Effect of Time Delay in the Stability Analysis of Cholera Epidemic-Endemic Disease Model

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Abstract:
Cholera as a disease is a kind of acute diarrhea caused by bacteria Vibrio cholerae. A nonlinear delayed mathematical model with environmental factor for the spread of infectious disease cholera is proposed and analyzed. A mathematical model for cholera was improved by adding a time delay that represents the time between the instant at which an individual becomes infected and the instant at which he begins to have symptoms of cholera disease. It is assumed that all susceptible are affected by carrier population density. The model is analyzed by stability theory of differential equations and computer simulation. We prove that the delayed cholera model is biologically meaningful and analyze the local asymptotic stability of the equilibrium points for positive time delays. Both the disease-free (DFE) and endemic equilibria are found and their stability investigated using the Routh-Hurwitz stability criterion method. Next Generation Matrix (NGM) method was used to get the basic reproductive number $R_0$. The disease-free equilibrium point is locally asymptotically stable if $R_0 < 1$, while the endemic equilibrium point is locally asymptotically stable if $R_0 > 1$. 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: Delayed model, Cholera, Infectious diseases, Stability, Differential equations.

Introduction
Differential equations are important tools for every ongoing research with a capability of reformulating basic real-world problems and analyzing their solutions in the various field (see Davies et al., 2023; Davies and Jackreece, 2005 and Davies and Haas, 2015). It is considered as the most frequently used mathematical model in population dynamics and patterns of disease conditions in epidemics and multi-species population interaction in ecology, economics, engineering and other fields (see Davies et al., 2022). Mathematical models have been in use for a long time and many problems of real life have been formulated mathematically in recent years as a crucial general process in engineering, science, medicine, economics, environmental science, and other fields that translate a physical situation or some other observations into a mathematical model (see Adiela et al. 2023; Kreyszig, 2011; Davies and Jackreece, 2005). According to (Scott and Carly, 2021), mathematical models of disease transmission has provided researchers with critical insights into the progression, control and prevention of
disease spread. One of the most fundamental of these models is the SIR (Susceptible, Infectious and Recovered) differential equation model which can be used to model infectious disease. Infectious disease can spread and turn into epidemics, taking thousands of lives within a few days, one of those diseases is cholera.

Cholera is an acute diarrheal infection of the intestine caused by the ingestion of food or water contaminated with the toxigenic bacterium *Vibrio cholerae*. This disease is transmitted through drinking water which is contaminated from improper treatment of sewage. Its dynamics are complicated by the multiple interactions between the human host, the pathogen and the environment which contribute to both direct; human-to-human and indirect; environment-to-human transmission pathways (Wang and Liao, 2011). A study on cholera by (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. Many infectious diseases spread by carriers such as flies, ticks, mites and snails, which are present in the environment. Toxigenic *Vibrio cholerae* can survive in some aquatic environments for months to years, in association with zooplankton and other aquatic organisms. Under stress, this pathogen assumes a viable but not culturable state, becoming undetectable to traditional bacteriological techniques (Codeco, 2001; Colwell, 1996). Classical control strategies in terms of the sensitization of population and sanitation are integrated through the impulsive differential equations (Kolaye et al., 2020). More recently, (Martins et al., 2022) in their study of mathematical model for prevention and control of cholera transmission in a variable population developed an extended SIRB deterministic epidemiological model for cholera and strictly analyzed it to ascertain the impact of immigration in cholera transmission and to assess the suitability of the various control measures (also see Olaniyi and Ogbonna, 2021). The model was found to have two equilibria. The local stability of the disease-free equilibrium (DFE) and endemic equilibrium (EE) were found to be dependent on a certain epidemiological threshold known as the basic reproductive number (number of secondary infections resulting from the introduction of a single infected individual into a population). They found out that the impact of the control over the long and short cycle transmission routes were more effective than vaccination in combating the menace of cholera and the effects of immigration in the transmission of cholera were validated via numerical simulation using estimated and baseline parameter values. Similar to their findings was a research by (Mondal and Kar, 2013) where they presented a water-borne disease epidemic model amendable to cholera dynamics including multiple transmissions. They studied the sensitivity analysis of the system and discovered that the number of infected individual and concentration of pathogens are directly proportional to the two-type disease transmission rate. In a related study of sensitivity analysis of the parameters of a cholera model by (Tilahun et al., 2020; and Peter et al., 2018), they formulated a deterministic mathematical model to analyze the degree of sensitivity of some factors that aid cholera transmission and management. They obtained a disease-free equilibrium point and conducted the local stability of the disease-free and endemic equilibria of the model. The method of normalized forward sensitivity index is employed to determine the numerical value of the key model parameters with respect to the effective reproduction numbers to determine their relative importance to cholera transmission and management. Their result showed that the most important parameter to cholera transmission is the contact rate between susceptible and infectious individuals while the most crucial parameter to cholera management is the rate of cholera awareness.

