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Deterministic and Stochastic Dynamics of an Eco-epidemiological Model

Research Article

B.N.R.Karuna[1]*, K.Lakshmi Narayan[1] and B.Ravindra Reddy[2]

1 Department of Basic Science Humanities, Vignan Institute of Technology and Science, Deshmukhi (v), Hyderabad, India.

2 Department of Mathematics, JNTU College of Engineering, Kukatpally, Hyderabad, India.

Abstract: A three-compartmental eco-epidemiological model consisting of three species, namely, the susceptible prey, the infected prey and the predator population is considered. The SIR type deterministic epidemiological models are revisited and stochastic modeling for those models is introduced. The exponential and mean square stability for the stochastic model is derived and the considerable discrepancy between the results of stochastic and deterministic models is supported by numerical simulations with a hypothetical set of data.

Keywords: Eco-epidemiology, Stochastic stability, Predator-prey model, Lyapunov function.

1. Introduction

The mathematical modelling of epidemics is done by using deterministic compartmental models where the population amongst whom the disease is spreading is divided into several classes. The deterministic compartment model was first proposed by McKendrick [1] and Kermack and McKendrick [2]. They used deterministic differential equations (so called SIR models) which assume that the population is divided into three compartments, susceptible (S), infectious (I) and recovered (R). Various mathematical models have been developed earlier for predicting and preventing the infectious diseases. The analysis of the models is useful for understanding the mechanism involved in the spread of infectious diseases. In nature, two or more interacting species subjected to parasitism have more biological significance where the persistence-extinction threshold of each population can be studied. Meanwhile, a little attention has been given so far to merge the major fields of study in mathematical biology, namely predator-prey systems and models for transmissible diseases as reported in Haque and Venturino [3, 4], Haque and Greenhalgh [5], Liu et al. [6], Venturino [7], Xiao and Chen [8, 9] and Hethcote et al. [10]. In order to study the influence of disease on an environment, two or more interacting species should be presented. On the other hand, the mechanism of the spread of infectious diseases is probabilistic in nature and the intrinsic fluctuation or noise occurs in the time evolution of the number in each compartment. This fluctuation can play a critical role in the transmission dynamics of infectious diseases, especially when the size of the population is small. The intrinsic fluctuations when the size of population is small cannot be identified with the deterministic epidemic models. To capture the fluctuation and for an accurate description of disease transmission the stochastic approach can be used. A good introduction for stochastic

* E-mail: karunakrao@gmail.com

epidemic models can be found in the literature [11-13]. This approach can become an attractive tool for modelling many biomedical problems, since it is able to give a more realistic description of the phenomena of interest without excessively complicating the settings.

In this paper, we shall focus on an eco-epidemiological system consisting of three species, namely, the sound prey (which is susceptible), the infected prey (which becomes infective by some viruses) and the predator population. We considered the saturated incidence rate $g(I(t))S(t)$ given by Capasso and Serio [14], where $g(I(t)) = \beta I(t)/(1+\alpha I(t))$; $\beta I(t)$ measures the infection force of the disease and $1/(1+\alpha I(t))$ measures the inhibition effect from the behavioural change of the susceptible individuals when their number increases or from the crowding effect of the infective individuals. Numerous field studies show that infected prey is more vulnerable to predation compared with their non-infected counterpart as given by Hudson et al. [15], Lafferty and Morris [16] and Murray et al. [17]. The predation rates on infected prey may be thirty one times higher compared to that on susceptible prey as given by Lafferty et al. [16]. We consider the case when the predator mainly eats the infected prey with Leslie-Gower ratio dependent schemes as proposed by Song and Li [18], Nindjin et al. [19], Aziz-Alaoui et al. [20], Hsu et al. [21] and Aziz-Alaoui [22]. Therefore, the predator consumes the prey according to the ratio-dependent functional response and the predator grows logistically with intrinsic growth rate d and carrying capacity proportional to the prey populations size $I(t)$. We also propose a nonlinear stochastic differential equations model which allows describing the noise in the systems under investigation. The outline of the paper is as follows; we introduce deterministic models Also, we describe the stochastic modeling of the epidemic models.

