Available online at http://scik.org
Commun. Math. Biol. Neurosci. 2024, 2024:74
https://doi.org/10.28919/cmbn/8650
ISSN: 2052-2541

MATHEMATICAL MODELLING AND ANALYSIS OF CHOLERA DYNAMICS VIA VECTOR TRANSMISSION

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Abstract. In this study, a deterministic model for the transmission of cholera via fly vectors is derived and examined. We consider in detail the human population, vector/houseflies population, and the environmental reservoir. The study splits the class of infected individuals into symptomatic and asymptomatic infected individuals and incorporates the exposed compartment in the vector population to build a system of ordinary differential equations. Theoretically, the developed model is analysed by studying the stability of equilibrium points. The results of the analysis shows that there exist a locally stable disease free equilibrium point, E_0 when $R_0 < 1$ and endemic equilibrium, E^* when $R_0 > 1$. In an attempt to examine the effect of some parameters of the dynamic of the disease sensitivity analysis is employed. Finally, numerical simulations are also performed to verify the analytic results. The simulation study has revealed that reducing the rate of exposure to contaminated water and each infected vector's contribution to the aquatic environment is necessary to achieve a significant and effective control.

Keywords: infectious disease; mathematical model; basic reproduction number; stability analysis; numerical simulation.

2020 AMS Subject Classification: 92D30.

1. INTRODUCTION

Globally, the spread of enteric diseases is a serious threat to humanity. Enteric diseases are infections produced by bacteria or viruses that enter the body through the mouth or digestive

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Received May 21, 2024

tract. These infections are mainly brought on by consuming contaminated foods or liquids [1]. Food-borne diseases are the major global public health concern, outpacing all other diseases. Millions of individuals die from these diseases, and they have a significant negative influence on the social well-being of communities [2]. More than 400,000 people died from food-borne illnesses in 2015, according to a WHO report, with children under five accounting for one-third of all deaths [3]. When treating an infected individual, medical institutions typically consume a sizable percentage of budgets in most countries, which directly affects such countries ' economies [4]. Cholera is a food-borne enteric disease caused by a bacterium (*Vibrio cholera*). Globally, there are thought to be 1.6% to 3.5% cholera-related deaths and 1.3 to 4.0 million cases of the disease annually [5]. Food-borne diseases like cholera are more common in developing countries due to lack of education, unhygienic conditions, poor drainage system and abundance of carriers [6]. By promoting awareness of proper food storage practices and enhancing environmental sanitation, the development of food-borne diseases can be curbed [4].

Food-borne diseases are also spread by vectors, sometimes referred to as carriers, such as house flies, ticks, mites, etc. Some studies show that the common housefly, or flies in general, can act as mechanical vectors of many kind of pathogens such as bacteria [7], protozoa [8], and helminth eggs [9]. Cholera is also one of the vector-borne diseases which spread to human population due to the presence of house flies [10]. Numerous sources have indicated an increased risk of cholera during fly-filled periods of high fly density [7, 11]. It supports the notion that flies are also a significant carrier of bacteria that cause cholera. It should be mentioned that houseflies spread disease-causing germs to human food sources. Flies typically consume human food, trash, animal excrement, etc. They have the ability to absorb food in two different forms: liquid or solid, soluble in salivary gland secretions. House flies carry disease-causing germs on their bodies and feet when they rest on domestic waste. These bacteria then find their way into human food sources [12]. In essence, human food becomes contaminated when cholera-causing germs are carried by flies and introduced into the food supply [13, 14]. Susceptible population get infected with cholera as a result of eating this contaminated food [15]. Despite the lack of direct human-housefly interaction, the environment's housefly population nevertheless contributes to the overall number of affected people [4]. Approximately one thousand vibrios per

gram of stool are shed by the infected vector in their stool for only one day, according to certain studies [16]. Thus, vectors (e.g. house flies) can play a crucial role in the transmission of the cholera. The way house flies spread the cholera virus to people is by feeding, crawling on, and laying their eggs on human food [17].

A number of mathematical models have been developed to understand the complex dynamics of cholera [18, 19, 20, 21]. An environmental class for bacteria is included in the Susceptible (S) -Infectious (I) - Recovered (R) - Bacterial concentration in contaminated water (B) type model [18], which may be seen as the bacterial content in the water supply integrated into a *SIR* model to create a combined human - environment epidemiological system. This model was used to simulate the outbreaks in Zimbabwe and Haiti [22]. The Susceptible (S) - Symptomatic infectious (I) - Asymptomatic infectious (I) - Recovered (R) - Hyperinfectious Bacterial concentration (B) - Lowinfectious Bacterial concentration (B) model was developed by extending the *SIRB* model to include a hyperinfectious bacterial class and an asymptomatic infected human class [19]. Other Susceptible (S) - Symptomatic infectious (I) - Asymptomatic infectious (I) - Recovered (R) - Bacterial concentration (B) model [20] and the Susceptible (S) - Partial immunity class (S) - Symptomatic infectious (I) - Asymptomatic infectious (I) - Recovered from symptomatic infection (R) - Recovered from asymptomatic infection (R) - Bacterial concentration (B) model [21] were developed with the intent of having to replace the hyperinfectious bacterial class in the context of the *SIIRBB* model with person-to-person transmission and to allow for the possibility that the ability to obtain partial immunity may influence long-term cholera dynamics, respectively. The dynamics of cholera are also described mathematically in [23], along with how prevention measures including vaccination, treatment, sanitation, and awareness campaigns help keep the disease under control.

Many studies have been conducted in the past to investigate the dynamics of cholera transmission, both directly through human-to-human contact and indirectly through the consumption of contaminated water. However, the impact of fly vectors on the transmission of cholera diseases has received less attention. Fly vectors are among the species with high growth rate that are responsible for the worldwide spread of diseases like cholera [15]. Nowadays, food-borne diseases are a major threat to developing nations and they mainly spread due to the presence

of fly vectors (a kind of carrier). To understand the spread of these diseases, a mathematical model considering the role of houseflies in cholera transmission dynamics proposed in [1]. A single compartment is used in this mathematical model to represent an infected individual, and the exposed vector population is not taken into consideration. However, it could be preferable to split the infected individuals into asymptomatic and symptomatic infected individuals in order to observe the contribution of vibrio cholerae to the environment from each compartment. Since exposed vectors do not become infectious right away, adding the exposed compartment to the vector population to the model makes it somewhat more realistic and aids in understanding the dynamics of disease spread. In this study we extend the deterministic model developed in [1], by splitting infected compartment into symptomatic infected and asymptomatic infected individuals. Additionally, the exposed compartment is included in the vector population. The model also assumes the division of the environment as contaminated and uncontaminated or safe. Susceptible population will get infected from uncontaminated or safe environment as the vectors can transmit the bacterial pathogen to this environment.

2. MODEL FORMULATION

We formulate the basic model for the dynamics of the houseflies-cholera model with two host populations, the human (individuals) population (N_h) and the vector population (N_v). The model also incorporates the indirect environmental transmission, and the dynamics of the concentration of free-living cholera vibrios in contaminated/unsafe and uncontaminated/safe environments. The total human population (N_h) is subdivided into four compartments depending on the epidemiological status of individuals namely; susceptible individuals S_h, symptomatic infected individuals I_{sh}, asymptomatic infected individuals I_{ah} and recovered individuals R_h, and then $N_h = S_h(t) + I_{sh}(t) + I_{ah}(t) + R_h(t)$ at any given time t. Similarly the total vector population (N_v) is subdivided into three compartments; susceptible vectors S_v, exposed vectors E_v and infected vectors I_v and then $N_v = S_v(t) + E_v(t) + I_v(t)$ at any given time t. Bacterial concentrations in contaminated/unsafe water $C(t)$ and bacterial concentrations in non-contaminated/safe water $P(t)$ are the bacteria (pathogen) population. The housefly vector gets the infection from contaminated/unsafe environment then it transmits it to non-contaminated/safe environment. The following assumptions are imposed in formulating the model:

(1) There is neither immigration nor emigration; the population is closed.

(2) The contribution rate of shedding bacteria to the *Vibrio cholera* population in the aquatic environment is ξ_1 and ξ_2, respectively, for both symptomatic infected humans (I_{sh}) and asymptomatic infected humans (I_{ah}).

(3) The total human and vector population are constant.

(4) Human birth and death rates occur at different rates (i.e., b_h and μ_h) respectively.

(5) Vectors' birth and death rates occur at different rates (i.e., b_v and μ_v) respectively.

