistic. First, we have fixed some arbitrary parameters under which both the processess are stable and these values are taken to be the mean of the Gaussian distribution. Now, we can set the range of the variance in such a way that most of the random samples will fall in the positive plane. In other words, most of the 3σ limits of the Gaussian distribution of the parameters will lie in the positive plane and as a result, most of the random sample will fall in the positive plane with high probability. It is illustrated through Fig. 8 where almost all the histograms are defined in the positive x-plane. So, although the Gaussian distribution is defined in whole real line, it is not unrealistic to assume the underlying distribution of the model parameters to be Gaussian.

To draw the random sample, we adopt the most commonly and frequently used method of uncertainty and sensitivity analysis of parameters popularly known as LHS.

5.1 *Latin hypercube sampling*

LHS, a stratified random procedure, provides an efficient way of sampling variables from their distributions (Iman & Conover, 1980). The LHS involves sampling ns values from the prescribed distribution of each of k variables $X_1, X_2, \ldots, X_k$. The cumulative distribution for each variable is divided into N equiprobable intervals. A value is selected randomly from each interval. The N values obtained for each variable are paired randomly with the other variables. Unlike simple random sampling, this method ensures a full coverage of the range of each variable by maximally stratifying each marginal distribution. In our case, we have $(X_1, X_2, \ldots, X_k) = (c, r, k, a_1, a_2, b_1, b_2, e, f, d_1, d_2)$, i.e. the number of variables $k = 11$ and the number of random samples drawn $N = 10,000$.

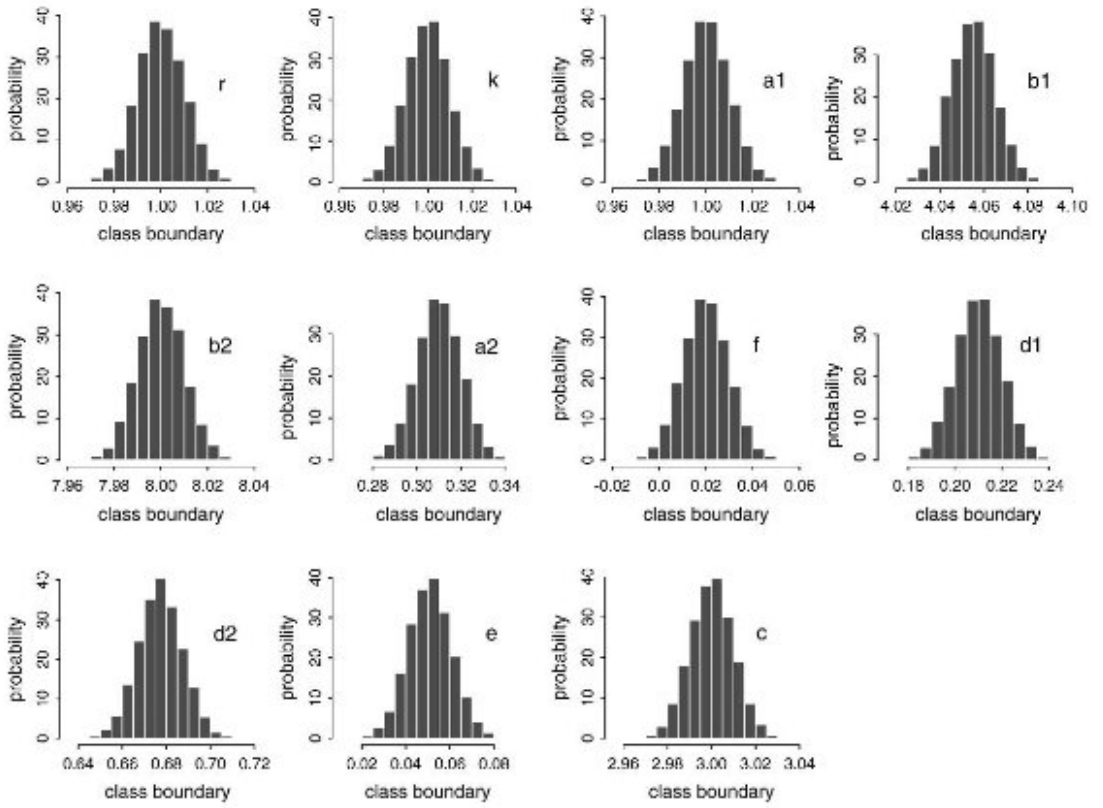

FIG. 8. Histogram of different parameters drawn randomly by LHS technique.

