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Bistable dynamics of an insect–pathogen model

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Abstract. We consider a model for insect–pathogen interaction where the insect population is divided into two groups, one group susceptible to disease and other resistant to disease. An individual born susceptible to or resistant to disease depends on the local population levels at the start of each generation. Here we consider density-dependent models of transmission because we characterize diseases that spread through environmental propagules or through random contact among individuals. We consider the case where the fraction of resistant individuals increases as the total population increases. White and Wilson (*Theor. Popul. Biol.* **56**, 163 (1999)) have reported the results of density-dependent monotonic increase of resistance class by choosing a particular type of function. In this paper, we have chosen a class of monotonic density-dependent resistance functions and studied their effects on insect–pathogen dynamics. In particular, we have investigated the effects of different types of monotonic density-dependent resistance on the bistable nature of the model. Numerical simulation results are presented and interpreted.

Keywords. Insect–pathogen model; bistable system; bifurcation.

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1. Introduction

There is a growing interest in the potential impact of pathogens on the dynamics of insects and other invertebrates. Part of the increased interest comes from technological advances in insect pathology that have revealed the ubiquity of infection of insects by virus, bacteria, and fungi, and have provided the tools for epidimiological study. At the same time, the importance of modelling insect–pathogen interactions has also been emphasized by population dynamic models. The application of simple ecological models to insect–pathogen dynamics starts with the work of Anderson and May [1,2]. In particular, Anderson and May have argued that the well-documented population cycles of forest insects may be due to interactions with pathogens. The majority of insect host–pathogen models assume that the number of new infections is linearly proportional to the density of susceptible hosts and the density of infectious agents. Some experimental evidences [3] suggest

that the infection process is nonlinear. According to Wilson and Reeson [4], for many insect pest species we can expect phenotypic plasticity to result in a decline in susceptibility to disease with increasing population density, due to an increased investment in pathogen-resistance mechanisms at high densities. However, some natural phenomena cannot be considered as continuous because they occur at certain moments of time only: for instance, the case for animals that reproduce seasonally or animals that are vulnerable to attacks during a certain period of their lifecycle only. These characteristics gave rise to discrete time models (difference equations) that were particularly developed by the consumer-resource modelling community with respect to host–parasitoid interactions [5]. One key factor appears to be whether transmission of the pathogen is a function of the absolute density of infected hosts (density-dependent), or whether it is a function of the proportion of the total population that is infected with the pathogen (frequency-dependent). Density-dependent models of transmission are generally used to characterize diseases which spread through environmental propagules or through random contact among individuals. The processes controlling disease resistance can strongly influence the population dynamics of insect outbreaks. Evidence that disease resistance is density-dependent is accumulating [6,7] but the exact form of this relationship is highly variable from species to species. It has been hypothesized that insects experiencing high population densities might allocate more energy to disease resistance than those at lower densities, because they are more likely to encounter density-dependent pathogens. In contrast, the increased stress of high-density conditions might leave insects more vulnerable to disease. Both scenarios have been reported for various outbreak of lepidoptera in [8].

Existence of some costs associated with maintaining and operating resistance mechanisms has been demonstrated in plants and bees [9,10]. Such costs have been demonstrated in a number of interactions between insects and their pathogens and parasites [11–13]. This fact motivated us to investigate the effects of cost of disease resistance on the stability properies of the insect–pathogen system.

White and Wilson [14] have reported the results of insect–pathogen interactions for a particular type of monotonic density-dependent resistance. In this paper, we have chosen a class of monotonic density-dependent resistance functions and studied their effects on insect–pathogen dynamics. We have discussed in detail the effects of constant (density-independent) resistance in insect–pathogen interactions. A bistable dynamical system is one that possesses two asymptotic stable states for a fixed set of parameters depending on initial conditions. In particular, we have investigated the effects of different types of monotonic density-dependent resistance on the bistable nature of the model. In the next section, model formulation is done with suitable assumptions. In §3 determination of steady-state population levels and their stability criteria are determined. In §4 numerical simulation results are presented and compared with the results of White and Wilson [14]. Finally, conclusion is drawn in §5.

2. Model

We formulate a model using the following assumptions:

(a) We use a discrete-time population model because many insects have non-overlapping generations.

