

## BIFURCATION AND SPATIAL PATTERN FORMATION IN SPREADING OF DISEASE WITH INCUBATION PERIOD IN A PHYTOPLANKTON DYNAMICS

RANDHIR SINGH BAGHEL, JOYDIP DHAR, RENU JAIN

**ABSTRACT.** In this article, we propose a three dimensional mathematical model of phytoplankton dynamics with the help of reaction-diffusion equations that studies the bifurcation and pattern formation mechanism. We provide an analytical explanation for understanding phytoplankton dynamics with three population classes: susceptible, incubated, and infected. This model has a Holling type II response function for the population transformation from susceptible to incubated class in an aquatic ecosystem. Our main goal is to provide a qualitative analysis of Hopf bifurcation mechanisms, taking death rate of infected phytoplankton as bifurcation parameter, and to study further spatial patterns formation due to spatial diffusion. Here analytical findings are supported by the results of numerical experiments. It is observed that the coexistence of all classes of population depends on the rate of diffusion. Also we obtained the time evaluation pattern formation of the spatial system.

### 1. INTRODUCTION

It is well known that the phytoplankton and zooplankton are single cell organisms that drift with the currents on the surface of open oceans. Further, the phytoplanktons are the staple items for the food web and they are the recycler of most of the energy that flows through the ocean ecosystem. It has a major role in stabilizing the environment and survival of living population as it consumes half of the universal carbon-dioxide and releases oxygen. So far, there is a number of studies which show the presence of pathogenic viruses in the plankton community [1, 11]. A good review of the nature of marine viruses and their ecological as well as their biological effects is given in [12]. Some researchers have shown using an electronic microscope that these viral diseases can affect bacteria and phytoplankton in coastal area and viruses are held responsible for the collapse of *Emiliana huxleyi* bloom in Mesocosms [2, 14]. Marine viruses infect not only plankton but cultivated stocks of Crabs, Oysters, Mussels, Clams shrimp, Salmon and Catfish, etc., are all susceptible to various kinds of viruses. We know that the viruses are nonliving organisms, in the sense, they have no metabolism when out side the host and they can reproduce only by infecting the living organisms. Viral infection of

---

2000 *Mathematics Subject Classification.* 34C11, 34C23, 34D08, 34D20, 35Q92, 92B05, 92D40.

*Key words and phrases.* Phytoplankton dynamics; reaction-diffusion equation; local stability; Hopf-bifurcation; diffusion-driven instability; spatial pattern formation.

©2012 Texas State University - San Marcos.

Submitted July 25, 2011. Published February 2, 2012.

the phytoplankton cell is of two types, namely, Lysogenic and Lytic. Moreover from literature, in lytic viral infection, when a virus injects its DNA into a cell, it hijacks the cell's replication machinery and produces large a number of viruses. As a result, they rupture the host and are released into the environment. On the other hand, in lysogenic viral infection, the DNA of the viruses do not use the machinery of the host themselves, but their genes are duplicated each time as the host cell divides. Many papers have already been developed which have used this kind of lysogenic viral infection [3, 6, 10, 17]. In this kind of infection, infected phytoplankton do not grow like susceptible phytoplankton but their number grows and it is proportional to the number of susceptible phytoplankton and infected phytoplankton. Moreover, we do not look on the viruses as mere pathogens that destroy others life rather, they produce the fuel, essential to the running of the marine engine by destroying phytoplankton; i.e., they produce the essential minerals which are required for the growth of phytoplankton. Secondly, we have introduced incubated class in between a susceptible class and an infected class, Unlike simple S-I-S models in mathematical epidemiology. Generally, we have been using direct shifting from susceptible to infected class, but this process is not regular, rather, phytoplankton stay for some definite period in an incubated class after leaving the susceptible class and joining the infected class. The period for which they stay in incubated class is termed as the incubation period. The incubation period is defined as the time from the exposure to the onset of disease, when they are exposed to infection. The incubation period is useful not only for making the rough guesses for finding the cause and source of infection, but also in developing treatment strategies to extend the incubation period, and for performing an early projection of the disease prognosis [12, 16].

In the previous thirty years, the pattern formation in predator-prey systems has been studied by many researchers. The spread of diseases in human populations can exhibit large scale patterns, underlining the need for spatially explicit approaches. The spatial component of ecological interactions has been identified as an important factor in a spatial world and it is a natural phenomenon that a substance goes from high density regions to low density regions. Also, spatial patterns are ubiquitous in nature. These patterns modify the temporal dynamics and stability properties of population densities at a range of spatial scales and their effects must be incorporated in temporal ecological models that do not represent space explicitly. The spatial component of ecological interactions has been identified as an important factor in how ecological communities are shaped [7, 9]. Patterns are present in the chemical and biological worlds and since Turing [19] first introduced his model of pattern formation, reaction-diffusion equations have been a primary means of predicting them. Similarly structured systems of ordinary differential equations govern the spatiotemporal dynamics of ecological population models, yet most of the simple models predict spatially homogeneous population distributions [5]. It is important to note that the above model takes into account the invasion of the prey species by predators but does not include stochastic effects or any influences from the environment. Nevertheless, a reaction-diffusion equation modeling predator-prey interaction show a wide spectrum of ecologically relevant behavior resulting from intrinsic factors alone, and is an intensive area of research. An introduction to research in the application of reaction-diffusion equations to population dynamics are available in [8, 15].

These numerical simulations reveal that a large variety of different spatiotemporal dynamics can be found in this model. The numerical results are consistent with our theoretical analysis. It should be noted that, if considered in a somewhat broader ecological perspective, our results have an intuitively clear meaning. There has been a growing understanding during the past years regarding the dynamics of real ecosystems. It is important to reveal different spatial dynamical regimes arising as a result of perturbation of the system parameters [18, 20].

