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AN S-I EPIDEMIC MODEL WITH SATURATION INCIDENCE : DISCRETE AND STOCHASTIC VERSION

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Abstract. A discrete-time susceptible-infected model with saturation in incidence is investigated.For this model, the existence and the stability of fixed points are analyzed.Stochastic analysis of the model suggests that it is asymptotically mean square stable for certain strengths of white noise.The bifurcation diagram and time series plots are obtained for different values of parameters for the model. Analytical results and numerical simulations show that some combinations of the saturation factors tend to have a stabilizing effect for the system.Our discrete model exhibits rich dynamics which is not present in both the continuous model and its stochastic version.

1. Introduction

The study of discrete-time models described by difference equations has now been paid great attention since these models are more reasonable than the continuous time models when populations have non-overlapping generations. Discrete-time models give rise to more efficient computational models for numerical simulations and also show rich dynamics compared to the continuous ones.In recent years,numerous papers have been published on the mathematical models of biology that discussed the system of difference equations generated from the associated system of differential equations as well as the associated numerical methods.Mathematical models of epidemics have created a major area of research interest during the last few decades.Examples of discrete and continuous time epidemic models can be found in [?, ?, ?, ?].Incidence rate refers to the rate of infection of susceptible individuals through contacts with infectives. In order to model the disease transmission process we consider a modified incidence function of the form $\frac{\beta SI}{1+\alpha_1 S+\alpha_2 I}$ studied in a continuous-time differential equations model proposed in [?]to analyze the effects of saturation factor (refer to α_1) stemming from epidemic control [?, ?] public response to outbreak severity [?, ?],crowding of infective individuals, or due to intervention measures to protect susceptible individuals [?, ?]. It is important to note that $\frac{\beta SI}{1+\alpha_1 S+\alpha_2 I}$ becomes the saturated incidence rate $\frac{\beta SI}{1+\alpha_2 I}$ if $\alpha_1 = 0$ and the saturated incidence rate $\frac{\beta SI}{1+\alpha_1 S}$ if $\alpha_2 = 0$. Moreover,it becomes the bilinear incidence rate βSI

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if $\alpha_1 = \alpha_2 = 0$.

Kaddar [**?**] studied a delayed SIR epidemic model with modified saturated incidence rate, where results on local stability and the existence of Hopf bifurcation were obtained. He concluded that delayed SIR epidemic model with a bilinear incidence rate ($\alpha_1 = 0 = \alpha_2$) or a saturated incidence rate ($(\alpha_1 = \alpha_2) \neq (0, 0)$) generate the same asymptotic properties if ($\alpha_1 = \alpha_2$) is sufficiently close to $(0, 0)$. However, with large values of the inhibitory effect α_1 and/or α_2 this equivalence was not true. Moreover, for large values of α_1 and/or α_2 the dynamics obtained by the modified saturated incidence rate is not equivalent to the following three forms: βSI,$\frac{\beta SI}{1+\alpha_1 S}$, and $\frac{\beta SI}{1+\alpha_2 S}$.

In view of the above, using article [**?**] we are now interested to investigate the dynamics of the discrete and stochastic version of the following continuous model:

$$\begin{aligned} \frac{dS}{dt} &= A - \mu S - \frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I}, \\ \frac{dI}{dt} &= \frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I} - (\mu + \alpha + r)I. \end{aligned} \tag{1.1}$$

where S is the number of susceptible individuals at time t, I is the number of infective individuals at time t, A is the recruitment rate, μ is the natural death rate, α is the disease induced death rate, β is the disease transmission rate, $\alpha_i (i = 1, 2)$ are the parameters that measure the inhibitory effect and r is the recovery rate of infected individuals.