(b) Assuming that no major density-dependent effects remain after larval insect stage of insect development we consider only the larval insect stage in our model. The state variables relating to larval population are defined as

S_i = density of susceptible larvae at the start of the ith generation.

R_i = density of resistant larvae at the start of the ith generation.

(c) We assume that individuals are born into susceptible or resistant classes and remain within that class throughout their development. Particularly,the fraction of the individuals giving birth to susceptibles, $f(N)$, decreases with the total density of larvae which have survived the previous generation to reproduce. This is the same as in White and Wilson [14]. Only difference is that White and Wilson [14] have chosen $f(N) = \alpha/(1 + \gamma N)$ but there is no biological reason for choosing this particular type of monotonic decreasing function. To investigate the effects of different types of monotonic decreasing density-dependent susceptible class we first choose function $f(N)$ as $f(N) = \alpha/(1 + \gamma N^{\beta})$, where γ = constant $> 0, 0 < \alpha$ = constant $< 1,\ 0 \leq \beta < \infty$ and N is the number of surviving individuals at the end of a generation. Notice that this $f(N)$ is the same as that in White and Wilson [14] for $\beta = 1$. We have also considered the case where $f(N) = \alpha/\mathrm{e}^{\gamma N}$ where $\alpha,\ \gamma$ and N have the same meaning. This $f(N)$ is similar to the $f(N)$ of White and Wilson [14] for very small N limit.

(d) We assume that resistance has a phenotypically plastic response to host density (same assumption as in White and Wilson [14]).

(e) We assume all diseased individuals die in a single generation.

(f) After the death of infected individuals some free-living disease pathogen propagules are produced which have some constant survival probability during any generation. Here we include the state variable P_i = density of free-living pathogen particles at the start of the ith generation.

(g) The fraction of the susceptible larvae surviving to the end of their generation is a decreasing function of the density of free-living pathogen particles. $\sigma_{\mathrm{S}}(P_i)$ is the density-dependent survival of susceptibles, given as

$$\sigma_{\mathrm{S}}(P_i) = \tau \left(1 + \frac{a P_i}{k}\right)^{-k} \tag{1}$$

(same as in White and Wilson [14]) where $\tau,\ a,\ k$ = constants > 0.

We use a negative binomial distribution of pathogen attacks with the parameter k, an inverse measure of the degree of aggregation of such attacks [15].

(h) Costs of resistance are fundamentally important in epidemiology and in the ecology and evolution of host–pathogen interactions. We assume that the cost associated with resistance to disease has the effect of reducing larval survival for the resistant class in the absence of disease. The differential survival arising from costs associated wih host resistance to disease is included in the model by assuming that $\sigma_{\mathrm{S}}(0) = \tau > \sigma_{\mathrm{R}}$ where σ_{R} is the density-independent survival of resistants. Survival of susceptibles may fall below that of some non-trivial population density (i.e. $\sigma_{\mathrm{S}}(P_i > 0) < \sigma_{\mathrm{R}}$) due to the disease.

Under the above assumptions we obtain the following model:

$$\begin{aligned} S_{i+1} &= \Lambda_S(f(N_i)N_i) \\ R_{i+1} &= \Lambda_R(1 - f(N_i))N_i \\ P_{i+1} &= \sigma_P P_i + \lambda(\sigma_S(0) - \sigma_S(P_i))S_i \end{aligned} \tag{2}$$

where

$$N_i = (\sigma_S(P_i)S_i + \sigma_R R_i)$$

is the total number of individuals which survive to the end of the ith generation and Λ_S is the number of susceptibles born per surviving individual, Λ_R is the number of resistants born per suviving individual, λ is the number of pathogen propagules produced per infected death, σ_P is the density-independent survival of pathogen propagules.

3. Equilibrium points and their stability

In this section we find the equilibrium points of the model and discuss their stability. We consider the complete model (2) and find its steady states and determine the conditions for the stability of its steady states.