The algorithm used here to find which incidence function tends to yield greater stability using LHS can be summarized as follows:

(1) Give the mean value of each parameter and its standard deviation. In our case, we observe from Table 1 that both the systems are stable around the positive steady state for $c = 3$ with other parameters the same as in the text. So, we have considered these parameter values as the mean values of the parameters. We have taken the standard deviation to be 0.01 because this is the maximum standard deviation for which all the parameters fall in the positive region (see Fig. 8).

(2) Then, LHS is used to draw a random sample. The LHS involves the following steps:

   (a) Divide the cumulative distribution of each variable into N equiprobable intervals.

   (b) From each interval, selecting a value randomly, for the ith interval, the sampled cumulative probability can be written as (Wyss & Jorgensen, 1998) $\text{Prob}_i = (1/N)r_u + (i-1)/N$, where r_u is a normally distributed random number. r_u is an uniformly distributed random number ranging from 0 to 1.

   (c) Transform the probability values sampled into the value x using the inverse of the distribution function F^{-1}, where F is the cumulative density function of the normal distribution:

$$x = F^{-1}(\text{Prob}).$$

(d) The N values obtained for each variable x are paired randomly (equally likely combinations) with the N values of the other variables.

(3) Using Steps (1) and (2), we have drawn 10,000 random samples (each of which is actually a set of all the parameter values) from the 11-dimensional parameter space. Then, we have collected those random samples in set 'S' for which all the parameter values are positive.

(4) Then, we have used the rejection technique. Within the random samples collected in the set 'S', those sample values for which System (2.1) is stable are collected in set 'A' and others are rejected. Finally, the probability of a sample parameter value falling in a region of parameter space where System (2.1) is stable around the positive steady state, $P(A)$, was obtained by

$$P(A) = \frac{n(A)}{n(S)},$$

where $n(A)$ is the number of elements in set 'A' and $n(S)$ is the number of elements in set 'S'. Similarly, a probability $P(B)$ is also obtained for System (2.2) from

$$P(B) = \frac{n(B)}{n(S)},$$

where $n(B)$ is the number of elements in set 'B'.
We subtract these two probabilities to obtain a probability difference.

(5) Repeat Steps (3) and (4) for 1000 times. Find the average of all these probability differences. We observe that the mean of $(P(B) - P(A)) > 0$ (see Fig. 9).

We observe that when the parameters are random, then also the system following standard incidence is more stable than mass action incidence in probabilistic sense. We obtain the above result by setting the standard deviation at 0.01. In the second stage, we slowly increase the standard deviation up to 0.1 and

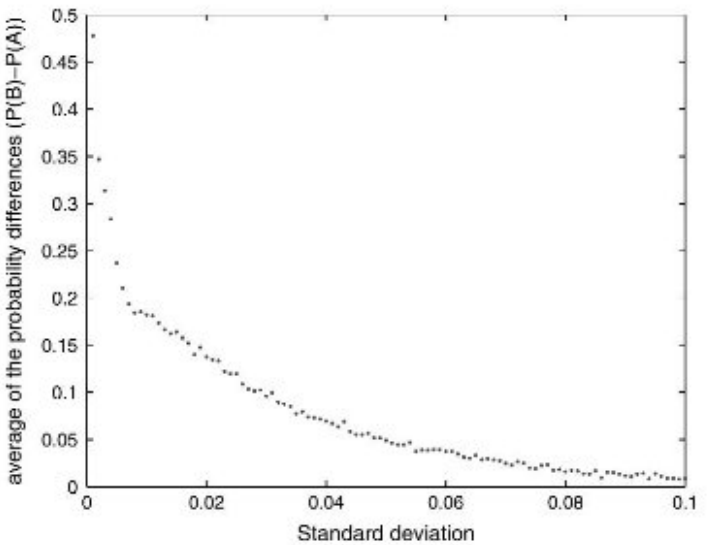

FIG. 9. Figure depicting the relation between the probability difference between the two systems and the standard deviation.

observe that the probability of System (2.1) to be stable is still more than System (2.2). But obviously, the probability differences decrease when the standard deviation increases.