The objective of the paper is to consider the Bifurcation and spatial pattern formation in a Phytoplankton dynamics. In section 2 and 3, we have developed mathematical model and analyzed dynamical behavior of system. In section 4, studied the Hopf bifurcation mechanisms analytical and numerically, in section 5, studied the spatial pattern formation and numerical simulation of two dimensional systems and finally in section 6, we summarize our results and discuss the relative importance of different mechanisms of bifurcation and spatial pattern formation.

## 2. MATHEMATICAL MODEL

Now, we consider the case of viral infection of phytoplankton population, the shifting from susceptible to infected class is not regular. In fact, the susceptible phytoplankton stay for some definite period in the incubated class after leaving the susceptible class and joining the infected class. Taking the population densities of susceptible and infected phytoplankton as  $P_s$  and  $P_i$  respectively, at any instant of time  $T$ . The population of susceptible phytoplankton is assumed to be growing logistically with intrinsic growth rate  $r$  and carrying capacity  $K$ . Now taking  $P_{in}$  as the population density of population in incubated class. Here, we will use nonlinear Holling Type II functional responses for disease spreading because the disease conversion rates become saturated as victim densities increase. Let  $\alpha$  be the disease contact rate and it is volume-specific encounter rate between susceptible and infected phytoplankton, which is equivalent to the inverse of the average search time between successful spreading of disease. The coefficients  $\delta$  and  $\beta$  are the total removal of phytoplankton from the infected and incubated class because of the death (including recovered) from disease and due to natural causes respectively. Again,  $\gamma_1$  be the fraction of the population recovered from infected phytoplankton population and joined in the susceptible phytoplankton population and  $\beta_1$  is the fraction of the incubated class population which will move to the infected class. Therefore, quantitatively  $\delta > \gamma_1$  and  $\beta > \beta_1$ . Keeping in view the above, the mathematical model for a viral infected phytoplankton population with an incubated class is governed by the following set of differential equations:

$$\frac{dP_s}{dT} = rP_s\left(1 - \frac{P_s}{K}\right) - \frac{\alpha P_s P_i}{P_s + 1} + \gamma_1 P_i, \quad (2.1)$$

$$\frac{dP_{in}}{dT} = \frac{\alpha P_s P_i}{P_s + 1} - \beta P_{in}, \quad (2.2)$$

$$\frac{dP_i}{dT} = \beta_1 P_{in} - \delta P_i, \quad (2.3)$$

$$P_s(0) > 0, \quad P_i(0) > 0, \quad P_{in}(0) > 0. \quad (2.4)$$

The Holling type-II the functional response  $\frac{\alpha P_s P_i}{P_s + 1}$  is used [4] and many other researchers. In this section, we have studied the dynamical behavior of the system

(2.1)-(2.3), with initial population; i.e.,  $P_s(0) > 0$ ,  $P_{in}(0) > 0$ ,  $P_i(0) > 0$  and the total population at any instant  $t$  is  $N(t) = P_s(t) + P_{in}(t) + P_i(t)$ .

Now, on rescaling the above system (2.1)-(2.3) using the change of variables:  $x = \frac{P_s}{K}$ ,  $y = \frac{P_{in}}{K}$ ,  $z = \frac{P_i}{K}$ ,  $t = rT$ , we obtain

$$\frac{dx}{dt} = x(1-x) - \frac{axz}{x+1} + cz, \quad (2.5)$$

$$\frac{dy}{dt} = \frac{axz}{x+1} - dy, \quad (2.6)$$

$$\frac{dz}{dt} = d_1y - ez, \quad (2.7)$$

where  $a = \frac{\alpha K}{r}$ ,  $c = \frac{\gamma_1}{r}$ ,  $d = \frac{\beta}{r}$ ,  $d_1 = \frac{\beta_1}{r}$ ,  $e = \frac{\delta}{r}$ ,  $x(0) > 0$ ,  $y(0) > 0$  and  $z(0) > 0$ .

### 3. BOUNDEDNESS OF THE SYSTEM

Now, we will study the existence of all possible steady states of the system and the boundedness of the solutions. There are three biologically feasible equilibria for the system (2.5)-(2.7), (i)  $E_0 = (0, 0, 0)$  is the trivial steady state; (ii)  $E_1 = (1, 0, 0)$  is the disease free steady state and (iii)  $E^* = (x^*, y^*, z^*)$  is endemic equilibrium state, where

$$x^* = \frac{ed}{d_1a - ed}, \quad y^* = \frac{e^2d(ad_1 - 2ed)}{(ed - d_1c)(ad_1 - ed)^2}, \quad z^* = \frac{edd_1(ad_1 - 2ed)}{(ed - d_1c)(ad_1 - ed)^2}.$$

Further, it is clear from the above expression that  $E^* \in R_+^3$ , if  $a > \frac{de}{d_1} > c$ . Now, we will show that all the solutions of the system (2.5)-(2.7) are bounded in a region  $B \subset R_+^3$ . We consider the function

$$w(\tau) = x(\tau) + y(\tau) + z(\tau), \quad (3.1)$$

and substituting the values from (2.5)-(2.7), we obtain

$$\frac{dw}{d\tau} = x(1-x) - (d-d_1)y - (e-c)z.$$

If we choose a positive real number  $\eta = \min\{d-d_1, e-c\}$ , then

$$\frac{dw(\tau)}{d\tau} + \eta w(\tau) \leq x(1+\eta) - x^2 = f(x).$$

Moreover,  $f(x)$  has maxima at  $x = (1+\eta)/2$  and  $f(x) \leq (1+\eta)^2/4 = M$  (say). Hence,  $\dot{w}(\tau) + \eta w(\tau) \leq M$ . Now, using comparison theorem, as  $\tau \rightarrow \infty$ , we obtain  $\sup w(\tau) = \frac{M}{\eta}$ . Therefore,  $0 \leq x(\tau) + y(\tau) + z(\tau) \leq \frac{M}{\eta}$ . and let us consider the set  $B = \{(x, y, z) \in R_+^3 : 0 \leq x(\tau) + y(\tau) + z(\tau) \leq M/\eta\}$ , hence, The system (2.5)-(2.7) is uniformly bounded in the region  $B \subset R_+^3$ .

