Article

Role of environmental factors on the spread of bacterial diseases: A modeling study

Sandhya Rani Verma¹, Ram Naresh¹, Manju Agarwal², Shyam Sundar³

¹Department of Mathematics, School of Basic & Applied Sciences, Harcourt Butler Technical University, Kanpur-208002, India
 ²Department of Mathematics & Astronomy, University of Lucknow, Lucknow-226007, India
 ³Department of Mathematics, P. S. Institute of Technology, Kanpur-208020, India
 E-mail: verma.sandhya.15@gmail.com, ramntripathi@yahoo.com, manjuak@yahoo.com, ssmishra15@gmail.com

Received 18 January 2020; Accepted 22 February 2020; Published 1 June 2020

Abstract

In this paper, a nonlinear mathematical model is proposed and analyzed to study the role of cumulative environmental degradation on the spread of bacterial diseases. In the modeling process, it is assumed that the disease is not only transmitted directly from the infective population to susceptible population but also indirectly by the bacteria present in the conducive degraded environment. The cumulative density of the bacteria population is assumed to be governed by a generalized logistic model, and is also dependent on conducive environmental degradation. The cumulative density of environmental degradation is assumed to be dependent on human population-related factors. The analysis of the model is performed by using the stability theory of differential equations and numerical simulation. The model analysis reveals that the increased rate of conducive environmental degradation increases the density of bacteria population which leads to fast spread of the bacterial diseases.

Keywords mathematical modeling; stability; bacterial diseases; environmental degradation; simulation.

Computational Ecology and Software ISSN 2220-721X URL: http://www.iaees.org/publications/journals/ces/online-version.asp RSS: http://www.iaees.org/publications/journals/ces/rss.xml E-mail: ces@iaees.org Editor-in-Chief: WenJun Zhang Publisher: International Academy of Ecology and Environmental Sciences

1 Introduction

Bacteria are single-cell microorganisms that flourish in various environments. They comprise a large domain of prokaryotic microorganisms found in soil, air, water and inside the human being. Bacteria may be beneficial for us, for example, in curdling milk into yogurt, in our digestion etc. The harmful bacteria which help in spreading the infection and cause disease are called pathogenic bacteria. In developing countries, dumping of solid wastes is very much responsible for ruining the environmental conditions. Environmental factors such as poor supply of water, sanitation facilities, food and climate spread communicable diseases that are prone to epidemics and increase the spread of infectious diseases. There are certain diseases caused by bacterial infection include tuberculosis, typhoid fever, diphtheria, measles, etc. In case of many infectious diseases, infection is directly transmitted to susceptibles by infectives whereas in some diseases infection is also indirectly transmitted by flow of bacteria present in the environment (Gonzalez, 1989). For example, tuberculosis spreads from person to person as a result of direct contact with infectives by inhaling tubercle bacilli during breathing, talking etc. and indirectly via droplets containing tubercle bacilli released by TB infected person during coughing, singing or sneezing etc. These bacteria often settle down on human clothes, plants, flower pots etc. or remain airborne. The poor environmental conditions provide conducive environment to the growth of bacteria population which helps to further escalate the infectious diseases. An increase in bacteria population further degrades our environment leading to fast spread of infectious diseases. Due to continuous increase in human population and related activities, environmental degradation is more likely to take place. Thus, increasing demand for more food, shelter etc. results in pollution, deforestation, decline of natural resources which further add to environmental degradation. The degraded environmental conditions, caused by human activities, enhance the chances of bacteria population to grow resulting in fast spread of infectious diseases.

Several studies have been made for the spread of infectious diseases where direct transmission of disease has been considered without taking into account the role of bacteria population and environmental aspects (Agarwal and Verma, 2012b; Anderson and May, 1979; Gonzalez, 1989; Hethcote, 2000; Hsu and Zee, 2004; Nadjafikhah and Shagholi, 2017; Ugwa et al., 2013). However, to capture the realistic dynamics, in recent years, the spread of infectious diseases has been modeled by considering the role of bacteria population in the unhygienic environmental and ecological conditions in the habitat (Agarwal and Verma, 2012a; Ghosh et al., 2005, 2006; Naresh and Pandey, 2009a, b, 2012; Nthiiri et al., 2016; Pandey et al., 2018; Shukla et al., 2011; Singh et al., 2005). In particular, Ghosh et al. (2005) studied an SIS model for infectious disease spread by bacteria present in the degraded environment of poor people who work as service providers. They have assumed the growth rate and the carrying capacity of the bacteria population as constant in a structured population where service providers are drawn from an environmentally degraded region. Further, Ghosh et al. (2006) studied a model by assuming the logistic growth of both the human population and the bacteria population. It was shown that due to an increase in environmental discharge by human sources, the spread of infectious disease increases. Naresh and Pandey (2009a) proposed a mathematical model to analyze the effect of ecological factors in the habitat on the spread of TB in the human population. Their study shows that due to the increase in environmental degradation, the spread of tuberculosis is faster due to presence of bacteria in the conducive environment. As pointed earlier, environmental degradation is a serious issue to be considered.