2. The Mathematical Model

The model equations are

$$\begin{aligned} \frac{dS(t)}{dt} &= b - ds - \frac{\beta SI}{1+\alpha I} \\ \frac{dI(t)}{dt} &= \frac{\beta SI}{1+\alpha I} - (eP+d)I \\ \frac{dP(t)}{dt} &= \mu P\left(1 - \frac{nP}{I}\right) \end{aligned} \tag{1}$$

where $S(t)$ is the density of the susceptible prey population, $I(t)$ is the density of the infective prey population and $P(t)$ is the density of their predator population. $\frac{\beta SI}{1+\alpha I}$ is the saturated incidence rate, b is the birth rate of the sound prey, β is the infection rate, α is the half saturation constant of infection, e is the search rate, μ is the intrinsic growth rate of the predator and n is the maximum value of the percapita reduction rate of prey due to predator.

2.1. Equilibrium Points

The system (1) has the two equilibrium points:

(1). the boundary equilibrium $E_1(S_1, I_1, 0)$, where $S_1 = \frac{d}{\beta}(1+\alpha I_1)$ and $I_1 = \frac{b\beta - d^2}{d(\beta + d\alpha)}$

(2). the positive equilibrium $E_2(S_2, I_2, P_2)$ where $P_2 = \frac{I_2}{n}$, $S_2 = \frac{1}{n\beta}((eI_2 + dn)(1+\alpha I_2))$ and I_2 is the positive root of $A_1 I^2 + A_2 I + A_3 = 0$, where $A_1 = e\beta + d\alpha e$, $A_2 = \beta dn + de + d^2 n\alpha$, $A_3 = n(d^2 - b\beta)$.

Clearly, E_2 is positive if $A_3 < 0$, i.e., $\beta > \frac{d^2}{b}$ and $I_2 = \frac{-A_2 + \sqrt{(A_2^2 - 4A_1A_3)}}{2A_1}$. Let $E^*(S^*, I^*, P^*)$ be any arbitrary equilibrium. Then the characteristic equation about E^* is given by

$$\begin{vmatrix} -d-\frac{\beta I^*}{1+\alpha I^*}-\lambda & \frac{-\beta S^*}{(1+\alpha I^*)^2} & 0 \\ \frac{\beta I^*}{1+\alpha I^*} & \frac{\beta S^*}{(1+\alpha I^*)^2}-(eP^*+d)-\lambda & -eI^* \\ 0 & \frac{\mu n {P^*}^2}{{I^*}^2} & \mu-\frac{2\mu n P^*}{I^*}-\lambda \end{vmatrix} = 0 \tag{2}$$

For equilibrium E_2, (2) reduces to

$$\Delta = \begin{vmatrix} -d-\lambda & \frac{-\beta S_2}{(1+\alpha I_2)^2} & 0 \\ \frac{\beta I_2}{1+\alpha I_2} & \frac{\beta S_2}{(1+\alpha I_2)^2}-(eP_2+d)-\lambda & -eI_2 \\ 0 & \frac{\mu}{n} & -\mu-\lambda \end{vmatrix} = 0 \tag{3}$$

that is

$$\Delta(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0. \tag{4}$$

where,

$$\begin{aligned} a_1 &= \left(2d+\mu+eP_2-\frac{\beta S_2 I_2}{(1+\alpha I_2)^2}\right) \\ a_2 &= \left(\frac{\beta S_2 d}{(1+\alpha I_2)^2}+eP_2 d+d^2+\frac{\beta S_2 I_2}{(1+\alpha I_2)^3}+2eP_2\mu+2d\mu-\frac{\beta S_2 \mu}{(1+\alpha I_2)^3}\right) \\ a_3 &= \left(2eP_2 d\mu+d^2\mu+\frac{\beta S_2 I_2 \mu}{(1+\alpha I_2)^3}-\frac{\beta S_2 d\mu}{(1+\alpha I_2)^2}\right). \end{aligned}$$

We see that $a_1 > 0$, $a_3(a_1a_2 - a_3) > 0$. Hence, by Routh-Hurwitz criterion, the positive equilibrium E_2 is locally asymptotically stable.

