



Modeling and Analysis of the Spread of an Infectious Disease Cholera with Environmental Fluctuations

Manju Agarwal and Vinay Verma

Department of Mathematics & Astronomy

University of Lucknow

Lucknow-226007, India

manjuak@yahoo.com; vinay.verma09@rediffmail.com

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Abstract

A nonlinear delayed mathematical model with immigration for the spread of an infectious disease cholera with carriers in the environment is proposed and analyzed. It is assumed that all susceptible are affected by carrier population density. The carrier population density is assumed to follow the logistic model and grows due to conducive human population density related factors. The model is analyzed by stability theory of differential equations and computer simulation. Both the disease-free (DFE), (CFE) and endemic equilibria are found and their stability investigated. Bifurcation analyses about endemic equilibrium are also carried out analytically using the theory of differential equations. The model study shows that the spread of the infectious disease cholera increases due to growth of carriers in the environment and disease becomes more endemic due to immigration. Numerical simulations are also carried out to investigate the influence of certain parameters on the spread of disease, to support the analytical results of the model.

Keywords: Nonlinear delayed model; Cholera; Carriers; Infectious diseases; Stability; Bifurcation analysis; Numerical simulation

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1. Introduction

A recent study on cholera Emch et al. (2008) reveals that local environmental parameters are intensely associated with cholera dynamics. In particular, increase in ocean chlorophyll concentration; sea surface temperature and river height play a significant role on the occurrence of cholera and the magnitude of the epidemic. Cholera, a man-environment disease is transmitted through drinking water which is contaminated from improper treatment of sewage. Many infectious diseases spread by carriers such as flies, ticks, mites and snails, which are present in the environment. For example, air-borne carriers or bacteria spread diseases such as tuberculosis and measles; while water-borne carriers or bacteria are responsible for the spread of dysentery, diarrhea, cholera, etc. The last few years have witnessed many cholera outbreaks in developing countries, including India (2007), Congo (2008), Iraq (2008), Zimbabwe (2008-2009) Mukandavire et al. (2009, Vietnam), Nigeria (2010), and Haiti (2010). In the year 2010 alone, it is estimated that cholera affected 3-5 million people and caused 100000-130000 deaths in the world (WHO). Particularly, cholera represents a significant public health burden to developing countries and cholera continues to receive worldwide attention.

If the degree of infectivity increases due to sociological or other mechanisms saturation of infectives takes place Busenberge et al. (1993). It is therefore important to determine the effect of environmental fluctuations by considering the saturation incidence term $kSI/(1+k'I)$. The form of $kSI/(1+k'I)$ tends to kSI when k' is small and tends to kS/k' for very large value of I . In this study we revisit the carrier dependent cholera model already studied in Das et al. (2005) to investigate the effect of random fluctuation to the model system (2.1). We first consider a delayed *SIRS* model with a period of immunity of fixed length τ after recovery from the disease (the limiting case $\tau \rightarrow 0$ is an *SIS* model, and the limiting case $\tau \rightarrow \infty$ is an *SIRS* model). It is known that for such a single patch model with constant population size [Hethcote et al. (1981)] or with recruitment-death demographics and standard incidence [Genik et al. (1999)].

The classical disease transmission compartmental models assume homogeneous mixing of members of the population being studied. In the SARS epidemic of 2002-2003, infection was carried to many places by international travelers, with the disease becoming established in some locations but not in others Riley et al. (2003). A more realistic way of looking at the spread of disease may be to think of populations in several locations, or 'patches' with some travel between patches. For the formulation and analysis of the multi patches model, see for example, the review articles [Arino et al. (2006) and Wang et al. (2007) references therein.] This patch approach is analogous to a social network of contacts in which there is a small number of long distance contacts; see, for example, Meyers et al. (2007), Meyers et al. (2005) and Newman (2002).

Epidemic models have been studied by several researchers [Anderson et al. (1979), Bailey (1975) and Hsu et al. (2004)]. The effects of the presence of bacteria and carriers in the environment on the spread of infectious disease have not been studied using mathematical models [Gonzalez-Guzmem (1989)]. Hethcote (1976) discussed an epidemic model in which the carrier population is assumed to be constant. But in general, the size of the carrier population is not constant and depends on the environmental conditions in the habitat. Singh et al. (2003) and Ghose et al.

(2004) have considered variable growth of carrier population which depends on the household emissions other population density related factors in their models. Although, Codeço et al. (2006) have discussed “Trends in Cholera Epidemiology” and Ghose et al. (2005) and Shukla et al. (2006) have studied the spread of infectious diseases with bacteria in the environment they have ignored the role of carriers present in the environment. In this paper, a delayed *SIRS* model with immigration for the spread of the infectious disease cholera is proposed and analyzed by considering that all susceptible population are affected by carrier population density in the environment. Also, suppose that a recovered stays in the recovered class for a fixed finite time period τ , during which time natural death may occur. After time τ , immunity wanes and individuals still alive return to the susceptible class. Thus, we assume a constant period of temporary immunity, i.e., a step function distribution in contrast to the more common assumption of a negative exponential distribution.

The organization of this paper is as follows: In Section 2, we introduce our mathematical model. In Section 3, we present positivity and boundedness of solutions, respectively. In Section 4 and 5, we analyze our model with regard to equilibria and their stabilities. In Section 6, we establish bifurcation criterion to show the regulating impact of time delay. Computer simulations are performed to illustrate the feasibility of our analytical findings in Section 7. In the last Section 8, we present the conclusion based on our analysis.

2. The Mathematical Model

The population with size denoted by $N(t)$, is divided into three disjoint classes of individuals that are susceptible, infective and temporarily recovered, with class sizes denoted by $S(t)$, $I(t)$ and $R(t)$, respectively. It is assumed that all susceptible are affected by carrier population density $C(t)$, which is governed by a general logistic law. For $t > \tau$, the model is described by the system of delay differential equations (DDEs),

$$\begin{aligned}\frac{dS}{dt} &= A - \frac{kSI}{1+k'I} - \lambda SC - dS + \gamma e^{-d\tau} I(t-\tau), \\ \frac{dI}{dt} &= \frac{kSI}{1+k'I} + \lambda SC - (d + \varepsilon + \gamma)I, \\ \frac{dR}{dt} &= \gamma I - \gamma e^{-d\tau} I(t-\tau) - dR, \\ \frac{dC}{dt} &= (N + n_0 - s_0 C - s_1)C,\end{aligned}\tag{2.1}$$

where $N = S + I + R$. The associated initial condition is $S(0) > 0$, $I(0) > 0$, $R(0) \geq 0$, $C(0) \geq 0$.