In most of the above studies, the growth of bacteria population is taken to be directly proportional to the infective population or is governed by a logistic model with constant intrinsic growth rate and carrying capacity. However, as the human population density increases, the resulting environmental degradation leads to further growth in the density of bacteria population. This results in the fast spread of bacterial diseases. Thus, the growth rate and carrying capacity of bacteria population density must be taken as a function of cumulative density of environmental discharges (Naresh and Pandey, 2012).

Our main objective, in this paper, is to model and analyze the spread of bacterial diseases due to cumulative environmental degradation. In the modeling process, the density of bacteria population is assumed to grow logistically with the growth rate and carrying capacity taken as dependent on the density of cumulative environmental degradation, which further depend on human population related factors enhancing the growth of bacteria population density.

2 Mathematical Model

61

In this section, a nonlinear mathematical model dealing with the spread of bacterial diseases due to environmental degradation is proposed. Here, total human population N(t) is divided into two subclasses namely, susceptibles X(t) and infectives Y(t) at any time t. Further, B(t) and $E_m(t)$ represent cumulative bacteria population density and the density of environmental degradation at any time t respectively.

For simplicity we use X(t) = X, Y(t) = Y, B(t) = B and $E_m(t) = E_m$. The model is governed by following system of nonlinear ordinary differential equations:

$$\frac{dX}{dt} = A - \beta XY - \lambda BX - dX + \nu Y ;$$

$$\frac{dY}{dt} = \beta XY + \lambda BX - dY - \alpha Y - \nu Y;$$

$$\frac{dB}{dt} = s(E_m) \left(1 - \frac{B}{L(E_m)}\right) B + s_1 Y - s_0 B + s_2 BE_m ;$$

$$\frac{dE_m}{dt} = Q_0 - \theta_0 E_m + \theta_1 (A - dN).$$
(1)

 $\int_{dt} -Q_0 = 0 \int_{0}^{0} L_m + 0 \int_{0}^{1} (A - uN).$ where, $X(0) > 0, Y(0) > 0, B(0) > 0, E_m > 0.$

It is assumed that the infectious diseases not only spread directly from infective population to susceptibles but also indirectly from the bacteria present in the environment. The parameters representing the transmission rate are β and λ respectively, due to direct interaction of infectives with susceptibles and indirectly by bacteria present in the environment.

In the first equation of the model, the parameter A is the constant rate of immigration of population in the region. The natural mortality rate d is same for all individuals and the rate of infected individuals who take treatment and get themselves recovered is represented by the parameter ν . The parameter α is the rate at which the infected individuals die due to disease.

In third equation of the model system (1), the growth of bacteria population density is taken as logistic and its intrinsic growth $s(E_m)$ and carrying capacity $L(E_m)$ is assumed to be dependent on the cumulative density of environmental degradation.

Since the bacteria population is also assumed to grow due to its release from infective population, it is taken as directly proportional to infective population. The parameter s_1 represents the growth rate of bacteria population due to its release from infective population, the rate at which the bacteria population declines naturally or due to some other factors is represented by s_0 . The parameter s_2 is the rate of increase in the density of bacteria population due to cumulative environmental degradation. Since we assume that the growth rate of bacteria population increases due to the cumulative density of environmental degradation, we have,

$$s(0) = s$$
 and $s'(E_m) \ge 0$

where, s is the value of $s(E_m)$ at $E_m = 0$ and $s'(E_m)$ denotes the derivative of the function with respect to its argument. We assume that the modified carrying capacity increases with the cumulative density of environmental degradation, so that

 $L(0) = l \qquad \text{and} \qquad L'(E_m) \ge 0$

where, l is the value of $L(E_m)$ when $E_m = 0$.

The last equation of model system (1) represents the cumulative density of environmental degradation

(2)

(3)

which is taken to increase at a constant rate Q_0 and it is also assumed to be directly proportional to the growth of human population. Further, the parameter θ_0 represents the rate of decrease of cumulative density of environmental degradation due to some factors.

