Stability Analysis of the Vector-Host Epidemic Model for Cholera

Sukanya Isuntier and Ekkachai Kunnawuttipreechachan

***Abstract*—A mathematical model of cholera disease is a valuable tool for studying the dynamics and simulating effects of possible treatment. In this work, we analyze the cholera disease model with both vector and host populations. The purpose of this project is to thoroughly investigate stability properties of the models equilibria related to the basic reproduction number, $\mathcal{R}_0$. The Lyapunov function method is applied to find the conditions in which the disease-free equilibrium is asymptotically stable. The result shows that if $\mathcal{R}_0$ is less than unity, then the cholera disease will eventually die out. Moreover, the models parameters could be controlled in order to prevent the further spread of a cholera outbreak.**

***Index Terms*—Equilibrium, Basic Reproduction Number, Lyapunov Function, Stability Analysis.**

I. Introduction

CHOLERA is one of many vector-borne diseases, causing extreme diarrhea that can eventually lead to death because of a massive loss of bodily fluids. 80% of cholera cases today can be prevented by the ingestion of rehydration salts. In the past, people believed that the disease was caused by miasma; a noxious form of "bad air", but we now know that the true cause of the disease is by a strain of bacteria called Vibrio cholera. This bacterium is transmitted through the intake of contaminated food and water. The bacterium can possibly infect and develop into developed cholera infection in as little as two hours. Researchers have estimated that each year there are 1.3 to 4.0 million cases of cholera, and 1.6% to 3.5% deaths worldwide [1].

The first outbreak (1817-1823) was in Kishnagur, India. The second outbreak, (1826-1837) spread from India, across Asia and Europe, and by 1834 had even reached most major cities in the United States and Canada. The third and fourth outbreaks (1846-1863 and 1865-1875 respectively) spread much like the second outbreak in India but with much more aggressive virulence. In 1854, during the third outbreak, 23,000 people in England and Wales were killed by the disease and even more in southern Europe [2].

During the 19th and early 20th centuries a total of six cholera pandemics occurred, ending in 1923. The seventh outbreak affecting mostly the southern hemisphere, began in South Asia (1961), and reached Africa (1971) and the Americas (1991). Recent years have shown cholera outbreaks in developing countries, including Haiti (2010-2011), Cameroon (2010-2011), Kenya (2010), Vietnam (2009), Zimbabwe (2008-2009), Iraq (2008), the Democratic Republic of Congo (2008) and India (2007). Due to its huge impact on public health, cholera has been the subject of extensive studies in clinical, experimental and theoretical fields. It remains an important global cause of illness and mortality, capable of causing periodic epidemic disease outbreaks. Cholera is an example of a bacterial disease whose primary mode of infection is indirect, which is caused when individuals ingest fecal-contaminated water containing the bacteria V. cholera. Transmission between humans and reservoirs of pathogens implies that disease transmission includes an indirect route rather than human-to-human contacts and continues to this day [3], [4].

There are many research based studies and published literature about cholera. This model was rigorously analyzed in [5]. Joh, et al. [6] in 2009: Modified Codecos model; by a threshold pathogen density for infection with a careful focus on human-environment contact and in-reservoir pathogen dynamics. This model was meticulously investigated in a proposed model cholera outbreak in Zimbabwe study in $2008-2009$. It took into consideration both human-to-human and environment-to-human transmission pathways. The results in this work are of great importance in regards to the human-to-human transmission in cholera epidemics, Mukandavire et al [7].

In 2010 Tien and Earn [8] published a water-borne disease model, which also included the dual transmission pathways. A rigorous global stability analysis was conducted in [9] many of the afore-mentioned models. In addition, in 2010, Neilan et al. [10] modified the cholera model proposed by Hartley, Morris and Smith [7] and added several control measures into the model. They consequently analyzed the optimal intervention strategies and conducted numerical simulations based on their model. In 2011 Abid Ali Lashari and Gul Zamanb [11] published "Global dynamics of vector-borne diseases with a horizontal transmission in host population", they extended the model of Cai and Li [12] to include exposed individuals, disease induced death rate and time dependent total population size in both host and vector population.