Here, A is the constant immigration rate of human population from outside the region under consideration. The parameters k and λ are the transmission coefficients due to infective and carrier population respectively. Further d is the natural death rate, ε is the disease related death rate and γ be the rate at which infective individuals recover. The constant s_1 is the death rate coefficient of carriers due to natural factors as well as by control measures. We may note that if the growth rate and death rate are carrier population are be balanced then it may approach to zero. Here, k' and n_0 are saturation constant of incidence rate and birth rate of carriers population respectively. s_0 is the intraspecific coefficient of competition of carrier population. Suppose that a recovered stays in the recovered class for a fixed finite time period τ , during which time natural death may occur. All parameters assumed to be positive except ε and τ which are non-negative. After time τ , immunity wanes and individuals still alive return to the susceptible class. Thus, we assume a constant period of temporary immunity, i.e., a step function distribution in contrast to the more common assumption of a negative exponential distribution. Thus, the third equation of model (2.1) is equivalent to,

$$R = \gamma \int_{t-\tau}^t I(S) e^{-d(t-s)} ds. \tag{2.2}$$

For a given set of non- negative initial conditions, there is a unique solution of system (2.1) for $t \in [0, \tau]$. Note that if $\tau = 0$, the system (2.1) reduces to an *SIS* model, and the limit $\tau \rightarrow \infty$, it reduces to an *SIRS* model. To analyze model system (2.1), we consider the following reduced system (since $N = S + I + R$),

$$\begin{aligned} \frac{dI}{dt} &= \frac{k(N - I - R)I}{1 + k'I} + \lambda(N - I - R)C - (d + \varepsilon + \gamma)I, \\ \frac{dR}{dt} &= \gamma I - \gamma e^{-d\tau} I(t - \tau) - dR, \\ \frac{dN}{dt} &= A - dN - \varepsilon I, \\ \frac{dC}{dt} &= (N + n_0 - s_0 C - s_1)C. \end{aligned} \tag{2.3}$$

3. Boundedness of Solutions

Continuity of the right hand side of system (2.3) and its derivative imply that the model is well posed for $N > 0$. The invariant region where solution exists is obtained as follows:

$$\frac{A}{(d + \varepsilon)} \leq \liminf N(t) \leq \limsup N(t) \leq \frac{A}{d} \quad (\text{as } t \rightarrow \infty),$$

since $N(t) > 0$ for all $t \geq 0$. Therefore, $N(t)$ cannot blow up to infinity in finite time and consequently, the model system is dissipative (solutions are bounded). Hence, the solution exists for all $t > 0$ in the invariant and compact set,

$$\Omega = \left\{ (I, R, N, C) \in \mathbb{R}_+^4 : 0 \leq I, R \leq N \leq \frac{A}{d} + \eta, 0 \leq C \leq C_m \right\},$$

which is a region of attraction for any arbitrary small constant $\eta > 0$.

Here, $C_m = \frac{\left(\frac{A}{d} + n_0 - s_1 \right)}{s_0}$, provided $s_1 < \frac{A}{d} + n_0$.

As $N(t)$ tends to zero, $S(t)$, $I(t)$ and $R(t)$ also tends to zero. Hence, each of these subpopulations tends to zero as $N(t)$ does. It is therefore natural to interpret these terms as zero at $N(t) = 0$.

3.1. Positivity of Solutions

Let the initial data be $I(0) = I_0 \geq 0, N(0) = N_0 > 0, R(0) = R_0 \geq 0$ and $C(0) = C_0 \geq 0$ for all $t \geq 0$. Then, the solution $[I(t), N(t), R(t), C(t)]$ of the model remain positive for all time $t \geq 0$. From the first equation of model (2.3) we get, $I'(t) \geq -(d + \varepsilon + \gamma)I(t)$, which gives $I(t) \geq c_1 e^{-(d+\varepsilon+\gamma)t}$.

Here, c_1 is a constant of integration. A similar reasoning for the remaining equations shows that they are always positive in Ω for $t > 0$. We assume that at $t = 0$, $N(t), I(t), R(t)$ and $C(t)$ are all non-negative and that $N(0) > 0$.

We notice that,

$$\frac{A}{(d + \varepsilon)} \leq \liminf N(t) \leq \limsup N(t) \leq \frac{A}{d}, \text{ this implies that } S(t) > 0 \text{ for all } t.$$

4. Equilibrium Analysis

In this section we investigate the existence of equilibria of system (2.3). Solving the right hand side of the model system (2.3) by equating it to zero, we obtain the following biologically relevant equilibria.

(1) Disease-Free Equilibrium, $E_0\left(0, \frac{A}{d}, 0, 0\right)$, exists without any condition. The existence of E_0 is obvious. This equilibrium implies that if the carrier population, which serves as a medium of transport of cholera, does not participate in the system then the equilibrium level of human population will reach the value $\frac{A}{d}$. It may also be noted that in the absence of carrier population, the infected human population will become zero.

(2) Carrier-Free Equilibrium, $E_1(\bar{I}, \bar{R}, \bar{N}, 0)$. The equilibrium values of different variables will be given by \bar{I}, \bar{R} and \bar{N} . We prove the existence of the second equilibrium E_1 by setting right hand side of equations (2.3) to zero and solving the resulting algebraic equations, we get,

$$k(\bar{N} - \bar{I} - \bar{R})\bar{I} - (\varepsilon + d + \gamma)\bar{I}(1 + k\bar{I}) = 0. \tag{4.1}$$

$$\bar{N} = \left(\frac{A - \varepsilon\bar{I}}{d} \right). \tag{4.2}$$

$$\bar{R} = \frac{\gamma\bar{I}(1 - e^{-d\tau})}{d}. \tag{4.3}$$

Now using equation (4.2) and (4.3) in equation (4.1) we get,

$$\bar{I} = \frac{kA - d(d + \varepsilon + \gamma)}{k(\varepsilon + d) + dk'(d + \varepsilon + \gamma) + k\gamma(1 - e^{-d\tau})}. \tag{4.4}$$