Since N(t) = X(t) + Y(t), the above model system (1) can be rewritten as follows,

$$\frac{dY}{dt} = \beta(N-Y)Y + \lambda B(N-Y) - (d + \alpha + \nu)Y;$$

$$\frac{dN}{dt} = A - dN - \alpha Y;$$

$$\frac{dB}{dt} = s(E_m) \left(1 - \frac{B}{L(E_m)}\right)B + s_1Y - s_0B + s_2BE_m$$

$$\frac{dE_m}{dt} = Q_0 - \theta_0 E_m + \theta_1 (A - dN).$$
(4)

dt co o m ic y

To show the feasibility of the model system (1), we show that all the variables are non-negative for all time t. From the first equation of model system (1), we have

$$\frac{dX}{dt} = A + \nu Y - (\beta Y + \lambda B + d)X.$$

The above equation can be written as

$$\frac{dX(t)}{dt}\exp\left(\int_{0}^{t}f_{1}(p)dp\right) + f_{1}(t)X(t)\exp\left(\int_{0}^{t}f_{1}(p)dp\right) = (A + \nu Y)\exp\left(\int_{0}^{t}f_{1}(p)dp\right),$$

where, $f_{1}(p) = \beta Y(p) + \lambda B(p) + d.$
$$\Rightarrow \frac{d}{dt}(X(t)\exp\left(\int_{0}^{t}f_{1}(p)dp\right)) = (A + \nu Y(t))\exp\left(\int_{0}^{t}f_{1}(p)dp\right),$$

and hence, we obtain,

$$X(t) = X(0)\exp\left(-\int_0^t f_1(p)dp\right) + \exp\left(-\int_0^t f_1(p)dp\right)\left[\int_0^t (A + vY(p))\exp\left(\int_0^t f_1(u)du\right)dp\right].$$

This shows that $X(t) > 0 \forall t > 0$.

Similarly, it can be shown that $Y(t) > 0, B(t) > 0 \& E_m(t) > 0, \forall t > 0$. Boundedness: From the second equation of model system (4), we have

$$\frac{dN}{dt} \le A - dN,$$

From above, we get,

$$\frac{d}{dt}(Ne^{dt}) \le Ae^{dt},$$

Now, integrating above equation from 0 to t, we obtain

$$N(t) \le \frac{A}{d} + \left(N(0) - \frac{A}{d}\right)e^{-dt}$$

Therefore, by using the theory of differential inequality (Lakshmikantham and Leela, 1969), we have

$$\lim_{t\to\infty} \sup N(t) \leq \frac{A}{d} \; .$$

and thus, $0 \le N(t) \le \frac{A}{d}$ for large t > 0.

From the third equation of model system (4), and using the fact that $Y(t) < N(t) \le \frac{A}{d}$ for large t > 0, we

;

have

$$\frac{dB(t)}{dt} - \left(s(E_m)_m \left(1 - \frac{B}{L(E_m)_m}\right) - s_0 + s_2(E_m)_m\right)B \le s_1 \frac{A}{d}$$

From the theory of differential inequality, we obtain $\lim_{t\to\infty} supB(t) \le B_m$ (say).

where,

$$B_m = \frac{L(E_m)_m}{2s(E_m)_m} \left[s(E_m)_m - s_0 + s_2(E_m)_m + \sqrt{(s(E_m)_m - s_0 + s_2(E_m)_m)^2 + \frac{4s(E_m)_m}{L(E_m)_m} \frac{s_1A}{d}} \right]$$
(5)

This implies that $0 < B(t) \le B_m$ for large t > 0.

Further, from the fourth equation of model system (4), we obtain

$$\frac{dE_m}{dt} + \theta_0 E_m \le Q_0 + \theta_1 A,$$

and hence,

 $\lim_{t\to\infty} \sup E_m(t) \le (E_m)_m \text{ (say).}$ where,

$$(E_m)_m = \frac{Q_0 + \theta_1 A}{\theta_0}$$

(6)

Now the following lemma is stated which is required for stability analysis of the equilibria of model system (4). **Lemma**

The region of attraction for the solution of the model system (4) is given as follows:

$$\Omega = \left\{ (Y(t), N(t), B(t), E_m(t)) \in \mathbb{R}_+^4 : 0 < Y < N < \frac{A}{a}, 0 < B < B_m, 0 < E_m < (E_m)_m \right\}.$$

which is positively invariant and all solutions stay in Ω , (Freedman and So, 1985). Here, B_m and $(E_m)_m$ are defined in eqns. (5) and (6) respectively.