6. Conclusion

In this paper, we have modified the model proposed by Chatterjee *et al.* (2006). The main objective of this paper is to compare the different modes of disease transmission giving special emphasis to chaotic dynamics. The mode of transmission is crucially important for two reasons. First, it determines the probable response of the disease to control. Second, the objective in many models of eco-epidemiology is to predict what will happen when the infection is introduced into a system in which it does not currently exist. So, if we know the threshold for disease (i.e. the minimum population size or population density of susceptible hosts necessary for the disease to increase), there is a possibility to control the disease (McCallum *et al.*, 2001). Another important aspect of a dynamical system is the occurrence of the chaos. It is already known to us that though chaos is rare, it may occur in nature for some realistic parameter values (Hastings & Powell, 1991). In the present study, we have also tried to find a relation between the occurrence of the chaos and the mode of disease transmission.

We have first shown the boundedness of the solution and worked out analytically the conditions for the local stability criteria of different equilibrium points and the conditions for the persistence of both the prey and the predator species. Since the structure of the model presented here is a complex one, our main results are based on the numerical simulations. Moreover, numerical simulations help us to find the long-term behaviour of the system. With the help of numerical integration, we have shown different dynamical behaviour exhibited by the considered model, e.g. stable population distribution, limit cycle, quasi-periodic oscillation and chaotic behaviour. To confirm the presence of the chaos, we have calculated the Lyapunov exponent of the system and the largest Lyapunov exponent is found to be positive. It is seen during the chaotic behaviour that though for a short period of time the behaviour may be fairly regular, over a long period of time the irregularities occur in the behaviour of the system, and the sensitivity to the initial condition and unpredictability becomes more visible.

We observe that if the system follows standard incidence as the mode of disease transmission, then the chance for the occurrence of chaos decreases and it becomes easier to make that system stable around the positive steady state. These observations were obtained by varying the parameter c and holding the other parameter values fixed. Finally, using the method of LHS, we have shown that the system with standard incidence yields more stability even when all the parameters are varied randomly. Exploring the entire parameter space, we have observed that the stability region increases in the case of standard incidence.

Finally, our observations may be summarized as follows:

(a) The phenomenon of non-occurrence or rarity of chaos in nature is well defined by the model where the mode of disease transmission follows standard incidence rate.
(b) The stability region is larger in the case of standard incidence than the simple mass action.

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Appendix A

Proof of Lemma 3.3.2. We consider first $S(t) + I(t) \geqslant 1$ for all $t \geqslant 0$. From the first two equations of (2.3), we get

$$\frac{\mathrm{d}}{\mathrm{d}t}(S(t) + I(t)) = S(1 - S - I) - \frac{(S + \gamma I)P}{1 + \alpha S + \beta I} - \delta I. \tag{A.1}$$

Hence, for all $t \geqslant 0$, we have that $\frac{\mathrm{d}S(t)}{\mathrm{d}t} + \frac{\mathrm{d}I(t)}{\mathrm{d}t} \leqslant 0$. Let

$$\lim_{t\to\infty} S(t) + I(t) = \eta. \tag{A.2}$$

If $\eta > 1$, then by the Barbalat lemma, we have

$$\begin{aligned} 0 = \lim_{t\to\infty} \frac{\mathrm{d}}{\mathrm{d}t}(S(t) + I(t)) &= \lim_{t\to\infty} \left[S(t)(1 - S(t) - I(t)) - \frac{(S(t) + \gamma I(t))P(t)}{1 + \alpha S(t) + \beta I(t)} - \delta I(t) \right] \\ &\leqslant \lim_{t\to\infty} [S(t)(1 - S(t) - I(t)) - \delta I(t)] \\ &= \lim_{t\to\infty} [S(t)(1 - \eta) - \delta I(t)] \\ &\leqslant -\min\{(\eta - 1), \delta\} \lim_{t\to\infty} (S(t) + I(t)) \\ &= -\min\{(\eta - 1), \delta\} < 0. \end{aligned}$$

This contradiction shows that $\eta = 1$, i.e.