Developing countries are the most affected by cholera disease due to inadequate sanitation, improper treatment of reservoirs and lack of safe water supply. Thus, this epidemic is still endemic in many parts of Africa and Asia (Misra and Singh, 2012). Even though, many clinical and theoretical studies with administrative and
intervention efforts have been made in recent years (see Wand and Liao, 2011; Brauer et al., 2013 and Fitriyani et al., 2020), it remains a public health treat in these continents. For example, (Wang and Liao, 2012) carried out a study of epidemic cholera models and obtained a basic reproduction number by computing the spectral radius of the next generation matrix. They also incorporated the Volterra-Lyapunov matrix theory into Lyapunov functions to facilitate the analysis of the global endemic stability. Their result showed that despite the incorporation of the environmental components, there exists a forward transcritical bifurcation for the combined human-environment epidemiological model under biologically reasonable conditions.

In a similar method (Fitriyani et al. 2020) studied the spread of cholera model in the form of non-linear differential equation systems with four variables SIRB, where they analyzed the local stability of the equilibrium point of the dynamic population by the Routh-Hurwitz stability criterion and bifurcation method. The result of their numerical simulations indicates that the disease-free equilibrium point is locally asymptotically stable and increasing the rate of vaccination and disinfection can reduce the population of susceptible, infected and bacteria of *Vibrio cholerae*. However, the dynamics of transmission of cholera could have more appropriate prevention and control strategies if time delays are incorporated in the logistic equations of epidemic and endemic models. Time delay is responsible for the non-linear behavior of many phenomena in ecological, economical, engineering and other natural systems (Davies and Haas, 2019; Davies, 2006).

The existence of time delay in systems has been known to be a source of poor system performances and even instability; studies involving different time delays can be found in (Davies and Haas, 2019). The influence of time delays on cholera models has been considered by few in recent years in order to accurately describe some characteristics of cholera dynamics (see Wang and Wei, 2013) and independent analysis obtained on providing solution to the health challenge posed by cholera. For example, in (Wang and Wei, 2013), the global dynamics of cholera model having time delay was considered and a basic reproduction number based on the model was found to be the determining factor for its global dynamics by using the Lyapunov functional method. It was established that, when the reproduction number is less than one, the infection-free equilibrium is globally asymptotically stable and the cholera dies out; when the reproduction number is greater than one, the unique endemic equilibrium is globally asymptotically stable and the infection is considered to persist. However, the effect of the delay does not lead to periodic oscillations from their results.

(Misra and Singh, 2012), studied a SIRS model to explore the dynamics of water borne diseases like cholera by incorporating delay in using disinfectants to control the disease. Here, they analyzed the model by looking for the possibility of Hopf bifurcation and the Routh-Hurwitz criterion where they established two equilibria, the disease-free equilibrium and the endemic equilibrium. Their analysis shows that under certain conditions, the cholera disease may be controlled by using disinfectants but a longer delay in their use may destabilize the system.

(Shuai et al., 2012) investigated the stability of a mathematical model for cholera that incorporates hyper-infectivity and temporary immunity with distributed delays by first looking at the existence, uniqueness and continuity of the solution and by using the LaSalle-Lyapunov theorem. Their result show that, the value of the basic reproduction number obtained gives threshold that determines state of the disease; that is, if the reproduction number is greater than one, the unique endemic equilibrium may lose stability.

Motivated by the works in (Wang and Wei 2013; Misra and Singh, 2012; and Shuai et al., 2012), we untangle the role of delays in the dynamics of cholera by studying the effect of time delay in the stability analysis of cholera epidemic-endemic disease model. This will extend the earlier works of (Wang and Wei, 2013), where the delay is added in the dynamics of pathogen *Vibrio cholerae* to the aquatic environment, (Misra and Singh, 2012) where the delay is considered between the
sampling of water for the measurement of density of *Vibrio cholera* and introduction of disinfectants and (Shuai *et al.*, 2012), where the delay is in the differential infectivity since the pathogen was shed to develop a model where the delay is the instant an individual becomes infected and time the symptoms begins to show. The interest here is to investigate the stability property of the complex cholera dynamics in both the epidemic and endemic dynamics where the delay is added the water reservoir using the Routh-Hurwitz criterion and the Rouche’s theorem.

**Model Description and Formulation**

Many mathematical models have been developed for studying problems related to cholera. A mathematical model based on a system of ordinary differential equations for the dynamics of cholera and endemic-epidemic disease incorporating delay was formulated. The assumptions used in the process of modeling the spread of cholera are as follows:

i. The value of the birth rate equals $\Lambda$.

ii. The value of the death rate equals $\mu$.

iii. The two ways of transmission are from human to human and environment to human.

iv. The populations of susceptible individuals increase are due to the natural birth rate at a constant rate of $\Lambda$.

v. The susceptible individuals reduce due to interactions with *Vibrio cholera*, with transmission rate as $\beta_e$ and the interactions with infected individuals, with transmission rate as $\beta_h$.

vi. Infected individuals increase due to interactions between susceptible individual and *Vibrio cholera* and interactions with infected individuals.

vii. Infected individuals reduce due to recovery rate from itself and natural deaths.

We revisit the dynamical model studied in (Wang and Liao, 2012) and introduce a time delay $\tau \geq 0$, which represents the time between the instant in which an individual becomes infected and the instant in which he begins to show symptoms. Thus, we propose a time-delayed model that involves a SIRB (susceptible, infected, recovered, reservoir) and that also considers a class of bacterial concentration in the dynamics of cholera.