**3.1. Local stability of the system.** We have already established that the system (2.5)-(2.7) has three equilibrium points, namely,  $E_0 = (0, 0, 0)$ ,  $E_1 = (1, 0, 0)$  and  $E^* = (x^*, y^*, z^*)$  in the previous section. The general variational matrix corresponding to the system is given by

$$J = \begin{bmatrix} 1 - 2x^* - \frac{az^*}{(x^*+1)^2} & 0 & -\frac{ax^*}{(x^*+1)} + c \\ \frac{az^*}{(x^*+1)^2} & -d & \frac{ax^*}{(x^*+1)} \\ 0 & d_1 & -e \end{bmatrix}$$

Now, corresponding to the trivial steady state  $E_0 = (0, 0, 0)$  the Jacobian  $J$  has the following eigenvalues  $\lambda_i = 1, -d, -e$ . Hence,  $E_0$  is repulsive in  $x$ -direction and attracting in  $y - z$  plane. Clinically, it means that when there is no susceptible population then, there will be no mass in incubated and in the infected class. Hence,  $E_0$  is a saddle point. The corresponding to the disease free equilibrium point  $E_1 = (1, 0, 0)$ , we have following  $\lambda_1 = -1$  and  $\lambda_{2,3}$  are the roots of the quadratic equation  $\lambda^2 + (d + e)\lambda + (de - \frac{ad_1}{2}) = 0$  when  $de > d_1a/2$ , then both the roots have a negative real parts and thus  $E_1(1, 0, 0)$  is locally stable in this case.

Further, from the existence of  $E^*$  and the stability condition of  $E_1$ , it is clear that the instability of the disease free equilibrium will lead to the existence of the endemic equilibrium. Now, we will examine the local behavior of the flow of the system around the endemic equilibrium  $E^*$ . The characteristic equation corresponding to the equilibrium is

$$P(\lambda) = \lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0, \quad (3.2)$$

where

$$\begin{aligned} A_1 &= 2x^* + \frac{az^*}{(x^* + 1)^2} + d + e - 1, \\ A_2 &= 2x^*e + 2x^*d + ed - e - d - \frac{ad_1x^*}{(x^* + 1)} + \frac{aez^*}{(x^* + 1)^2} + \frac{adz^*}{(x^* + 1)^2}, \\ A_3 &= \frac{eadz^*}{(x^* + 1)^2} + \frac{ad_1x^*}{(x^* + 1)} - \frac{2ad_1x^{*2}}{(x^* + 1)} - \frac{acd_1z^*}{(x^* + 1)^2} + 2xed - ed. \end{aligned}$$

Hence, using Routh-Hurwitz criteria  $E^*$  is locally asymptotically stable, if  $A_i > 0$ ,  $i = 1, 2, 3$  and  $A_1A_2 > A_3$ .

#### 4. HOPF-BIFURCATION ANALYSIS

In this section, we obtain the Hopf-bifurcation criteria of above system (2.5)-(2.7), taking "e" (i.e., total removal of phytoplankton from the infected population) as the bifurcation parameter. Now, the necessary and sufficient condition for the existence of the Hopf-bifurcation can be obtained if there exists  $e = e_0$ , such that (i)  $A_i(e_0) > 0$ ,  $i = 1, 2, 3$ , (ii)  $A_1(e_0)A_2(e_0) - A_3(e_0) = 0$  and (iii)  $\frac{d}{de}(u_i) \neq 0$ ,  $i = 1, 2, 3$ , where  $u_i$  is the real part of the eigenvalues of the characteristic equation (3.2). After substituting of the values of  $A_i$  for  $i = 1, 2, 3$  in equation  $A_1A_2 - A_3 = 0$  and solving it for  $e$ , it reduces to

$$e^7B_1 + e^6B_2 + e^5B_3 + e^4B_4 + e^3B_5 + e^2B_6 + eB_7 + B_8 = 0, \quad (4.1)$$

where

$$\begin{aligned} B_1 &= (d^6 - 2d^5d_1a + 8d^6d_1a), \\ B_2 &= (2d^4d_1^2a^2 + 2d^4d_1^2ac - 2d^6d_1a + 2d^5d_1a + d^7 - 2d^5d_1c - 12d^5d_1^2a^2 \\ &\quad - 16d^5d_1^2ac + 8d^2d_1a), \\ B_3 &= (2d^5d_1^2ac - 2d^4d_1a + 9d^3d_1^3a^2c + 2d^6d_1a - 4d^4d_1^2ac - 2d^4d_1^2a^2 \\ &\quad + 4d^3d_1^3a^3 - d^5d_1^2a^2 + d^4d_1^2c^2 + 4d^2d_1^2ac - 2d^6d_1c + 4d^4d_1^3a^3 \\ &\quad + 8d^4d_1^3ac^2 + 24d^4d_1^3a^2c - 12d^6d_1^2a^2 - 16d^6d_1^2ac), \end{aligned}$$