We now study the dynamics of discrete S-I epidemic model which has the following two difference equations:

$$\begin{aligned} S_{n+1} &= S_n + A - \mu S_n - \frac{\beta S_n I_n}{1 + \alpha_1 S_n + \alpha_2 I_n}, \\ I_{n+1} &= I_n + \frac{\beta S_n I_n}{1 + \alpha_1 S_n + \alpha_2 I_n} - (\mu + \alpha + r)I_n. \end{aligned} \tag{1.2}$$

2. Fixed Points and Stability

In this section, we first determine the existence of fixed points of system (2) and then investigate their stability by calculating the eigenvalues for the variation matrix of (2) at each fixed point. We have two nonnegative fixed points which are as follows:

1) Disease-free fixed point $E_1(\frac{A}{\mu}, 0)$.
2) Interior endemic fixed point $E_2(S^*, I^*)$ where $S^* = \frac{A[(\mu+\alpha+r+\alpha_2 A]}{\mu[(\mu+\alpha+r)R_0+\alpha_2 A]}$ and $I^* = \frac{A(R_0-1)}{[(\mu+\alpha+r)R_0+\alpha_2 A]}$ where, $R_0 = \frac{A[\beta-(\mu+\alpha+r)\alpha_1]}{\mu(\mu+\alpha+r)}$.

Now, we investigate the local behavior of the model around each of the above fixed points. The local stability analysis of the model can be studied by computing the variation matrix corresponding to each fixed point. The Jacobian matrix $J(S,I)$ of system at the state variable is given by

$$J(S,I) = \begin{pmatrix} 1-\mu-\frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2} & -\frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2} \\ \\ \frac{\beta I(1+\alpha_2 I)}{(1+\alpha_1 S+\alpha_2 I)^2} & 1+\frac{\beta S(1+\alpha_1 S)}{(1+\alpha_1 S+\alpha_2 I)^2}-(\mu+\alpha+r) \end{pmatrix}.$$

Hence the characteristic equation of Jacobian matrix is $\lambda^2 - Tr(J)\lambda + Det(J) = 0$,(3) where $Tr(J)$ and $Det(J)$ are the trace and determinant of the Jacobian matrix $J(S,I)$, respectively.

Let λ_1 and λ_2 be the two roots of Eq.(3), which are the eigenvalues of system (2) at a fixed point (S,I). We recall the definitions of topological types for a fixed point (S,I) The fixed point (S,I) is called a sink if $|\lambda_1|<1$ and $|\lambda_2|<1$, so the sink is locally asymptotically stable. (S,I) is called a source if $|\lambda_1|>1$ and $|\lambda_2|>1$, so the source is locally unstable. Also (S,I) is called a saddle if $|\lambda_1|<1$ and $|\lambda_2|>1$ (or $|\lambda_1|>1$ and $|\lambda_2|<1$). Further the fixed point (S,I) is non-hyperbolic if either $|\lambda_1|=1$ or $|\lambda_2|=1$.

Theorem 2.1. *The disease-free fixed point E_1*

i) is a sink if $\mu<2$ and $\frac{\beta A}{\mu+\alpha_1 A}<(\mu+\alpha+r)<2+\frac{\beta A}{\mu+\alpha_1 A}$

ii) is a source if $\mu>2$ and $(\mu+\alpha+r)>2+\frac{\beta A}{\mu+\alpha_1 A}$

iii) is non-hyperbolic if $\mu=2$ or $\frac{\beta A}{\mu+\alpha_1 A}=(\mu+\alpha+r)$

iv) is a saddle for other values of parameters except those values as described in (i)-(iii).

Proof. We calculate the eigenvalues of Jacobian matrix for E_1 which is given by

$$J(E_1) = \begin{pmatrix} 1-\mu & -\frac{\beta A}{(\mu+\alpha_1 A)} \\ \\ 0 & 1+\frac{\beta A}{(\mu+\alpha_1 A)}-(\mu+\alpha+r) \end{pmatrix}.$$

The eigenvalues of the matrix, $J(E_1)$, are $\lambda_1=1-\mu$ and $\lambda_2=1+\frac{\beta A}{\mu+\alpha_1 A}-(\mu+\alpha+r)$. It is easy to see that, E_1 is a sink if $\mu<2$ and $\frac{\beta A}{\mu+\alpha_1 A}<(\mu+\alpha+r)<2+\frac{\beta A}{\mu+\alpha_1 A}$; E_1 is a source if $\mu>2$ and $(\mu+\alpha+r)<2+\frac{\beta A}{\mu+\alpha_1 A}$; and E_1 is non-hyperbolic if $\mu=2$ or $\frac{\beta A}{\mu+\alpha_1 A}=(\mu+\alpha+r)$. Furthermore, E_1 is a saddle point for all other values of the parameters. This complete the proof of the theorem.