In this work, we modify the model of Lashari and Zaman [11] cholera model. Our objective is to formulate and analyze a model for cholera that takes into consideration both human and vector transmission pathways as the SEIR model and the SI model respectively. This model was meticulously investigated in a proposed model cholera outbreak in Zimbabwe study in 2008-2009 [7]. It took into consideration both human-to-human and environment-to-human transmission pathways. The results in this work are of great importance in regards to the human-to-human transmission in cholera epidemics.

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II. Model formulation

In this section, we consider the chorela model with two groups, host and vector populations are described by SEIR and SI model respectively.

$N_h(t)$	total host population size at time t
$S_h(t)$	Number of susceptible hosts at time t
$E_h(t)$	Number of exposed hosts at time t
$I_h(t)$	Number of infections hosts at time t
$R_h(t)$	Number of recovered hosts at time t
$N_v(t)$	total vector population size at time t
$S_v(t)$	Number of susceptible vectors at time t
$I_v(t)$	Number of infections vectors at time t

In this diagram can represent the relation of each subclasses.

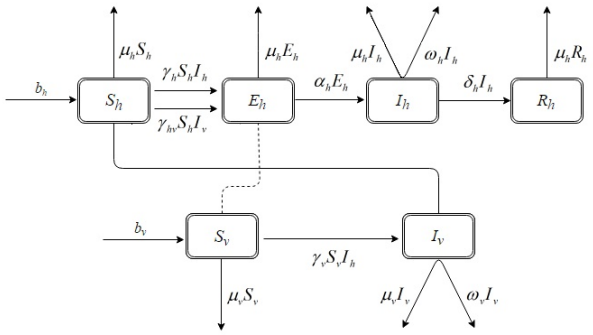

Fig. 1. The flow chart represents the interaction and transfer diagram of both human and vector.

The definitions of the non-negative parameters are given:

TABLE I
Definitions of the model's parameters

Parameters	Meaning	Unit
b_h	recruited rate of the host population	individual $\cdot$ time^{-1}
b_v	recruited rate of the vector population	individual $\cdot$ time^{-1}
μ_h	natural death rate of the host population	time^{-1}
μ_v	natural death rate of the vector population	time^{-1}
γ_h	rate of direct transmission	individual^{-1} $\cdot$ time^{-1}
γ_{hv}	rate of carrying pathogen infectious vector	individual^{-1} $\cdot$ time^{-1}
γ_v	rate of infective vector	individual^{-1} $\cdot$ time^{-1}
α_h	rate of host populations from E_h to I_h	time^{-1}
ω_h	disease-related death of host population	time^{-1}
ω_v	disease-related death of vector population	time^{-1}
δ_h	recovery rate of host population	time^{-1}

Where the $N_h = S_h + E_h + I_h + R_h$, $N_v = S_v + I_v$. We assume that vertical transmission in the host population does not occur so that all newly recruited individuals are susceptible. The recovered individuals are assumed to acquire permanent immunity and there is no transfer from the R class back to the S class. Susceptible hosts can get infected via two routes of transmission, directly denote the rate as γ_h, through a contact with an infected individual, and indirectly denote the rate as γ_{hv}.

Furthermore, we assume that all newborn vector population are susceptible and vertical transmission can be unconsidered. Susceptible vectors start carrying the pathogen after getting into contact an infective host.