$$\begin{aligned}
B_4 &= (20d^4d_1^3a^2c + 2d^6d_1^2ac + 2d^6d_1^2a^2 - 4d^5d_1^2ac - 8d^5d_1^2a^2 - 22d^3d_1^3a^2c \\
&\quad + 2d^4d_1^3ac^2 + 4d^4d_1^3a^3 - 2dd_1^4a^3c - 3d^2d_1^4a^2c^2 + 6d^5d_1^2a^2 - 2d^3d_1^3ac^2 \\
&\quad + d^5d_1^2c^2 - 2d^3d_1^3a^3 + 4d^5d_1^2ac - 12d^3d_1^4a^2c^2 - 8d^3d_1^4a^3c \\
&\quad + 4d^5d_1^3a^3 + 24d^5d_1^3a^2c + 8d^5d_1^3ac^2), \\
B_5 &= (9d^5d_1^3a^2c - 12d^4d_1^3a^2c + 10d^4d_1^3a^3 - 12d^2d_1^4a^3 + 4dd_1^5a^3c^2 - 12d^3d_1^4a^3c \\
&\quad + 7d^2d_1^4a^2c^2 - 8d^4d_1^3a^2c - 4d^4d_1^3a^3 + 8dd_1^4a^3c - dd_1^4a^4 - 3d^3d_1^4a^2c^2 \\
&\quad + dd_1^5a^4c - 2d^4d_1^3ac^2 + d^2d_1^4a^4 + 4d^2d_1^5a^3c^2 - 8d^4d_1^4a^3c - 12d^4d_1^4a^2c^2), \\
B_6 &= (d^4d_1^4a^4 - 4d^3d_1^4a^3c - 2d^3d_1^3a^4 - 12d^2d_1^5a^3c^2 - 2d^5d_1^4a^2c^2 - 6dd_1^5a^3c^2 \\
&\quad + d^2d_1^4a^3c + 11d^3d_1^4a^2c^2 + 4d^2d_1^5a^4c - d^4d_1^4a^2c^2 - d^6d_1^4c), \\
B_7 &= (4d^3d_1^5a^3c^2 - 6d^2d_1^5a^3c^2 - dd_1^6a^4c^2 + d^3d_1^5a^4c + d_1^6a^4c^2), \\
B_8 &= (dd_1^6a^4c^2 - d^2d_1^6a^4c^2).
\end{aligned}$$

For better understanding of the above result, taking an example values of the parameters  $c = 0.01$ ,  $d = 0.11$ ,  $d_1 = 0.1$  and  $a = 7.15$ , we obtain a positive root  $e = 0.074$  of the quadratic equation (4.1). Therefore, the eigenvalues of the characteristic equation (4.1) at  $e = 0.074$  are of the form  $\lambda_{1,2} = \pm iv$  and  $\lambda_3 = -w$ , where  $v$  and  $w$  are positive real numbers.

Now, we will verify the condition (iii) of Hopf-bifurcation. Put  $\lambda = u + iv$  in (3.2), we obtain

$$(u + iv)^3 + A_1(u + iv)^2 + A_2(u + iv) + A_3 = 0. \quad (4.2)$$

On separating the real and imaginary parts and eliminating  $v$  between real and imaginary parts, we obtain

$$8u^3 + 8A_1u^2 + 2(A_1^2 + A_2)u + A_1A_2 - A_3 = 0. \quad (4.3)$$

Now, we have  $u(e_0) = 0$  as  $A_1(e_0)A_2(e_0) - A_3(e_0) = 0$ . Further,  $e = e_0$ , is the only positive root of  $A_1(e)A_2(e) - A_3(e) = 0$ , and the discriminate of  $8u^2 + 8A_1u + 2(A_1^2 + A_2) = 0$  is  $64A_1^2 - 64(A_1^2 + A_2) < 0$ . Here, differentiating (4.3) with respect to  $e$ , we have  $(24u^2 + 16A_1u + 2(A_1^2 + A_2)) \frac{du}{de} + (8u^2 + 4A_1u) \frac{dA_1}{de} + 2u \frac{dA_2}{de} + \frac{d}{de}(A_1A_2 - A_3) = 0$ . Now, since at  $e = e_0$ ,  $u(e_0) = 0$ , we obtain

$$\left[ \frac{du}{de} \right]_{e=e_0} = \frac{-\frac{d}{de}(A_1A_2 - A_3)}{2(A_1^2 + A_2)} \neq 0,$$

which ensures that the above system has a Hopf-bifurcation. It is shown graphically in figure 1.

## 5. SPATIOTEMPORAL MODEL

Now, we consider the phytoplankton dynamics with movement (i.e., diffusion). Therefore the population densities; i.e.,  $P_s$ ,  $P_i$  and  $P_{in}$  become space and time dependent. Keeping in view of the above, our mathematical model can be stated by the reaction diffusion system

$$\frac{\partial u}{\partial t} = u(1 - u) - \frac{auw}{u + 1} + cw + D_a \nabla^2 u, \quad (5.1)$$

$$\frac{\partial v}{\partial t} = \frac{auw}{u + 1} - dv + D_b \nabla^2 v, \quad (5.2)$$