Also, using equation (4.4) in equation (4.2) and (4.3) we get,

$$\bar{N} = \frac{Ak[d + \gamma(1 - e^{-d\tau})] + d(d + \varepsilon + \gamma)(Ak' + \varepsilon)}{d[k(\varepsilon + d + \gamma(1 - e^{-d\tau})) + dk'(d + \varepsilon + \gamma)]}. \tag{4.5}$$

and

$$\bar{R} = \frac{\gamma(1 - e^{-d\tau})}{d} \left[\frac{kA - d(d + \varepsilon + \gamma)}{k(\varepsilon + d) + dk'(d + \varepsilon + \gamma) + k\gamma(1 - e^{-d\tau})} \right]. \tag{4.6}$$

Hence, carrier-free equilibrium point $E_1(\bar{I}, \bar{R}, \bar{N}, 0)$ exist if $k > \frac{d(d + \varepsilon + \gamma)}{A}$.

(3) Endemic Equilibrium, $E_2(I^*, R^*, N^*, C^*)$. This equilibrium implies that if the carrier population is present in the system, then the infection will be transmitted to the human population. The equilibrium values of different variables will be given by I^*, R^*, N^* and C^* . These equilibrium values are explicitly given by equations (4.8-4.10). We prove the existence of

endemic equilibrium E_2 by setting right hand side of equations (2.3) to zero and solving the resulting algebraic equations, we get,

$$k(N - I - R)I + \lambda(N - I - R)C(1 + k'I) - (d + \varepsilon + \gamma)I(1 + k'I) = 0, \quad (4.7)$$

$$I^* = \frac{A - dN^*}{\varepsilon}, \quad (4.8)$$

$$R^* = \frac{\gamma(A - dN^*)(1 - e^{-d\tau})}{d}, \quad (4.9)$$

$$C^* = \frac{(N^* + n_0 - s_1)}{s_0}, \quad (4.10)$$

In the equilibrium E_2 , N^* is the positive root of the following equation, which can be obtained from equation (4.7) after using I^* , R^* and C^* from equations (4.8), (4.9) and (4.10), respectively. Using this value of $N = N^* > 0$ in equations (4.8-4.10) we obtain other equilibrium values,

$$\begin{aligned} F(N) = & k \left[N - \frac{(A - dN)}{\varepsilon} - \frac{\gamma(A - dN)(1 - e^{-d\tau})}{\varepsilon d} \right] \left(\frac{A - dN}{\varepsilon} \right) \\ & + \lambda \left[\frac{N + n_0 - s_1}{s_0} \right] \left[N - \frac{(A - dN)}{\varepsilon} - \frac{\gamma(A - dN)(1 - e^{-d\tau})}{\varepsilon d} \right] \left[1 + \frac{k'(A - dN)}{\varepsilon} \right] \\ & - (d + \varepsilon + \gamma) \left(\frac{A - dN}{\varepsilon} \right) \left[1 + \frac{k'(A - dN)}{\varepsilon} \right]. \end{aligned} \quad (4.11)$$

It would be sufficient if we show that $F(N) = 0$ has one and one only root. From equation (4.11), if we note that $F\left(\frac{A}{(d + \varepsilon)}\right) < 0$ and $F\left(\frac{A}{d}\right) > 0$. This implies that there exists a root N^* of $F(N) = 0$ in $\frac{A}{d + \varepsilon} < N < \frac{A}{d}$.

Also, $F'(N) > 0$, provided $\lambda\varepsilon \left[1 + \frac{k'(A - dN)}{\varepsilon} \right] > s_0 d \left[k + \frac{\lambda k'(N + n_0 - s_1)}{s_0} \right]$ in $\frac{A}{d + \varepsilon} < N < \frac{A}{d}$.

Thus, there exists a unique positive root of $F(N) = 0$, (say N^*) in $\frac{A}{d + \varepsilon} < N < \frac{A}{d}$. Knowing the value of N^* , the value of I^* , R^* and C^* can be computed from equations (4.8-4.10).

5. Stability Analysis

Now, we analyze the stability of equilibria E_0, E_1 and E_2 and the stability results of these equilibria are stated in the following theorems.

Theorem 5.1.

- (i) The disease-free equilibrium (DFE), E_0 is locally asymptotically stable if $k < d(d + \varepsilon + \gamma) / A$, $s_1 - n_0 > A / d$. Again E_0 is unstable if either $k > d(d + \varepsilon + \gamma) / A$, or $s_1 - n_0 < A / d$.
- (ii) The carrier-free equilibrium (CFE), E_1 is locally asymptotically stable if $s_1 - n_0 > \bar{N}$ and it is unstable if $s_1 - n_0 < \bar{N}$.

Proof:

- (i) The variational matrix V_0 for the system (2.3) corresponding to equilibrium $E_0\left(0, \frac{A}{d}, 0, 0\right)$ is given by,

$$V_0 = \begin{bmatrix} \frac{kA}{d} - (d + \varepsilon + \gamma) & 0 & 0 & \frac{\lambda A}{d} \\ -\varepsilon & -d & 0 & 0 \\ \gamma(1 - e^{-(\lambda+d)\tau}) & 0 & -d & 0 \\ 0 & 0 & 0 & \frac{A}{d} + n_0 - s_1 \end{bmatrix}.$$

The eigenvalues of V_0 are $\lambda_1 = \frac{kA}{d} - (d + \varepsilon + \gamma)$, $\lambda_2 = -d$, $\lambda_3 = -d$ and $\lambda_4 = \frac{A}{d} + n_0 - s_1$. Since all the model parameters are assumed to be nonnegative, it follows that $\lambda_i (i = 1, 2, 3, 4) < 0$. The stability of E_0 will be depend on the sign of λ_1 and λ_4 . Thus, the disease-free equilibrium (DFE) E_0 is locally asymptotically stable if $k < d(d + \varepsilon + \gamma) / A$, and $s_1 - n_0 > A / d$. Also, disease-free equilibrium (DFE) E_0 is unstable if either $k > d(d + \varepsilon + \gamma) / A$, or $s_1 - n_0 < A / d$.