Let $N(t)$ be the total human population and is sub-divided into three compartments of susceptible $S(t)$ infected $I(t)$ and recovered $R(t)$. Thus:

$$N(t) = S(t) + I(t) + R(t)$$

The concentration of *Vibrio cholerae* in the environment (contaminated water reservoir) is denoted by $B$. $\mu$ denotes the natural death rate, $\beta_e$ is the rate of ingestion of *Vibrio cholerae* from environment, while $\beta_h$ is the rate of human-to-human transmission. $k$ is the pathogen concentration that yields 50% chance of contracting cholera, $\gamma$ is the rate of recovery from cholera, $\delta$ is the rate of human contribution to *Vibrio cholerae* and $\xi$ is the death rate of *Vibrio cholerae*. Our model for the cholera dynamics can be described by a set of differential equations based on the combination of a regular SIRB model and an environmental component:

$$\frac{dS}{dt} = \Lambda - \beta_e S(t) \frac{B(t)}{k+B(t)} - \beta_h S(t)I(t) - \mu S(t)$$

$$\frac{dI}{dt} = \beta_e S(t) \frac{B(t)}{k+B(t)} + \beta_h S(t)I(t) - \gamma I(t) - \mu I(t)$$  

(2.1)

$$\frac{dR}{dt} = \gamma I(t) - \mu R(t)$$

$$\frac{dB}{dt} = \delta I(t) - \xi B(t)$$
For simplicity, we let $S(t) = S$, $I(t) = I$, $R(t) = R$ and $B(t) = B$, so that our new model system becomes;

**Without Delay**

$$\frac{dS}{dt} = \Lambda - \beta e \frac{B}{k+B} - \beta h I - \mu S$$

$$\frac{dI}{dt} = \beta e \frac{B}{k+B} + \beta h S - \gamma I - \mu I \quad (2.2)$$

$$\frac{dR}{dt} = \gamma I - \mu R$$

$$\frac{dB}{dt} = \delta I - \xi B$$

**With Delay**

$$\frac{dS}{dt} = \Lambda - \beta e \frac{B}{k+B} - \beta h S - \mu S$$

$$\frac{dI}{dt} = \beta e \frac{B}{k+B} + \beta h S - \gamma I - \mu I \quad (2.3)$$

$$\frac{dR}{dt} = \gamma I - \mu R$$

$$\frac{dB}{dt} = \delta I(t - \tau) - \xi B$$

**Model Analysis**

We first establish the well-posedness of model (2.1) by showing that its solutions are non-negative and bounded. Then, we proceed to give the expressions of the disease-free equilibrium and endemic equilibrium points.

**Non-negativity of Solutions**

Since the model monitors human population, we therefore need to show that all the state variables in (2.1) are non-negative at all times for all $t \geq 0$. As in (Lemo-Paiao, 2017), we assume that the initial conditions of system (2.1) are non-negative.

**Lemma 3.1**: Let $\Omega = \{(S, I, R, B) \in \mathbb{R}_+^4 : S(0) > 0, I(0) > 0, R(0) > 0, B(0) > 0\}$ then the solutions of $S(t), I(t), R(t), B(t)$ of the system equations (2.1) – (2.3) are positive for all $t \geq 0$.

**Proof:**

$$\frac{dS}{dt} = \Lambda - \beta e \frac{B}{k+B} - \beta h S - \mu S$$

$$\frac{dI}{dt} \geq -\left(\beta e \frac{B}{k+B} + \beta h I + \mu\right) S$$

$$\log_e S(t) \geq -\left(\beta e \frac{B}{k+B} + \beta h I + \mu\right) t + c_1$$

$$S(t) \geq Ae^{-\left(\beta e \frac{B}{k+B} + \beta h I + \mu\right)t}, \quad \text{where} \ A = e^{c_1}$$

$S(0) = A$, so that,

$$S(t) \geq S(0)e^{-\left(\beta e \frac{B}{k+B} + \beta h I + \mu\right)t} \quad (3.1)$$

Therefore, $S(t) \geq 0$ for all $t \geq 0$.

Similarly,

$$\frac{dI}{dt} = \beta e \frac{B}{k+B} + \beta h S - \gamma I - \mu I$$

$$\frac{dI}{dt} \geq -(\gamma + \mu) I$$

$$\log_e I(t) \geq -(\gamma + \mu)t + c_2$$

$$I(t) \geq Be^{-(\gamma + \mu)t}, \quad \text{where} \ B = e^{c_2}$$
\( I(0) = B \), so that; \( I(t) \geq I(0) e^{-(\gamma + \mu)t} \)  
(3.2)

\( I(t) \geq 0 \) for all \( t \geq 0 \).

Also,

\[
\frac{dR}{dt} = \gamma I - \mu R
\]

\[
\frac{dR}{dt} + \mu R \geq \gamma I
\]

\[
e^{\mu t} \frac{dR}{dt} + e^{\mu t} \mu R(t) \geq e^{\mu t} \gamma I
\]

\[
\frac{d}{dt}(e^{\mu t} R) \geq e^{\mu t} \gamma I
\]

\[
\int \frac{d}{dt}(e^{\mu t} R) dt \geq \int e^{\mu t} \gamma I dt
\]

\[ R(t) \geq \frac{\gamma I}{\mu} + c_3 e^{-\mu t} \]

Therefore,

\[ R(t) \geq \frac{\gamma I}{\mu} + (R(0) - \frac{\gamma I}{\mu}) e^{-\mu t} \geq 0 \]  
(3.3)

Thus,

\[ R(t) \geq 0 \] for all \( t \geq 0 \)

Finally,

\[
\frac{dB}{dt} = \delta I - \xi B
\]

It is therefore proven that all state variables are positive for all time \( t \). The solutions are therefore non-negative for \( t > 0 \).