(6) The population is homogeneously mixed i.e., each individual within the population is susceptible to disease.

The schematic of the model is presented in Figure 1, and the description of model parameters is in Table 1.

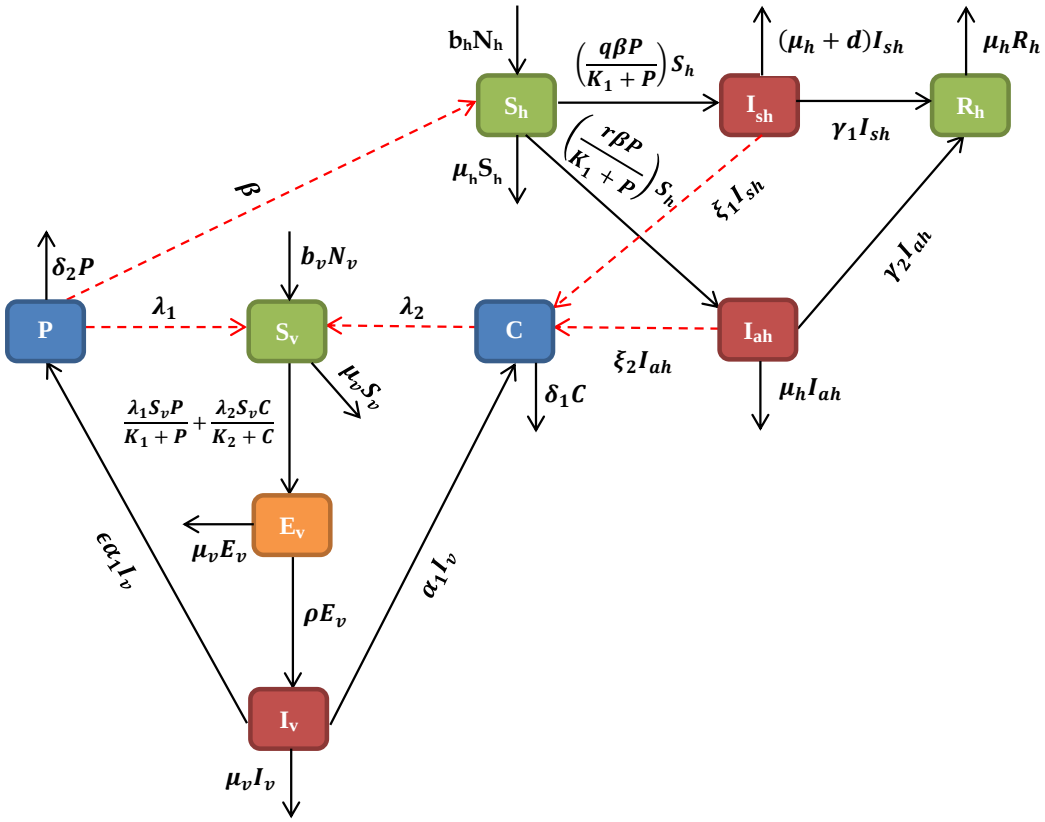


FIGURE 1. Model flowchart showing the compartments.

TABLE 1. Parameters and their descriptions of the model.

Parameter	Description
b_h	Birth or recruitment rate by human
b_v	Birth or recruitment rate by vector
μ_h	Natural human death rate
μ_v	Natural vector death rate
β	Rates of ingesting vibrios from the safe environment by human
k_1	Concentration of the bacteria i.e. vibrio cholerae in pure/safe water
k_2	Concentration of the bacteria i.e. vibrio cholerae in unsafe water
ξ_1	Rate of shedding bacteria from $I_s(t)$ into the environment
ξ_2	Rate of shedding bacteria from $I_a(t)$ into the environment
α_1	Rate of contribution to (*Vibrio cholera*) in the both environments by vectors
ε	Modification parameter
d	Disease induced death rate
γ_1	Recovery rate of symptomatic infected individuals
γ_2	Recovery rate of asymptomatic infected individuals
q	Probability of new infected from S_h to be symptomatic
r	Probability of new infected from S_h to be asymptomatic in which $r = 1-q$
ρ	Infectious rate of a vector
λ_1	Rates of ingesting vibrios from the non-contaminated/safe environment to vectors
λ_2	Rates of ingesting vibrios from the contaminated/unsafe environment to vector
δ_1	Vibrios net death rate in the contaminated/unsafe environment
δ_2	Vibrios net death rate in the non-contaminated/safe environment

It is assumed that the susceptible individuals who contract disease at rate β acquire infection with cholera due to the environment-to-human transmission represented by a logistic function. The vector gets the infection from contaminated/unsafe environment then it transmits it to non-contaminated/safe environment. Using the flowchart of the model framework (Figure 1), we formulate the following ordinary differential equations (ODEs) for the model:

$$\frac{dS_h}{dt} = b_h N_h - \frac{\beta P S_h}{k_1 + P} - \mu_h S_h, \tag{1}$$

$$\frac{dI_{sh}}{dt} = \frac{q\beta P S_h}{k_1 + P} - (\mu_h + d + \gamma_1) I_{sh}, \tag{2}$$

$$\frac{dI_{ah}}{dt} = \frac{r\beta P S_h}{k_1 + P} - (\mu_h + \gamma_2) I_{ah}, \tag{3}$$

$$\frac{dR_h}{dt} = \gamma_1 I_{sh} + \gamma_2 I_{ah} - \mu_h R_h, \tag{4}$$

$$\frac{dS_v}{dt} = b_v N_v - \frac{\lambda_1 P S_v}{k_1 + P} - \frac{\lambda_2 C S_v}{k_2 + C} - \mu_v S_v, \tag{5}$$

$$\frac{dE_v}{dt} = \frac{\lambda_1 P S_v}{k_1 + P} + \frac{\lambda_2 C S_v}{k_2 + C} - (\mu_v + \rho) E_v, \tag{6}$$

$$\frac{dI_v}{dt} = \rho E_v - \mu_v I_v, \tag{7}$$

$$\frac{dC}{dt} = \xi_1 I_{sh} + \xi_2 I_{ah} + \alpha_1 I_v - \delta_1 C, \tag{8}$$

$$\frac{dP}{dt} = \varepsilon \alpha_1 I_v - \delta_2 P. \tag{9}$$

In the above Equations (1) to (9), we assume that each susceptible individual has an equal chance of acquiring cholera through the recruitment rate b_h and consuming water with *Vibrio cholera* in the reservoir at the force of infection $\frac{\beta P}{k_1+P}$ and each susceptible vector has an equal chance of acquiring cholera through the recruitment rate b_v and consuming water with *Vibrio cholera* in the reservoir at the force of infection $\frac{\lambda_1 P}{k_1+P}$ and $\frac{\lambda_2 C}{k_2+C}$, where $\frac{P}{k_1+P}$ and $\frac{C}{k_2+C}$ are the ratios of *Vibrio cholera* concentration and k_1 and k_2 are the concentrations of *Vibrio cholera* in the water reservoir that will make a possibility of 50% of the susceptible population infected [24].

3. Mathematical Analysis of the Model

First, in order to assess the biological significance of the model, we establish the positivity and boundedness of the solution. We then demonstrate that all solutions of Equations (1) to (9) are positive for all $t \geq 0$ and are drawn to that region.

3.1. Positivity and Boundedness of Solutions. In the following, we show that the solutions of system Equations (1) to (9) are positive with the non-negative initial conditions. We also, show that the feasible solutions are bounded in a region such that $\Phi = (S_h, I_{sh}, I_{ah}, R_h, S_v, E_v, I_v, C, P) \in R_+^9$.