The dynamics of this class of infectious disease can be described by the following system of nonlinear differential equations.

$$\begin{aligned}
\frac{dS_h}{dt} &= b_h - \mu_h S_h - \gamma_h S_h I_h - \gamma_{hv} S_h I_v, \\
\frac{dE_h}{dt} &= \gamma_h S_h I_h + \gamma_{hv} S_h I_v - \mu_h E_h - \alpha_h E_h, \\
\frac{dI_h}{dt} &= \alpha_h E_h - \delta_h I_h - \mu_h I_h - \omega_h I_h, \\
\frac{dR_h}{dt} &= \delta_h I_h - \mu_h R_h, \\
\frac{dS_v}{dt} &= b_v - \mu_v S_v - \gamma_v S_v I_h, \\
\frac{dI_v}{dt} &= \gamma_v S_v I_h - \mu_v I_v - \omega_v I_v.
\end{aligned} \tag{1}$$

For some reasonable biological assumptions on the parameters of the model, we assumed that all parameters are positive. The following lemma for the suitable feasible region Ω is Let $(S_h, E_h, I_h, R_h, S_v, I_v)$ be the solution of (1) with the initial conditions

$$S_h(0) \geq 0, E_h(0) \geq 0, I_h(0) \geq 0, R_h(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0.$$

The feasible region Ω is defined by

$$\begin{aligned}
\Omega &= \{(S_h, E_h, I_h, R_h, S_v, I_v) \in \mathbb{R}^6_+ | S_h + E_h + I_h + R_h \leq \frac{b_h}{\mu_h}, \\
&\qquad S_v + I_v \leq \frac{b_v}{\mu_v}\}.
\end{aligned}$$

is positively invariant set for the system (1)

III. The equilibria and their stability

In the model we find the disease-free equilibrium point and the endemic equilibrium point. We analyze the basic reproduction numbers ($\mathcal{R}_0$) of the model by the using estimation method. We analyze the global stability by using the Lyapunov function at the disease-free equilibrium point.

A. The existence of equilibria

For simplicity, the first third and fith-sixth in (1) are independent with the variable $R_h(t)$ Therefore, the system (1) can be reduced to the following system of equations

$$\begin{aligned}
\frac{dS_h}{dt} &= b_h - \mu_h S_h - \gamma_h S_h I_h - \gamma_{hv} S_h I_v, \\
\frac{dE_h}{dt} &= \gamma_h S_h I_h + \gamma_{hv} S_h I_v - A_1 E_h, \\
\frac{dI_h}{dt} &= \alpha_h E_h - A_2 I_h, \\
\frac{dS_v}{dt} &= b_v - \mu_v S_v - \gamma_v S_v I_h, \\
\frac{dI_v}{dt} &= \gamma_v S_v I_h - A_3 I_v,
\end{aligned} \tag{2}$$

where $A_1 = \alpha_h + \mu_h$, $A_2 = \omega_h + \delta_h + \mu_h$ and $A_3 = \omega_v + \mu_v$. The equilibrium points of system (2) are given by

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the constant solutions of the algebraic system obtained by setting the derivatives equal zero. To find these solutions, We write all possible variables in terms of I_h. So we have disease free euilibium point E_0 when $I_h^* = 0$

$$E_0 = (S_h^0, 0, 0, S_v^0, 0), \text{where } S_h^0 = \frac{b_h}{\mu_h}, S_v^0 = \frac{b_v}{\mu_v}. \quad (3)$$

If $I_h^* \neq 0$ there exists a unique endemic equilibrium $E_1 = (S_h^*, E_h^*, I_h^*, S_v^*, I_v^*)$ in the interior of Ω given by

$$S_h^* = \frac{I_h^* A_1 A_2 - \alpha_h b_h}{\alpha_h \mu_h}, \quad E_h^* = \frac{S_h^*(I_h^* \gamma_h + I_v^* \gamma_{hv})}{A_1}, \quad (4)$$

$$S_v^* = \frac{b_v}{I_h^* \gamma_v + \mu_v}, \quad I_v^* = \frac{I_h^* b_v * \gamma_v}{(I_h^* \gamma_v + \mu_v) A_3}. \quad (5)$$

the following quadratic equation:

$$f(I_h) = a_1 I_h^2 + a_2 I_h + a_3 = 0, \quad (6)$$

where

$$\begin{aligned} a_1 &= A_1 A_2 A_3 \gamma_h \gamma_v, \\ a_2 &= A_1 A_2 A_3 (\gamma_h \mu_v + \gamma_v \mu_h) + A_1 A_2 b_h \gamma_{hv} \gamma_v \\ &\quad - A_3 \gamma_h \gamma_v b_h \alpha_h, \\ a_3 &= A_1 A_2 A_3 \mu_h \mu_v (1 - \mathcal{R}_0). \end{aligned}$$