3 Equilibrium Analysis

To study the qualitative behavior of the model system (4), we carry out the equilibrium analysis of the model. Two non-negative equilibria are found to be feasible, which are given below:

1. $E_0\left(0, \frac{A}{d}, 0, \frac{Q_0}{\theta_0}\right)$. This is disease-free equilibrium.

2. $E^*(Y^*, N^*, B^*, E_m^*)$. This is endemic equilibrium. The existence of E_0 is obvious.

 $\beta(N-Y)Y + \lambda B(N-Y) - (d + \alpha + \nu)Y = 0 ,$

We prove the existence of endemic equilibrium E^* by setting right hand side of equations in model (4) to zero and solving the resulting algebraic equations,

$$s(E_m)\left(1-\frac{B}{L(E_m)}\right)B + s_1Y - s_0B + s_2BE_m = 0,$$

(9)

 $A - dN - \alpha Y = 0,$

$$Q_0 - \theta_0 E_m + \theta_1 (A - dN) = 0$$

From (8) we have,

$$N = \frac{A - \alpha Y}{d}.$$

From (10) and (11) we get,

$$E_m = \frac{Q_0 + \theta_1 \alpha Y}{\theta_0}.$$

Now, using (11) in (7)

$$(A - (\alpha + d)Y)(\beta Y + \lambda B) - d(d + \alpha + \nu)Y = 0.$$

$$Y = \left[\frac{\theta_0}{s_2\theta_1\alpha B + s_{1\theta_0}}\right] \left[\frac{s(E_m)\theta}{L(E_m)}B^2 - \left(s(E_m) - s_0 + s_2\frac{Q_0}{\theta_0}\right)B\right].$$

Now we show the existence of Y^* and B^* from (13) and (14), and the corresponding values of N^* and E_m^* can be obtained from (11) and (12).

From (13), we have

(i) For B = 0,

We get,
$$Y = 0$$
 or $Y = \frac{\beta A - d(d + \alpha + \nu)}{\beta(\alpha + d)} = \tilde{Y}$ (say),

 \tilde{Y} is positive, if $\beta A > d(d + \alpha + \nu)$ and negative otherwise.

(ii) At (0, 0), the slope of (13) is given by,

$$\frac{dY}{dB} = -\frac{\lambda A}{\beta A - d(d + \alpha + \nu)}.$$

which is positive or negative depending upon \tilde{Y} being negative or positive, respectively. (iii) At $(0, \tilde{Y})$, the slope of (13) is given by,

$$\frac{dY}{dB} = \frac{\lambda d(d+\alpha+\nu)}{\beta(\beta A - d(d+\alpha+\nu))}.$$

which is positive or negative depending upon \tilde{Y} being positive or negative, respectively. From (14), we observe the following points,

(i) When Y = 0,

IAEES

We get,
$$B = 0$$
 or $B = \frac{L(E_m)}{s(E_m)} \left(s(E_m) - s_0 + \frac{s_2 Q_0}{\theta_0} \right) = \tilde{B}$ (say).

(ii) At (0, 0), the slope of (14) is given by,

$$\frac{dY}{dB} = -\frac{1}{s_1} \left(s(E_m) - s_0 + \frac{s_2 Q_0}{\theta_0} \right) < 0.$$

(iii) At $(\tilde{B}, 0)$, the slope of (14) is given by,

$$\frac{dY}{dB} = \frac{L^2(E_m)\theta_0\left(s(E_m) - s_0 + \frac{s_2Q_0}{\theta_0}\right)}{s_1\theta_0 L^2(E_m) + \alpha\theta_1 \bar{B}\left[L^2(E_m)s_2 + L(E_m)L'(E_m)s(E_m) + \left(s_0 - \frac{s_2Q_0}{\theta_0}\right)r\right]} > 0.$$

provided r > 0 where, $r = \frac{L(E_m)}{s(E_m)} (L(E_m)s'(E_m) - s(E_m)L'(E_m)).$

(10)

(11)

(12)

(13)

(14)

Thus, after plotting Y and B corresponding to (13) and (14) in Figs 1 & 2, we see that there are two intersecting points (0,0) and (Y^*, B^*) . After finding Y^* and B^* , we can calculate N^* and E_m^* using (11) and (12).





4 Stability Analysis

4.1 Local stability analysis

The local stability behavior of the equilibrium E_0 is presented here after computing the variational matrix about E_0 . However, for stability of E^* , Lyapunov's method is used in the linearized system.