Steady states of the complete model are solutions of the following system:

$$\begin{aligned} S_i &= \Lambda_S f(N_i)N_i, \\ R_i &= \Lambda_R(1 - f(N_i))N_i, \\ P_i &= \sigma_P P_i + \lambda(\sigma_S(0) - \sigma_S(P_i))S_i. \end{aligned} \tag{3}$$

The complete system has non-trivial steady-states obtained from the implicit equation

$$f\left[\frac{\omega_2(P)}{\omega_1(P)}\right] - \omega_1(P) = 0, \tag{4}$$

where

$$\omega_1(P) = \frac{1 - \sigma_R\Lambda_R}{\sigma_S(P)\Lambda_S - \sigma_R\Lambda_R}$$

and

$$\omega_2(P) = \frac{P(1 - \sigma_P)}{\lambda\Lambda_S(\sigma_S(0) - \sigma_S(P))}.$$

As $0 < f(N) < 1$, the non-trivial steady state exists only if $0 < \omega_1(P) < 1$. This requires that either $\sigma_S(P)\Lambda_S > 1$ and $\sigma_R\Lambda_R \leq 1$ or $\sigma_S(P)\Lambda_S \leq 1$ and $\sigma_R\Lambda_R > 1$.

The steady-state values of the susceptibles, resistants and total host levels are then given by the following relations:

$$\begin{aligned} S &= \Lambda_S\omega_2(P), \\ R &= \Lambda_R[1 - \omega_1(P)]\frac{\omega_2(P)}{\omega_1(P)}, \\ N &= \frac{\omega_2(P)}{\omega_1(P)}, \end{aligned} \tag{5}$$

all of which can be determined once P is known.

Case 1: Density-independent resistance, i.e., $f(N) = \alpha < 1$

If $f(N) = \text{constant} = \alpha$, then using (4) we obtain

$$\sigma_S(P) = \frac{1 - (1-\alpha)\sigma_R\Lambda_R}{\alpha\Lambda_S}. \tag{6}$$

As $0 < \sigma_S(P) < 1$, we require that $0 < 1 - (1-\alpha)\sigma_R\Lambda_R < \alpha\Lambda_S$ for the existence of non-trivial steady state.

Case 2: Density-dependent resistance, i.e., $f(N) = \alpha/(1+\gamma N^\beta)$

Steady-state value of P are given by the solutions of the following equation:

$$\frac{\alpha\omega_1^\beta(P)}{\omega_1^\beta(P) + \gamma\omega_2^\beta(P)} = \omega_1(P) \tag{7}$$

and then the steady-state values of S and R are given by (5).

Case 3: Density-dependent resistance, i.e., $f(N) = \alpha/e^{\gamma N}$

Steady-state value of P are given by the solutions of the following equation:

$$\frac{\alpha}{e^{\omega_2(P)/\omega_1(P)}} = \omega_1(P) \tag{8}$$

and then the steady-state values of S and R are given by (5).

The Jacobian at the non-trivial steady state is

$$J = \begin{pmatrix} \sigma_P - \lambda\sigma_S'(P)S & \lambda(\sigma_S(0) - \sigma_S(P)) & 0 \\ \Lambda_S\sigma_S'(P)S\eta(N) & \Lambda_S\sigma_S(P)\eta(N) & \Lambda_S\sigma_R\eta(N) \\ \Lambda_R\sigma_S'(P)S(1-\eta(N)) & \Lambda_R\sigma_S(P)S(1-\eta(N)) & \Lambda_R\sigma_R(1-\eta(N)) \end{pmatrix}, \tag{9}$$

where $\eta(N) = f(N) + Nf'(N)$.

The characteristic equation is the cubic plynomial of the form

$$A(\chi) = \chi^3 + a_1\chi^2 + a_2\chi + a_3 = 0, \tag{10}$$

where the coefficients a_i are given by the relations

$$\begin{aligned} a_1 &= -\sigma_P + \lambda\sigma_S'(P)S - \Lambda_R\sigma_R(1-\eta(N)) - \Lambda_S\sigma_S(P)\eta(N), \\ a_2 &= -\lambda(\sigma_S(0) - \sigma_S(P))\Lambda_S\sigma_S'(P)S\eta(N), \\ &\quad +(\sigma_P - \lambda\sigma_S'(P)S)(\Lambda_R\sigma_R(1-\eta(N)) + \Lambda_S\sigma_S(P)\eta(N)), \\ a_3 &= 0. \end{aligned} \tag{11}$$

As $a_3 = 0$, by Jury's conditions for linear stability ($-1 < \chi < 1$) are given by

$$\begin{aligned} &A(1) = 1 + a_1 + a_2 > 0, \\ &-A(-1) = 1 - a_1 + a_2 > 0, \\ &1 > |a_2|. \end{aligned} \tag{12}$$

We do not discuss the special case with no resistance class and the case with no pathogen because detailed analysis of these cases are reported by White and Wilson [14].