Let $\mathcal{R}_0 = \frac{b_h}{\mu_h}(\frac{b_v \gamma_{hv} \gamma_v \alpha_h}{\mu_v A_1 A_2 A_3} + \frac{\gamma_h \alpha_h}{A_1 A_2})$ [13] If $\mathcal{R}_0 > 1$, then $a_3 < 0$, and (6) has a unique positive root.

B. Locally stability of the disease-free equilibrium

In this section, we check local stability of the disease-free equilibrium point by showing that the real parts of eigenvalues of the Jacobian matrix at the E_0 are negative for $\mathcal{R}_0 < 1$ and at least one real part is positive for $\mathcal{R}_0 > 1$. These conditions on the eigenvalues of the Jacobian follow from the linearization test for local stability of the disease-free equilibrium point.

Theorem 1: If $\mathcal{R}_0 < 1$, the disease-free equilibrium of system (2) is locally asymptotically stable and is unstable if $\mathcal{R}_0 > 1$.

Proof: We linearize system (2) around the disease-free equilibrium E_0. The matrix of the linearization at E_0 is given by

$$J_0^* = \begin{bmatrix} -\mu_h & 0 & -\frac{\gamma_h b_h}{\mu_h} & 0 & -\frac{\gamma_{hv} b_h}{\mu_h} \\ 0 & -A_1 & \frac{\gamma_h b_h}{\mu_h} & 0 & \frac{\gamma_{hv} b_h}{\mu_h} \\ 0 & \alpha_h & -A_2 & 0 & 0 \\ 0 & 0 & -\frac{\gamma_v b_v}{\mu_v} & -\mu_v & 0 \\ 0 & 0 & \frac{\gamma_v b_v}{\mu_v} & 0 & -A_3 \end{bmatrix} \quad (7)$$

The characteristic equation of (7) is

$$(\lambda + \mu_h)(\lambda + \mu_v)(\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3) = 0 \quad (8)$$

where

$$\begin{aligned} a_1 &= A_1 + A_2 + A_3, \\ a_2 &= A_1 A_2 + A_1 A_3 + A_2 A_3 - \frac{\gamma_h b_h \alpha_h}{\mu_h}, \quad (9) \\ a_3 &= A_1 A_2 A_3 (1 - R_0) \end{aligned}$$

we can see two of the eigenvalues of J_0^* are $-\mu_h$ and $-\mu_v$ have negative real parts. The remaining 3 eigenvalues also have negative real parts if satisfy the Routh-Hurwitz criterion. The disease-free equilibrium point will be local asymptotically stable if $a_1 > 0$, $a_3 > 0$ and $a_1 a_2 > a_3$ if $R_0 < 1$. The disease-free equilibrium is unstable is $R_0 > 1$.

C. Locally stability of the endemic equilibrium

In section, we show that the real parts of eigenvalues of the Jacobian matrix at endemic equilibrium point that exist when $\mathcal{R}_0 > 1$ are negative for for all positive parameters. These conditions on the eigenvalues of the Jacobian follow from the linearization test for local stability of the endemic equilibrium point.

Theorem 2: If $\mathcal{R}_0 > 1$, the endemic equilibrium of system (2) is locally asymptotically stable.

