Mathematics Research Developments

Ordinary and Partial Differential Equations

Raymond Brewer
Editor

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ORDINARY AND PARTIAL DIFFERENTIAL EQUATIONS

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PREFACE

Disease in the prey population increases the risk of prey outcomes in predation or to be harvested. In this book, an eco-epidemiological model consisting of predator-prey model with SIS disease in the prey population is proposed and analyzed. Furthermore, the authors discuss a mathematical S-E-I-L (Susceptible-Latently infected-Infected-Lost of sight) model for the spread of a directly transmitted infectious disease in an age-structured population; examine how starting from the classical Chebyshev ordinary differential equation (ODE), a generic realization of its Lie algebra of point symmetries sl(3;R) is obtained in terms of the Chebyshev polynomials of first and second kind; and give a comparative summary of different recent contributions to the theme of the linear stability and nonlinear dynamics of solitary waves in the nonlinear Dirac equation in the form of the Gross-Neveu model.

In Chapter 1, an eco-epidemiological model consisting of predator-prey model with SIS disease in the prey population is proposed and analyzed. Selective harvesting and time delay are taken into account. In the authors'model, the authors also consider two generalized Holling response functions of type III, both in the predator and the prey equations. Qualitative mathematical analysis of the authors' model is performed. The authors investigate the positivity and the boundedness of solutions of the model. Important thresholds are identified and their implications are explained. Existence and stability analysis of equilibria are carried out: monostabilities and bistability. A Hopf bifurcation exists both in the presence of zero and non-zero time lag. A dynamically consistent nonstandard finite difference scheme is designed and numerical simulations that illustrate the theory are provided.

In chapter 2, the authors consider a mathematical S-E-I-L (Susceptible-Latently infected-Infected-Lost of sight) model for the spread of a directly transmitted infectious disease in an age-structured population; taking into account the demographic process. First, the authors establish the mathematical well-posedness of the time evolution problem by using the integrated semigroup approach. Next the authors prove that the basic reproduction ratio R_0 is given as the spectral radius of a positive operator, and an endemic state exist if and only if the basic reproduction ratio R_0 is greater than unity, while the disease-free equilibrium is locally asymptotically stable if $R_0<1$. The authors also show that the endemic steady states are forwardly bifurcated from the disease-free steady state when R_0 cross the unity. Finally, the authors examine the conditions for the local stability of the endemic steady states.

Starting from the classical Chebyshev ordinary differential equation (ODE), a generic realization of its Lie algebra of point symmetries $\mathfrak{sl}(3,\mathbb{R})$ is obtained in terms of the Chebyshev polynomials of first and second kind. In Chapter 3 it is shown that the corresponding structure tensor of the symmetry algebra does not depend on the parameter n of the Chebyshev equation. A slight modification of the ansatz enables us to obtain a generic realization of the point symmetries of linear homogeneous second-order ODEs admitting fundamental solutions of trigonometric and hyperbolic type. The problem of constructing orthogonal systems of functions in terms of point symmetries is considered. Finally, the variational symmetries of homogeneous ODEs with maximal symmetry are analyzed. It is shown that non-linear deformations of an ODE preserving a certain subalgebra of Noether symmetries are deeply related to the symmetry breaking problem.

In Chapter 4, the authors give a comparative summary of different recent contributions to the theme of the linear stability and nonlinear dynamics of solitary waves in the nonlinear Dirac equation in the form of the Gross-Neveu model. The authors indicate some of the key controversial statements in publications within the past few years and we attempt to address them to the best of our current understanding. The conclusion that the authors reach is that the solitary wave solution of the model is spectrally stable in the cubic nonlinearity case, however, it may become unstable through an instability amounting to the violation of the Vakhitov-Kolokolov criterion for higher exponents. The authors find that for the Dirac model, the interval of instability is narrower. Furthermore, contrary to what is the case in the nonlinear Schrödinger analogue of the model, the unstable dynamical evolution, does *not* lead to collapse (blowup) and hence it appears that the relativistic nature of the model mitigates the collapse instability. Various issues associated with

different numerical schemes are highlighted and some possibilities for future alleviation of these is suggested.