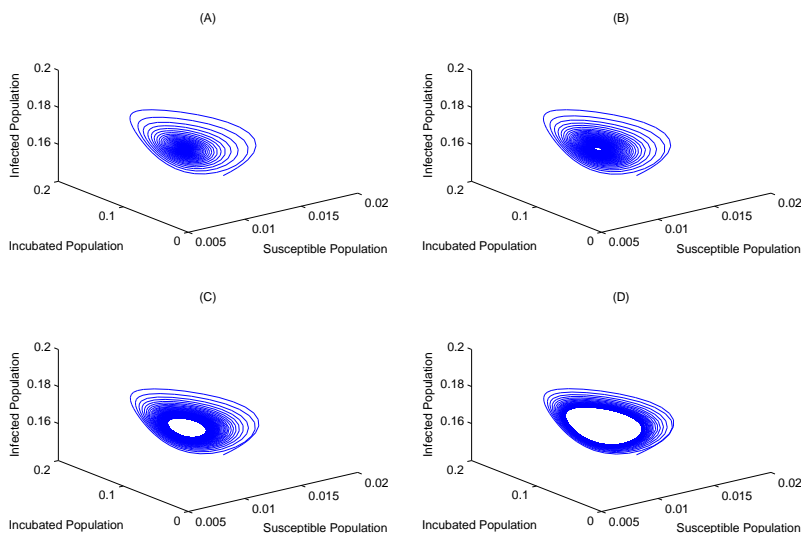


FIGURE 1. Phase plane representation of three species around the endemic equilibrium, taking  $c = 0.01$ ,  $d = 0.11$ ,  $d_1 = 0.1$ ,  $a = 7.15$ : (A)  $e = 0.073$ , (B)  $e = 0.074$ , (C)  $e = 0.075$ , (D)  $e = 0.076$

$$\frac{\partial w}{\partial t} = d_1 v - e w + D_c \nabla^2 w, \quad (5.3)$$

where  $(x, y)$  is the position in space for two dimensional on a bounded domain and  $D_a$ ,  $D_b$  and  $D_c$  are diffusion coefficients for susceptible phytoplankton, incubated phytoplankton and infected phytoplankton population, respectively. The no-flux boundary conditions were used.

Now, we will explore the possibility of diffusion-driven instability with respect to the steady state solution, i.e., the spatially homogenous solution  $(u^*, v^*, w^*)$  of the reaction diffusion system.

We assume that  $E^*$  is stable in the temporal system and unstable in spatiotemporal system, which means that the spatially homogeneous equilibrium is unstable with respect to spatially homogeneous perturbations.

We obtain the conditions for the diffusion instability to occur in system (5.1)-(5.3), one should check how small heterogeneous perturbations of the homogeneous steady state develop in the large-time limit. For this purpose, we consider the perturbation

$$u(x, y, t) = u^* + \epsilon \exp((kx + ky)i + \lambda_k t), \quad (5.4)$$

$$v(x, y, t) = v^* + \eta \exp((kx + ky)i + \lambda_k t), \quad (5.5)$$

$$w(x, y, t) = w^* + \rho \exp((kx + ky)i + \lambda_k t), \quad (5.6)$$

where  $\epsilon$ ,  $\eta$  and  $\rho$  are chosen to be small and  $k = (k_x, k_y)$  is the wave number. Substituting (5.4)-(5.6) into (5.1)-(5.3), linearizing the system around the interior equilibrium  $E^*$ , we obtain the characteristic equation as follows:

$$|J_k - \lambda_k I_2| = 0, \quad (5.7)$$

with

$$J_k = \begin{bmatrix} 1 - D_a k^2 - 2u^* + \frac{au^*w^*}{(1+u^*)^2} - \frac{aw^*}{1+u^*} & 0 & c - \frac{au^*}{1+u^*} \\ -\frac{au^*w^*}{(1+u^*)^2} + \frac{aw^*}{1+u^*} & -(d + D_b k^2) & \frac{au^*}{1+u^*} \\ 0 & d_1 & -(e + D_c k^2) \end{bmatrix},$$

where  $I_3$  and  $k$  are third order identity matrix and wave number respectively. The characteristic equation following form:

$$\lambda^3 + P_2\lambda^2 + P_1\lambda + P_0 = 0, \quad (5.8)$$

where

$$P_2 = -\left(1 - d - e - D_a k^2 - D_b k^2 - D_c k^2 - 2u^* + \frac{au^*w^*}{(1+u^*)^2} - \frac{aw^*}{1+u^*}\right),$$

$$\begin{aligned} P_1 = & -\left(d + e - de - dD_a k^2 + D_b k^2 + D_c k^2 - dD_c k^2 - D_a e k^2 - D_b e k^2 \right. \\ & - D_a D_b k^4 - D_a D_c k^4 - D_b D_c k^4 - 2du^* - 2eu^* - 2D_b k^2 u^* - 2D_c k^2 u^* \\ & + \frac{ad_1 u^*}{(1+u^*)} + \frac{adu^* w^*}{(1+u^*)^2} + \frac{aeu^* w^*}{(1+u^*)^2} + \frac{aD_b k^2 u^* w^*}{(1+u^*)^2} + \frac{aD_c k^2 u^* w^*}{(1+u^*)^2} \\ & \left. - \frac{adw^*}{1+u^*} - \frac{aew^*}{1+u^*} - \frac{aD_b k^2 w^*}{1+u^*} - \frac{aD_c k^2 w^*}{1+u^*}\right), \end{aligned}$$