Theorem 3.1. *The solutions* $(S_h(t), I_{sh}(t), I_{ah}(t), R_h(t), S_v(t), E_v(t), I_v(t), C(t), P(t))$ *of model of Equations* (1) *to* (9) *are non-negative for all* $t > 0$ *with the non-negative initial conditions.*

Proof. System of Equations (1) to (9) can be put into the matrix form:

$$X' = M(X), \tag{10}$$

where $X = (S_h, I_{sh}, I_{ah}, R_h, S_v, E_v, I_v, C, P)^T \in R^9$ and $M(X)$ is given by

$$M(X) = \begin{pmatrix} M_1(X) \\ M_2(X) \\ M_3(X) \\ M_4(X) \\ M_5(X) \\ M_6(X) \\ M_7(X) \\ M_8(X) \\ M_9(X) \end{pmatrix}$$

$$= \begin{pmatrix} b_h N_h - \frac{\beta P S_h}{k_1+P} - \mu_h S_h \\ \frac{q\beta P S_h}{k_1+P} - (\mu_h + d + \gamma_1) I_{sh} \\ \frac{r\beta P S_h}{k_1+P} - (\mu_h + \gamma_2) I_{ah} \\ \gamma_1 I_{sh} + \gamma_2 I_{ah} - \mu_h R_h \\ b_v N_v - \frac{\lambda_1 P S_v}{k_1+P} - \frac{\lambda_2 C S_v}{k_2+C} - \mu_v S_v \\ \frac{\lambda_1 P S_v}{k_1+P} + \frac{\lambda_2 C S_v}{k_2+C} - (\mu_v + \rho) E_v \\ \rho E_v - \mu_v I_v \\ \xi_1 I_{sh} + \xi_2 I_{ah} + \alpha_1 I_v - \delta_1 C \\ \varepsilon \alpha_1 I_v - \delta_2 P \end{pmatrix}. \tag{11}$$

We have

$$\frac{dS_h}{dt}|_{S_h=0} = b_h N_h > 0,$$
$$\frac{dI_{sh}}{dt}|_{I_{sh}=0} = \frac{q\beta P S_h}{k_1+P} \geq 0,$$
$$\frac{dI_{ah}}{dt}|_{I_{ah}=0} = \frac{r\beta P S_h}{k_1+P} \geq 0,$$
$$\frac{dR_h}{dt}|_{R_h=0} = \gamma_1 I_{sh} + \gamma_2 I_{ah} \geq 0,$$
$$\frac{dS_v}{dt}|_{S_v=0} = b_v N_v > 0,$$

$$\frac{dE_v}{dt}|_{E_v=0} = \frac{\lambda_1 PS_v}{k_1+P} + \frac{\lambda_2 CS_v}{k_2+C} \geq 0,$$
$$\frac{dI_v}{dt}|_{I_v=0} = \rho E_v \geq 0,$$
$$\frac{dC}{dt}|_{C=0} = \xi_1 I_{sh} + \xi_2 I_{ah} + \alpha_1 I_v \geq 0,$$
$$\frac{dP}{dt}|_{P=0} = \varepsilon\alpha_1 I_v \geq 0,$$

Therefore,

$$M_i(X)|_{X_i(t)=0}, X_i \in C_+^9 \geq 0, i = 1,2,3,4,5,6,7,8,9. \tag{12}$$

Due to lemma 2 in [25], any solution of system of Equations (1) to (9) is such that $X(t) \in R_+^9$ for all $t \geq 0$. This completes the proof of Theorem 3.1.

Theorem 3.2. *The solutions for the model system of Equations* (1) *to* (9) *are contained and remain in the region* Φ *for all time* $t \geq 0$.

Proof. Consider the total human population

$N_h(t) = S_h(t) + I_{sh}(t) + I_{ah}(t) + R_h(t)$

its time derivative satisfies

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dI_{sh}}{dt} + \frac{dI_{ah}}{dt} + \frac{dR_h}{dt}. \tag{13}$$

Substituting the derivatives from Equations (1) to (9) to Equation (13) we get

$$\begin{aligned}\frac{dN_h(t)}{dt} &= b_h N_h - \mu_h S_h - \mu_h I_{sh} - \mu_h I_{sh} - \mu_h I_{ah} - \mu_h R_h \\ &= \hat{b}_h - (S_h(t) + I_{sh}(t) + I_{ah}(t) + R_h(t))\mu_h - \mu_h I_{sh} \\ &= \hat{b}_h - N_h(t)\mu_h - \mu_h I_{sh} \\ &\leq \hat{b}_h - N_h(t)\mu_h \\ \frac{dN_h(t)}{dt} &+ N_h(t)\mu_h \leq \hat{b}_h.\end{aligned}$$

The integration factor **I.F** = $e^{\mu_h t}$. The solution becomes $N_h(t) \leq \frac{\hat{b}_h}{\mu_h} + Ce^{-\mu_h t}$, where C is the constant. Then

$\lim_{t\to\infty} N_h(t) \leq \frac{\hat{b}_h}{\mu_h}$.

Consider the total vector population

$N_v(t) = S_v(t) + E_v(t) + I_v(t)$

its time derivative satisfies

$$\frac{dN_v(t)}{dt} = \frac{dS_v}{dt} + \frac{dE_v}{dt} + \frac{dI_v}{dt}. \tag{14}$$

Substituting the derivatives from Equations (1) to (9) to Equation (14) we get

$$\begin{aligned}
\frac{dN_v(t)}{dt} &= b_v N_v - \mu_v S_v - \mu_v E_v - \mu_v I_v \\
&= \hat{b}_v - (S_v(t) + E_v(t) + I_v(t)) \\
&= \hat{b}_v - N_v(t)\mu_v \\
&\leq \hat{b}_v - N_v(t)\mu_v \\
\frac{dN_v}{dt} + N_v(t)\mu_v &\leq \hat{b}_v.
\end{aligned}$$

The integration factor **I.F** = $e^{\mu_v t}$. The solution becomes $N_v(t) \leq \frac{\hat{b}_v}{\mu_v} + Ce^{-\mu_v t}$, where C is the constant. Then

$\lim_{t\to\infty} N_v(t) \leq \frac{\hat{b}_v}{\mu_v}$.

However, for the pathogen population the boundedness are shown as follows

$\frac{dC}{dt} = \xi_1 I_{sh} + \xi_2 I_{ah} + \alpha_1 I_v - \delta_1 C$, but $I_{sh} \leq \frac{\hat{b}_h}{\mu_h}$, $I_{ah} \leq \frac{\hat{b}_h}{\mu_h}$, and $I_v \leq \frac{\hat{b}_v}{\mu_v}$. Then

$\frac{dC}{dt} \leq \xi_1 \frac{\hat{b}_h}{\mu_h} + \xi_2 \frac{\hat{b}_h}{\mu_h} + \alpha_1 \frac{\hat{b}_v}{\mu_v} - \delta_1 C$

By integrating this equation (**I.F** = $e^{\delta_1 t}$), we get this solution

$C(t) \leq \frac{(\xi_1+\xi_2)\hat{b}_h}{\delta_1\mu_h} + \frac{(\alpha_1)\hat{b}_v}{\delta_1\mu_v} + Ae^{\delta_1 t}$, where A is a constant. Then $\lim_{t\to\infty} C(t) \leq \frac{(\xi_1+\xi_2)\hat{b}_h}{\delta_1\mu_h} + \frac{(\alpha_1)\hat{b}_v}{\delta_1\mu_v}$.

$\frac{dP}{dt} = \varepsilon\alpha_1 I_v - \delta_2 P$, but $I_v \leq \frac{\hat{b}_v}{\mu_v}$. Then

$\frac{dP}{dt} \leq \varepsilon\alpha_1 \frac{\hat{b}_v}{\mu_v} - \delta_2 P$

By integrating this equation (**I.F** = $e^{\delta_2 t}$), we get this solution

$P(t) \leq \frac{(\varepsilon\alpha_1)\hat{b}_v}{\delta_2\mu_v} + Ae^{\delta_2 t}$, where A is a constant. Then $\lim_{t\to\infty} P(t) \leq \frac{(\varepsilon\alpha_1)\hat{b}_v}{\delta_2\mu_v}$.

Hence, we have that

$0 \leq N_h(t) \leq \frac{\hat{b}_h}{\mu_h}, 0 \leq N_v(t) \leq \frac{\hat{b}_v}{\mu_v}, 0 \leq C(t) \leq \frac{(\xi_1+\xi_2)\hat{b}_h}{\delta_1\mu_h} + \frac{(\alpha_1)\hat{b}_v}{\delta_1\mu_v}$, and $0 \leq P(t) \leq \frac{(\varepsilon\alpha_1)\hat{b}_v}{\delta_2\mu_v}$ which implies that N_h , N_v all other variables $(S_h, I_{sh}, I_{ah}, R_h, S_v, E_v, I_v, C, P)$ of model Equations (1) to (9) are bounded and all the solutions starting in Φ approach, enter or stay in Φ. This completes the proof.