4. Results

In this section we present numerical simulation results of the model (2) for different sets of parameter values and initial conditions. In all the figures the blue lines represent the susceptible population levels S_i, the green lines represent the resistant population levels R_i, the red lines represent the parasite population levels P_i. In figure 1 the dynamics of density-independent resistance (i.e., $f(N) = \alpha$) to disease are presented. In figure 1, model parameters are $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R = 0.5$. In figures 1a, 1b and 1c the time evolution of the population is plotted for $\alpha = 0.6, 0.8$ and 0.9 respectively. The bifurcation diagram of the model with respect to α is shown in figure 1d. Therefore, the model may have complex dynamic behaviour depending on the value of α which is clear from the bifurcation diagram with respect to α. Now we consider the density-dependent function $f(N) = \alpha/(1 + \gamma N^\beta)$ and effects of β variation in the bistable nature is shown in figures 2 and 3. In figure 2 the model parameters are $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R = 0.5, \beta = 1.0$. In figure 2a the initial condition is (10, 1, 10) and in figure 2b the initial condition is (20, 1, 20) and for both the cases $\gamma = 0.25$. In figure 2c the initial condition is (10, 1, 10) and in figure 2d the initial condition is (20, 1, 20) and for both the cases $\gamma = 0.001$. It is clear that the system reaches two different steady states depending on the initial conditions. Therefore, the system has bistable nature which is reported by White and Wilson [14]. Now we vary β and show that for different values of β bistability exists in the model. In figure 3, the model parameters are $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R =$

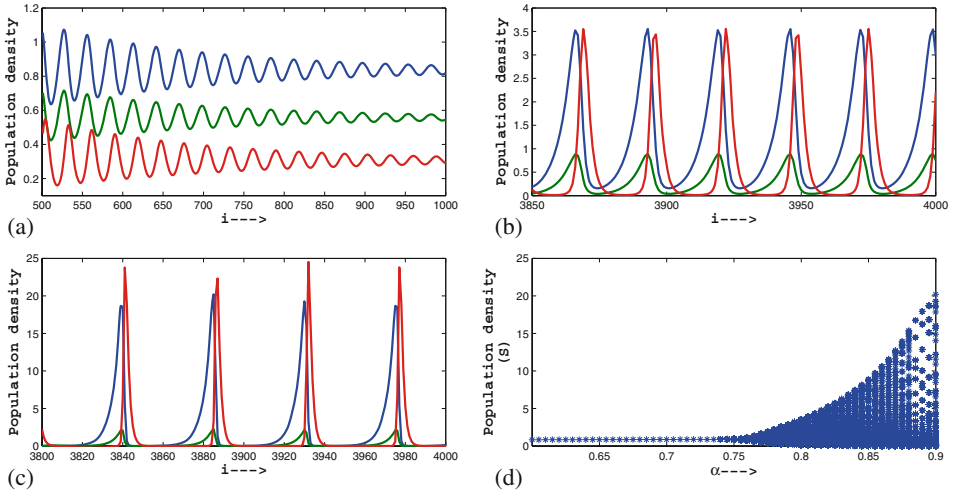

Figure 1. Dynamics of density-independent resistance to disease. **(a)** Time evolution of the population for $\alpha = 0.6$, **(b)** $\alpha = 0.8$ and **(c)** $\alpha = 0.9$. **(d)** Bifurcation diagram with respect to α. In all figures the blue lines give the susceptible population levels S_i, the green lines give the resistant population levels R_i, the red lines give the parasite population levels P_i. Model parameters common to all figures are: $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \gamma = 0.25, \sigma_R = 0.5, \beta = 1.0$.