$$\begin{aligned} P_0 = & \left(-de - dD_c k^2 + dD_a e k^2 - D_b e k^2 + dD_a D_c k^4 - D_b D_c k^4 + D_a D_b e k^4 \right. \\ & + D_a D_b D_c k^6 + 2deu^* + 2dD_c k^2 u^* + 2D_b e k^2 u^* + 2D_b D_c k^4 u^* + \frac{ad_1 u^*}{1+u^*} \\ & - \frac{ad_1 D_a k^2 u^*}{1+u^*} - \frac{2ad_1 u^{*2}}{1+u^*} + \frac{acd_1 u^* w^*}{(1+u^*)^2} - \frac{adeu^* w^*}{(1+u^*)^2} - \frac{adD_c k^2 u^* w^*}{(1+u^*)^2} \\ & - \frac{aD_b e k^2 u^* w^*}{(1+u^*)^2} - \frac{aD_b D_c k^4 u^* w^*}{(1+u^*)^2} - \frac{acd_1 w^*}{1+u^*} + \frac{adew^*}{1+u^*} + \frac{adD_c k^2 w^*}{1+u^*} \\ & \left. + \frac{aD_b e k^2 w^*}{1+u^*} + \frac{aD_b D_c k^4 w^*}{1+u^*}\right). \end{aligned}$$

According to the Routh-Hurwitz criterium all the eigenvalues have negative real parts if and only if the following conditions hold:

$$P_2 > 0, \quad (5.9)$$

$$P_0 > 0, \quad (5.10)$$

$$Q = P_0 - P_2 P_1 < 0. \quad (5.11)$$

This is best understood in terms of the invariants of the matrix and of its inverse matrix

$$J_k^{-1} = \frac{1}{\det(J_k)} \begin{pmatrix} M_{11} & M_{12} & M_{13} \\ M_{21} & M_{22} & M_{23} \\ M_{31} & M_{32} & M_{33} \end{pmatrix},$$

where  $M_{11} = de - \frac{ad_1 u}{(u+1)}$ ,  $M_{12} = -d_1 c - \frac{ad_1 u}{u+1}$ ,  $M_{13} = d(c - \frac{au}{u+1})$ ,  $M_{21} = \frac{aew}{(u+1)^2}$ ,  $M_{22} = -e(1 - 2u - \frac{aw}{(u+1)^2})$ ,  $M_{23} = -(\frac{au}{(u+1)}(1 - 2u - \frac{aw}{(u+1)^2}) - \frac{aw}{(u+1)^2}(c - \frac{au}{(u+1)}))$ ,  $M_{31} = \frac{ad_1 w}{(u+1)^2}$ ,  $M_{32} = -d_1(1 - 2u - \frac{aw}{(u+1)^2})$ ,  $M_{33} = -d(1 - 2u - \frac{aw}{(u+1)^2})$ . Here, matrix  $M_{ij}$  is the adjunct of  $J_k$ .



We obtain the following conditions of the steady-state stability (i.e. stability for any value of  $k$ ):

- (i) All diagonal cofactors of matrix  $J_k$  must be positive.
- (ii) All diagonal elements of matrix  $J_k$  must be negative.

The two above condition taken together are sufficient to ensure stability of a give steady state. It means that instability for some  $k > 0$  can only be observed if at least one of them is violated. Thus we arrive at the following necessary condition for the Turing Instability [13]: (i) The largest diagonal element of matrix  $J_k$  must be positive and/or (ii) the smallest diagonal cofactor of matrix  $J_k$  must be negative.

By the Routh-Hurwitz criteria, instability takes place if and only if one of the conditions (5.9)-(5.11) is broken. We consider (5.10) for instability condition:

$$P_0(k) = D_a D_b D_c k^6 - (D_a D_b a_{33} + D_b D_c a_{11} + D_a D_c a_{22}) k^4 + (D_a M_{11} + D_b M_{22} + D_c M_{33}) k^2 - \det J.$$

According to Routh-Hurwitz criterium  $P_0(k^2) < 0$  is sufficient condition for matrix  $J_k$  being unstable. Let us assume that  $M_{33} < 0$ . If we choose  $D_a = 0, D_b = 0$  and

$$\begin{aligned} P_0(k^2) &= D_c M_{33} k^2 - \det(J_k) \\ &= - \left[ d D_c k^2 \left( 1 - 2u^* - \frac{aw^*}{(1+u^*)^2} \right) + \frac{1}{(1+u^*)^2} (1+u^*)(2u^*-1) \right. \\ &\quad \times \left( ed + D_c dk^2 + edu^* + D_c du^* k^2 - ad_1 u^* \right) \\ &\quad \left. + (edaw^* + D_c daw^* k^2 - cad_1) \right] < 0. \end{aligned} \tag{5.12}$$

Hence, in this system diffusion-driven instability occur.

Now, we obtain the eigenvalues of the characteristic equation (5.8) numerically of the spatial system (5.1)-(5.3). Here, we choose some parametric values of  $a = 0.4$ ,  $c = 0.01$ ,  $d = 0.11$ ,  $d_1 = 0.1$ ,  $e = 0.08$ . In this set of values  $P_0(k^2) < 0$ , for all  $k > 0$ , hence from (5.12), we can observe diffusion driven instability of the system (see Fig.2).

**5.1. Pattern formation.** Now, we will study numerical system (5.1)-(5.3) for the pattern formation of two dimensional space with zero-flux boundary conditions is used. We choose the initial spatial distributions of each species are random and the numerical results are obtained using finite difference method for figure 3-4 and we use the parametric values same as above section.

Now, we obtain that the spatial distributions of phytoplankton dynamics in the time evaluation in figures 3-4. By varying coupling parameters, we observed that one parameter value change then spatial structure change over the times of the spatial system. In figure 3-4 have observed well organized structures for the spatial distribution population also observed that time  $T$  increase from 10 to 300 the density of different classes of population become uniform throughout the space. Finally, all these figure are shown that the qualitative changes spatial density distribution of the spatial system for the each species.