$$\lim_{t\to\infty} (S(t) + I(t)) = 1. \tag{A.3}$$

Let us denote $g(t) = S(t) + I(t)$ for $t \in [0, \infty)$. Of course, $g(t)$ is differentiable and $g'(t)$ is uniformly continuous for $t \in (0, +\infty)$. Thus, with (A.3) all the assumptions of the Barbalat lemma hold true and, therefore,

$$\lim_{t\to\infty} \frac{\mathrm{d}}{\mathrm{d}t}(S(t) + I(t)) = 0. \tag{A.4}$$

Since from the first two equations of (2.3)

$$\frac{\mathrm{d}}{\mathrm{d}t}(S(t) + I(t)) = S(t)(1 - S(t) - I(t)) - \frac{(S(t) + \gamma I(t))P(t)}{1 + \alpha S(t) + \beta I(t)} - \delta I(t), \tag{A.5}$$

then (A.3) implies that

$$\begin{aligned} \lim_{t\to\infty} \frac{\mathrm{d}}{\mathrm{d}t}(S(t) + I(t)) &= \lim_{t\to\infty} \left[S(t)(1 - S(t) - I(t)) - \frac{(S(t) + \gamma I(t))P(t)}{1 + \alpha S(t) + \beta I(t)} - \delta I(t) \right] \\ &= -\lim_{t\to\infty} \left[\frac{(S(t) + \gamma I(t))P(t)}{1 + \alpha S(t) + \beta I(t)} + \delta I(t) \right]. \end{aligned} \tag{A.6}$$

Hence, (A.4) and (A.6) are in agreement if and only if $\lim_{t\to\infty} I(t) = 0$ and $\lim_{t\to\infty} P(t) = 0$, which jointly from (A.3) implies that $\lim_{t\to\infty} S(t) = 1$. This completes the case (i).

Suppose that assumption (i) is violated. Then, there exists $t_0 > 0$ at which for the first time $S(t_0) + I(t_0) = 1$. According to (A.5), we have

$$\frac{\mathrm{d}}{\mathrm{d}t}(S(t) + I(t)) \Big|_{t=t_0} = -\left[\frac{(S(t_0) + \gamma I(t_0))P(t_0)}{1 + \alpha S(t_0) + \beta I(t_0)} + \delta I(t_0) \right] < 0.$$

This implies that once a solution with $S + I$ has entered into the interval $(0, 1)$, then it remains bounded there for all $t > t_0$, i.e. $S(t) + I(t) < 1$ for all $t > t_0$.

Finally, if $S(t_0) + I(t_0) < 1$, then applying the previous argument it follows that $S(t) + I(t) < 1$ for all $t > 0$, i.e. (iii) holds true. This completes the proof.

Proof of Lemma 3.3.3. Lemma 3.3.2 implies that for any $(S(t_0), I(t_0), P(t_0))$ such that $S(t_0) + I(t_0) \geqslant 1$, either a time $t_0 > 0$ exists for which $S(t) + I(t) < 1$ for all $t > t_0$ or $\lim_{t\to\infty} S(t) = \frac{B}{\delta}$ and $\lim_{t\to\infty} I(t) = 0$. Furthermore, if $S(t_0) + I(t_0) < 1$, then $S(t) + I(t) \leqslant 1$ for all $t > 0$. Hence, in any case a non-negative time, say t^*, exists such that $I(t) < 1$, $S(t) < 1$, for all $t > t^*$.

Set $W = S(t) + I(t) + P(t)$.

Calculating the derivative of W along the solution of System (2.3), we find for $t > t^*$,

$$\begin{aligned}
\dot{W} &= S(t)(1 - S(t) - I(t)) - \frac{(S(t) - e_1 S(t) + \gamma I(t) - e_2 I(t))P(t)}{1 + \alpha S(t) + \beta I(t)} - \delta I(t) - \mu P(t) \\
&\leqslant S(t)1 - \delta I(t) - \mu P(t) \qquad (\because e_1 < 1, e_2 < \gamma) \\
&\leqslant 1 - \min\{1, \delta, \mu\}(S(t) + I(t) + P(t)) \\
&= 1 - \xi W,
\end{aligned}$$

where $\xi = \min\{1, \delta, \mu\}$.

Thus, there exists a positive constant M such that $W(t) < M$ for all large t. The assertion of Lemma 3.3.2 now follows and the proof is completed.

Let Ω be the following subset of $R^3_{0,+}$:

$$\Omega = \{(S, I, P) \varepsilon R^3_{0,+} : S + I \leqslant 1, P \leqslant M\} \tag{A.7}$$

Proof of Theorem 3.3.1. Due to Lemmas 3.3.2 and 3.3.3, for all initial conditions in $R^3_{+,0}$ such that $(S(t_0), I(t_0), P(t_0))$ does not belong to Ω, either there exists a positive time, say T, $T = \max\{t_1, t^*\}$, such that the corresponding solution $(S(t), I(t), P(t) \in \text{int}\ \Omega$ for all $t > T$ or the corresponding solution is such that $(S(t), I(t), P(t)) \to E_1(1, 0, 0)$ as $t \to +\infty$. But, $E_1 \in \partial\Omega$. Hence, the global attractivity of Ω in $R^3_{0,+}$ has been proved.