Proof: We first find the Jacobian matrix at E_1 as follows

$$J_1^* = \begin{bmatrix} -B_1 - \mu_h & 0 & B_2 & 0 & B_3 \\ B_1 & -A_1 & -B_2 & 0 & -B_3 \\ 0 & \alpha_h & -A_2 & 0 & 0 \\ 0 & 0 & -B_4 & -I_h \gamma_v - \mu_v & 0 \\ 0 & 0 & B_4 & -I_h \gamma_v & -A_3 \end{bmatrix} \quad (10)$$

where

$$\begin{aligned} B_1 &= I_h \gamma_h + \frac{I_h b_v \gamma_{hv} \gamma_v}{(I_h \gamma_v + \mu_v) A_3}, \quad B_2 = \gamma_h \frac{(I_h^* A_1 A_2 - \alpha_h b_h)}{\alpha_h \mu_h}, \\ B_3 &= \gamma_{hv} \frac{(I_h^* A_1 A_2 - \alpha_h b_h)}{\alpha_h \mu_h}, \quad B_4 = \frac{b_v \gamma_v}{I_h^* \gamma_v + \mu_v}. \end{aligned}$$

We applied Gaussian elimination for J_1^* to acquire

$$J_1^* = \begin{bmatrix} -B_1 - \mu_h & 0 & B_2 & 0 & B_3 \\ 0 & -A_1 & -\frac{B_2 \mu_h}{B_1 + \mu_h} & 0 & -\frac{B_3 \mu_h}{B_1 + \mu_h} \\ 0 & 0 & -\frac{C_1}{A_1(B_1 + \mu_h)} & 0 & -\frac{\alpha_h B_3 \mu_h}{A_1(B_1 + \mu_h)} \\ 0 & 0 & 0 & -I_h \gamma_v - \mu_v & -\frac{\mu_h \gamma_h B_3 B_4}{C_1} \\ 0 & 0 & 0 & 0 & J_{1,55}^* \end{bmatrix}$$

where

$$\begin{aligned} J_{1,55}^* &= -(\frac{A(B_1 + \mu_h)}{C_1} + \frac{\alpha_h \mu_h A_3 B_2}{C_1} + \frac{\alpha_h \mu_h \mu_v B_3 B_4}{C_1 (I_h \gamma_v + \mu_v)}), \\ A &= A_1 A_2 A_3, \\ C_1 &= A_1 A_2 B_1 + A_1 A_2 \mu_h + B_2 \alpha_h \mu_h. \end{aligned}$$

The endemic equilibrium will be locally asymptotically stable if the real parts of all eigenvalues of J_1^* are negative. The eigenvalues of a triangular matrix are the entries on its main diagonal. There are

$$\begin{aligned} \lambda_1 &= -(B_1 + \mu_h), \quad \lambda_2 = -A_1, \\ \lambda_3 &= -\frac{C_1}{A_1(B_1 + \mu_h)}, \quad \lambda_4 = -(I_h \gamma_v + \mu_v), \end{aligned}$$

$$\lambda_5 = -(\frac{A(B_1 + \mu_h)}{C_1} + \frac{\alpha_h \mu_h A_3 B_2}{C_1} + \frac{\alpha_h \mu_h \mu_v B_3 B_4}{C_1 (I_h \gamma_v + \mu_v)}).$$

Since all parameters are positive so it easy to consider $Re(\lambda_i) < 0$ for $i = 1, .., 5$, which show that the endemic equilibrium is locally asymptotically stable.

D. Globally stability of the disease-free equilibrium

To show that the system is globally asymptotically stable, we use the Lyapunov function theory for present the global stability of the disease-free equilibrium.

Theorem 3: If $\mathcal{R}_0 \leq 1$, the disease-free equilibrium E_0 of the system (2) is globally asymptotically stable on Ω.