**Conclusion.** In this paper, we have studied a phytoplankton dynamics with viral infection. We observed that in a three dimensional phytoplankton system with the Holling-II response function, there exit Hopf bifurcation with respect to remove rate including nature death of infected phytoplankton. In the qualitative analysis, we

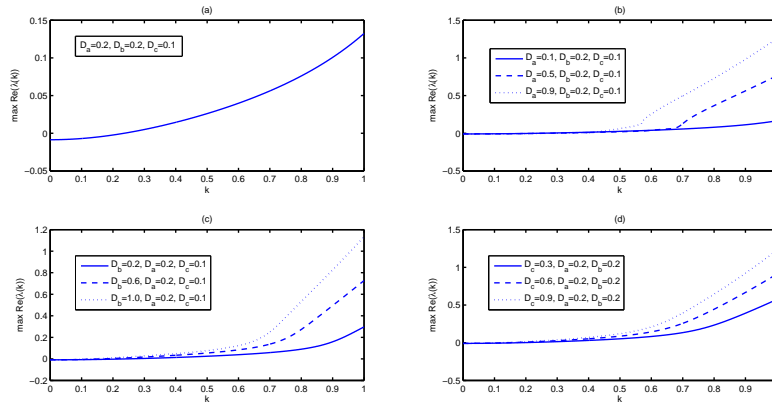


FIGURE 2. Plot of  $\max \operatorname{Re}(\lambda(k))$  against  $k$ . The other parametric values are given in text

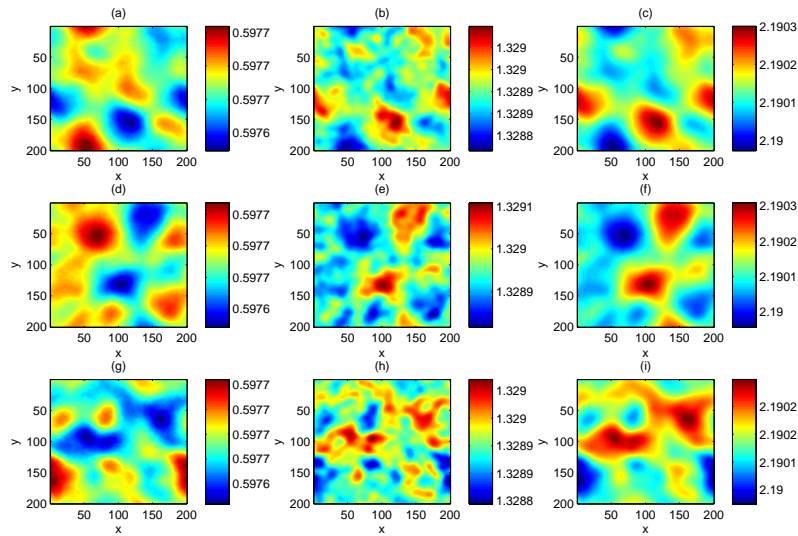


FIGURE 3. Spatial distribution of susceptible phytoplankton [first column], incubated phytoplankton [second column] and infected phytoplankton [third column] population density of the model system (5.1)-(5.3). The diffusivity coefficient values are:  $D_a = 0.02$ ,  $D_b = 0.2$ ,  $D_c = 5$ . Spatial patterns are obtained at different time levels: for plot  $T = 10$  (a, b, c),  $T = 40$  (d, e, f),  $T = 80$  (g, h, i)

studied the boundedness, dynamical behavior and local stability of the system. It is established that the rate of total removal of phytoplankton from the infected class; i.e.,  $e$ , crossed its threshold value,  $e = e_0$ , then susceptible, incubated and infected

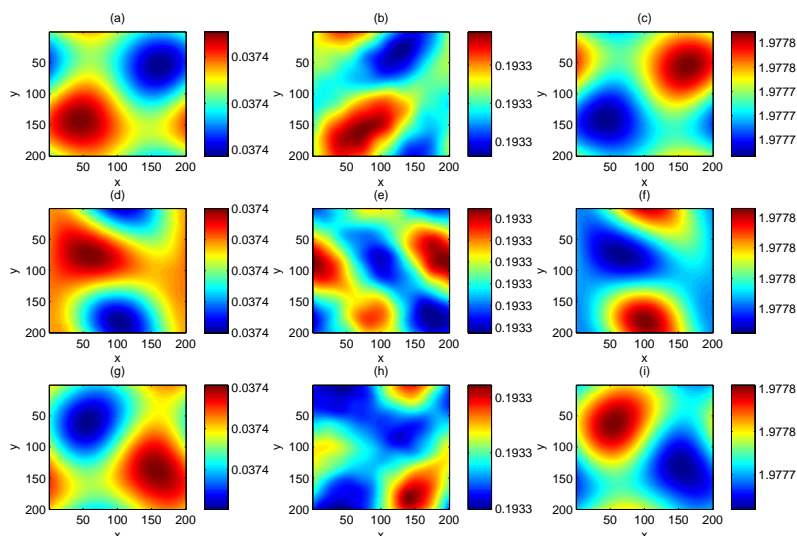


FIGURE 4. Spatial distribution of susceptible phytoplankton [first column], incubated phytoplankton [second column] and infected phytoplankton [third column] population density of the model system (5.1)-(5.3). The diffusivity coefficient values are:  $D_a = 0.02$ ,  $D_b = 0.2$ ,  $D_c = 5$ . Spatial patterns are obtained at different time levels: for plot  $T = 100$  (a, b, c),  $T = 200$  (d, e, f),  $T = 300$  (g, h, i)

phytoplankton population started oscillating around the endemic equilibrium. The above result has been shown numerically in figure 1 for different values of  $e$ . In particular, in figure 1(A), we observed that the endemic equilibrium was stable, when  $e < 0.074$ , but when it crossed the threshold value of  $e = 0.074$ , the above system showed Hopf-bifurcation, shown in figure 1(B, C, D). We have also observed spatially ordered structures of patterns in spatial systems and the solutions of the spatial system converges to its equilibrium as time  $T$  increase from 10 to 300 in the two-dimensional pattern formation, shown in figures 3-4. It is numerically established that with slight change in a time  $T$  parameter of the system (5.1)-(5.3), can lead to dramatic changes in the qualitative behavior of the system.