3.2. Existence of the disease-free equilibrium. In this section, we analysed system of Equations (1) to (9) in order to obtain the equilibrium points of the system and its stability. Let $E = (S_h^*, I_{sh}^*, I_{ah}^*, R_h^*, S_v^*, E_v^*, I_v^*, C^*, P^*)$ be the steady-state of system of Equations (1) to (9). Then, the equilibrium points are obtained by setting the right hand sides of system Equations (1) to (9) to zero, that is;

$$
\begin{aligned}
b_h N_h - \frac{\beta P S_h}{k_1 + P} - \mu_h S_h &= 0, \\
\frac{q\beta P S_h}{k_1 + P} - (\mu_h + d + \gamma_1) I_{sh} &= 0, \\
\frac{r\beta P S_h}{k_1 + P} - (\mu_h + \gamma_2) I_{ah} &= 0, \\
\gamma_1 I_{sh} + \gamma_2 I_{ah} - \mu_h R_h &= 0, \\
b_v N_v - \frac{\lambda_1 P S_v}{k_1 + P} - \frac{\lambda_2 C S_v}{k_2 + C} - \mu_v S_v &= 0, \\
\frac{\lambda_1 P S_v}{k_1 + P} + \frac{\lambda_2 C S_v}{k_2 + C} - (\mu_v + \rho) E_v &= 0, \\
\rho E_v - \mu_v I_v &= 0, \\
\xi_1 I_{sh} + \xi_2 I_{ah} + \alpha_1 I_v - \delta_1 C &= 0, \\
\varepsilon \alpha_1 I_v - \delta_2 P &= 0.
\end{aligned} \tag{15}
$$

The population will never be extinct as long as the human recruitment term $b_h N_h$ and the housefly birth term $b_v N_v$ are not zero. This implies that there is no trivial equilibrium point, thus $(S_h^*, I_{sh}^*, I_{ah}^*, R_h^*, S_v^*, E_v^*, I_v^*, C^*, P^*) \neq (0,0,0,0,0,0,0,0,0)$. The model system of Equations (1) to (9) has a steady state in the absence of cholera disease, that is, $I_{sh}^* = I_{ah}^* = S_v^* = E_v^* = I_v^*$. Hence, the disease-free equilibrium (DFE) denoted as E_0, of the model system Equations (1) to (9) is given by

$$
\begin{aligned}
E_0 &= (S_h^*, I_{sh}^*, I_{ah}^*, R_h^*, S_v^*, E_v^*, I_v^*, C^*, P^*) \\
&= (\frac{b_h N_h}{\mu_h}, 0, 0, 0, \frac{b_v N_v}{\mu_v}, 0, 0, 0, 0).
\end{aligned} \tag{16}
$$

3.3. Reproduction number. In epidemiology, a key parameter is the basic reproduction number (sometimes called basic reproductive rate, basic reproductive ratio) denoted by R_0, defined

as the average number of secondary infectious cases transmitted by a single primary infectious cases introduced into a whole susceptible population [26].

To compute R_0, we use the next generation matrix approach as described in [27] and obtained by taking the largest (dominant) eigenvalue value (spectral radius) of

$$\left[\frac{\partial F_i(E_0)}{\partial X_i}\right]\left[\frac{\partial V_i(E_0)}{\partial X_i}\right]^{-1}, \tag{17}$$

where F_i is the rate of appearance of new infection in compartment i, V_i is the net transition between compartments, E_0 is the disease free equilibrium and X_i stands for the terms in which the infection is in progression. In an attempt to calculate R_0 for system of Equations (1) to (9), we start from the infected compartments for both populations; $I_{sh}, I_{ah}, E_v, I_v, C, P$ and then followed by the uninfected classes; S_h, R_h, S_v. The two populations thus give

$$\begin{aligned}
\frac{dI_{sh}}{dt} &= \frac{q\beta PS_h}{k_1+P} - (\mu_h + d + \gamma_1)I_{sh},\\
\frac{dI_{ah}}{dt} &= \frac{r\beta PS_h}{k_1+P} - (\mu_h + \gamma_2)I_{ah},\\
\frac{dE_v}{dt} &= \frac{\lambda_1 PS_v}{k_1+P} + \frac{\lambda_2 CS_v}{k_2+C} - (\mu_v + \rho)E_v,\\
\frac{dI_v}{dt} &= \rho E_v - \mu_v I_v,\\
\frac{dC}{dt} &= \xi_1 I_{sh} + \xi_2 I_{ah} + \alpha_1 I_v - \delta_1 C,\\
\frac{dP}{dt} &= \varepsilon\alpha_1 I_v - \delta_2 P,\\
\frac{dS_h}{dt} &= b_h N_h - \frac{\beta PS_h}{k_1+P} - \mu_h S_h,\\
\frac{dR_h}{dt} &= \gamma_1 I_{sh} + \gamma_2 I_{ah} - \mu_h R_h,\\
\frac{dS_v}{dt} &= b_v N_v - \frac{\lambda_1 PS_v}{k_1+P} - \frac{\lambda_2 CS_v}{k_2+C} - \mu_v S_v.
\end{aligned} \tag{18}$$

From Equation (18), we show the rate of appearance of new infections in compartments; I_sh, I_ah, and E_v using the next generation matrix as

$$f = \begin{bmatrix} \frac{q\beta PS_h}{k_1+P} \\ \frac{r\beta PS_h}{k_1+P} \\ \frac{\lambda_1 PS_v}{k_1+P} + \frac{\lambda_2 CS_v}{k_2+C} \\ 0 \\ 0 \\ 0 \end{bmatrix}. \tag{19}$$

Differentiating the matrix above, with respect to the model variables using Jacobian matrix method at the disease-free equilibrium point E_0, where $N_h(t) \leq \frac{\hat{b}_h}{\mu_h}$ and $N_v(t) \leq \frac{\hat{b}_v}{\mu_v}$ to get Jacobian matrix;

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & \frac{q\beta S_h^*}{k_1} \\ 0 & 0 & 0 & 0 & 0 & \frac{r\beta S_h^*}{k_1} \\ 0 & 0 & 0 & 0 & \frac{\lambda_2 S_v^*}{k_2} & \frac{\lambda_1 S_v^*}{k_1} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \tag{20}$$

where $S_h^* = \frac{\hat{b}_h}{\mu_h} = \frac{b_h N_h}{\mu_h} and S_v^* = \frac{\hat{b}_v}{\mu_v} = \frac{b_v N_v}{\mu_v}$.

Calculating the transfer of individuals out of the compartments of the system Equation (18) by all other means, we have

$$v = \begin{bmatrix} (\mu_h + d + \gamma_1) I_{sh} \\ (\mu_h + \gamma_2) I_{ah} \\ (\mu_v + \rho) E_v \\ \mu_v I_v - \rho E_v \\ \delta_1 C - [\xi_1 I_{sh} + \xi_2 I_{ah} + \alpha_1 I_v] \\ \delta_2 P - \varepsilon \alpha_1 I_v \end{bmatrix}. \tag{21}$$

Hence, the Jacobian matrix of V evaluated at E_0 is given by

$$V = \begin{bmatrix} a_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & a_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & a_3 & 0 & 0 & \\ 0 & 0 & -\rho & \mu_v & 0 & 0 \\ -\xi_1 & -\xi_2 & 0 & -\alpha_1 & \delta_1 & 0 \\ 0 & 0 & 0 & -\varepsilon\alpha_1 & 0 & \delta_2 \end{bmatrix}, \tag{22}$$

where, $a_1 = \mu_h + d + \gamma_1, a_2 = \mu_h + \gamma_2, a_3 = \mu_v + \rho$. Using the next-generation matrix approach, the basic reproduction number R_0 of the model is computed as the spectral radius $\rho\left(F \times V^{-1}\right)$ of the next generation matrix, i.e.

$R_0 = \rho\left(F \times V^{-1}\right)$.

Now the expression for the basic reproduction number,R_0 , becomes the sum of two quantities, i.e.

$R_0 = R_{0h} + R_{0v}$

where, quantities $R_{0h} = \sqrt{\frac{\alpha_1 \rho b_h \beta \varepsilon \lambda_2 (a_1 r \xi_2 + a_2 q \xi_1)}{K_1 K_2 a_1 a_2 \delta_1 \delta_2}}$ and $R_{0v} = \frac{\alpha_1 \rho (K_1 \delta_2 \lambda_2 + K_2 \delta_1 \varepsilon \lambda_1) \sqrt{(b_v)^3}}{K_1 K_2 \delta_1 \delta_2 \sqrt{(a_3 \mu_v)^3}}$ are contributions of the human and vector/houseflies infectious classes, respectively.