- (ii) The variational matrix V_1 for the system (2.3) corresponding to equilibrium $E_1(\bar{I}, \bar{R}, \bar{N}, 0)$ is given by,

$$V_1 = \begin{bmatrix} \frac{k(\bar{N} - \bar{I} - \bar{R}) - k\bar{I}(1 + k\bar{T})}{(1 + k\bar{T})^2} - (d + \varepsilon + \gamma) & \frac{k\bar{I}}{(1 + k\bar{T})} & \frac{-k\bar{I}}{(1 + k\bar{T})} & \lambda(\bar{N} - \bar{I} - \bar{R}) \\ -\varepsilon & -d & 0 & 0 \\ \gamma(1 - e^{-(\lambda+d)\tau}) & 0 & -d & 0 \\ 0 & 0 & 0 & \bar{N} + n_0 - s_1 \end{bmatrix}.$$

One eigenvalue of V_1 are $\lambda_1 = \bar{N} + n_0 - s_1$. The stability of E_0 will be depend on the sign of λ_1 . The carrier-free equilibrium (CFE), E_1 is locally asymptotically stable if $s_1 - n_0 > \bar{N}$ and it is unstable if $s_1 - n_0 < \bar{N}$.

The term " $s_1 - n_0$ " biologically represents the net decay rate of carrier population. It is to be noted here that E_1 becomes stable when the intrinsic growth rate of carrier population at the equilibrium density is negative otherwise it is unstable.

(iii) *Stability analysis of endemic equilibrium, $E_2(I^*, R^*, N^*, C^*)$.* The variational matrix V_2 for the system (2.3) corresponding to equilibrium $E_2(I^*, R^*, N^*, C^*)$ is given by,

$$V_2 = \begin{bmatrix} J_1 & J_2 & -J_3 & J_4 \\ -\varepsilon & -d & 0 & 0 \\ J_5 & 0 & -d & 0 \\ 0 & 0 & C^* & -J_6 \end{bmatrix},$$

where

$$J_1 = \frac{k(N^* - I^* - R^*) - kI^*(1 + kI^*)}{(1 + kI^*)^2} - \lambda C^* - (d + \varepsilon + \gamma), \quad J_2 = \frac{kI^*}{(1 + kI^*)} + \lambda C^*,$$

$$J_3 = \frac{kI^*}{(1 + kI^*)} + \lambda C^*, \quad J_4 = \lambda(N^* - I^* - R^*), \quad J_5 = \gamma - \gamma e^{-(\lambda+d)\tau}, \quad J_6 = (N^* + n_0 - s_1).$$

The characteristic equation of equilibrium E_2 is

$$\lambda^4 + \lambda^3 B_1 + \lambda^2 B_2 + \lambda B_3 + B_4 - (\lambda^2 B_5 + \lambda B_6 + B_7)e^{-\lambda\tau} = 0, \quad (5.2)$$

where,

$$B_1 = J_1 + J_6 - 2d,$$

$$B_2 = \{\varepsilon J_2 + 2dJ_1 - d^2 - \gamma J_3 + J_6(J_1 - 2d)\},$$

$$B_3 = \{J_1 d^2 + d\varepsilon J_2 + J_4 \varepsilon C^* + J_6(\varepsilon J_2 + 2dJ_1 - d^2 - \gamma J_3) - J_3 d\gamma\},$$

$$B_4 = \{J_4 \varepsilon C^* d + (d\varepsilon J_1 + d^2 J_1 - J_3 \gamma d)J_6\},$$

$$B_5 = J_3 \gamma e^{-d\tau} = J_3 \{(\gamma I^* - dR^*) / I^*\},$$

$$B_6 = J_3 d \gamma e^{-d\tau} = J_3 d \left\{ (\gamma I^* - dR^*) / I^* \right\},$$

$$B_7 = J_3 J_6 d \gamma e^{-d\tau} = J_3 J_6 d \left\{ (\gamma I^* - dR^*) / I^* \right\},$$

Let $\phi(\lambda, \tau) = \lambda^4 + \lambda^3 B_1 + \lambda^2 B_2 + \lambda B_1 + B_4 - (\lambda^2 B_5 + \lambda B_6 + B_7) e^{-\lambda\tau} = 0.$ (5.3)

To show the positive equilibria $E_2(I^*, R^*, N^*, C^*)$ is locally asymptotically stable for all $\tau > 0$, we use the following Theorem 5.4., Begon (1995).

Theorem 5.4. A set of necessary and sufficient conditions for $E_2(I^*, R^*, N^*, C^*)$ to be asymptotically stable for all $\tau \geq 0$ is

- (i) The real part of all roots of $\phi(\lambda, 0) = 0$ are negative.
- (ii) For all real ω_0 and $\tau \geq 0$, $\phi(i\omega_0, \tau) \neq 0$ where $i = \sqrt{-1}$.

Theorem 5.5. The endemic equilibrium point E_2 exist if

$$\lambda \varepsilon \left[1 + \frac{k'(A-dN)}{\varepsilon} \right] > s_0 d \left[k + \frac{\lambda k'(N+n_0-s_1)}{s_0} \right]$$

holds is locally asymptotically stable for all $\tau \geq 0$.

Proof:

We now apply Theorem (5.4) to prove Theorem (5.5). We prove this theorem in two steps.

Step 1. Substituting $\tau = 0$ in equation (5.3), we get

$$\phi(\lambda, 0) = \lambda^4 + \lambda^3 B_1 + \lambda^2 B_2 + \lambda B_1 + B_4 - (\lambda^2 B_5 + \lambda B_6 + B_7) = 0,$$

$$\phi(\lambda, 0) = \lambda^4 + P_1 \lambda^3 + P_2 \lambda^2 + P_3 \lambda + P_4 = 0, \tag{5.6}$$

If, $P_1 = B_1 > 0$, $P_2 = (B_2 - B_5) > 0$, $P_3 = (B_1 - B_6) > 0$, $P_4 = (B_4 - B_7) > 0$ and $P_1 P_2 P_3 > P_3^2 + P_1^2 P_4$, then by Routh-Hurwitz criterion, all roots of equation (5.6) have negative real parts. Hence condition (i) of Theorem (5.4) is satisfied and E_2 is a locally asymptotically stable equilibrium in the absence of delay.

Step 2. Suppose that $\phi(i\omega_0, \tau) = 0$ holds for some real ω_0 .