#### REFERENCES

- [1] B. Dubey, B. Das, J. Hussain; *A model for two competing species with self and cross-diffusion*, Indian Journal of Pure and Applied Mathematics **33** (2002), no. 6, 847–860.
- [2] S.A. Gourley; *Instability in a predator-prey system with delay and spatial averaging*, IMA journal of applied mathematics **56** (1996), no. 2, 121–132.
- [3] F. M. Hilker, H. Malchow, M. Langlais, S.V. Petrovskii; *Oscillations and waves in a virally infected plankton system:: Part ii: Transition from lysogeny to lysis*, Ecological complexity **3** (2006), no. 3, 200–208.
- [4] C. Holling; *Some characteristics of simple types of predation and parasitism*, Can. Entomol. **91** (1959), 385 – 398.

- [5] P. P. Liu, Z. Jin, *Pattern formation of a predator-prey model*, Nonlinear Analysis: Hybrid Systems **3(3)** (2009), no. 3, 177–183.
- [6] H. Malchow, F. M. Hilker, R. R. Sarkar, K. Brauer; *Spatiotemporal patterns in an excitable plankton system with lysogenic viral infection*, Mathematical and computer modelling **42** (2005), no. 9-10, 1035–1048.
- [7] A. B. Medvinsky, S. V. Petrovskii, I. A. Tikhonova, H. Malchow, B.L. Li, *Spatiotemporal complexity of plankton and fish dynamics*, Siam Review **3(44)** (2002), 311–370.
- [8] A. Morozov, S. Petrovskii, B. L. Li; *Bifurcations and chaos in a predator-prey system with the allee effect*, Proceedings of the Royal Society of London. Series B: Biological Sciences **271** (2004), no. 1546, 1407.
- [9] J. D. Murray; *Mathematical biology ii: Spatial models and biomedical applications, 3rd ed., in: Biomathematics*, Springer, New York, 2003.
- [10] S. Petrovskii, H. Malchow; *Wave of chaos: new mechanism of pattern formation in spatiotemporal population dynamics.*, Theor. Pop. Bio. **59** (2001), 157 – 174.
- [11] S. V. Petrovskii, H. Malchow; *Wave of chaos: new mechanism of pattern formation in spatiotemporal population dynamics*, Theoretical Population Biology **59** (2001), no. 2, 157–174.
- [12] M. A. Pozio; *Behaviour of solutions of some abstract functional differential equations and application to predator-prey dynamics*, Nonlinear Analysis **4** (1980), no. 5, 917–938.
- [13] H. Qian, J. D. Murray; *A simple method of parameter space determination for diffusion-driven instability with three species*, Appl. Math. Lett. **14** (2003), 405–411.
- [14] S. Ruan, X. Z. He; *Global stability in chemostat-type competition models with nutrient recycling*, SIAM Journal on Applied Mathematics **31** (1998), 170–192.
- [15] H. K. Pak, S. H. Lee, H. S. Wi; *Pattern dynamics of spatial domains in three-trophic population model*, Journal Korean Physical Society **44(3)** (2004), no. 1, 656–659.
- [16] R. A. Satnoian and M. Menzinger; *Non-turing stationary pattern in flow-distributed oscillators with general diffusion and flow rates*, Physical Review. E **62(1)** (2000), 113–119.
- [17] B. K. Singh, J. Chattopadhyay, S. Sinha; *The role of virus infection in a simple phytoplankton zooplankton system*, Journal of theoretical biology **231** (2004), no. 2, 153–166.
- [18] G. Q. Sun, G. Zhang, Z. Jin, L. Li; *Predator cannibalism can give rise to regular spatial pattern in a predator-prey system*, Nonlinear Dynamics **58** (2009), no. 1, 75–84.
- [19] A. M. Turing; *The chemical basis of morphogenesis*, Philos. Trans. R. Soc. London B **237** (1952), 37–72.
- [20] J. Xiao, H. Li, J. Yang, G. Hu; *Chaotic turing pattern formation in spatiotemporal systems*, Frontiers of Physics in China **1** (2006), no. 2, 204–208.

RANDHIR SINGH BAGHEL

SCHOOL OF MATHEMATICS AND ALLIED SCIENCE, JIWAJI UNIVERSITY, GWALIOR (M.P.)-474011, INDIA

*E-mail address:* randhirsng@gmail.com

JOYDIP DHAR

DEPARTMENT OF APPLIED SCIENCES, ABV-INDIAN INSTITUTE OF INFORMATION TECHNOLOGY AND MANAGEMENT, GWALIOR-474010,INDIA

*E-mail address:* jdhar@iiitm.ac.in

RENU JAIN

SCHOOL OF MATHEMATICS AND ALLIED SCIENCE, JIWAJI UNIVERSITY, GWALIOR (M.P.)-474011, INDIA

*E-mail address:* renujain3@rediffmail.com