Assume now that $(S(t_0), I(t_0), P(t_0)) \in \text{int}\ \Omega$. Then, Lemma 3.3.2 implies that $S(t) + I(t) < 1$ for all $t > 0$ and also by Lemma 3.3.3, we know that $P(t) < M$ for all large t. Let us remark that if $(S(t_0), I(t_0), P(t_0)) \in \partial\Omega$, because $S(t_0) + I(t_0) = 1$ or $P(t_0) = M$ or both, then still the corresponding solutions $(S(t), I(t), P(t))$ must immediately enter int Ω or coincide with E_1.

Appendix B. Behaviours of the system around $E_0(0, 0, 0)$

At the trivial equilibrium point E_0, the Jacobian matrix (4.1) is not defined. We have analysed the stability of the system around the trivial steady state following the technique used by Arino *et al.* (2004). Let us now for a moment consider in a general context, i.e. to say we consider a system in R^N,

$$\frac{dX}{dt} = H(X(t)) + Q(X(t)), \tag{B.1}$$

in which H is C^1-outside the origin and is continuous and homogenous of degree 1.

$$H(sX) = s\,H(X)$$

for all $s \geqslant 0$, $X \in R^N$, and Q is a C^1-function such that

$$Q(X) = o(X)$$

in the vicinity of the origin. Throughout the section, $\|\cdot\|$ denotes the Euclidian norm on R^N and $(\cdot, \cdot)$ the associated inner product. In the case of our model, $N = 3$,

$$X = (x_1, x_2, x_3) = (S, I, P),$$

$$H(X) = (H_1(X), H_2(X), H_3(X)),$$

$$Q(X) = (Q_1(X), Q_2(X), Q_3(X)).$$

The function H_i and Q_i $(i = 1, 2, 3)$ are given by

$$H_1(X) = S - \frac{\lambda SI}{S + I},$$

$$H_2(X) = \frac{\lambda SI}{S + I} - \delta I,$$

$$H_3(X) = -\mu P,$$

$$Q_1(X) = -\frac{SP}{1 + \alpha S + \beta I},$$

$$Q_2(X) = -\frac{\gamma IP}{1 + \alpha S + \beta I},$$

$$Q_3(X) = \frac{(e_1 S + e_2 I) P}{1 + \alpha S + \beta I}.$$

Let $X(t)$ be a solution of System (B.1). Assume that $\liminf_{t \to \infty} \|X(t)\| = 0$ and X is bounded. One can extract from the family $(X(t + \cdot))_{t \geqslant 0}$ sequences $X(t_n + \cdot)$, $t_n \longrightarrow \infty$, such that $X(t_n + \cdot) \longrightarrow 0$ locally uniformly on $s \in R$. Define

$$y_n(s) = \frac{X(t_n + s)}{\|X(t_n + s)\|}. \tag{B.2}$$

Recall that

$$Q(X) = o(X)$$

in the vicinity of the origin. We can then write Q as

$$Q(X) = (\|X\|)^2 o(1). \tag{B.3}$$

We have

$$\frac{dX(t_n + s)}{ds} = H(X(t_n + s)) + Q(X(t_n + s)). \tag{B.4}$$

From (B.2), we have

$$X(t_n + s) = y_n(s)\|X(t_n + s)\| = y_n(s) \cdot \langle X(t_n + s), X(t_n + s)\rangle^{\frac{1}{2}}. \tag{B.5}$$

Now, using the derivative of $\langle X(t_n + s), X(t_n + s)\rangle$ with respect to s

$$\frac{\mathrm{d}}{\mathrm{d}s}(\langle X(t_n + s), X(t_n + s)\rangle) = 2\left\langle X(t_n + s), \frac{\mathrm{d}X(t_n + s)}{\mathrm{d}s}\right\rangle$$

in (B.5), we obtain

$$\frac{\mathrm{d}X(t_n + s)}{\mathrm{d}s} = \frac{\mathrm{d}y_n(s)}{\mathrm{d}s}(\|X(t_n + s)\|) + \frac{y_n(s)}{\|X(t_n + s)\|}\left\langle X(t_n + s), \frac{\mathrm{d}X(t_n + s)}{\mathrm{d}s}\right\rangle.$$