**Boundedness of the Solutions**

We show that the system of equation (2.1) has solutions which are bounded in the invariant region \( \Omega \) as contained in the feasible region:

\[ \Omega = \{ (S, I, R, B) : N < \frac{A}{\mu} \} \]

**Lemma 3.2:** The solutions of the model are contained in the feasible region

\[ \Omega = \{ (S, I, R, B) \in \mathbb{R}_+^4 : 0 \leq S, 0 \leq I, 0 \leq R, 0 \leq B; S + I + R \leq \frac{A}{\mu} \} \]

**Proof:**

Consider the total population at a time \( t \) given by \( N(t) = S(t) + I(t) + R(t) \).

Then, taking the time derivative of \( N(t) \) from equation (2.2), we have;
\[
\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} \quad (3.5)
\]
\[
\frac{dN}{dt} = \Lambda - \mu S - \mu I - \mu R
\]
\[
\frac{dN}{dt} + \mu N \leq \Lambda
\]
\[
N(t) \leq \frac{\Lambda}{\mu} + c_5 e^{-\mu t}
\]
\[
N(0) - \frac{\Lambda}{\mu} \leq c_5, \quad (3.6)
\]
Thus,
\[
N(t) \leq \frac{\Lambda}{\mu} + (N(0) - \frac{\Lambda}{\mu}) e^{-\mu t} \quad (3.7)
\]
Where \(N(0)\) is the initial population.
As \(t \to \infty\) and taking
\[
\lim_{t \to \infty} N(t) \leq \frac{\Lambda}{\mu} + (N(0) - \frac{\Lambda}{\mu}) e^{-\mu t} \quad (3.8)
\]
Thus; \(0 \leq N < \frac{\Lambda}{\mu}\)
The solutions are therefore bounded in the invariant region \(\Omega\).

**Stability Analysis of the Model**

Equilibrium is defined as a constant solution of a model system. The equilibrium point is a condition where there are no changes in each population over time. At equilibrium points, the rate of change of the model system are obtained by setting the right-hand side of the differential equations to zero and solving each to get a constant solution. These points are also referred to as steady state solutions. Epidemiological models usually have two equilibrium points namely, the disease-free equilibrium and the endemic equilibrium.

**Disease free Equilibrium point (DFE)**

We now analyze the equilibria of the system (2.2) to understand the essentials regarding the transmission dynamics of the disease. This is obtained by setting the model equation to zero since there are no infectious individuals in the population and therefore no disease to recover from. This means that;

\[
\frac{dS}{dt} = \frac{di}{dt} = \frac{dR}{dt} = \frac{dB}{dt} = 0
\]

So that,
\[
\Lambda - \beta eS \frac{B}{k+B} - \beta_h SI - \mu S = 0
\]
\[
\beta eS \frac{B}{k+B} + \beta_h SI - \gamma I - \mu I = 0 \quad (4.1)
\]
\[
\gamma I - \mu R = 0
\]
\[
\delta I - \xi B = 0
\]
\[
\Lambda - 0 - 0 - \mu S = 0
\]
Therefore, \(S = \frac{\Lambda}{\mu}\)

Hence, there exists a disease-free equilibrium point given as;

\[
(S_0, I_0, R_0, B_0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right) \quad (4.2)
\]

**Endemic Equilibrium point (EE)**

We now analyze the endemic equilibrium of the system (2.1). This is obtained by setting the model equation to zero. Thus;
\[
\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = \frac{dB}{dt} = 0
\]

So that,
\[
\Lambda - \beta_e S \frac{B}{k+B} - \beta_h SI - \mu S = 0
\]
\[
\beta_e S \frac{B}{k+B} + \beta_h SI - \gamma I - \mu I = 0 \quad (4.3)
\]
\[
\gamma I - \mu R = 0
\]
\[
\delta I - \xi B = 0
\]

Hence, the endemic equilibrium point is given as;
\[
S^* = \frac{\Lambda(B+k)}{B\mu + k\mu + B\beta_e + B\beta_h + k\beta_h}
\]
\[
I^* = \frac{SB\beta_e}{(k+B)(\gamma+\mu-S\beta_h)} \quad (4.4)
\]
\[
R^* = \frac{\gamma I}{\mu}
\]
\[
B^* = \frac{\delta(t-\tau)}{\xi}
\]
\[
E^* = (S^*, I^*, R^*, B^*) \quad (4.5)
\]

**Basic Reproduction Number**

The basic reproduction number denoted by \( R_0 \) is the average number of secondary infections caused by an infectious individual during his/her entire period of infectiousness. The \( R_0 \) is an important quantity in epidemiology as it sets the threshold in the study of a disease both for predicting its outbreak and for evaluating its control strategies. Thus, whether a disease becomes persistent or dies out in an environment depends on the value of the reproduction number \( R_0 \).