□

3. Local stability and Bifurcation around Interior Fixed Point

Now, we shall discuss the stability and bifurcations of interior (endemic) fixed point E_2. E_2 is stable if it satisfies the following criteria:

$i)1 + Tr(J(E_2)) + Det(J(E_2)) > 0,$

$ii)1 - Tr(J(E_2)) + Det(J(E_2)) > 0,$

$iii)1 - Det(J(E_2)) > 0$, where $J(E_2)$ is given by

$$J(S,I) = \begin{pmatrix} 1-\mu-\frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} & -\frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} \\ \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} & 1+\frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}-(\mu+\alpha+r) \end{pmatrix}.$$

The above conditions are well known (e.g., [**?**]), and are sufficient for the local stability of the fixed point and necessary for the roots $\lambda_{1,2}$ of the characteristic equation to be inside the unit circle of the complex plane.

Now,$Tr(J(E_2)) = 2(1-\mu) - (\alpha + r) + \beta[\frac{S^*(1+\alpha_1 S^*) - I^*(1+\alpha_2 I^*)}{1+\alpha_1 S^*+\alpha_2 I^*)^2}]$

$Det(J(E_2)) = (1-\mu)[(1-\mu-\alpha-r+\frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}] - \frac{(1-\mu-\alpha-r)\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}.$

Since $\frac{\beta S^*}{(1+\alpha_1 S^*+\alpha_2 I^*)} = \mu+\alpha+r$ it can be shown that

$Tr(J(E_2) = 2-\mu-\frac{\beta\alpha_2 S^* I^*}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} - \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}$ and

$Det(J(E_2) = (1-\mu)[1-\frac{\beta\alpha_2 S^* I^*}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}] - \frac{(1-\mu-\alpha-r)\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}$

Define $p = \frac{\beta\alpha_2 S^* I^*}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}$ and $q = \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}$

Now $1 + Tr(J(E_2)) + Det(J(E_2)) = 4 - 2\mu - 2p - 2q + \mu p + q(\mu+\alpha+r) > 0$

if $4 + \mu p + q(\mu+\alpha+r) > 2(\mu+p+q)$.

Again we have $1 - Tr(J(E_2)) + Det(J(E_2)) = \mu p + q(\mu+\alpha+r) > 0$ and

$1 - Det(J(E_2)) = \mu + p + q - \mu p - (\mu+\alpha+r)q > 0$ if $\mu + p + q > \mu p + (\mu+\alpha+r)q$.

Thus we have the following theorem.

Theorem 3.1. *The fixed point E_2 is asymptotically stable if*

$\mu p + (\mu+\alpha+r)q < \mu + p + q < \frac{4+\mu p+q(\mu+\alpha+r)}{2}$.

The next result follows from Theorem 3.1 immediately.

Corollary. The fixed point E_2 is unstable if either $\mu + p + q < \mu p + (\mu + \alpha + r)q$ or $2(\mu + p + q) > 4 + \mu p + q(\mu + \alpha + r)$.

We can now state a result on the bifurcation phenomenon of system (2) for some critical value of inhibitory effect.

Theorem 3.2. *Suppose that* $2(\mu + p + q) < 4 + \mu p + q(\mu + \alpha + r)$. *Then system (2) undergoes a Hopf bifurcation when the saturation parameter* α_1 *passes through a critical value* α_{1c} *where* $\mu p + (\mu + \alpha + r)q = \mu + p + q$ *is satisfied at* $\alpha_1 = \alpha_{1c}$. *In that case* $|J(E_2)| = 1$ *so bifurcation arises and it is of Neimark-Sacker type.*

It is important to note here that inhibitory effect has no crucial role in continuous-time epidemic model, whereas in discrete system it gives rise to bifurcation for some of its critical value.