Firstly, when $\omega_0 = 0$,

$$\phi(0, \tau) = B_4 - B_7 \neq 0.$$

Secondly, suppose $\omega_0 \neq 0$,

$$\phi(i\omega_0, \tau) = \omega_0^4 - i\omega_0^3 B_1 - \omega_0^2 B_2 + i\omega_0 B_3 + B_4 - (-\omega_0^2 B_5 + i\omega_0 B_6 + B_7)e^{-i\omega_0 \tau}. \quad (5.7)$$

Equating real and imaginary parts of equation (5.7), we get

$$\omega_0^4 - \omega_0^2 B_2 + B_4 = (B_7 - \omega_0^2 B_5) \cos \omega_0 \tau + \omega_0 B_6 \sin \omega_0 \tau, \quad (5.8)$$

$$- \omega_0^3 B_1 + \omega_0 B_3 = \omega_0 B_6 \cos \omega_0 \tau - (B_7 - \omega_0^2 B_5) \sin \omega_0 \tau, \quad (5.9)$$

Squaring and adding equation (5.8) and (5.9), we get

$$\omega_0^8 + q_1 \omega_0^6 + q_2 \omega_0^4 + q_3 \omega_0^2 + q_4 = 0, \quad (5.10)$$

where

$$q_1 = (B_1^2 - 2B_2) > 0, \quad q_2 = (B_2^2 - 2B_1 B_3 + 2B_4 - B_5^2) > 0,$$

$$q_3 = (B_3^2 - 2B_2 B_4 + 2B_5 B_7 - B_6^2) > 0 \quad \text{and} \quad q_4 = (B_4^2 - B_7^2) > 0.$$

It follows that

$$\omega_0^8 + q_1 \omega_0^6 + q_2 \omega_0^4 + q_3 \omega_0^2 + q_4 > 0.$$

This contradicts (5.10). Hence, $\phi(i\omega_0, \tau) \neq 0$. For any real ω_0 , it satisfies condition (ii) of Theorem (5.4). Therefore, the unique positive equilibrium $E_2(I^*, R^*, N^*, C^*)$ is locally asymptotically stable for all $\tau \geq 0$ and the delay is harmless in this case.

6. Bifurcation Analysis

Substituting $\lambda = a(\tau) + ib(\tau)$ in (5.3) and separating real and imaginary parts, we obtain the following transcendental equations

$$\begin{aligned} a^4 - 6a^2 b^2 + b^4 + (a^3 - 3ab^2)B_1 + (a^2 - b^2)B_2 + aB_3 + B_4 \\ - e^{-a\tau} \left\{ (a^2 - b^2)B_5 + aB_6 + B_7 \right\} \cos b\tau - e^{-a\tau} (2abB_5 + bB_6) \sin b\tau = 0. \end{aligned} \quad (6.1)$$

$$(4ba^3 - 4ab^3) + (3a^2b - b^3)B_1 + 2abB_2 + bB_3 - e^{-a\tau} \{(2abB_5 + bB_6)\} \cos b\tau - e^{-a\tau} \{(a^2 - b^2)B_5 + aB_6 + B_7\} \sin b\tau = 0. \quad (6.2)$$

where a and b are functions of τ . We are interested in the change of stability of E_2 which will occur at the values of τ for which $a = 0$ and $b \neq 0$.

Let $\hat{\tau}$ be such that for which $a(\hat{\tau}) = 0$ and $b(\hat{\tau}) = \hat{b} \neq 0$. then equation (6.1) and (6.2) becomes

$$b^4 - b^2B_2 + B_4 - (B_7 - b^2B_5)\cos b\tau - bB_6\sin b\tau = 0. \quad (6.3)$$

$$-b^3B_1 + bB_3 - bB_6\cos b\tau + (B_7 - b^2B_5)\sin b\tau = 0. \quad (6.4)$$

Now eliminating $\hat{\tau}$ from (6.3) and (6.4), we get

$$\hat{b}^8 + (B_1^2 - 2B_2)\hat{b}^6 + (B_2^2 - 2B_1B_3 + 2B_4 - B_5^2)\hat{b}^4 + (B_3^2 - 2B_2B_4 + 2B_5B_7 - B_6^2)\hat{b}^2 + (B_4^2 - B_7^2) = 0. \quad (6.5)$$

To analyze the change in the behavior of stability of E_2 with respect to τ , we examine the sign of $\frac{da}{d\tau}$ as it crosses zero. If this derivative is positive (negative), then clearly a stabilization (destabilization) cannot take place at that value of τ . Differentiating equations (6.1) and (6.2) with respect to τ , then setting $\tau = \hat{\tau}$, $a = 0$ and $b = \hat{b}$, we get

$$\theta_1 \frac{da}{d\tau}(\hat{\tau}) + \theta_2 \frac{db}{d\tau}(\hat{\tau}) = g, \quad (6.6)$$

$$-\theta_2 \frac{da}{d\tau}(\hat{\tau}) + \theta_1 \frac{db}{d\tau}(\hat{\tau}) = h, \quad (6.7)$$

where

$$\begin{aligned} \theta_1 &= -3\hat{b}^2B_1 + B_3 + (B_7 - \hat{b}^2B_5)\hat{\tau} \cos \hat{b}\hat{\tau} - B_6 \cos \hat{b}\hat{\tau} + B_6\hat{b}\hat{\tau} \sin \hat{b}\hat{\tau} - 2B_5\hat{b} \sin \hat{b}\hat{\tau}, \\ \theta_2 &= 4\hat{b}^3 - 2\hat{b}B_2 + (B_7 - \hat{b}^2B_5)\hat{\tau} \sin \hat{b}\hat{\tau} + 2B_5\hat{b} \cos \hat{b}\hat{\tau} - \hat{b}B_6\hat{\tau} \cos \hat{b}\hat{\tau} - B_6 \sin \hat{b}\hat{\tau}, \\ g &= \hat{b}^2B_6 \cos \hat{b}\hat{\tau} - (B_7 - \hat{b}^2B_5)\hat{b} \sin \hat{b}\hat{\tau}, \\ h &= -B_6\hat{b}^2 \sin \hat{b}\hat{\tau} - (B_7 - \hat{b}^2B_5)\hat{b} \cos \hat{b}\hat{\tau}. \end{aligned} \quad (6.8)$$