Therefore, the basic reproduction number is gotten using the Next Generation Matrix approach described in (Van den Driessche and Watmough, 2002).

Let’s consider the matrix \( H \), consisting of two \( n \times n \) matrices \( F \) and \( V \) such that;
\[
H = FV^{-1}
\]

And
\[
F = \left[ \frac{\partial F_i(E_0)}{\partial x_j} \right], \quad \text{and}
\]
\[
V = \left[ \frac{\partial V_i(E_0)}{\partial x_j} \right] \quad (4.6)
\]

The basic reproduction number \( R_0 \) is therefore, given as the dominant eigen-value or the spectral radius of matrix \( H \).

Thus, \( R_0 = \| FV^{-1} \| \) \quad (4.7)

We obtain matrix \( F \) and \( V \) at DFE as;
\[
F = \left[ \begin{array}{cc}
\beta_e & \Lambda \\
0 & \mu
\end{array} \right], \quad V = \left[ \begin{array}{ccc}
\gamma + \mu & 0 \\
\delta & -\xi
\end{array} \right]
\]

So that the inverse of matrix \( V \) can be obtained as;
\[
V^{-1} = \frac{1}{-\gamma+\mu} \left[ \begin{array}{cc}
-\xi & 0 \\
-\delta & \gamma + \mu
\end{array} \right] = \left[ \begin{array}{cc}
\frac{1}{\gamma+\mu} & 0 \\
\delta & \frac{1}{\gamma+\mu} \xi
\end{array} \right]
\]

Next, we obtain \( FV^{-1} \) as;
\[ FV^{-1} = \begin{bmatrix} \beta_h \frac{A}{\mu} & \beta_e \left( \frac{A}{\mu k} \right) \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{(\gamma+\mu)\delta} & 0 \\ \frac{\delta}{(\gamma+\mu)\xi} & -\frac{1}{\xi} \end{bmatrix} \]

\[ FV^{-1} = \begin{bmatrix} \left( \beta_h \frac{A}{\mu} \right) \left( \frac{1}{\gamma+\mu} \right) + \left( \beta_e \frac{A}{\mu k} \right) \left( \frac{\delta}{(\gamma+\mu)\xi} \right) & \left( \beta_e \frac{A}{\mu k} \right) \left( \frac{1}{\xi} \right) \\ 0 & 0 \end{bmatrix} \]  

(4.8)

Furthermore, the largest or dominant eigenvalue of matrix \( FV^{-1} \) gives the basic reproduction number \( R_0 = \frac{\lambda}{bFV^{-1}} \). Therefore,

\[ R_0 = \frac{\lambda}{\mu(\gamma+\mu)} \left( \beta_h + \beta_e \frac{\delta}{\xi k} \right) \]  

(4.9)

Local Stability of the Disease-Free Equilibrium

Here we investigate the local stability of the Disease-Free Equilibrium point \( E_0(S_0, I_0, B_0) \).

**Theorem 4.1:** The Disease-Free Equilibrium is locally asymptotically stable if \( R_0 < 1 \) and is unstable if \( R_0 > 1 \).

**Proof:**

The Jacobian matrix of model system (2.2) can be obtained as;

\[ U = \begin{bmatrix} -\mu & \beta_h \frac{A}{\mu} & 0 & -\beta_e \frac{A}{\mu k} \\ 0 & \beta_h \frac{A}{\mu} - \gamma - \mu & 0 & \beta_e \frac{A}{\mu k} \\ 0 & \gamma & -\mu & 0 \\ 0 & \delta & 0 & -\xi \end{bmatrix} \]

By solving for the eigenvalues of \( U \) we get the characteristics equation as;

\[ |U - I\lambda| = \begin{vmatrix} -\mu & \beta_h \frac{A}{\mu} & 0 & -\beta_e \frac{A}{\mu k} \\ 0 & \beta_h \frac{A}{\mu} - \gamma - \mu & 0 & \beta_e \frac{A}{\mu k} \\ 0 & \gamma & -\mu & 0 \\ 0 & \delta & 0 & -\xi \end{vmatrix} - \begin{bmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{bmatrix} \lambda \]

\[ = \begin{vmatrix} -(\mu + \lambda) & \beta_h \frac{A}{\mu} & 0 & -\beta_e \frac{A}{\mu k} \\ 0 & \beta_h \frac{A}{\mu} - \gamma - \mu - \lambda & 0 & \beta_e \frac{A}{\mu k} \\ 0 & \gamma & -(\mu + \lambda) & 0 \\ 0 & \delta & 0 & -(-\xi + \lambda) \end{vmatrix} \]

\[ |U - I\lambda| = (-\mu - \lambda)(-\mu - \lambda) \begin{vmatrix} \beta_h \frac{A}{\mu} - \gamma - \lambda & \beta_e \frac{A}{\mu k} \\ \delta & -(\xi + \lambda) \end{vmatrix} \]  

(4.10)

From equation (4.10), we easily observe that \( \lambda_1 = \lambda_2 = -\mu \). We then proceed to find the other eigenvalues of the reduced block matrix given by;

\[ A = \begin{bmatrix} \beta_h \frac{A}{\mu} - \gamma - \mu & \beta_e \frac{A}{\mu k} \\ \delta & -\xi \end{bmatrix} \]  