CONTENTS

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Chapter 1

SELECTIVE HARVESTING AND TIME DELAY IN A PREDATOR-PREY MODEL WITH INFECTIOUS PREYS

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Abstract

Disease in the prey population increases the risk of prey outcomes in predation or to be harvested. In this paper, an eco-epidemiological model consisting of predator-prey model with SIS disease in the prey population is proposed and analyzed. Selective harvesting and time delay are taken into account. In our model, we also consider two generalized Holling response functions of type III both in the predator and the prey equations. Qualitative mathematical analysis of our model is performed. We

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investigate the positivity and the boundedness of solutions of the model. Important thresholds are identified and their implications are explained. Existence and stability analysis of equilibria are carried out: monostabilities and bistability. A Hopf bifurcation exists both in the presence of zero and non-zero time lag. A dynamically consistent nonstandard finite difference scheme is designed and numerical simulations that illustrate the theory are provided.

Keywords: Predator-prey; disease; harvesting; time delay; stability; Hopf bifurcation; nonstandard numerical scheme

1. Introduction

It has been considered that a transmissible disease in the prey population has negative consequences on predator-prey dynamics, both in terms of growth and reproduction. Initially, models for ecological interactions and models for contagious diseases were developped separately (Lotka-Volterra, 1920 and Kermac-McKendric, 1927 [1]). However, in the natural world, it is relevant to merge these two important areas of research. Together with spreading the disease, species also compete with other species for food and space and are also predated by some other species, simultaneously. Eco-epidemiology which is a relatively new branch of study in theoretical biology, tackles such situations by dealing with both ecological and epidemiological issues. Mathematical models have been used extensively to study the effects of a disease in the prey population on the dynamics of the predator-prey system. Hethcote et al. (2004) [2] have studied a predator-prey system in which only preys are infected. Chattopadhyay et al. (1999) [3] considered a prey-predator model with non-selective harvesting and infection in the prey population (see also Xiao and Chen (2001) [4]). However, those models do not predict periodic solutions (limit cycle) around the coexistence equilibrium where prey populations survive with disease in presence of predator. Conversly, our model allows a Hopf bifurcation. The biological interpretation of Hopf bifurcation is that the predators coexist with susceptible preys and infected preys, exhibiting oscillatory balance behaviour.

An important aspect which should be kept in mind while formulating an eco-epidemiology model is the fact that harvesting will be taken into account (see Tewa et al. 2012 [5]). One other aspect is to maintain a time delay in harvesting by restricting harvested preys above a given age or health. It is relevant for a commercial point of view (see Kar 2003 [6]). In the present study, we consider

the problem of selective harvesting in a predator-prey model with generalized Holling response function of type III incorporating time delay in the harvesting term with SIS disease in the prey population. The model consists of the interaction of three populations namely susceptible preys, infected preys and the predator population. We consider that SIS diseases caused by pathogens (such as viruses and bacteria) affect only preys. There are several examples where a transmissible disease spreads among one population size in Gulland (1995) [7]. An example is the common seal (Phoca vitulina) and striped dolphin (Stenella coeruleoaba) when they are infected by Phocine Distemper Virus (PDV). The epidemic of PDV infections are found among harbor seals in 1988. PDV was identified in 1988 as the cause of death of 18000 harbor seals (P. vitulina) and 300 gray seals (Halichoerus grypus) along the northern European coast; in these case fishes are considered as preys. We subdivide the prey population in two classes: susceptible preys and infected preys. In our model, we consider that, the contact rate between infected and susceptible preys follows the law of mass action and that infected preys could recover and become susceptible again.

In population dynamics, the choice of the functional response is important. According to Heesterbeek and Metz (1993) [8] who suggest that a Holling type function is more appropriate to describe the contact process, we consider the Holling functional response of type III.