Solving (6.6) and (6.7), we get

$$\frac{da}{d\tau}(\hat{\tau}) = \frac{g\theta_1 - h\theta_2}{\theta_1^2 + \theta_2^2}. \quad (6.9)$$

From (6.9), it is clear that $\frac{da}{d\tau}(\hat{\tau})$ has the same sign as $g\theta_1 - h\theta_2$. From (6.8), after simplification, and solving (6.3) and (6.4), we get

$$g\theta_1 - h\theta_2 = \hat{b}^2 \left\{ \begin{aligned} &4\hat{b}^6 + 3\hat{b}^4(B_1^2 - 2B_2) + 2\hat{b}^2(B_2^2 - 2B_1B_3 + 2B_4 - B_5^2) \\ &+ (B_3^2 - 2B_2B_4 + 2B_5B_7 - B_6^2). \end{aligned} \right\}. \quad (6.10)$$

Let

$$G(u) = u^4 + S_1u^3 + S_2u^2 + S_3u + S_4, \quad (6.11)$$

where,

$$S_1 = (B_1^2 - 2B_2), S_2 = (B_2^2 - 2B_1B_3 + 2B_4 - B_5^2), S_3 = (B_3^2 - 2B_2B_4 + 2B_5B_7 - B_6^2) \text{ and}$$

$$S_4 = (B_4^2 - B_7^2).$$

From (6.11), we note that $G(u)$ is the left hand side of equation (6.5) with $\hat{b}^2 = u$. Therefore,

$$G(\hat{b}^2) = 0. \quad (6.12)$$

Now

$$\begin{aligned} \frac{dG(\hat{b}^2)}{du} &= 4\hat{b}^6 + 3S_1\hat{b}^4 + 2S_2\hat{b}^2 + S_3 \\ &= 4\hat{b}^6 + 3(B_1^2 - 2B_2)\hat{b}^4 + 2(B_2^2 - 2B_1B_3 + 2B_4 - B_5^2)\hat{b}^2 + (B_3^2 - 2B_2B_4 + 2B_5B_7 - B_6^2). \\ &= \frac{g\theta_1 - h\theta_2}{\hat{b}^2} = \frac{\theta_1^2 + \theta_2^2}{\hat{b}^2} \frac{da}{d\tau}(\hat{\tau}). \end{aligned}$$

This implies that

$$\frac{da}{d\tau}(\hat{\tau}) = \frac{\hat{b}^2}{\theta_1^2 + \theta_2^2} \frac{dG(\hat{b}^2)}{du}. \quad (6.13)$$

Hence, the criterion for instability (stability) of E_2 are

- (1) If the polynomial $G(u)$ has no positive root, there can be no change of stability.
- (2) If $G(u)$ is increasing (decreasing) at all of its positive roots, instability (stability) is preserved.

Now in this case, if

- (i) $S_4 < 0$, $G(u)$ has unique positive real root then it must increase at that point (since $G(u)$ is a cubic in u , $\lim_{u \rightarrow \infty} G(u) = \infty$).
- (ii) $S_4 > 0$, then (1) is satisfied, i.e. there can be no change of stability.

Therefore, we have the following theorems.

Theorem 6.1. If $S_4 < 0$ and E_2 is unstable for $\tau = 0$, it will remain unstable for $\tau > 0$.

Theorem 6.2. If $S_4 < 0$ and E_2 is asymptotically stable for $\tau = 0$, it is impossible that it remain stable for $\tau > 0$. Hence there exist a $\hat{\tau} > 0$, such that for $\tau < \hat{\tau}$, E_2 is asymptotically stable for $\tau > \hat{\tau}$, E_2 is unstable and as τ increases together with $\hat{\tau}$, E_2 bifurcates into small amplitude periodic solutions of Hopf type Anderson (1979). The value of $\hat{\tau}$ is given by the following equation

$$\hat{\tau} = \frac{1}{\hat{b}} \text{Sin}^{-1} \left[\frac{(\hat{b}^4 - \hat{b}^2 B_2 + B_4)\hat{b}B_6 + (\hat{b}^2 B_5 - B_7)(-\hat{b}^3 B_1 + \hat{b}B_3)}{(\hat{b}^2 B_5 - B_7)^2 + (\hat{b}B_6)^2} \right].$$

7. Numerical Simulation

Analytical studies always remain incomplete without numerical verification of the results. To facilitate the interpretation of our mathematical findings by numerical simulations, we consider the set of parameters values as $k = 1 \text{ day}^{-1}$, $\lambda = 0.02 \text{ day}^{-1}$, $A = 10 \text{ day}^{-1}$, $d = 0.5 \text{ day}^{-1}$, $\gamma = 2 \text{ day}^{-1}$, $\varepsilon = 1 \text{ day}^{-1}$, $k' = 10 \text{ day}$, $\tau = 2 \text{ day}$, $n_0 = 1 \text{ day}^{-1}$, $s_0 = 20 \text{ day}^{-1}$, $s_1 = 10 \text{ day}^{-1}$.

For the above set of parameter values, the equilibrium E_2 is given by, $I^* = 0.4499$, $N^* = 19.1002$, $R^* = 1.1376$, $C^* = 0.4050$.

Again with the set of parameters given above it can be verified that all the conditions of local stabilities are satisfied. We find here, in the absence of carrier ($C = 0$), the equilibria $E_1(\bar{I}, \bar{N}, \bar{R}, 0)$ for different values of delayed time τ is given by the following table:

Table 1.

τ	\bar{I}	\bar{N}	\bar{R}
2	0.40712	19.18575	1.02939
4	0.39798	19.20402	1.37650
6	0.39472	19.21054	1.50030

Table 1 shows that in the absence of carrier population, the infective human population and recovered population decreases and increases with τ respectively.

The variation of infective human population and recovered population with respect to time for different values of rate of dissemination τ is shown in Figures 1 and 2 respectively. From these figures, it can be noted that as the rate of dissemination τ increases, infective human population and recovered population decreases and increases respectively.

From Figures 3-4 the effect of various parameters, i.e., γ and k' on the infective human population have been shown. It is noted that these figures that as these parameters values increases, the infective human population decreases. In Figure 5 shows that infective human population increases for some time, reaches the peak, then starts decreasing and finally attains its equilibrium level. From this figure we also note that infective human population remain constant at its equilibrium level as k increases but the amplitude and timing of the peak decreases with increases in k . Also, in Figure 6 shows the effect of constant immigration rate of human population i.e., A on the infective population with time. From this figure we concluded that as the growth rate of constant immigration rate of human population increases, the infective human population increases. In Figure 7 shows that the transmission coefficient due to carrier population increases, the number of infective population increases.