(4.11)
Let $Tr$ be the Trace of $A$ and $\alpha$ be the determinant of $A$ and considering the linear system $x'(t) = Ax(t)$, the following conditions can be shown;

a) If $\alpha < 0$, the characteristic roots of $A$ will have opposite signs.

b) If $\alpha > 0$ and $\Delta = Tr^2 - 4\alpha \geq 0$, the characteristic roots of matrix $A$ will have the same sign. The roots will be negative if $Tr < 0$ and positive if $Tr > 0$.

c) If $\alpha > 0$, $\Delta < 0$ and $Tr \neq 0$ then, the characteristic roots of matrix $A$ will be imaginary with negative real part if $Tr < 0$ and a positive real part if $Tr > 0$.

d) If $\alpha > 0$ and $Tr = 0$ then, matrix $A$ will have purely imaginary roots.

The eigenvalues of matrix $A$ are obtained from the characteristic equation:

$$\lambda^2 - (\alpha + d)\lambda + (ad + bc) = 0$$

$$\lambda^2 - Tr\lambda + \alpha = 0$$

$$\lambda = \frac{Tr \pm \sqrt{Tr^2 - 4\alpha}}{2}$$

(4.12)

Thus,

a*) If $\alpha < 0$, there exist two real Eigen values of opposite signs.

b*) If $\alpha > 0$ and $\Delta \geq 0$, there exist two real eigenvalues of the same sign as the Trace.

c*) If $\alpha > 0$, $\Delta < 0$ and $Tr \neq 0$, there exist two complex conjugate eigenvalues $\lambda = p \pm ir$.

d*) If $\alpha > 0$ and $Tr = 0$, there exist two purely imaginary complex conjugate eigenvalues.

Now considering condition (b), we can therefore determine the signs of the other eigenvalues. For the remaining two eigenvalues to be negative then, $\alpha > 0$ and $Tr < 0$. We now proceed to find the conditions that makes the determinant positive and the Trace negative. From the reduced block matrix, the determinant is given by;

$$\alpha = -\xi \left( \beta_h \frac{\Lambda}{\mu} - \gamma - \mu \right) - \delta \left( \beta_e \frac{\Lambda}{\mu_k} \right)$$

(4.13)

But for the determinant to be positive, we let;

$$\xi \left( \beta_h \frac{\Lambda}{\mu} \right) + \delta \left( \beta_e \frac{\Lambda}{\mu_k} \right) < (\gamma + \mu)\xi$$

(4.14)

$$\frac{\Lambda}{\mu(\gamma + \mu)} \left( \beta_h + \frac{\beta_e\delta}{\xi_k} \right) < 1$$

(4.15)

Then, equation (4.15) becomes;

$R_0 < 1$.

Also, the Trace of the reduced block matrix is given by;

$$Tr = \beta_h \frac{\Lambda}{\mu} - \xi - (\gamma + \mu)$$

(4.16)

If we make $(\gamma + \mu)$ the subject of formula from equation (4.9) we get;

$$(\gamma + \mu) = \frac{\Lambda}{\mu R_0} \left( \beta_h + \frac{\beta_e\delta}{\xi_k} \right)$$

(4.17)

By substituting equation (4.17) into equation (4.16) gives;

$$Tr = \beta_h \frac{\Lambda}{\mu} - \xi - \frac{\Lambda}{\mu R_0} \left( \beta_h + \frac{\beta_e\delta}{\xi_k} \right)$$

(4.18)

The Trace of the reduced matrix needs to have negative Eigen values. Since all the other parameters are negative, we find the condition that makes $\beta_h \frac{\Lambda}{\mu}$ to be negative.
\[ \beta_h \frac{A}{\mu} - \frac{A}{\mu R_0} \beta_h = 0 \]  

(4.19)

Simplifying equation (4.19) gives;

\[ \beta_h \frac{A}{\mu} \left(1 - \frac{1}{R_0}\right) < 0 \]  

(4.20)

From equation (4.20) we see that if \( R_0 < 1 \) then,  
the equation becomes negative. Hence, the 
Jacobian matrix of the disease-free equilibrium  
has negative eigenvalues only when  
\( R_0 < 1 \) which implies that the disease-free equilibrium is  
locally asymptotically stable. The result of the  
thorem also confirms the conditions for local  
stability as outlined in (Van den Driessche and  
Watmough, 2002). In terms of the disease  
spread, this means that if there is a small  
perturbation on the system, the system will still  
return to the disease-free equilibrium.