Theorem-1 The equilibrium E_0 is unstable and the endemic equilibrium E^* is locally asymptotically stable provided the following conditions are satisfied,

$$(\lambda(N^* - Y^*) + s_1)^2 < \left(\beta Y^* + \frac{\lambda B^* N^*}{Y^*}\right) \left(\frac{s_1 Y^*}{B^*} + \frac{s(E_m^*)}{L(E_m^*)} B^*\right)$$

$$\theta_1^2 \left(s'(E_m^*) B^* - \frac{L(E_m^*) s'(E_m^*) - L'(E_m^*) s(E_m^*)}{L^2(E_m^*)} \right)^2 < \frac{\theta_0^2}{\alpha d} (\beta Y^* + \lambda B^*).$$
(16)

(For proof see Appendix-A)

4.2 Nonlinear stability analysis

The result of nonlinear stability analysis of endemic equilibrium E^* is presented in the following theorem.

Theorem-2 The endemic equilibrium E^* is nonlinearly asymptotically stable in the region Ω provided the following conditions are satisfied,

$$\left(\lambda\left(\frac{N^*-Y^*}{Y^*}\right)+s_1\right)^2 < \beta\psi(B^*, E_m^*)$$
(17)

$$\theta_1^2 \left(\left(B_m - \frac{B_m^2}{L(E_m^*)} \right) p + B_m^2 s((E_m)_m) \frac{q}{L_0^2} + s_2 B_m \right)^2 < \frac{\theta_0^2}{\alpha d} \left(\beta + \frac{\lambda B_m}{Y^*} \right) \psi(B^*, E_m^*), \tag{18}$$

where, $\psi(B^*, E_m^*) = \left(\frac{s(E_m^*)B^*}{L(E_m^*)} + s_0 - s_2 E_m^* - s(E_m^*)\right).$

(For proof see Appendix-B)

5 Numerical Experiments

We give here the numerical simulation of the model system (4) to show the existence of equilibrium values and to check the feasibility of stability conditions.

In the model, $s(E_m)$ and $L(E_m)$ are the growth rate and modified carrying capacity of the bacteria population as a function of cumulative density of environmental degradation E_m . Thus, for numerical simulation it is assumed that $s(E_m)$ and $L(E_m)$ are linear functions of E_m , i.e., $s(E_m) = s + aE_m$ and $L(E_m) = l + bE_m$, satisfying conditions (2) and (3). We integrate the system (4) by fourth-order Runge-Kutta method using MATLAB with the following set of parameter values,

 $A = 100, \beta = 0.002, \lambda = 0.000005, \nu = 0.02, d = 0.15, \alpha = 0.2, s = 0.85, s_0 = 0.3, s_1 = 0.0001, s_2 = 0.001, Q_0 = 25, \theta_1 = 0.001, \theta_0 = 0.1, a = 0.001, b = 0.01 and l = 10000.$

The equilibrium values of endemic equilibrium are computed as,

 $Y^* = 214.37, N^* = 380.83, B^* = 9551.94, E_m^* = 250.4.$

The eigenvalues of the variational matrix corresponding to the endemic equilibrium are $-0.0977, -0.3327 \pm 0.2513i, -1.0513$.

It can be seen that all the eigenvalues are either negative or have negative real part, therefore, for the above set of parameter values the endemic equilibrium is locally asymptotically stable. The results of the model are displayed graphically in Figures 3-10. Figure 3 shows that the endemic equilibrium E^* is nonlinearly asymptotically stable. The figure shows that the total human population N, infective population Y and the density of bacteria population B approach to the equilibrium point regardless of the initial values of N, Y

and *B*. The initial starts of all trajectories to reach the equilibrium point are given below:

- (1) $Y(0) = 100, N(0) = 300, B(0) = 7000, E_m(0) = 250.$ (2) $Y(0) = 300, N(0) = 600, B(0) = 5500, E_m(0) = 250.$
- (2) $Y(0) = 300, N(0) = 350, B(0) = 5000, E_m(0) = 250.$



















Fig. 4 shows the variation of infective population with time for different values of λ , the transmission rate due to density of bacteria population present in the environment. It is seen from the figure that with increase in the value of λ infective population increases.

Figs 5 and 6 show the role of release of bacteria from the infected individual at a rate s_1 on infectives and bacteria population density respectively with time. It is seen from the figure that the bacteria population increases in the environment as s_1 increases (Fig. 5). This increase in the bacteria population ultimately increases the infective population (Fig. 6). This implies that higher infective population releases more bacteria enhancing the density of bacteria population which further helps in inducing fast spread of bacterial diseases.