2.2. Global Stability

We will now establish the global stability of the positive equilibrium E_2. Let (S_2, I_2, P_2) be any positive solution of system (1). Consider the following Lyapunov functional;

$$V(t) = \left(S - S_2 - S_2 ln\frac{S}{S_2}\right) + \left(I - I_2 - ln\frac{I}{I_2}\right) + \left(P - P_2 - ln\frac{P}{P_2}\right)$$

Calculating the time derivative of this functional V(t) along the trajectories of system (1) yields

$$\begin{aligned} \frac{dV}{dt} &= \left(1-\frac{S_2}{S}\right)\frac{dS}{dt}+\left(1-\frac{I_2}{I}\right)\frac{dI}{dt}+\left(1-\frac{P_2}{P}\right)\frac{dP}{dt} \\ &= \left(\frac{S-S_2}{S}\right)\left[b-dS-\frac{\beta SI}{1+\alpha I}\right]+\left(\frac{I-I_2}{I}\right)\left[\frac{\beta SI}{1+\alpha I}-(eP+d)\,I\right]+\left(\frac{P-P_2}{P}\right)\left[\mu P\left(\frac{I-nP}{I}\right)\right] \end{aligned}$$

Substituting,

$$\begin{aligned} b-\frac{\beta SI}{1+\alpha I} &= dS_2 \\ \frac{\beta SI}{1+\alpha I} - ePI &= dI_2 \\ \mu P\left(1-\frac{nP}{I}\right) &= \mu P\left(1-\frac{P}{P_2}\right) \end{aligned}$$

We have,

$$\begin{aligned} \frac{dV}{dt} &= \left(\frac{S-S_2}{S}\right)[dS_2-dS]+\left(\frac{I-I_2}{I}\right)[dI_2-dI]+\left(\frac{P-P_2}{P_2}\right)[\mu\,(P_2-P)] \\ &\le -\frac{d}{S}(S-S_2)^2-\frac{d}{I}(I-I_2)^2-\frac{\mu}{P_2}(P-P_2)^2 < 0. \end{aligned}$$

Thus the system is globally asymptotically stable.

2.3. Dynamics of the Stochastic Differential Equation

Under stochastic perturbations system (1) yields

$$\begin{aligned}\frac{dS}{dt} &= b - dS - \frac{\beta SI}{1+\alpha I} + \sigma_1 (S - S^*)\, d\xi_1^1 \\ \frac{dI}{dt} &= \frac{\beta SI}{1+\alpha I} - (eP + d)\, I + \sigma_2 (I - I^*)\, d\xi_1^2 \\ \frac{dP}{dt} &= \mu P \left(1 - \frac{nP}{I}\right) + \sigma_3 (P - P^*)\, d\xi_1^3\end{aligned} \tag{5}$$

where, s_i's $(i = 1, 2, 3)$ are real constants, ξ_1^i $(i = 1, 2, 3)$; are independent from each other is a standard Wiener process. The system (5) has the same equilibrium as that of (1). Hence the stochastic differential equation (5) can be centered at its positive equilibrium point E_2 by the change of variables

$$u_1 = S - S^*, \quad u_2 = I - I^*, \quad u_3 = P - P^* \tag{6}$$

Stochastic stability of the model at the positive equilibrium E_2: The linearised stochastic differential equation around E_2 takes the form

$$du(t) = f(u(t))dt + g(u(t))d\xi(t) \tag{7}$$

where $u(t) = col[u_1(t), u_2(t), u_3(t)]$ and

$$f(u(t)) = \begin{bmatrix} -\left(d + \frac{\beta I}{1+\alpha I}\right) & -\frac{\beta S}{(1+\alpha I)^2} & 0 \\ \frac{\beta I}{(1+\alpha I)} & \frac{\beta S}{(1+\alpha I)^2} - (eP + d) & -eI \\ 0 & \frac{\mu n P^2}{I^2} & \mu - \frac{2\mu n P}{I} \end{bmatrix} \tag{8}$$

$$g(u) = \begin{bmatrix} \sigma_1 u_1 & 0 & 0 \\ 0 & \sigma_2 u_2 & 0 \\ 0 & 0 & \sigma_3 u_3 \end{bmatrix}$$