This paper is organized as follows. In section 2, we introduce a predator/prey model that allows for the incorporation of selective harvesting and SIS disease. To make a better used of delay in our model, we shall first study the model without delay. Section 3 focuses on the study of model without delay and section 4 concerns the full model which includes time delay in harvesting. Boundedness and existence of various solutions are discussed thoroughly. Stabilities of equilibria are analyzed. Existence of Hopf bifurcation is investigated both in presence of zero and non-zero time lag. In section 5, we provide a nonstandard numerical scheme complying with our mathematical analysis. Numerical simulations are presented in section 6. Discussions of results are given in section 7. The mathematical details are included in appendix.

2. The Model

Let $H(t)$ represents the number of preys and $P(t)$ denotes the number of predators which interact with preys at the time t. The following basic assumptions hold in our model.

(A1) : In the absence of disease, the prey population grows according to the logistic law with intrinsic growth rate $r > 0$ and the carrying capacity $K > 0$.

(A2) : In the presence of disease, the prey population is divided into two classes, namely, the susceptible preys (X) and the infected preys (Y). Therefore, at any time t, the total density of prey population is $H(t) = X(t) + Y(t)$.

(A3) : The disease is not genetically inherited. Infected populations do not become immune. Infected preys can recover and become susceptible again.

(A4) : It is assume that only susceptible preys X are capable of reproducing with logistic law. Infected preys are removed before having the possibility of reproducing. However, the infected preys Y still contribute with X to population growth toward the carrying capacity.

(A5) : The predator has a death rate constant $c > 0$ and the predation coefficient of susceptible preys is $m > 0$ (the search rate).

(A6) : It is assume that, infected preys are more vulnerable to predation.

(A7) : We assume that only susceptible preys are subjected to harvesting with constant effort $E > 0$.

(A8) : The delay $\tau \geq 0$ is a constant which represents the assumption that the harvesting begins at a given age or health.

(A9) : The functional response of predators is assumed to be a Holling type III function.

Set

$$f_1(X) = \frac{X^2}{\alpha_1 X^2 + \beta_1 X + 1}, \text{ and } f_2(Y) = \frac{Y^2}{\alpha_2 Y^2 + \beta_2 Y + 1}, \tag{1}$$

two predator response functions. They are increasing and bounded functions of X and Y.

The mathematical realization of all previous nine assumptions is given into the following system:

$$\begin{cases} \dot{H} = r\left(1 - \dfrac{H}{K}\right) H - m f_1(X) P - qmP f_2(Y) - hEX(t-\tau), \\ \dot{X} = \left(b - \dfrac{\theta r H}{K}\right) H - \left[\mu + \dfrac{(1-\theta) r H}{K}\right] X - \lambda XY + \delta Y - m f_1(X) P - hEX(t-\tau), \\ \dot{Y} = \lambda XY - \delta Y - \left[\mu + \dfrac{(1-\theta) r H}{K}\right] Y - qmP f_2(Y), \\ \dot{P} = amP f_1(X) + lqmP f_2(Y) - cP, \end{cases} \tag{2}$$

where initial conditions are
$X(0) = X_0 > 0, Y(0) = Y_0 > 0, H(0) = H_0 = X_0 + Y_0$, and $P(0) = P_0 > 0$. The parameters b and μ are the natural birth and death rates coefficients, the parameter $0 \leq \theta \leq 1$ is such that $b - \dfrac{\theta\, r\, H}{K}$ is the birth rate coefficient, $\mu + \dfrac{(1-\theta)\, r\, H}{K}$ is the mortality rate, $r = b - \mu$. The restricted growth in the logistic equation is due to a density-dependent per capita death rate when $\theta = 0$, to a density-dependent per capita birth rate when $\theta = 1$, and to a combination of these two if $0 < \theta < 1$. δ is the recovering rate of infected preys, to become susceptible. λ is the adequate contact rate between susceptible and infected preys., therefore λXY is the total number of new cases per unit time. This type of incidence is called standard incidence and is formulated by Anderson and May (1982) [9]. The factor $q \geq 1$ reflects that infected preys are more vulnerable to predation. The coefficients of conversing prey into predator are $l > 0$, $a > 0$ and $h > 0$ is the catchability coefficient of the predator species.