In Figs 7 and 8, the effect of s_2 , the rate of increase in the bacteria population density due to environmental degradation is shown on the bacteria population and infective population respectively with time. It is seen that as the environment degrades due to some environmental factors, the bacteria population increase in the environment (Fig. 7), which ultimately increases the infective population (Fig. 8). Thus, the conducive environmental degradation helps in increasing the bacteria population in the environment leading to fast spread of bacterial diseases.

The variation of bacteria population and infective population is shown in Figs 9 and 10 with time for distinct values of Q_0 , respectively. It is seen that as the growth rate of environmental degradation increases, bacteria population also increases (Fig. 9). This increment in bacteria population, increases the spread of bacterial diseases in infective population (Fig. 10).

It is, therefore, concluded that the environmental degradation plays an important role in increasing the density of bacteria population in the environment. This increase in bacteria population further contributes in enhancing the spread of infectious diseases.

6 Conclusion

In the present study, a nonlinear SIS model is proposed and analyzed to study the spread of bacterial diseases due to cumulative environmental degradation. In the model, the total human population is divided into two subclasses viz susceptibles and infectives. The direct and indirect interaction of susceptibles with infectives and with the bacteria present in the environment is considered here. The intrinsic growth rate and carrying capacity of bacteria population is assumed to be dependent on cumulative environmental degradation. It is also assumed that the growth rate of bacteria population density is directly proportional to the infective population. The cumulative density of environmental degradation depends upon human population-related factors. The model has been analyzed using the stability theory of differential equations and numerical simulation. The analysis of the model shows that the cumulative environment degradation helps in increasing the bacteria population in the environment which leads to fast spread of the infectious bacterial diseases. Thus, if the environment remains clean the density of bacteria population may decline and hence the spread of bacterial infectious diseases can be slowed down.

Appendix-A

The general variational matrix M for model system (4) is computed as follows:

$$M = \begin{bmatrix} \beta N - 2\beta Y - \lambda B - (d + \alpha + \nu) & \beta Y + \lambda B & \lambda N - \lambda Y & 0\\ -\alpha & -d & 0 & 0\\ s & 0 & -(s_0 - s_1 E_m) & s_1 B\\ 0 & -\theta_1 d & 0 & -\theta_0 \end{bmatrix}.$$

The variational matrix M₀ of model (4) corresponding to $E_0\left(0, \frac{A}{d}, 0, \frac{Q_0}{\theta_0}\right)$ is

$$M_{0} = \begin{bmatrix} \frac{\beta A}{d} - (d + \alpha + \nu) & 0 & \frac{\lambda A}{d} & 0 \\ -\alpha & -d & 0 & 0 \\ s & 0 & -\left(s_{0} - s_{1}\frac{Q_{0}}{\theta_{0}\theta}\right) & 0 \\ 0 & -\theta_{1}d & 0 & -\theta_{0} \end{bmatrix}.$$

The characteristics polynomial of above matrix is given by,

 $(d + \mu)(\theta_0 + \mu)(\mu^2 - h_1\mu - h_2) = 0,$ where,

$$h_1 = \left(\frac{\beta A}{d} - (d + \alpha + \nu)\right) - \left(s_0 - s_1 \frac{Q_0}{\theta_0}\right),$$

$$h_2 = \left(\frac{\beta A}{d} - (d + \alpha + \nu)\right) \cdot \left(s_0 - s_1 \frac{Q_0}{\theta_0}\right) + \frac{\lambda A s}{d}$$

Using Routh-Hurwitz criteria, we get that E_0 is unstable if $\left(\frac{\beta A}{d} - (d + \alpha + \nu)\right) > 1$ and stable if

$$\left(\frac{\beta A}{d} - (d + \alpha + \nu)\right) < 1.$$

To compute the local stability of endemic equilibrium E^* , we linearize the model system (4) using small perturbation *y*, *n*, *b*, e_m about E^* , defined as $Y = y + Y^*$, $N = n + N^*$, $B = b + B^*$ and $E_m = e_m + E_m^*$. Consider the following positive definite function:

$$U_1 = \frac{1}{2}(m_0 y^2 + m_1 n^2 + m_2 b^2 + m_3 e_m^2),$$

where, $m_i(i = 0, 1, 2, 3)$ are positive constants to be chosen appropriately. Differentiating above equation with respect to 't' and using linearized system of model (4) corresponding to E^* , we get,