Let $C^{1,2}\left([0, +\infty) \times R^2, R^+\right)$ be the family of non-negative functions. $W(t, u)$ defined on $[0, +\infty) \times \mathrm{R}^2$ is continuously differentiable function with respect to t and twice with respect to u. We define the differential operator L for a function $W(t, u)$ by

$$\begin{aligned} LW(t,u) &= \frac{\partial W(t,u)}{\partial t} + f^T(u) \frac{\partial W(t,u)}{\partial u} + \frac{1}{2} Tr \left[g^T(u) \frac{\partial^2 W(t,u)}{\partial u^2} g(u)\right] \\ \frac{\partial W}{\partial u} &= col \left[\frac{\partial W}{\partial u_1}, \frac{\partial W}{\partial u_2}, \frac{\partial W}{\partial u_3}\right] \quad \text{and} \\ \frac{\partial^2 W(t,u)}{\partial u^2} &= \frac{\partial^2 W}{\partial u_i \partial u_j}; \quad i, j = 1, 2 \end{aligned} \tag{9}$$

and T means transposition. With reference to the book authored by Afanasev et al. [23] the following theorem holds.

Theorem 2.1. *Suppose there exists a function* $W(t, u) \in C^{1,2}\left([0, +\infty) \times R^2, R^+\right)$ *satisfying the following inequalities*

$$K_1 |u|^p \le W(t, u) \le K_2 |u|^p \tag{10}$$

$$LW(t, u) \le -K_3 |u|^p$$

where, K_1*,* K_2*,* K_3 *and* p *are positive constants. Then the trivial solution of equation (7) is exponentially p-stable for* $t \ge 0$*. Moreover, if in equations (10) and (11),* $p = 2$*, the trivial solution of equation (7) is globally asymptotically stable in probability.*

Theorem 2.2. *Suppose that* $\sigma_1^2 \leq 2\left(d+\frac{\beta I}{1+\alpha I}\right), \sigma_2^2 \leq 2\left(eP+d-\frac{\beta S}{(1+\alpha I)^2}\right), \sigma_3^2 \leq 2\left(\frac{2\mu n P}{I}-\mu\right)$ *hold, then the trivial solution of equation* (7) *is asymptotically mean square stable.*

Proof. Consider the Lyapunov function

$$W(u)=\frac{1}{2}\left[w_1u_1^2+w_2u_2^2+w_3u_3^2\right] \tag{11}$$

where, w_1, w_2, w_3 are non-negative constants to be chosen in the following. It is easy to check that inequalities in equation (10) hold true for $p=2$.

$$\begin{aligned} LW(u) = w_1\left[-\left(d+\frac{\beta I}{1+\alpha I}\right)u_1-\frac{\beta S}{(1+\alpha I)^2}u_2\right]u_1+w_2\left[\left(\frac{\beta I}{1+\alpha I}\right)u_1+\left(\frac{\beta S}{(1+\alpha I)^2}-(eP+d)\right)u_2-eIu_3\right]u_2 \\ +w_3\left[0u_1+\frac{\mu}{n}u_2-\mu u_3\right]u_3 \end{aligned} \tag{12}$$