Proof: Now we construct the Lyaponov function [13].

$$L_0 = M_1 S_h^0 F(\frac{S_h}{S_h^0}) + M_2 E_h + M_3 I_h + M_4 S_v^0 F(\frac{S_v}{S_v^0}) + M_5 I_v \quad (11)$$

where M_i for $i = 1, ..., 5$ are some positive constants to be chosen later. We use advantages from the properties of the function $F(x) = x - 1 - \ln(x)$. We have:

$$\begin{aligned} L_0 &= M_1 (S_h - S_h^0 - S_h^0 ln \frac{S_h}{S_h^0}) + M_2 E_h + M_3 I_h \\ &\quad + M_4 (S_v - S_v^0 - S_v^0 ln \frac{S_v}{S_v^0}) + M_5 I_v \quad (12) \end{aligned}$$

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The derivative of L_0 along with the solution of (2) is given by;

$$L_0^{'} = M_1(1-\frac{S_h^0}{S_h})S_h^{'} + M_2E_h^{'} + M_3I_h^{'} + M_4(1-\frac{S_v^0}{S_v})S_v^{'} + M_5I_v^{'}$$

computed $L_0^{'}$ along the system (2) and some rearranged terms, we obtain

$$\begin{aligned} L_0^{'}(t) &= -\mu_h M_1\Big(\frac{S_h-S_h^0}{S_h}\Big)^2 + E_h[-M_2A_1 + M_3\alpha_h] \\ &\quad +I_h\Big[-M_3A_2 + \frac{M_1\gamma_h b_h}{\mu_h} + \frac{M_4\gamma_v b_v}{\mu_v}\Big] \\ &\quad -\mu_v M_4\Big(\frac{S_v-S_v^0}{S_v}\Big)^2 + I_v\Big[-M_5A_3 + \frac{M_1\gamma_{hv}b_h}{\mu_h}\Big] \\ &\quad +(M_2-M_1)[\gamma_h S_h I_h - \gamma_{hv}S_hI_v] \\ &\quad +(M_5-M_4)\gamma_v S_v I_h. \end{aligned} \tag{13}$$

We choose the suitable positive M_i for $i = 1, ..., 5$ to make (13) more easy to analyse. Then substitue $M_1 = M_2 = \frac{\alpha_h}{A_1}, M_3 = 1, M_4 = M_5 = \frac{\alpha_h\gamma_{hv}b_h}{A_1A_3\mu_h}$ into (13) with rearrangement, we get

$$\begin{aligned} L_0^{'}(t) &= -\frac{\mu_v\alpha_h\gamma_{hv}b_h}{A_1A_2\mu_h}\left(\frac{S_v-S_v^0}{S_v}\right)^2 \\ &\quad -\frac{\mu_h\alpha_h}{A_1}\left(\frac{S_h-S_h^0}{S_h}\right)^2 - A_2I_h\,(1-R_0) \end{aligned} \tag{14}$$

Thus $L_0^{'}(t)$ is negative if $\mathcal{R}_0 \leq 1$. Also note that, $L_0^{'}(t) = 0$ if and only if $S_h = S_h^0, E_h = 0, I_h = 0, S_v = S_v^0$ and $I_v = 0$. Therefore the largest compact invariant set in $\{(S_h,\ E_h,\ I_h,\ S_v,\ I_v) \in \Omega : L'(t) = 0\}$ involves $\{E_0\}$, where E_0 is the disease-free equilibrium point. Hence LaSalles invariant principle [14] then implies that E_0 is globally asymptotically stable in Ω. This proves the theorem.

IV. Conclusion

In this work we have studied cholera that takes into consideration both human and vector transmission pathways as the SEIR model and the SI model respectively. Mathematical analyzes of the model equations was done with regards to invariance of non-negativity, boundedness of solutions and the model having two steady states; a disease-free steady state and an endemically infected steady state. We have shown that the disease-free equilibrium is locally asymptotically stable when $\mathcal{R}_0 < 1$. Subsequently, we applied the Lyapunov function to present the global stability of the disease-free equilibrium. It has been proved that the global dynamics are completely determined by $\mathcal{R}_0$. If $\mathcal{R}_0 \leq 1$, the disease-free equilibrium is globally stable, otherwise unstable. When $\mathcal{R}_0 > 1$ then at least one of the infections will be present in the population. If $\mathcal{R}_0 > 1$, it is shown that unique endemic equilibrium exist and is locally asymtotically stable.

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