**Stability of Delayed Model**

Consider the following co-ordinate transformation; 
\( x_1(t) = S(t), x_2(t) = I(t), x_3(t) = R(t), x_4(t) = B(t) \) where  
\( (S, I, R, B) \) denotes the equilibrium points of  
equations (2.3). The linearized system of (2.3)  
takes the form;

\[ X(t) = A_0 X(t) + A_1 X(t - \tau). \]  

(4.21)

Where \( X = (x_1, x_2, x_3, x_4)^T \)

\[
A_0 = \begin{bmatrix}
-\beta_{e,k+B} - \beta_h I - \mu & \beta_h S & 0 & -\beta_e \frac{S}{K} \\
\beta_{e,k+B} & \beta_h I & \beta_h \Lambda_I - \gamma - \mu & 0 \\
0 & \gamma & -\mu & 0 \\
0 & \delta & 0 & -\xi
\end{bmatrix}
\]

\[
A_1 = \begin{bmatrix}
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 \\
0 & \delta & 0 & 0
\end{bmatrix}
\]

The characteristic equation becomes

\[ \Delta(y) = |yI_{4x4} - A_0 - A_1 e^{-\tau y}| \]

From above, we have;

\[ \Delta(y) = \begin{bmatrix}
y & 0 & 0 & 0 \\
0 & y & 0 & 0 \\
0 & 0 & y & 0 \\
0 & 0 & 0 & y
\end{bmatrix} - \begin{bmatrix}
-\beta_{e,k+B} & -\beta_h I - \mu & \beta_h S & 0 & -\beta_e \frac{S}{K} \\
\beta_e \frac{B}{k+B} + \beta_h I & \beta_h \Lambda_I - \gamma - \mu & 0 & \beta_e \frac{S}{K} \\
0 & \gamma & -\mu & 0 \\
0 & \delta & 0 & -\xi
\end{bmatrix} \]

Evaluating \( \Delta(y) \) at DFE yields;

\[ \Delta(y) = \begin{bmatrix}
y + \mu & -\beta_h \Lambda_I & 0 & \beta_e \Lambda \mu^{-k} \\
0 & y - \beta_h \Lambda_I + \gamma + \mu & 0 & -\beta_e \Lambda \mu^{-k} \\
0 & -\gamma & y + \mu & 0 \\
0 & -\delta + \delta e^{-\tau y} & 0 & y + \xi
\end{bmatrix} \]  

(4.22)

**Theorem 2:** if \( R_0 < 1 \), then the disease-free  
equilibrium \( E_0 \) is locally asymptotically stable for  
any time-delay \( \tau \geq 0 \). If \( R_0 > 1 \), then the  
disease-free equilibrium is unstable for any time-  
delay \( \tau \geq 0 \).

Proof:

The characteristic equation at the disease-free  
equilibrium is given by;

\[ P(y, \tau) = (y + \mu)^2 \left( y^2 + \left( \gamma + \mu + \xi - \beta_h \Lambda \mu^{-k} \right) y + \gamma_1 + \gamma_2 \right) = 0 \]  

(4.23)

Where \( \gamma_1 = \delta \beta_e \Lambda \mu^{-k} e^{-\tau y}, \quad \gamma_2 = \xi (y + \mu) - \xi \beta_h \Lambda \mu^{-k} - \delta \beta_e \Lambda \mu^{-k} \xi \).
Case 1: Let $\tau = 0$

Then, equation (4.23) becomes;

$$P(y, 0) = (y + \mu)^2 \left(y^2 + (y + \mu + \xi - \beta_h \frac{A}{\mu}) + \delta \beta_e \frac{A}{\mu} + \gamma_2\right) = 0 \quad (4.24)$$

We need to prove that all roots of the characteristic equation have negative real parts. It is quite easy to see from (4.24) that $y_1 = y_2 = -\mu$ are roots of (4.24) and are all negative. Thus, we just need to analyze the second term of (4.24), here denoted by $P_1$.

Thus,

$$P_1(y, 0) = \left(y^2 + (y + \mu + \xi - \beta_h \frac{A}{\mu})\right) + \delta \beta_e \frac{A}{\mu} + \gamma_2 = 0$$

Using the Routh-Hurwitz criterion, we know that all roots of $P_1(y, 0)$ have negative real parts if and only if the co-efficient of $P_1(y, 0)$ are strictly positive. In this case, we have $\left(y + \mu + \xi - \beta_h \frac{A}{\mu}\right) > 0$ if and only if $\gamma + \mu + \xi > \beta_h \frac{A}{\mu}$ and $\gamma_2 = \xi(y + \mu) - \xi(\beta_h \frac{A}{\mu} - \delta \beta e \frac{A}{\mu} k)$ if and only if $R_0 < 1$, since;

$$\xi(\beta h \frac{A}{\mu} - \delta \beta e \frac{A}{\mu} k) = \frac{\Lambda}{\mu(y + \mu)} \left(\beta h - \beta e \frac{\delta}{\xi k}\right) \xi(y + \mu) = R_0(\xi(y + \mu))$$

This implies that;

$$\gamma_2 = \xi(y + \mu) - R_0(\xi(y + \mu))$$

Clearly, $\gamma_2$ is positive only if $R_0 < 1$.