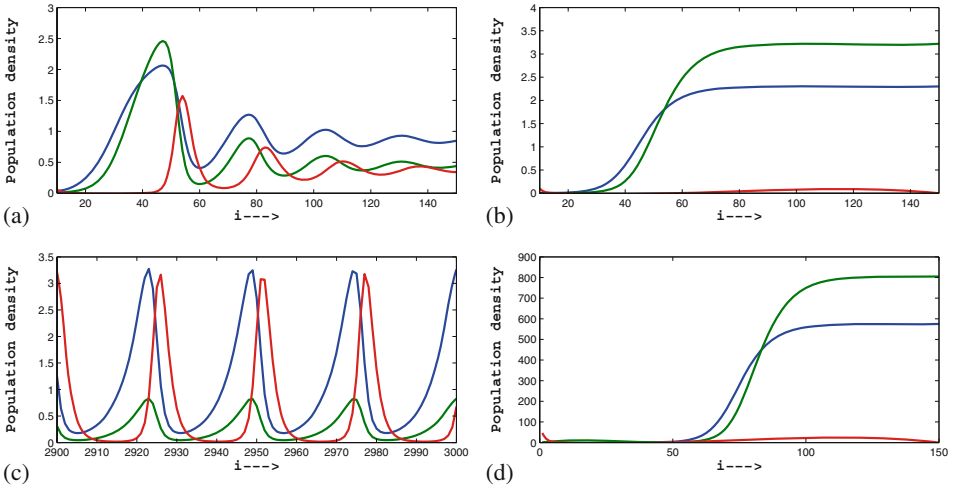

Figure 2. Bistable nature of the system is shown. In **(a)** initial condition is (10, 1, 10) and in **(b)** the initial condition is (20, 1, 20) and $\gamma = 0.25$. In **(c)** initial condition is (10, 1, 10) and in **(d)** the initial condition is (20, 1, 20) and $\gamma = 0.001$. In all the figures the blue lines give the susceptible population levels S_i, the green lines give the resistant population levels R_i, the red lines give the parasite population levels P_i. Model parameters common to all figures are: $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R = 0.5, \beta = 1.0$.

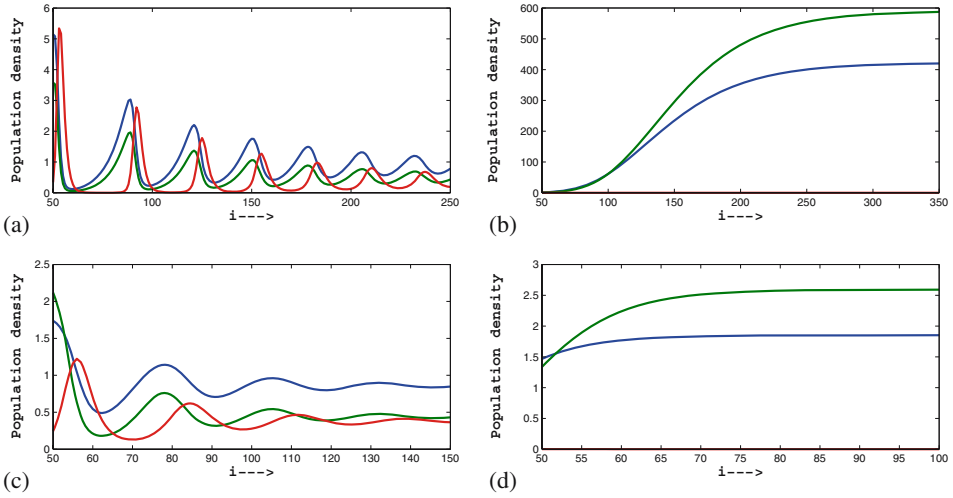

Figure 3. Bistable nature of the system is shown. In **(a)** initial condition is (10, 1, 10) and in **(b)** the initial condition is (20, 1, 20) and $\beta = 0.2$. In **(c)** initial condition is (10, 1, 10) and in **(d)** the initial condition is (20, 1, 20) and $\beta = 1.2$. In all the figures the blue lines give the susceptible population levels S_i, the green lines give the resistant population levels R_i, the red lines give the parasite population levels P_i. Model parameters common to all figures are: $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R = 0.5, \gamma = 0.25$.