3.4. Global stability analysis of disease-free equilibrium (DFE). Lemma. The DFE system of Equations (1) to (9) is locally asymptotically stable (LAS) whenever $R_0 < 1$, and unstable whenever $R_0 > 1$.

This suggests that when $R_0 < 1$ and the initial sizes of the host populations in the model are in the DFE's basin of attraction, the cholera can be eradicated from the community. However, to guarantee that the disease will be eliminated independently of the initial sizes of host populations, the DFE must be global asymptotically stable (GAS) of the DFE when $R_0 < 1$ as showing in the following theorem.

Theorem 3.3. *The DFE P_0 system of Equations* (1) *to* (9) *is GAS if $R_0 < 1$ in the compact set* Γ.

Proof. Using Castillo-Chavez theorem [28], model of Equations (1) to (9) can be written in the form:

$$\frac{dX}{dt} = F(X,Z),$$

$$\frac{dZ}{dt} = G(X,Z) = G(X,0) = 0, \tag{23}$$

where X and Z denote the uninfected and infected compartments respectively, that is, $X = (S_h, R_h, S_v)$ and $Z = (I_{sh}, I_{ah}, E_v, I_v, C, P)$. We begin by showing condition i of Castillo-Chavez theorem [28] as:

$$F(X,0) = \begin{bmatrix} b_h N_h - \mu_h S_h \\ -\mu_h R_h \\ b_v N_v - \mu_v S_v \end{bmatrix}, \tag{24}$$

and solving these three ordinary differential equations gives

$$S_h(t) = \frac{b_h N_h}{\mu_h} + S(0)e^{-\mu_h t},$$

$$R_h(t) = R(0)e^{-\mu_h t},$$

$$S_v(t) = \frac{b_v N_v}{\mu_v} + S(0)e^{-\mu_v t}.$$

Thus, $S_h(t) \to \frac{b_h N_h}{\mu_h}, S_v(t) \to \frac{b_v N_v}{\mu_v}$, and $R_h(t) \to 0$ as $t \to \infty$, regardless of the values of initial conditions. Thus, P_0 is globally asymptotically stable. Next, applying Castillo-Chavez theorem [28] to our cholera model of Equations (1) to (9) to show condition ii:

$$G(X,Z) = \begin{bmatrix} \frac{q\beta P S_h}{k_1+P} - (\mu_h + d + \gamma_1) I_{sh} \\ \frac{r\beta P S_h}{k_1+P} - (\mu_h + \gamma_2) I_{ah} \\ \frac{\lambda_1 P S_v}{k_1+P} + \frac{\lambda_2 C S_v}{k_2+C} - (\mu_v + \rho) E_v \\ \rho E_v - \mu_v I_v \\ \xi_1 I_{sh} + \xi_2 I_{ah} + \alpha_1 I_v - \delta_1 C \\ \varepsilon \alpha_1 I_v - \delta_2 P \end{bmatrix} \tag{25}$$

and

$$A = \begin{bmatrix} -(\mu_h + d + \gamma_1) & 0 & 0 & 0 & 0 & \frac{q\beta b_h N_h}{K_1 \mu_h} \\ 0 & -(\mu_h + \gamma_2) & 0 & 0 & 0 & \frac{r\beta b_h N_h}{K_1 \mu_h} \\ 0 & 0 & -(\mu_v + \rho) & 0 & \frac{\lambda_2 b_v N_v}{K_2 \mu_v} & \frac{\lambda_1 b_v N_v}{K_1 \mu_v} \\ 0 & 0 & \rho & -\mu_v & 0 & 0 \\ \xi_1 & \xi_2 & 0 & \alpha_1 & -\delta_1 & 0 \\ 0 & 0 & 0 & \varepsilon\alpha_1 & 0 & -\delta_2 \end{bmatrix}$$

which is clearly an M-matrix. Meanwhile, we find

$$\hat{G}(X,Z) = \begin{bmatrix} \frac{q\beta P b_h N_h}{k_1 \mu_h} - \frac{q\beta P S_h}{k_1 + P} \\ \frac{r\beta P b_h N_h}{k_1 \mu_h} - \frac{r\beta P S_h}{k_1 + P} \\ \frac{\lambda_2 b_v N_v C}{k_2 \mu_h} - \frac{\lambda_2 S_v C}{k_2 + C} + \frac{\lambda_1 b_v N_v P}{k_1 \mu_v} - \frac{\lambda_1 S_v P}{k_1 + P} \\ 0 \\ 0 \\ 0 \end{bmatrix},$$

and since $0 \leq S_h \leq \frac{b_h N_h}{\mu_h}$ and $0 \leq S_v \leq \frac{b_v N_v}{\mu_v}$, then it follows that $\hat{G}(X,Z) \geq 0$. Thus P_0 is GAS whenever $R_0 < 1$. This completes the proof.

3.5. Existence and stability of the endemic equilibrium Point. The endemic equilibrium points of the model system of Equations (1) to (9) is given by $E^* = \left(S_h^*, I_{sh}^*, I_{ah}^{,*}, S_v^*, E_v^*, I_v^*, C^*, P^*\right)$ with $I_{sh} \neq 0, I_{ah} \neq 0, E_v \neq 0, I_v \neq 0, C \neq 0$ and $P \neq 0$. It can be obtained by equating the RHS of each equation of the model system Equations (1) to (9) equal to zero, which exists for $R_0 > 1$.

At equilibrium, $\frac{dS_h}{dt} = \frac{dI_{sh}}{dt} = \frac{dI_{ah}}{dt} = \frac{dR_h}{dt} = \frac{dS_v}{dt} = \frac{dE_v}{dt} = \frac{dI_v}{dt} = \frac{dC}{dt} = \frac{P}{dt} = 0$,we therefore equate Equations (1) to (9) to zero.

$S_h^* = \frac{b_h N_h}{\mu_h} - \frac{(\mu_h + d + \gamma_1) I_{sh}^*}{q\mu_h}$

$I_{sh}^* = \frac{b_h N_h - \mu_h S_h^* - (\mu_h + \gamma_2) I_{ah}^*}{\mu_h + d + \gamma_1}$

$I_{ah}^* = \frac{b_h N_h - \mu_h S_h^* - (\mu_h + d + \gamma_1) I_{sh}^*}{\mu_h + \gamma_2}$

$R_h^* = \frac{\gamma_1 I_{sh}^* + \gamma_2 I_{ah}^*}{\mu_h}$

$S_v^* = \frac{b_v N_v}{\mu_v} - \frac{(\mu_v + \rho) E_v^*}{\mu_v}$

$E_v^* = \frac{b_v N_v}{\mu_v+\rho} - \frac{\mu_v S_v^*}{\mu_v+\rho}$
$I_v^* = \frac{\rho E_v^*}{\mu_v}$
$C^* = \frac{\xi_1 I_{sh}^* + \xi_2 I_{ah}^* + \alpha_1 I_v^*}{\delta_1}$
$P^* = \frac{\varepsilon \alpha_1 I_v^*}{\delta_2}$

Stability of the endemic equilibrium can be proved by considering the Lyaponuv function defined as;

$$\left.\begin{aligned} L(S_h^*, I_{sh}^*, I_{ah}^*, {}^*, S_v^*, E_v^*, I_v^*, C^*, P^*) = \left(S_h - S_h^* - S_h^* \ln\left(\frac{S_h^*}{S_h}\right)\right) + \left(I_{sh} - I_{sh}^* - I_{sh}^* \ln\left(\frac{I_{sh}^*}{I_{sh}}\right)\right) \\ + \left(I_{ah} - I_{ah}^* - I_{ah}^* \ln\left(\frac{I_{ah}^*}{I_{ah}}\right)\right) + \left(R_h - R_h^* - R_h^* \ln\left(\frac{R_h^*}{R_h}\right)\right) \\ + \left(S_v - S_v^* - S_v^* \ln\left(\frac{S_v^*}{S_v}\right)\right) + \left(E_v - E_v^* - E_v^* \ln\left(\frac{E_v^*}{E_v}\right)\right) \\ + \left(I_v - I_v^* - I_v^* \ln\left(\frac{I_v^*}{I_v}\right)\right) + \left(C - C^* - C^* \ln\left(\frac{C^*}{C}\right)\right) \\ + \left(P - P^* - P^* \ln\left(\frac{P^*}{P}\right)\right) \end{aligned}\right\}$$