Case 2: Let $\tau > 0$

We use Rouche’s theorem to prove that all roots of the characteristic equation (4.23) cannot intersect the imaginary axis i.e the characteristics equation cannot have pure imaginary roots. Suppose the contrary, that is, suppose there exist $\omega \in \mathbb{R}$ such that $y = \omega i$ is a solution of equation (4.24). Replacing $y = \omega i$ in the second term of equation (4.23), we get;

$$-\omega^2 + \left(y + \mu + \xi - \beta_h \frac{A}{\mu}\right) \omega i + \left(\xi(y + \mu) - \left(\xi(\beta h \frac{A}{\mu} + \delta \beta e \frac{A}{\mu} k)\right) + \delta \beta e \frac{A}{\mu} k \cos(\tau \omega) - i \sin(\tau \omega)\right) = 0$$

Let;

$$\gamma_0 = \left(y + \mu + \xi - \beta_h \frac{A}{\mu}\right),$$

$$\gamma_1 = \delta \beta e \frac{A}{\mu} e^{-\tau y},$$

$$\gamma_2 = \xi(y + \mu) - \left(\xi(\beta h \frac{A}{\mu} + \delta \beta e \frac{A}{\mu} k)\right)$$

So that we get;

$$-\omega^2 + \gamma_0 \omega i + \gamma_2 + \gamma_1(\cos(\tau \omega) - i \sin(\tau \omega)) = 0$$

Separating the real and imaginary parts gives;

$$\left\{-\omega^2 + \gamma_2 = -\gamma_1 \cos(\tau \omega)\right\}$$

Clearly, $\gamma_2$ is positive only if $R_0 < 1$. 

By adding up the squares of both equations and using the fundamental trigonometric formula, we obtain:

$$\omega^4 + (\tau_0^2 - 2\tau_2)\omega^2 + \tau_2^2 - \tau_1^2 = 0$$  \hspace{1cm} (4.25)$$

Then,

$$\omega^2 = \frac{1}{2} \left[ -(\tau_0^2 - 2\tau_2) \pm \sqrt{(\tau_0^2 - 2\tau_2)^2 - 4(\tau_2^2 - \tau_1^2)} \right]$$

This implies that equation (4.25) has no positive roots when $R_0 < 1$ which shows that equation (4.23) has no imaginary roots for all $\tau > 0$. Hence, the DFE is locally asymptotically stable for any $\tau > 0$.

**Results**

In this section, we present some results to illustrate our analysis on the dynamics of the cholera model.

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**Figure 1. Dynamics of Susceptible Population with and without Delay**

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**Figure 2. Dynamics of Infectious Population with and without Delay**
Numerical simulation was performed on the system of differential equation of the model parameters to see the dynamics of the population of susceptible, infectious and recovered in the system. This was done to see how the population of the susceptible, infectious and the recovered change with time. The variation of infective human population and recovered population with respect to time for different values of rate of contamination of the susceptible population is zero at DFE. Therefore, the steady state solutions of the DFE depict that only the susceptible class will remain active at this point. The numerical simulations of the system in fig. 1–4 support the claim in the theoretical or qualitative analysis of the model. The endemic equilibrium of the delayed epidemic model is locally asymptotically stable. Comparing the system with and without delay, it can be seen that the proportion of susceptible population is higher in the delayed system. The analysis of fig. 1 clearly shows stability at $R_0 < 1$ for this combined human-environment epidemiological model. At DFE, there is stability for the susceptible population at 1000. The human population at this point remains unchanged and unaffected by any external factor. The rate of the infectious population as seen in fig. 2b shows the infected population declining at $t < 15$, thereafter remained stable at $t > 20$ without delay. While on the other hand in fig. 2a, after the delay ($\tau$) was introduced, the system was observed to be unstable at $t > 50$ but gradually attains stability at $t > 80$. fig. 3 represents the recovered population of the system. Clearly, we see that the delay in the system has further increased the recovery time of the infected persons. Thus, the system is
strongly imparted with the introduction of the delay ($\tau$) term and the rate of recovery tends to be faster in the system without delay. Considering the presence of \textit{Vibrio cholerae} in the system, fig. 4b shows that the cholera concentration in the population of the system gradually reduces with respect to time and attains stability at $t > 20$ given the absence of delay ($\tau$). However, with the introduction of the delay ($\tau$) term in the system, the cholera concentration in the system as seen in fig. 4a becomes unstable for a longer period of time and attains stability at $t > 60$. Thus, the introduction of delay ($\tau$) in the system strongly imparts the cholera concentration in the population.

**Conclusion**

The paper is devoted to the analysis of a time-delayed mathematical model for cholera. The considered model is a SIRB (Susceptible, Infectious, Recovered, Cholera Concentration) system, where an additional class B is considered: a class of bacterial concentration in the dynamics of cholera. The host population is divided into four classes: susceptible, infectious with symptoms, recovered and an additional compartment reflecting the bacterial concentration. The formulated model is analyzed, providing the non-negativity of the solutions for non-negative initial conditions, as well as the disease-free equilibrium, basic reproduction number, and endemic equilibrium. For positive time delays, the stability of the equilibrium points is also analyzed. We proved the positivity of the solutions of the delayed SIRB model and analyzed the local asymptotic stability of the equilibrium points. Our analysis shows that the ingestion rate of the bacteria through contaminated sources $\beta$ has an important influence on the stability of the endemic equilibrium and the endemic equilibrium of the system is locally asymptotically stable whenever $\tau$ is suitably small. Analysis of the model shows that there exists a region where the model is mathematically and epidemiologically well-posed because its solutions were positive and bounded. Computation of the basic reproduction number, which was the threshold parameter, was done using the next generation matrix approach. It was determined that when $R_0 < 1$, cholera does not spread. Stability analysis of the cholera model showed that the disease-free equilibrium is locally asymptotically stable when the basic reproduction number is less than unity. This shows that when $R_0 > 1$, the disease persists and spreads in the population.

**References**