3. Results of the Model without Delay

To make a better used of delay in our model, we first study the model without delay i.e. $\tau = 0$. We substitute $H = X + Y$ in the two second equations of (2) and we obtain

$$\begin{cases} \dot{X} = r\left(1 - \dfrac{X}{K} - \dfrac{(1+\theta)Y}{K}\right)X + \left(b - \dfrac{\theta r Y}{K}\right)Y - \lambda XY + \delta Y - mPf_1(X) - hEX, \\ \dot{Y} = \lambda XY - \delta Y + \dfrac{(1-\theta)r}{K}Y^2 - \left[\mu + \dfrac{(1-\theta)rX}{K}\right]Y - qmPf_2(Y), \\ \dot{P} = amPf_1(X) + lqmPf_2(Y) - cP, \end{cases} \tag{3}$$

To establish the biological validity of system 3, we first have to show that the solutions of system (3) are bounded.

Theorem 3.1. *Set*

$$\Omega = \left\{(X, Y, P) \in \mathbf{R}^3_+ / X + Y + P \leq \frac{(r+1)\sigma_1}{\xi}\right\},$$

where $\xi = \min\{1, c\}$ *and* $\sigma_1 = \max\{H_0, K\}$.

All solution of system (3) which starts in the compact Ω is uniformly bounded. Thus system (3) is dissipative.

Proof. A proof of theorem 3.1 is provided in the appendix A.

3.1. Equilibria

We investigate the existence of equilibria.

$$\text{Let } X_{100} = K\left(1 - \frac{hE}{r}\right), X_{101} = \frac{\frac{c}{a}\beta_1 + \sqrt{(\frac{c}{a}\beta_1)^2 + 4\frac{c}{a}\left(m - \frac{c}{a}\alpha_1\right)}}{2\left(m - \frac{c}{a}\alpha_1\right)},$$

$$P_{101} = \frac{ar}{cK}\left[X_{100} - X_{101}\right]X_{101}, R_0 = \frac{(1-\theta)r}{\lambda K}, a_0 = \lambda(1 - R_0),$$

$$a_1 = \delta + \mu > 0, a_2 = \frac{(1-\theta)r}{K} = \lambda R_0 > 0, b_0 = \left(\lambda + \frac{(1+\theta)r}{K}\right),$$

$$R_1 = \left(\frac{2a_1}{Ka_0} + \frac{hE\frac{a_2}{a_0}}{r\left(1 + \frac{a_2}{a_0}\right)}\right), R_2 = \left(\frac{hE}{r} + \frac{a_1}{Ka_0}\right),$$

$$A_0 = -\frac{r}{K}\left(\frac{a_2}{a_0} + 1\right)^2 < 0, A_1 = r(1 - R_1)\left(1 + \frac{a_2}{a_0}\right), A_2 = (1 - R_2)\frac{a_1 r}{a_0},$$

$$Y_{110} = \frac{-A_1}{2A_0}, Y'_{110} = \frac{-A_1 + \sqrt{(A_1)^2 - 4A_0A_2}}{2A_0}, \text{ and}$$

$$Y''_{110} = \frac{-A_1 - \sqrt{(A_1)^2 - 4A_0A_2}}{2A_0}.$$

Remark 3.1. *When there is no predator (P=0), we have $X = \frac{a_1}{a_0} + \frac{a_2}{a_0}Y$ of susceptible preys for all $Y > 0$ of infected preys.*

The following theorem holds.

Theorem 3.2. *Equilibria of system (3) are given as follow*

1. $E_{000} = (0; 0; 0)$ *always exists. It is the trivial equilibrium.*
2. *When* $\frac{hE}{r} < 1$, *then the X-direction equilibrium* $E_{100} = (X_{100}; 0; 0)$ *exists.*