4. Analysis of Stochastic Model

In this section we investigate the dynamics of stochastic version of the continuous model (1.1). Stochastic perturbations have been introduced in some of the important model parameters in the model equations (see, e.g., [**?**, **?**]). In this study, instead, we allow stochastic perturbations of the variables S and I around the positive equilibrium E_2, in the case when E_2 is feasible and locally asymptotically stable. Local stability of E_2 is implied by the existence condition of E_2. Hence in system (1.1), we assume that stochastic perturbations of the variables around E_2 are of white noise type, which are proportional to the respective distances of S and I from values S^* and I^*. So system (1.1) transformed into:

$$\begin{aligned} dS &= [A - \mu S - \frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I}]dt + \sigma_1(S - S^*)d\xi_t^1, \\ dI &= [\frac{\beta SI}{1 + \alpha_1 S + \alpha_2 I} - (\mu + \alpha + r)I]dt + \sigma_2(I - I^*)d\xi_t^2. \end{aligned} \tag{4.1}$$

where $\sigma_j, j = 1, 2$ are real constants, and $\xi_j^t, j = 1, 2$ are standard Wiener processes independent from each other [**?**]. We determine whether the dynamical nature of the continuous model (1.1) is robust with respect to such a kind of stochasticity by investigating the asymptotic stochastic stability behaviour of the equilibrium E_2 for (4.1) and by comparing the results with those obtained for system (1.1). We will consider (4.1) as the Ito stochastic differential system.

4.1. Stochastic stability of the interior equilibrium

The system of SDEs in (4.1) can be centered at its interior endemic equilibrium E_2 by the changes of the variables $U_1 = S - S^*, U_2 = I - I^*$ (4.2).
The linearized SDEs around E_2 take the form $du(t) = f(u(t))dt + g(u(t))d\xi(t)$ (4.3) where, $u(t) = col(u_1(t), u_2(t))$,

$$J(E_2) = \begin{pmatrix} -\mu - \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} & -\frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} \\ \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} & \frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} - (\mu+\alpha+r) \end{pmatrix},$$

and $g(u) = \begin{pmatrix} \sigma_1 u_1 & 0 \\ 0 & \sigma_2 u_2 \end{pmatrix}$.

Clearly the endemic equilibrium E_2 corresponds to the trivial solution $u(t) = 0$ in (4.3).Note that, with reference to (4.3),
$LW(t,u) = \frac{\delta W(t,u)}{\delta t} + f^T(u)\frac{\delta W(t,u)}{\delta t} + \frac{1}{2}Tr[g^T(u)\frac{\delta^2 W(t,u)}{\delta u^2 g(u)}]$, where $\frac{\delta W}{\delta u} = col(\frac{\delta W}{\delta u_1}, \frac{\delta W}{\delta u_2})$, $\frac{\delta^2 W(t,u)}{\delta u^2} = \frac{\delta^2 W}{\delta u_i \delta u_j}; i,j = 1,2$, and the superscript T represents transposition. With reference to Afanas'ev et al.[**?**], the following theorem holds.

Theorem 4.1. *Assume that there exists a function $W(t,u) \in C_0^2(U)$ satisfying the inequalities $K_1|u^p| \leq W(t,u) \leq K_2|u^p|$ (4.4) and $LW(t,u) \leq -K_3|u^p|$ (4.5), where K_i and p are positive constants. Then the trivial solution of (4.3) is exponentially p-stable for $t \geq 0$.*

It is important to note that, if p =2 in inequalities (4.4-4.5), then the trivial solution of (4.3) is globally asymptotically stable in probability. For definitions of stability used, we again refer to Afanas'ev et al. [**?**] .