0.5, $\gamma = 0.25$. In figure 3a initial condition is (10, 1, 10) and in figure 3b the initial condition is (20, 1, 20) and $\beta = 0.2$ for both the cases. In figure 3c the initial condition is (10, 1, 10) and in figure 3d the initial condition is (20, 1, 20) and $\beta = 1.2$ for both the cases. The bistable nature of the model for different β is clear from the figures. Now we consider another type of function $f(N) = \alpha/e^{\gamma N}$ and choose the model parameters as $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R = 0.5$ and depict the dynamics of the model for different γ in figure 4. In figure 4a initial condition is (10, 1, 10) and in figure 4b the initial condition is (20, 1, 20) and $\gamma = 0.25$ for both the cases. In figure 4c the initial condition is (10, 1, 10) and in figure 4d the initial condition is (20, 1, 20) and $\gamma = 0.005$ for both the cases. The bistable nature of the model exists for this type of density-dependent function also. Bifurcation diagram of the model with respect to γ for $f(N) = \alpha/e^{\gamma N}$ is plotted in figure 5. In all the figures the blue dots represent the susceptible population levels S_i, the green dots represent the resistant population levels R_i, the red dots represent the parasite population levels P_i. The doted circles and triangles represent results for different sets of initial conditions. Model parameters common to all figures are $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R = 0.5, \alpha = 0.8$. From the figure it is clear that there exists a critical γ below which the bistable nature of the model exists and above which it does not. At the critical γ two different stable states coincide. The interval of values of γ where bistability exists decreases with the decrease of α which is clear from the bifurcation (figure 6). In figure 6 all the parameters are the same except $\alpha = 0.6$. Different line styles represent results for different sets of initial conditions here. In figure 7 bifurcation diagram of the susceptible population of the model with respect to β is plotted for $f(N) = \alpha/(1 + \gamma N^{\beta})$ for $\alpha = 0.8$. Model parameters are

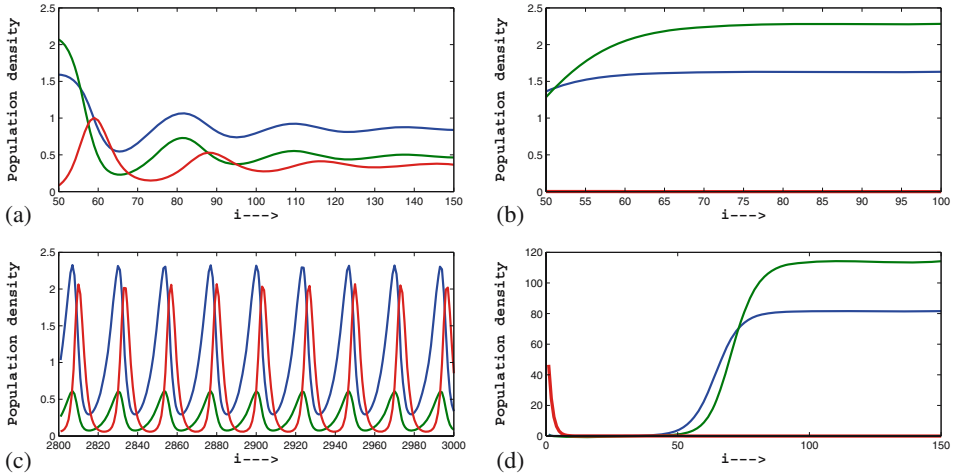

Figure 4. Bistable nature of the system is shown. In **(a)** initial condition is (10, 1, 10) and in **(b)** the initial condition is (20, 1, 20) and $\gamma = 0.25$. In **(c)** initial condition is (10, 1, 10) and in **(d)** the initial condition is (20, 1, 20) and $\gamma = 0.005$. In all the figures the blue lines give the susceptible population levels S_i, the green lines give the resistant population levels R_i, the red lines give the parasite population levels P_i. Model parameters common to all figures are: $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R = 0.5$.