8. Conclusion

In this paper, we have proposed and analyzed a non-linear delayed *SIRS* model with immigration for the spread of an infectious disease caused by discharges of carrier in the environment by infectives and their interactions with the susceptible. The carrier dependent infectious disease cholera is studied here by incorporating environmental fluctuations through additive white noise. It has been considered that the density of carriers follows the logistic model and the growth rate of which increases due to the conducive human density related factors. This system is analyzed for positivity, boundedness of solutions, and bifurcations of the model are studied analytically using theory of differential equations. The model has three non-negative equilibria, namely disease free (DFE), carrier free (CFE) and the endemic equilibrium. The stability analysis has shown that the (DFE) and (CFE) are locally asymptotically stable and unstable under certain condition. Also, we have to show that the endemic equilibrium is locally asymptotically stable. It has been shown that spread of disease increases, apart from direct contacts between susceptible and infectives, due to the presence of carrier in the environment, their interactions with susceptible directly as well as indirectly through transport mechanisms used by carriers. It has been found that the disease become more endemic due to immigration. By

computer simulation it is shown that when the growth rate of human population increases due to demographic changes, the infectious disease spreads even further and becomes more endemic. It is concluded from the analysis that if the growth of carrier population caused by conducive household discharges increases, the spread of the infectious disease increases. Also, when growth rate at which infective individuals recover increases, the infective human population decreases.

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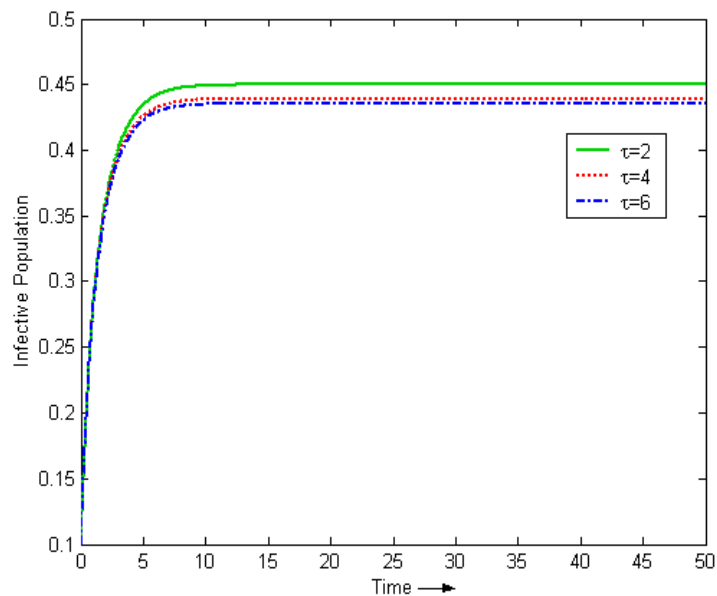


Figure 1. Variation of infective population with t for different τ in the presence of carrier population and other parameters values are same.

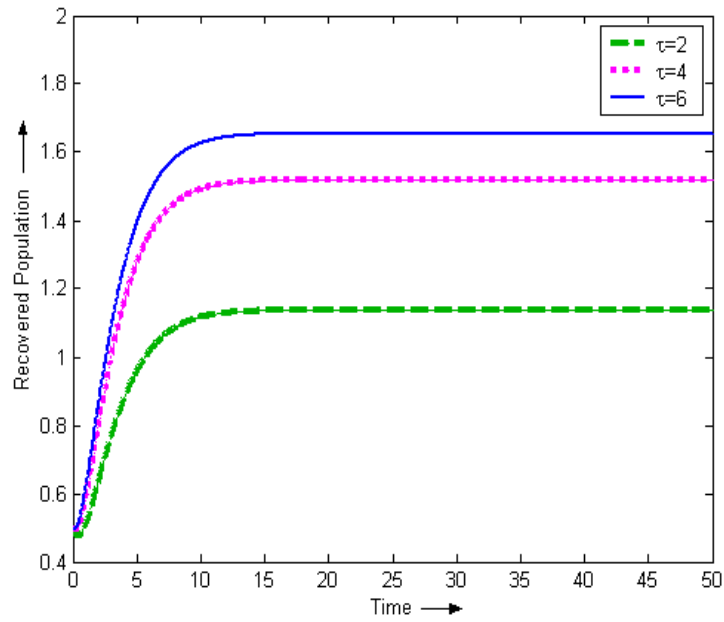


Figure 2. Variation of recovered population with t for different τ in the presence of carrier population and other parameters values are same.

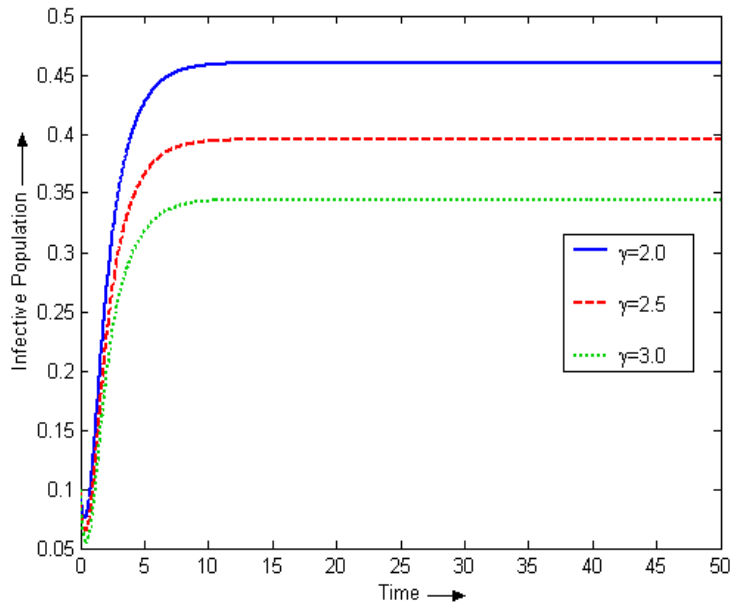


Figure 3. Variation of infective population with t for different γ in the presence of carrier population and other parameters values are same.