$$\begin{aligned} \frac{dU_1}{dt} &= -m_0 \left(\frac{\lambda B^* N^*}{Y^*} + \beta Y^* \right) y^2 - m_1 dn^2 - m_2 \left(\frac{s_1 Y^*}{B^*} + \frac{s \left(E_m^* \right)}{L(E_m^*)} B^* \right) b^2 - m_3 \theta_0 e_m^2 \\ &+ [m_0 (\beta Y^* + \lambda B^*) - m_1 \alpha] ny + [m_0 \lambda (N^* - Y^*) + m_2 s_1] by \\ &+ m_2 \left(s' \left(E_m^* \right) B^* - \frac{L(E_m^*) s' \left(E_m^* \right) - L'(E_m^*) s(E_m^*)}{L^2(E_m^*)} B^{*2} + s_2 B^* \right) be_m - m_3 \theta_1 dne_m \end{aligned}$$

After choosing $m_0 = 1, m_1 = \frac{\beta Y^* + \lambda B^*}{\alpha}, m_2 = 1$ and

$$\frac{\left(\frac{s_{\ell}(E_{m}^{*})B^{*} - \frac{L(E_{m}^{*})s_{\ell}(E_{m}^{*}) - L'(E_{m}^{*})s(E_{m}^{*})}{L^{2}(E_{m}^{*})}B^{*2} + s_{2}B^{*}\right)^{2}}{\theta_{0}\left(\frac{s_{1}Y^{*}}{B^{*}} + \frac{s(E_{m}^{*})}{L(E_{m}^{*})}B^{*}\right)} < m_{3} < \frac{\theta_{0}}{\theta_{1}^{2}d\alpha}\left(\beta Y^{*} + \lambda B^{*}\right).$$

we get $\frac{dU_1}{dt}$ to be negative definite showing that U_1 is Lyapunov function and hence E^* is locally asymptotically stable provided the conditions (15) and (16) are satisfied.

Appendix-B

Proof: Consider the following positive definite function, corresponding to the model system (4) about E^* ,

$$U_{2} = m_{0} \left(Y - Y^{*} - Y^{*} \ln \frac{Y}{Y^{*}} \right) + \frac{m_{1}}{2} (N - N^{*})^{2} + \frac{m_{2}}{2} (B - B^{*})^{2} + \frac{m_{3}}{2} (E_{m} - E_{m}^{*})^{2},$$

where, $m_i(i = 0,1,2,3)$ are positive constants to be chosen appropriately. Differentiating the above equation with respect to 't' and using system (4), we get

$$dU_2$$
 (2. λBN) (U_1 U_2) (U_2 U_2 U_2 U_3 U_4 U_2 $\left(S(E_2^m) = 1 + (2\pi T_2^m) \right)$ (R_1

$$\begin{aligned} \frac{de_{Z}}{dt} &= -m_0 \left(\beta + \frac{de_{T}}{YY^*}\right) (Y - Y^*)^2 - m_1 d(N - N^*)^2 - m_2 \left(\frac{de_{T}}{L(E_m^*)}B + \psi(B^*, E^*)\right) (B - B^*)^2 \\ &- m_3 \theta_0 (E_m - E_m^*)^2 + \left[m_0 \left(\beta + \frac{\lambda B}{Y^*}\right) - m_1 \alpha\right] (Y - Y^*) (N - N^*) \\ &+ \left[m_0 \lambda \left(\frac{N^* - Y^*}{Y^*}\right) + m_2 s\right] (Y - Y^*) (B - B^*) \\ &+ m_2 \left[\left(B - \frac{B^2}{L(E_m^*)}\right) f(E_m) + B^2 s(E_m) g(E_m) + s_2 B \right] (B - B^*) (E_m - E_m^*) \\ &- m_3 \theta_1 d(N - N^*) (E_m - E_m^*). \end{aligned}$$

where, $f(E_m)$ and $g(E_m)$ are defined as follows,

$$f(E_m) = \begin{cases} \frac{s(E_m) - s(E_m^*)}{E_m - E_m^*}, & E_m \neq E_m^* \\ \frac{ds}{dE_m}, & E_m = E_m^* \end{cases},$$
$$g(E_m) = \begin{cases} \frac{L(E_m) - L(E_m^*)}{L(E_m)L(E_m^*)(E_m - E_m^*)}, & E_m \neq E_m^* \\ \frac{1}{L_0^2} \frac{dL}{dE_m}, & E_m = E_m^* \end{cases}$$

By considering the assumptions of the theorem and the mean value theorem, we have,

$$|f(E_m)| \le p, |g(E_m)| \le \frac{q}{L_0^2}$$
.