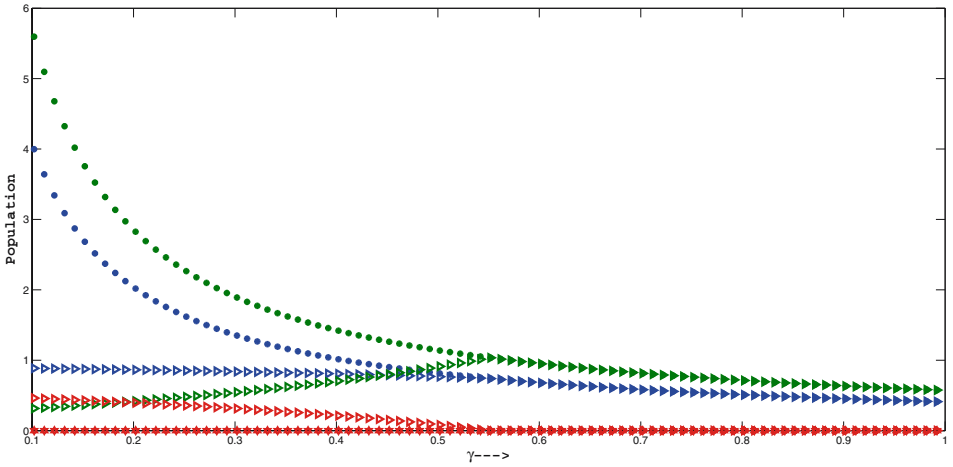

Figure 5. Bifurcation diagram of the model with respect to γ for $f(N) = (\alpha/e^{\gamma N})$. In all figures the blue dots give the susceptible population levels S_i, the green line gives the resistant population levels R_i, the red line gives the parasite population levels P_i. Model parameters common to all figures are: $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R = 0.5, \alpha = 0.8$.

$\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R = 0.5, \gamma = 0.25$. In this figure, the red line represents simulation results with initial condition (10, 1, 10) and blue line represents that with initial condition (20, 1, 40). From the figure it is clear that bistable

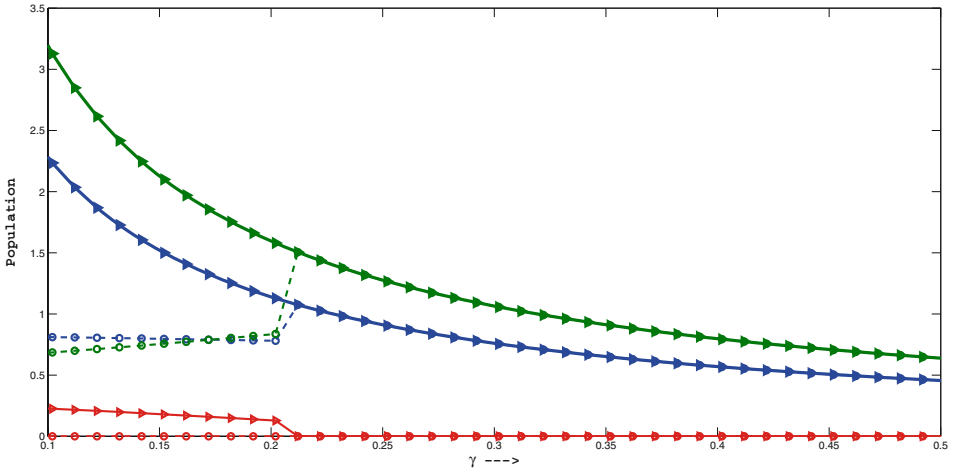

Figure 6. Bifurcation diagram of the model with respect to γ for $f(N) = \alpha/e^{\gamma N}$. In all figures the blue dots give the susceptible population levels S_i, the green line gives the resistant population levels R_i, the red line gives the parasite population levels P_i. Model parameters common to all figures are: $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R = 0.5, \alpha = 0.6$.

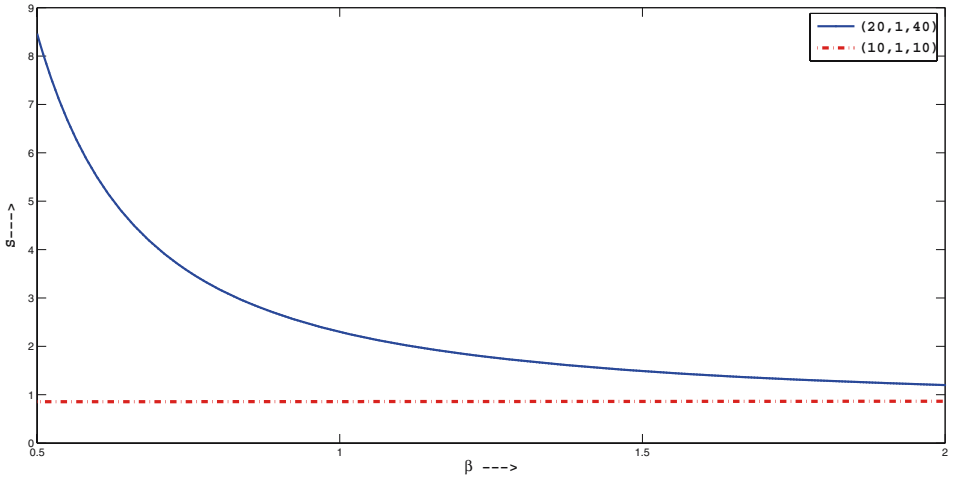

Figure 7. Bifurcation diagram of the susceptible population of the model with respect to β for $f(N) = \alpha/(1 + \gamma N^{\beta})$ for $\alpha = 0.8$. Model parameters common to all figures are: $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R = 0.5, \gamma = 0.25$.