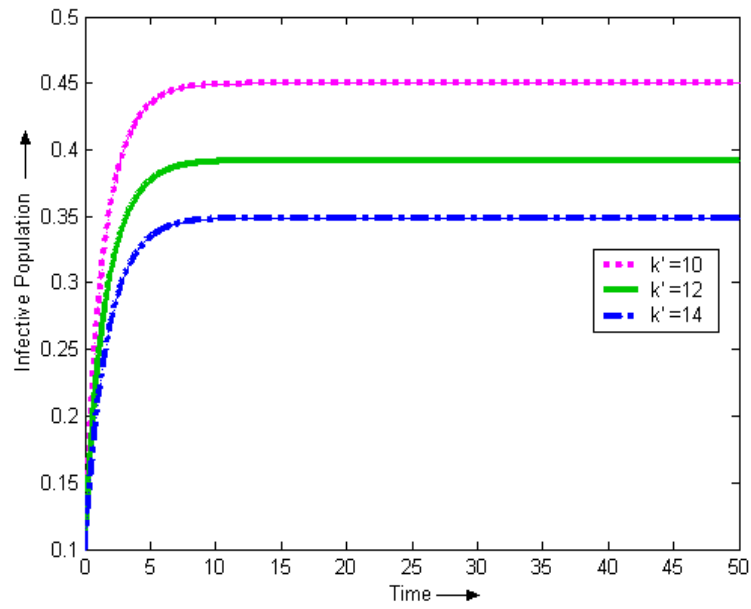


Figure 4. Variation of infective population with t for different k' in the presence of carrier population and other parameters values are same.

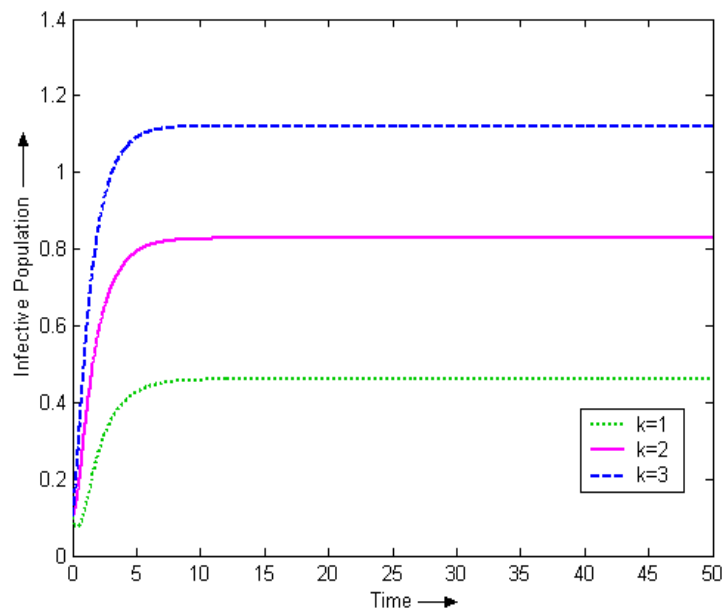


Figure 5. Variation of infective population with t for different k in the presence of carrier population and other parameters values are same.

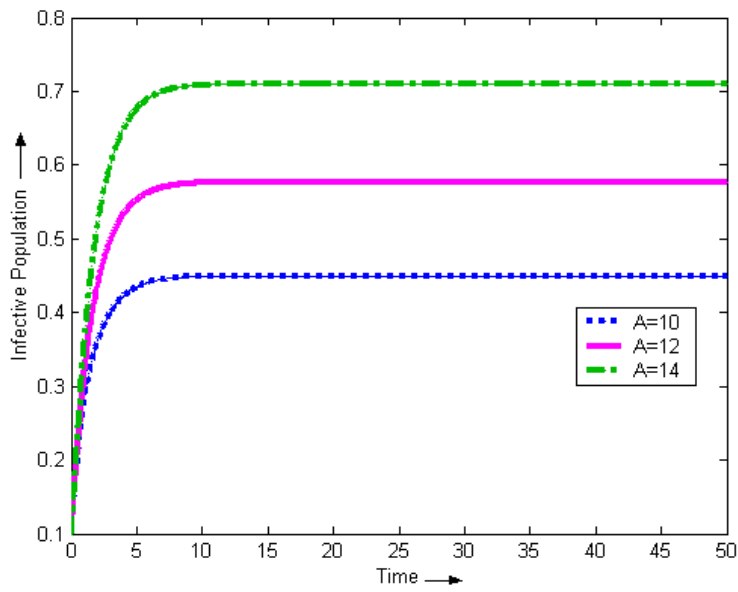


Figure 6. Variation of infective population with t for different A in the presence of carrier population and other parameters values are same.

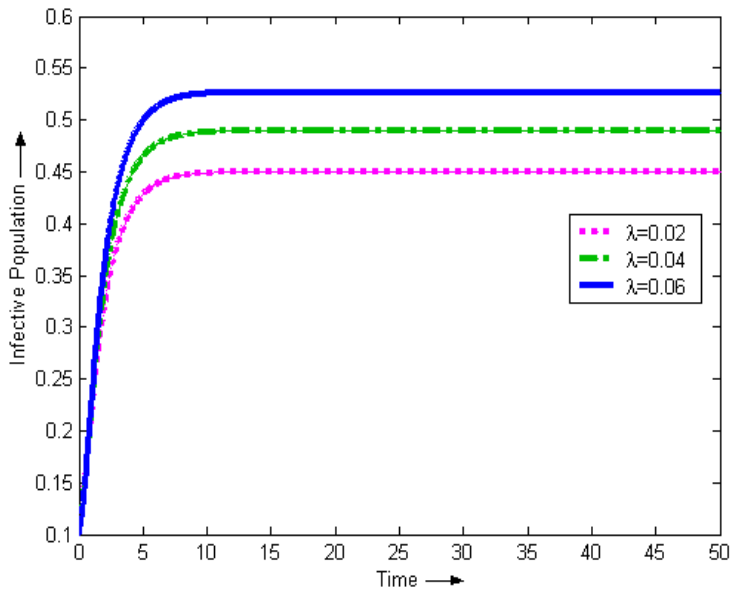


Figure 7. Variation of infective population with t for different λ and other parameters values are same.