After choosing $m_0 = 1$, $m_1 = \frac{1}{\alpha} \left(\beta + \frac{\lambda B_m}{Y^*} \right)$, $m_2 = 1$ and

$$\frac{\left(\left(B_m - \frac{B_m^2}{L(E_m^*)}\right)p + B_m^2 s((E_m)_m)\frac{q}{L_0^2} + s_2 B_m\right)^2}{\varphi(B^*, E_m^*)} < m_3 < \frac{\theta_0}{\alpha d \theta_1^2} \left(\beta + \frac{\lambda B_m}{Y^*}\right).$$

we get $\frac{dU_2}{dt}$ to be negative definite showing that U_2 is a Lyapunov function and hence E^* is nonlinearly asymptotically stable provided the conditions (17) and (18) are satisfied.

Acknowledgements: This research is financially supported by Technical Education Quality Improvement Programme, phase-II (TEQIP-II), HBTU Kanpur, to one of the authors (Sandhya Rani Verma) in the form of Research cum Teaching Fellowship through letter no. SPFU/TEQIP-II/2015-16/88 dated 23.09.2015.

References

Agarwal M, Verma V. 2012. Modeling and analysis of the spread of an infectious disease cholera with environmental fluctuations. Applications and Applied Mathematics: An International Journal, 7: 406-425

Agarwal M, Verma V. 2012. Stability analysis of an SIR model for the spread of malaria. International Journal

of Applied Mathematics and Computer Science, 4(1): 64-76

Anderson RM, May RM. 1979. Population biology of infectious diseases. Nature, 280: 361-367

- Freedman HI, So JWH. 1985. Global stability and persistence of simple food chains. Mathematical Biosciences, 76: 69-86
- Ghosh M, Chandra P, Sinha P. 2005. Modelling the spread of bacterial disease: effect of service providers from an environmentally degraded region. Applied Mathematics and Computation, 60: 615-647
- Ghosh M, Chandra P, Sinha P, Shukla JB. 2006. Modelling the spread of bacterial infectious disease with environmental effect in a logistically growing human population. Nonlinear Analysis: Real World Applications, 7: 341-363
- Gonzalez-Guzmem J. 1989. An epidemiological model for direct and indirect transmission of Typhoid fever. Mathematical Biosciences, 96: 33-46
- Hethcote HW. 2000. The mathematics of infectious diseases. Society for Industrial and Applied Mathematics, 42(4): 599-653
- Hsu S, Zee A. 2004. Global spread of infectious diseases. Journal of Biological Systems, 12(3): 289-300
- Lakshmikantham V, Leela S. 1969. Differential and Integral Inequalities; Theory and Applications. Academic Press, New York, USA
- Nadjafikhah M, Shagholi S. 2017. Mathematical modeling of optimized SIRS epidemic model and some dynamical behaviors of the solution. International Journal of Nonlinear Analysis and Applications, 2: 125-134
- Naresh R, Pandey S. 2009a. Modeling the cumulative effect of ecological factors in the habitat on the spread of tuberculosis. International Journal of Biomathematics, 3: 339-355
- Naresh R, Pandey S. 2012. Modeling the effect of environmental factors on the spread of bacterial disease in an economically structured population. Applications and Applied Mathematics: An International Journal, 1: 426-454
- Naresh R, Pandey S. 2009b. Modeling and analysis of the spread of Japanese encephalitis with environmental effects. Applications and Applied Mathematics: An International Journal, 4(1): 155-175
- Nthiiri JK, Lawi GO, Akinyi CO, Oganga DO, Muriuki WC, Musyoka J, Otieno PO, Koech L. 2016. Mathematical modelling of Typhoid fever disease incorporating protection against infection. British Journal of Mathematics and Computer Science, 14(1): 1-10
- Pandey S, Nanda S, Vutha A, Naresh R. 2018. Modeling the impact of biolarvicides on malaria transmission. Journal of Theoretical Biology, 454: 396-409
- Shukla JB, Singh V, Misra A.K. 2011. Modeling the spread of an infectious disease with bacteria and carriers in the environment. Nonlinear Analysis: Real World Applications, 12: 2541-2551
- Singh S, Shukla JB, Chandra P. 2005. Modelling and analysis of the spread of malaria: Environmental and ecological effects. Journal of Biological Systems, 13: 1-11
- Ugwa K, Agwu A, Agbanyim N. 2013. Mathematical analysis of the endemic equilibrium of the transmission dynamics of tuberculosis. International Journal of Scientific and Technology Research, 12: 263-269