Choose $w_1u_2=w_2u_1$ and $w_2u_3=w_3u_2$ i.e., $w_1.\frac{\beta S}{(1+\alpha I)^2}=w_2.\frac{\beta I}{1+\alpha I}$, $w_2.eI=w_3.\frac{\mu}{n}$ then we have,

$$\begin{aligned} \frac{1}{2}Tr\left[g^T(u)\frac{\partial^2 W(t,u)}{\partial u^2}g(u)\right] &= \frac{1}{2}\left[w_1u_1^2+w_2u_2^2+w_3u_3^2\right] \\ \Rightarrow LW(u) &= -\left[\left(d+\frac{\beta I}{1+\alpha I}\right)-\frac{1}{2}\sigma_1^2\right]w_1u_1^2-\left[(eP+d)-\frac{\beta S}{(1+\alpha I)^2}-\frac{1}{2}\sigma_2^2\right]w_2u_2^2-\left[\mu-\frac{1}{2}\sigma_3^2\right]w_3u_3^2 \\ &< 0. \end{aligned}$$

According to Theorem 2.1, we conclude that the trivial solution of system (7) is globally asymptotically stable. □

3. Numerical Simulations

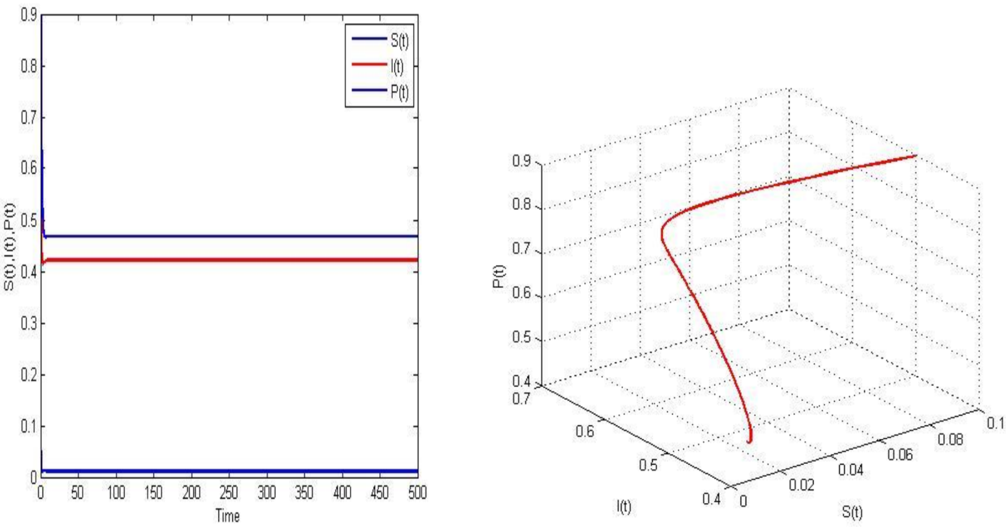

Figure 1: Numerical simulations for the system when $b=0.2$, $\beta=50$, $\alpha=0.614$, $c=0.528$, $d=0.221$, $\delta=0.9$ and $h=0.9$.

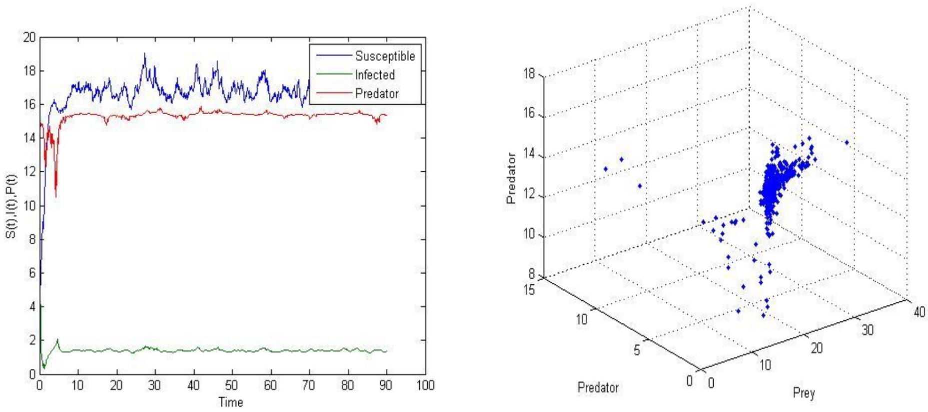

Figure 2: Numerical simulations for the system when $b = 10$, $\beta = 0.5$, $\alpha = 0.614$, $c = 0.28$, $d = 0.221$, $\delta = 0.08$, $h = 0.09$; $\sigma_1 = 0.01$, $\sigma_2 = 0.02$ and $\sigma_3 = 0.04$.