Therefore, we have

$$H(X(t_n + s)) + Q(X(t_n + s)) = \frac{\mathrm{d}y_n(s)}{\mathrm{d}s}\|X(t_n + s)\| + \frac{y_n(s)}{\|X(t_n + s)\|}\langle X(t_n + s), H(X(t_n + s)) + Q(X(t_n + s))\rangle.$$

Now, dividing by $\|X(t_n + s)\|$ and replacing $\frac{X(t_n+s)}{\|X(t_n+s)\|}$ by $y_n(s)$, we obtain

$$\frac{\mathrm{d}y_n(s)}{\mathrm{d}s} = H(y_n(s)) - \langle y_n(s), H(y_n(s))\rangle y_n(s) + \|X(t_n + s)\|\left[\frac{1}{\|X(t_n + s)\|}Q(X(t_n + s)) - \left\langle y_n(s), \frac{1}{\|X(t_n + s)\|}Q(X(t_n + s))\right\rangle y_n(s)\right],$$

which is equivalent to

$$\frac{\mathrm{d}y_n}{\mathrm{d}s} = [H(y_n(s)) - (y_n(s), H(y_n(s)))y_n(s)] + \|X(t_n + s)\|[Q(y_n(s)) - (y_n(s), Q(y_n(s)))y_n(s)].$$

Clearly, y_n is bounded, $\|y_n(s)\| = 1$ for any s and $\frac{\mathrm{d}y_n}{\mathrm{d}s} = 1$ is bounded too. So, applying the Ascoli–Arzela theorem (see, e.g. Brezis, 1983), one can extract from y_n a subsequence—also denoted by y_n—which converges locally, uniformly on R towards some function y such that $\|X(t_n+s)\|[Q(y_n(s))-(y_n(s), Q(y_n(s)))y_n(s)]_{t_n\to\infty} \to 0$ and y satisfies the following system:

$$\frac{\mathrm{d}y}{\mathrm{d}t} = H(y(t)) - (y(t), H(y(t)))y(t), \quad \|y(t)\| = 1, \ \forall t. \tag{B.6}$$

Equation (B.6) is defined for all $t \in R$.

Let us, for a moment, focus on the study of (B.6). The steady states of H are vectors V satisfying

$$H(V) = (V, H(V))V.$$

This is the so-called non-linear eigenvalue. Note that the equation can be alternatively written as

$$H(V) = \phi V,$$

with $\|V\| = 1$; it then holds that $\phi = (V, H(V))$.

These stationary solutions correspond to fixed direction that the trajectories of (B.6) may reach asymptotically:

$$[(\phi - 1)v_1 + (\phi - 1 + \lambda)v_2]v_1 = 0, \tag{B.7}$$

$$[(\phi - \lambda + \delta)v_1 + (\phi + \delta)v_2]v_2 = 0, \tag{B.8}$$

$$[(\phi + \mu)v_3]v_3 = 0. \tag{B.9}$$

Now, we are in a position to discuss in detail the possibility of reaching the origin following fixed direction.

Case 1. $v_1 = 0$.

(a) $v_2 = 0$ and $v_3 \neq 0$. In this case, there is a possibility of reaching the origin following the P-axis with $\phi = -\mu$.

(b) $v_2 \neq 0$ and $v_3 = 0$. In this case also, there is a possibility of reaching zero following the I-axis with $\phi = -\delta$.

(c) $v_2 \neq 0$ and $v_3 \neq 0$. In this case, there is a possibility to reach the origin either with $\phi = -\delta$ or with $\phi = -\mu$ following the PI-plane.

Case 2. $v_1 \neq 0$.

(a) $v_2 = 0, v_3 = 0$. In this case, we cannot reach the origin following the S-axis, i.e. to say that the S-axis is not a fixed direction that the trajectories can follow to reach the zero.

(b) $v_2 = 0$ and $v_3 \neq 0$. In this case, we have two possibilities:

  (i) with $\phi = -\mu$, there is a possibility to reach the origin;

  (ii) with $\phi = 1$, there is no possibility of reaching the origin following the SP-plane.

(c) $v_2 \neq 0, v_3 = 0$. In this case, there is no possibility of reaching the origin following the SI-plane.

(d) $v_2 \neq 0, v_3 \neq 0$.

  (i) With $\phi = -\mu$, there is a possibility for going to the origin following a fixed direction that is contained in the positive octant.