The derivative of L along the solution of the system is directly;

$$\begin{aligned} \frac{L}{dt} = \left(\frac{S_h - S_h^*}{S_h}\right)\frac{dS_h}{dt} + \left(\frac{I_{sh} - I_{sh}^*}{I_{sh}}\right)\frac{dI_{sh}}{dt} + \left(\frac{I_{ah} - I_{ah}^*}{I_{ah}}\right)\frac{dI_{ah}}{dt} \\ + \left(\frac{R_h - R_h^*}{R_h}\right)\frac{dR_h}{dt} + \left(\frac{S_v - S_v^*}{S_v}\right)\frac{dS_v}{dt} + \left(\frac{E_v - E_v^*}{E_v}\right)\frac{dE_v}{dt} \\ + \left(\frac{I_v - I_v^*}{I_v}\right)\frac{dI_v}{dt} + \left(\frac{C - C^*}{C}\right)\frac{dC}{dt} + \left(\frac{P - P^*}{P}\right)\frac{dP}{dt} \end{aligned}$$

$$\begin{aligned} \frac{L}{dt} = \left(\frac{S_h - S_h^*}{S_h}\right)\left[b_h N_h - \frac{\beta P S_h}{k_1 + P} - \mu_h S_h\right] + \left(\frac{I_{sh} - I_{sh}^*}{I_{sh}}\right)\left[\frac{q\beta P S_h}{k_1 + P} - (\mu_h + d + \gamma_1) I_{sh}\right] \\ + \left(\frac{I_{ah} - I_{ah}^*}{I_{ah}}\right)\left[\frac{r\beta P S_h}{k_1 + P} - (\mu_h + \gamma_2) I_{ah}\right] + \left(\frac{R_h - R_h^*}{R_h}\right)[\gamma_1 I_{sh} + \gamma_2 I_{ah} - \mu_h R_h] \\ + \left(\frac{S_v - S_v^*}{S_v}\right)\left[b_v N_v - \frac{\lambda_1 P S_v}{k_1 + P} - \frac{\lambda_2 C S_v}{k_2 + C} - \mu_v S_v\right] + \left(\frac{E_v - E_v^*}{E_v}\right)\left[\frac{\lambda_1 P S_v}{k_1 + P} + \frac{\lambda_2 C S_v}{k_2 + C} - (\mu_v + \rho) E_v\right] \\ + \left(\frac{I_v - I_v^*}{I_v}\right)[\rho E_v - \mu_v I_v] + \left(\frac{C - C^*}{C}\right)[\xi_1 I_{sh} + \xi_2 I_{ah} + \alpha_1 I_v - \delta_1 C] \\ + \left(\frac{P - P^*}{P}\right)[\varepsilon \alpha_1 I_v - \delta_2 P] \end{aligned}$$

By expansion and simplification;

$\frac{L}{dt} = P - Q$

where P are the positive terms and Q are the negative terms, such that;

$$\begin{aligned} P = b_h N_h - \frac{\beta P S_h^*}{k_1 + P} + \mu_h S_h^* + \frac{q\beta P S_h}{k_1 + P} + (\mu_h + d + \gamma_1) I_{sh}^* \\ + \frac{r\beta P S_h}{k_1 + P} + (\mu_h + \gamma_2) I_{sh}^* + \gamma_1 I_{sh} + \gamma_2 I_{ah} + \mu_h R_h^* \\ + b_v N_v - \frac{\lambda_1 P S_v^*}{k_1 + P} + \frac{\lambda_2 C S_v^*}{k_2 + C} + \mu_v S_v^* + \frac{\lambda_1 P S_v}{k_1 + P} \\ + \frac{\lambda_2 C S_v}{k_2 + C} + (\mu_v + \rho) E_v^* + \rho E_v + \mu_v I_v^* + \xi_1 I_{sh} \\ + \xi_2 I_{ah} + \alpha_1 I_v + \delta_1 C^* + \varepsilon \alpha_1 I_v + \delta_2 P^* \end{aligned}$$

$$\begin{aligned} Q = -\frac{\beta P S_h}{k_1 + P} - \mu_h S_h - \frac{b_h N_h S_h^*}{S_h} - (\mu_h + d + \gamma_1) I_{sh} - \frac{q\beta P S_h I_{sh}^*}{(k_1 + P) I_{sh}^*} \\ - (\mu_h + \gamma_2) I_{ah} - \frac{r\beta P S_h I_{ah}^*}{(k_1 + P) I_{ah}^*} - \mu_h R_h - \frac{\gamma_1 I_{sh} R_h^*}{R_h} - \frac{\gamma_2 I_{ah} R_h^*}{R_h} \\ - \frac{\lambda_1 P S_v}{k_1 + P} - \frac{\lambda_2 C S_v}{k_2 + C} - \mu_v S_v - \frac{b_v N_v S_v^*}{S_v} - (\mu_v + \rho) E_v \\ - \frac{\lambda_1 S_v P E_v^*}{(k_1 + P) E_v} - \frac{\lambda_2 S_v C E_v^*}{(k_2 + C) E_v} - \mu_v I_v - \frac{\rho E_v I_v^*}{I_v} - \delta_1 C \\ - \frac{\xi_1 I_{sh} C^*}{C} - \frac{\xi_2 I_{ah} C^*}{C} - \frac{\alpha_1 I_v C^*}{C} - \delta_2 P - \frac{\varepsilon \alpha_1 I_v P^*}{P} \end{aligned}$$

If $P < Q$, then $\frac{dL}{dt} \leq 0$.$\frac{dL}{dt} = 0$ if and only if $S_h = S_h^*, I_{sh} = I_{sh}^*, I_{ah} = I_{ah}^*, R_h = R_h^*, S_v = S_v^*, E_v = E_v^*, I_v = I_v^*, C = C^*, and P = P^*$.

The largest invariant set in $(S_h, I_{sh}, I_{ah}, R_h, S_v, E_v, I_v, C, P) \in \Theta : \frac{dL}{dt} = 0$ is a singleton of E^*, where E^* is the endemic equilibrium. This implies that the endemic equilibrium is globally asymptotically stable [29, 30].

4. Sensitivity Analysis

Sensitivity analysis is employed to ascertain the role that model parameters have in the spread of disease. In relation to the model parameters, we compute the sensitivity indices of the basic reproductive number R_0. With the use of these indices, we can determine the relative importance of each parameter for the spread of the disease and how to focus on intervention efforts. Since

errors are frequently made in data collection and assumed model parameter values, sensitivity analysis is primarily used to characterize how robustly a model forecasts to parameter values [31]. Here, we quantify the changes in the model parameters by doing a sensitivity analysis of the basic reproductive number, R_0. Now from this, we can identify the parameters that have a high impact on the basic reproduction number and on the spread of disease. Sensitivity indices allow us to quantify how much a state variable changes in relation to a changing parameter. Sensitivity analyses can be performed in a variety of methods, and the results yield sensitivity rankings that vary slightly [32]. The normalized forward sensitivity index, commonly known as elasticity, was employed. The ratio of the relative change in the R_0 to the relative change in the parameter is the normalized forward sensitivity index of a variable with regard to a parameter [32, 33]. The normalized forward sensitivity index of R_0 with a parameter P is defined as follows:

$$S_P^{R_0} = \left(\frac{\partial R_0}{\partial P}\right) \times \left(\frac{P}{R_0}\right). \tag{26}$$

The values shown in Table 3 for the parameters are considered baseline values and are utilized to evaluate the sensitivity indices of specific parameters that are accountable for the spread of cholera. Therefore, the following Table 2 lists the sensitivity indices showing the contribution of each parameter to the basic reproduction number (R_0) given that the reproduction number is less than unity.

From the above Table 2, it has been noted that these parameters have either positive or negative effects on the basic reproduction number (R_0). We can observe that the parameters b_v, α_1, ρ, and λ_2 respectively have the most positive influence on R_0. This means that the increase of these parameters while keeping other parameters constant will increase the value of the basic reproduction number (R_0) leading to an increase of the spread of cholera diseases and vice versa. For example, $S_{\alpha_1}^{R_0} = 0.8293$ means that increasing (or decreasing) α_1 by 10% increases (or decreases) the value of R_0 by 8.3%. We likewise observe that the parameters k_2, δ_1, δ_2, and k_1 respectively have the most negative impact on R_0. This implies that the increase of these parameters while keeping the other constant will decrease the value of the basic reproduction number (R_0), meaning that they will decrease the endemicity of the cholera diseases in the population and vice versa.