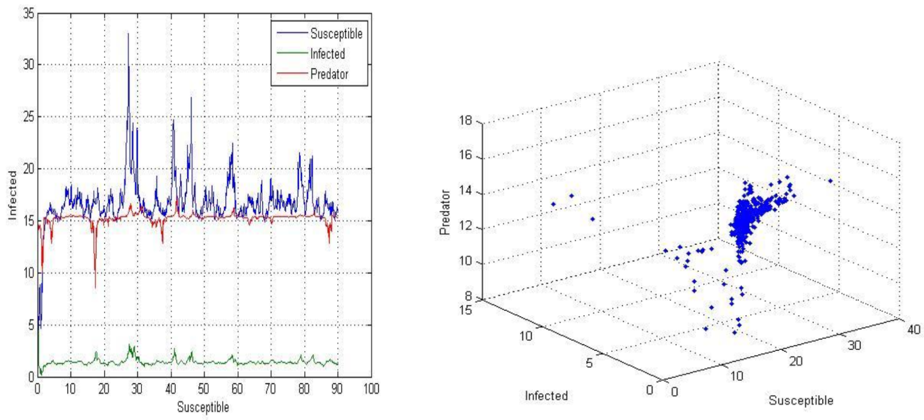

Figure 3: Numerical simulations for the system when $b = 10$, $\beta = 0.5$, $\alpha = 0.614$, $c = 0.28$, $d = 0.221$, $\delta = 0.08$, $h = 0.09$, $\sigma_1 = 0.03$, $\sigma_2 = 0.04$, $\sigma_3 = 0.06$.

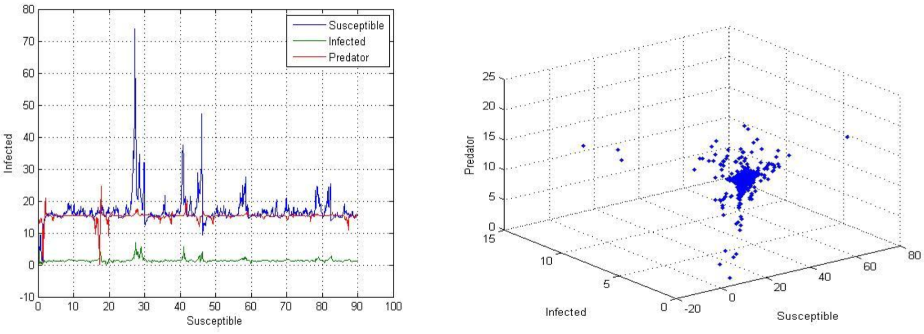

Figure 4: Numerical simulations for the system when $b = 10$, $\beta = 0.5$, $\alpha = 0.614$, $c = 0.28$, $d = 0.221$, $\delta = 0.08$, $h = 0.09$, $\sigma_1 = 0.05$, $\sigma_2 = 0.06$, $\sigma_3 = 0.09$.

4. Conclusion

A three-compartmental eco-epidemiological model consisting of a prey-predator system is studied. Here the prey population is divided into two groups, infected and non-infected. The system is analysed for its equilibria and their stability where the disease changes the external features or the behaviour of the prey so as to make infected individuals more vulnerable to predation. Local stability condition of E_2 is established which is very difficult to interpret biologically. The stochastic model is proved to be globally asymptotically stable which depends on the white noise. Our theoretical study has only captured the major mechanisms associated with an eco-epidemiological aspect where a predator selectively consumes infected prey. The processes prescribed by the model actually functioning in similar manner can be verified by experimental or field investigations in this direction. Lastly the numerical solutions are used to support the analytical results.

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