Theorem 4.2. *Assume that $\sigma_1^2 \prec 2[\mu + \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}]$ and $\sigma_2^2 \prec \frac{2\alpha_2 I^*(\mu+\alpha+r)}{(1+\alpha_1 S^*+\alpha_2 I^*)}$ hold. Then the zero solution of (4.3) is asymptotically mean square stable.*

Proof. Let us consider a Lyapunov function $W(u) = \frac{1}{2}(w_1 u_1^2 + w_2 u_2^2)$, where w_i are real positive constants to be chosen later. It is easy to check that inequalities (4.4) hold true with $p = 2$. Further,

$LW(U) = -[\mu + \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}]w_1 u_1^2 - \frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} w_1 u_1 u_2 + \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} w_2 u_1 u_2 -$
$[(\mu+\alpha+r) - \frac{\beta S^*(1+\alpha_1 S^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2}]w_2 u_2^2 + \frac{1}{2}Tr[g^T(u)\frac{\delta^2 W}{\delta u^2} g(u)]$ (4.6).

Now remark that $\frac{\delta^2 W}{\delta u^2} = \begin{pmatrix} w_1 & 0 \\ 0 & w_2 \end{pmatrix}$ and hence

$g^T(u)\frac{\delta^2 W}{\delta u^2} g(u) = \begin{pmatrix} w_1 \sigma_1^2 u_1^2 & 0 \\ 0 & w_2 \sigma_2^2 u_2^2 \end{pmatrix}$

with $\frac{1}{2}Tr[g^T(u)\frac{\delta^2 W}{\delta u^2} g(u)] = \frac{1}{2}(\omega_1 \sigma_1^2 u_1^2 + \omega_1 \sigma_1^2 u_1^2)$. (4.7)

If in (4.6) we choose $w_1 S^*(1+\alpha_1 S^*) = w_2 I^*(1+\alpha_2 I^*)$ from (4.7), it is easy to check that $LW(u) = -b_{11}u_1^2 - b_{22}u_2^2$, where $b_{11} = [\mu + \frac{\beta I^*(1+\alpha_2 I^*)}{(1+\alpha_1 S^*+\alpha_2 I^*)^2} - \frac{1}{2}\sigma_1^2]w_1, b_{12} = b_{21} = 0$ and $b_{22} = [\frac{\alpha_2 I^*(\mu+\alpha+r)}{1+\alpha_1 S^*+\alpha_2 I^*} - \frac{1}{2}\sigma_2^2]w_2$.

$LW(u) = -Y^T Z Y$, where $Y = (u_1, u_2)^T$ and $Z = [b_{ij}]_{2\times 2}$.Here, Z is positive definite if (i) $b_{ii} > 0, i = 1,2$; and (ii) $|Z| > 0$.

The inequality condition in (i) is satisfied by the assumptions of the theorem. Again, $|Z| > 0$. follows from the assumptions of the theorem. Hence according to Theorem 4.1, the proof is completed.

5. Numerical Examples and Simulations

In this section we provide comparative study with the following numerical examples for systems (1.1), (1.2) and (4.1), in order to illustrate their distinct dynamical natures.All numerical simulations are generated using MATLAB®(The Mathworks,Inc.,Version 7.10.0.499,R2010a)

Example 1. Choosing the model parameter values as follows:$A = 0.94, \mu = .0001, \alpha = 0.6, \beta = 0.1, r = 0.6, \alpha_1 = .01, \alpha_2 = 0.01$ and , we observe that the continuous system (1.1) is asymptotically stable (Figure 1a) whereas the discrete system (1.2) is unstable in nature (Figure 1b). Also the stochastic system (4.1) shows stable behavior (Figure 1c) for the same parameter values with $\sigma_1 = 0.5, \sigma_2 = 0.5$.

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Figure 1. Simulations of systems (1.1), (1.2) and (4.1) with $A = 0.94, \mu = .0001, \alpha = 0.6, \beta = 0.1, r = 0.6, \alpha_1 = .01, \alpha_2 = 0.01$. Figure1a: continuous system (1.1); Figure 1b: discrete system (1.2); Figure 1c: stochastic system (4.1) with $\sigma_1 = 0.5, \sigma_2 = 0.5$

Example 2. The model parameter values are chosen as follows:$A = 0.94, \mu = .0001, \alpha = 0.5, \beta = 0.1, r = 0.5, \alpha_1 = .01, \alpha_2 = 0.01$. The continuous system (1.1) is asymptotically stable (Figure 2a) and the discrete system (1.2) is stable in nature (Figure 2b). Furthermore, the stochastic system (4.1) exhibits stable behavior (Figure 2c) with the above choice of parameter values with $\sigma_1 = 0.5, \sigma_2 = 0.5$.