nature of the model exists for all β in this case. Keeping all other parameters fixed and setting $\alpha = 0.6$, we draw the bifurcation diagram of the model with respect to β in figure 8. In this figure the red dots represent simulation results with initial condition (20, 1, 40) and blue line represents that with initial condition (10, 1, 10). The most interesting result here is that the model does not show bistable nature at $\beta = 1$ but it shows bistability for a wide range of β lower than 1.

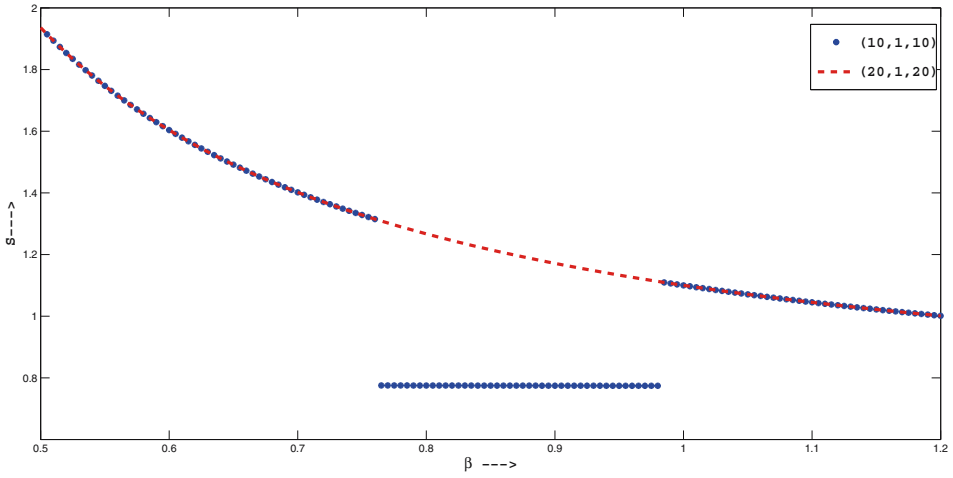

Figure 8. Bifurcation diagram of the susceptible population of the model with respect to β for $f(N) = \alpha/(1 + \gamma N^{\beta})$ for $\alpha = 0.6$. Model parameters common to all figures are: $\Lambda_S = \Lambda_R = \lambda = 1.5, \sigma_P = 0.5, \tau = 0.8, a = 0.1, \sigma_R = 0.5, \gamma = 0.25$.

5. Conclusion

White and Wilson [14] investigated an insect–pathogen model where pathogen resistance is density-dependent. They have chosen the function $f(N)$ (the fraction of individuals giving birth to susceptibles) in the form $f(N) = \alpha/(1 + \gamma N)$. But there is no biological reason for choosing this particular type of function. To investigate the effects of different types of monotonic density-dependent susceptible class we choose the generalized functional forms as $f(N) = \alpha/e^{\gamma N}$ and $f(N) = \alpha/(1 + \gamma N^{\beta})$. We have shown that different structures of monotonic nature of $f(N)$ significantly alter the model behaviour compared to the results reported by White and Wilson [14]. Main goal of this paper is to investigate the bistable nature of the model. We have shown that the generalized model with $f(N) = \alpha/(1 + \gamma N^{\beta})$ does not show bistable nature at $\beta = 1$ but it shows bistability for a wide a range of β lower than 1. We have also demonstrated that bistable nature of the model strongly depends on the value of γ for $f(N) = \alpha/e^{\gamma N}$. There exists a critical value of γ below which model shows bistability but above which it has monostable behaviour. Bistable nature of pathogen dynamics was observed experimentally by Malka *et al* [16]. Here, we were able to construct an insect–pathogen model which has bistable dynamics. This simple model may be helpful to identify the universal mechanisms which lead to bistability in a biological system. The control of bistable system is very difficult because the dynamics has sensitivity to initial conditions. Therefore, to control a disease in such cases we should have sufficient information about the multistable or monostable nature of the system.

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