TABLE 2. Sensitivity indices of R_0 with respect to the model parameters.

Parameter	Sensitivity Index (+ve/-ve)
b_h	0.0412
b_v	1.2294
μ_h	-0.0026
μ_v	-0.0068
β	0.0905
k_1	-0.1009
k_2	-0.8034
ξ_1	0.0849
ξ_2	0.0313
α_1	0.8293
ε	0.0911
d	-0.0075
γ_1	-0.0008
γ_2	-0.0107
q	0.0849
r	0.0313
ρ	0.6950
λ_1	0.0026
λ_2	0.1490
δ_1	-0.4161
δ_2	-0.3602

5. NUMERICAL RESULTS AND DISCUSSIONS

In this section, we perform a numerical simulation of model system Equations (1) to (9) to confirm our analytical results and to illustrate the asymptotic behaviour of the model. The systems of differential equations were solved over a specific period of time period using Range-Kutta method embedded in MATLAB. The parameter values used in the simulations are found in the Table 3 with the following initial conditions:$N_h(0) = 10,000$, $S_h(0) = 9,935$, $I_{sh}(0) = 15$,

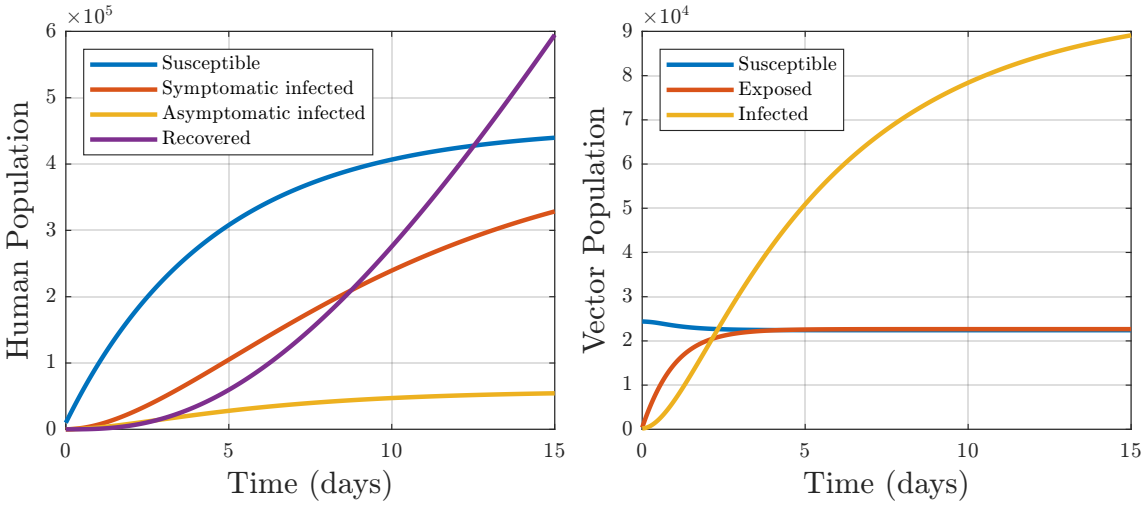
$I_{ah}(0) = 50$, $R_h(0) = 0$, $C(0) = 7000$, $P(0) = 2000$, $N_v(0) = 25,000$, $S_v(0) = 24,400$, $E_v(0) = 400$, $I_v(0) = 200$.

TABLE 3. Values for parameters of the cholera model.

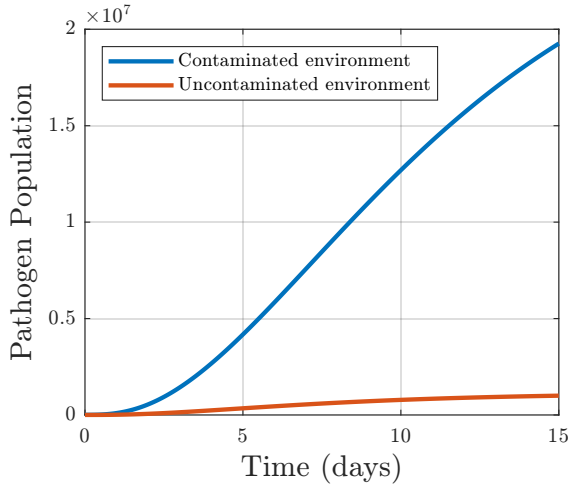
Parameter	Base line value($R_0 < 1$)	Base line value($R_0 > 1$)	Reference
b_h	10 day^{-1}	10 day^{-1}	[34]
b_v	1.066 day^{-1}	1.066 day^{-1}	Assumed
μ_h	0.005 $year^{-1}$	0.005 $year^{-1}$	[35]
μ_v	0.189 day^{-1}	0.189 day^{-1}	[36]
β	0.2143 day^{-1}	0.2143 day^{-1}	[37]
k_1	500 cells/day	500 cells/day	Assumed
k_2	500 cells/day	500 cells/day	[35]
ξ_1	20 cell/ml per day	100 cell/ml per day	[38]
ξ_2	20 cell/ml per day	100 cell/ml per day	Assumed
α_1	12 cells $mL^{-1}d^{-1}$ per vector	12 cells $mL^{-1}d^{-1}$ per vector	[1]
ε	0.4	0.89	Assumed
d	0.015	0.015	[34]
γ_1	0.14 per day	0.14 per day	[39]
γ_2	0.5 per day	0.5 per day	[39]
q	0.7	0.7	[24]
r	0.3	0.3	[24]
ρ	0.8	0.95	Assumed
λ_1	0.1	0.57	Assumed
λ_2	0.9	0.99	Assumed
δ_1	0.4 day^{-1}	0.4 day^{-1}	[40]
δ_2	0.4 day^{-1}	0.4 day^{-1}	Assumed

Population dynamics

We examine the population dynamics for both human and vector population with constant parameter values as shown in Figure 2 and Figure 3, respectively. We observe in Figure 2(a - c) that at disease-free equilibrium, the system converges and is globally stable whenever $R_0 < 1$. This implies that the population is eradicated and also evidence to Theorem 3.3. On the other hand, in Figure 3(a - c), the disease-free equilibrium of the system becomes unstable as $R_0 > 1$.



(a) (b)



(c)