Example 3. The model parameter values are chosen as follows:$A = 0.94, \mu = .0001, \alpha = 0.5, \beta = 0.1, r = 0.5, \alpha_1 = 0, \alpha_2 = 0$. System (1.2) is unstable in nature as limit cycle appears into the system (Figure 3a). With the same choice of above parameter values except $\alpha_1 = 0.01$, system (1.2) still exhibits unstable behavior (Figure 3b) whereas by letting $\alpha_1 = 0, \alpha_2 = 0.01$, and keeping other parameter values same as above, we observe that system (1.2) is stable in nature (Figure 3c).

Example 4. Choosing parameter values $A = 0.94, \mu = .0001, \alpha = 0.5, \beta = 0.1, r = 0.5, \alpha_1 = 0, \alpha_2 = 0$ we observe that the stochastic system (4.1) is stable (Figure 4a). With the same choice of above parameter values except $\alpha_1 = 0.01$, the system still is stable (Figure 4b) whereas by letting $\alpha_1 = 0, \alpha_2 = 0.01$, and keeping other parameter values same as above, we observe that the system remains stable in nature (Figure 4c).

Example 5. Choosing parameter values $A = 0.94, \mu = .0001, \alpha = 0.5, \beta = 0.1, r =$

$0.5, \alpha_1 = 0, \alpha_2 = 0$ we observe that the continuous system (1.1) is stable (Figure 5a). With the same choice of above parameter values except $\alpha_1 = 0.01$, the system still is stable (Figure 5b) whereas by letting $\alpha_1 = 0, \alpha_2 = 0.01$, and keeping other parameter values same as above, we observe that the system remains stable in nature (Figure 5c).

6. Discussion

In this paper we have proposed and analyzed a discrete-time susceptible-infected model together with its stochastic version. From the analysis it is evident that for $R_0 \leq 1$ only the disease-free fixed point exists and there is no meaningful endemic fixed point, but for $R_0 > 1$ disease is able to successfully invade the system. The analysis also shows that interior fixed point E_2 remains stable under certain range of values beyond which it is unstable. Moreover, Hopf-bifurcation occurs in the system for a critical value of α_1. Furthermore, the study of stochastic version suggests that if the strength of white noise is under some threshold value, then the system is asymptotic mean square stable and numerical findings indicate that the system approaches to its asymptotic level despite some initial moderate oscillations.

When the disease induced death rate and the recovery rate both increases, the discrete system is unstable; whereas at lower values for these rates the same discrete system is stable.Further if α_1 exists in absence of α_2(i.e. when $\alpha_1 > 0$ and $\alpha_2 = 0$), we observe that the discrete system (1.2) is unstable while with the sole presence of α_2 devoid of α_1 the system (1.2) is stable. But in absence of inhibitory effects, the system (1.2) is always unstable. On the other hand the continuous system (1.1) and the stochastic system (4.1) are both stable in nature for each of the above three cases. Hence the numerical simulations indicate that the inhibitory effect has a large impact only on the discrete system.

It is interesting to note that in a previous study with a continuous time SARS model for quarantine and saturation due to public response to avoid infection (which corresponds to α_2 here) [**?**], incorporating these above-mentioned factors in the model resulted in the occurrence of bistability in the system, which otherwise would not have been possible; while in this present discrete-time model, the presence of α_2 without α_1 (which accounts for saturation effect due to the infectives I_n and thus is similar to [**?**]) tends to have a stabilizing effect. Furthermore, for the continuous and stochastic version of the present model the saturation factors seem to have no effect on the stability of the system. Therefore, in applications the exact impact of saturation/inhibitory factor on the dynamics of system is highly model dependent, and thus must be examined separately for each particular model. In brief, our analytical results and numerical findings suggest that the discrete version of the continuous model in general exhibits richer and at times distinctively different dynamics than its continuous and stochastic analogous systems, and hence may be useful in describing the complex dynamics one often witnesses in the real world.

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