FIGURE 2. Population Dynamics for $R_0 = 0.75$

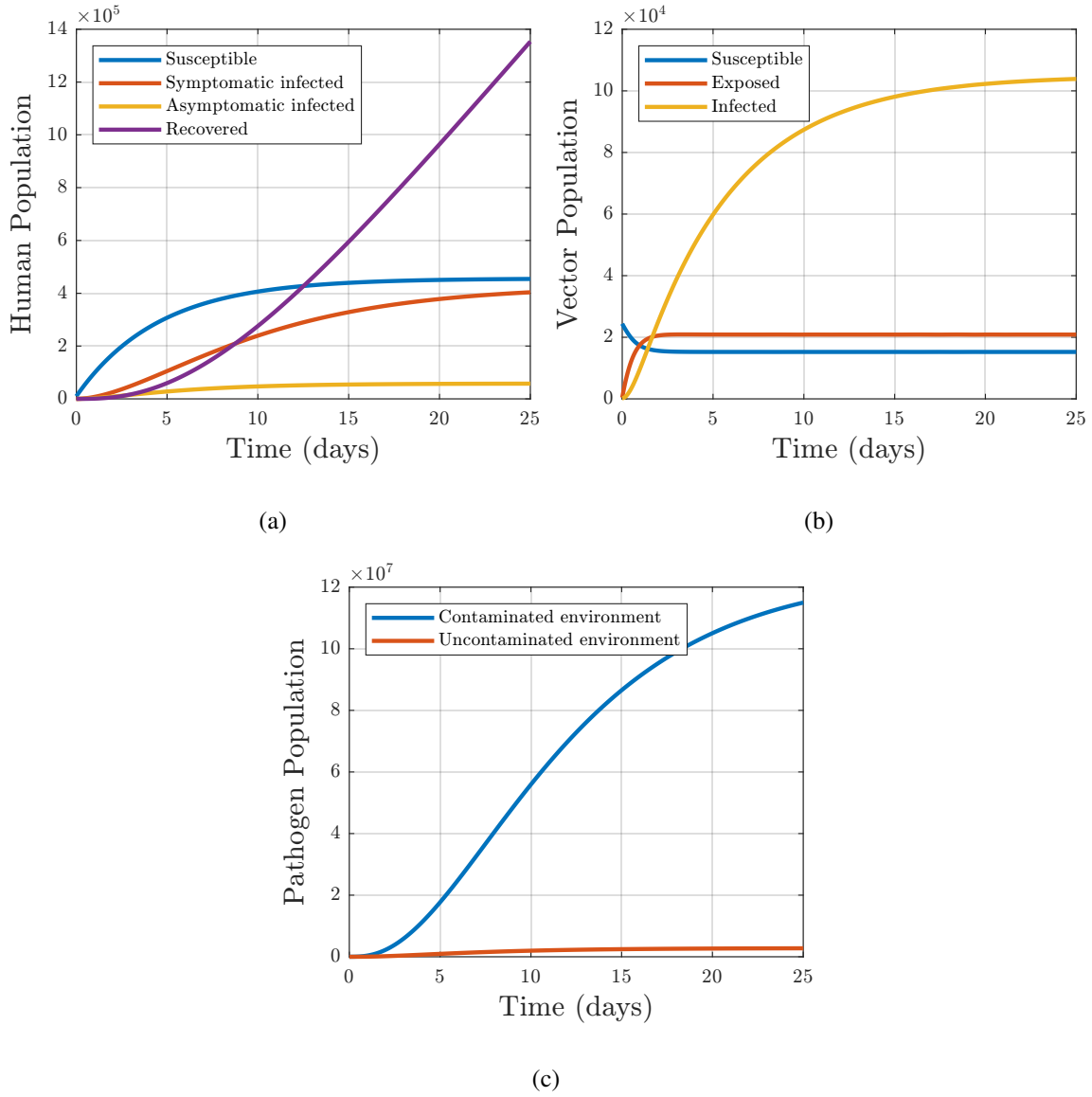


FIGURE 3. Population Dynamics for $R_0 = 1.46$

Effects of the values λ_1 and λ_2 (rates of ingesting vibrios from the non-contaminated/safe environment to vectors and rates of ingesting vibrios from the contaminated/unsafe environment to vector)

It can be seen from Figure 4 (a - b) that the effect of varying the values of λ_1 affects the exposed and infected vector populations positively with more effect in exposed vector population which is something predictable. On the other hand, the impact of increasing the value of λ_2 on the exposed vector population occurs after some time. In addition, after ingesting a

sufficient dose of *Vibrio cholera* vibrios by vectors then the infection starts to persist and hence the cholera transmission can become endemic. Consequently, the basic reproductive number is significantly increased over unity and hence this will affect the vector population (Figure 4 (c - d)).

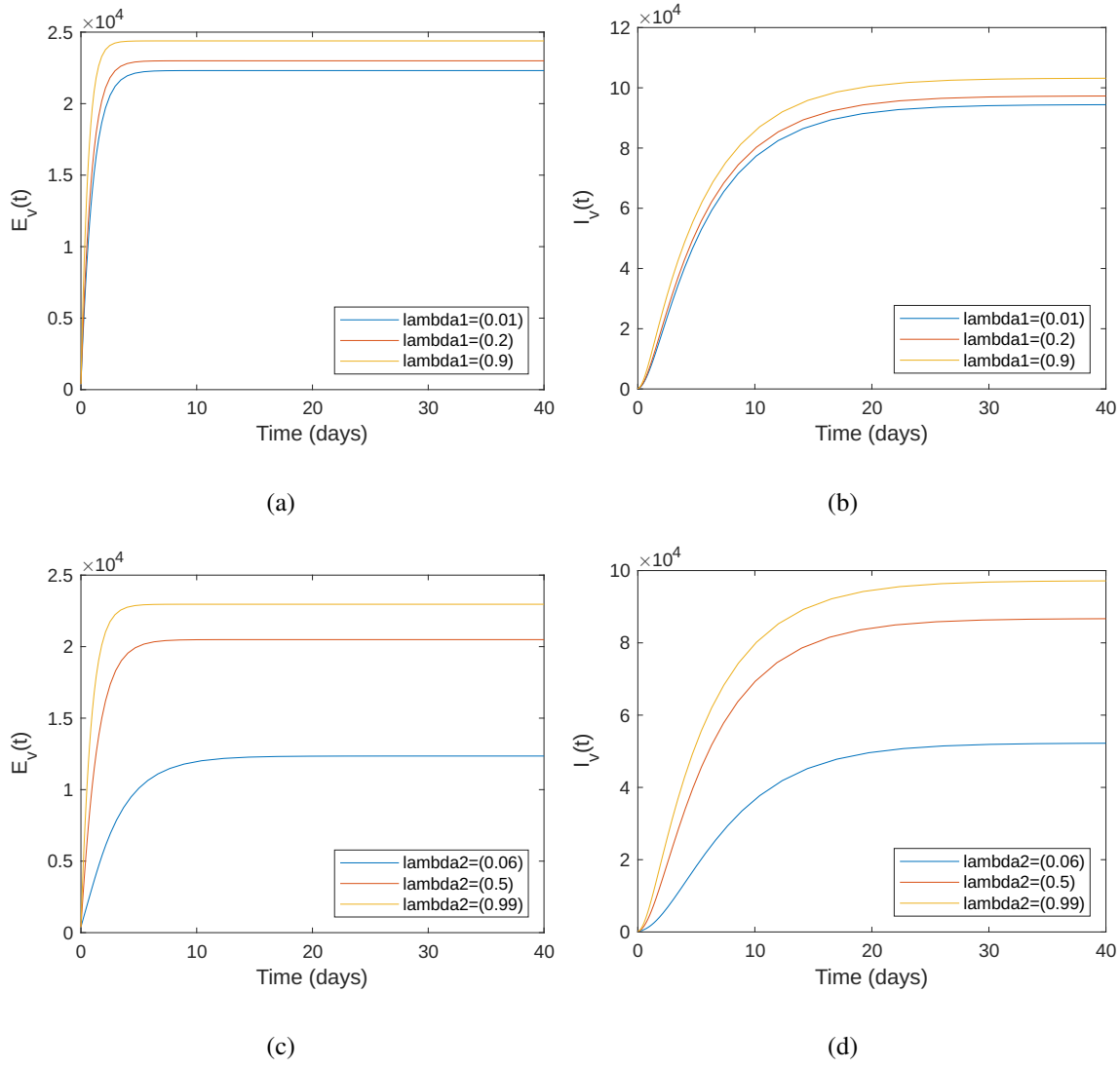


FIGURE 4. Effects of increasing or decreasing values of λ_1 and λ_2

6. CONCLUSION

We developed a compartmental model for cholera transmission involving the human host, fly vectors, and the environmental reservoir. The human host divided into four classes: susceptible, symptomatic infected, asymptomatic infected, recovered. The vector population divided

into three classes: susceptible, exposed, infected vectors and the environmental reservoir divided the environment into contaminated and uncontaminated sub-environments according to the concentration of *Vibrio cholera*. We established that the model is epidemiologically feasible and well-posed and we also showed the existence of the disease-free equilibrium. Furthermore, we employed the next generation matrix technique to derive the reproduction number R_0. We proved that the model has two equilibrium points; the disease-free equilibrium which is locally asymptotically stable whenever $R_0 < 1$, unstable otherwise giving rise to the existence of the endemic equilibrium for $R_0 > 1$. We performed the sensitivity analysis on the reproductive number, R_0. Our analyses revealed that the parameters birth or recruitment rate by vector, rate of contribution to *Vibrio cholera* in the aquatic environment, infectious rate of a vector, and the ingesting vibrios rate from aquatic environment by vectors have a highly positive influence on the reproduction number. Concentration of the bacteria i.e. *Vibrio cholera* in aquatic environment, and death rate of vibrios in aquatic environment are also highly sensitive over the reproduction number. This suggests that eradicating vector populations and disinfecting the aquatic environment is the most effective control method. We implemented and carried out the numerical simulations to confirm the theoretical analysis and explored more patterns of dynamical behaviours of our model. Numerical simulations were also used to examine the effect of the parameters of the model. The results showed that rates of ingesting vibrios from the safe environment by human and rates of ingesting vibrios from the contaminated/unsafe environment to vector have positive effects in disease transmission as the increase in their values contributes significantly to the spread of the cholera infections in the system. Consequently, the rate of exposure to contaminated water and the contribution of each infected vector to the aquatic environment must be decreased to achieve meaningful and effective control.

ACKNOWLEDGMENTS

Romain Glèlè Kakaï acknowledges the support of the Alexander von Humboldt Foundation, Germany through the SEMCA Research Hub.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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