LOCAL STABILITY AND SENSITIVITY ANALYSIS OF CHOLERA DISEASE DYNAMICS WITH LOGISTIC GROWTH

Tolulope D. Ogunniyi[1*], Jamiu A. Ademosu[2] and Idris A. Lawal[1]

[1]Department of Pure and Applied Mathematics, Ladoke Akintola University of Technology, Ogbomoso, Nigeria

[2]Department of Mathematics, Lagos State University of Science and Technology, Ikorodu, Nigeria

*Corresponding author: **Tolulope D. Ogunniyi**[1*]

ABSTRACT

Cholera remains a significant public health threat, particularly in regions with poor sanitation and limited healthcare access. This paper presents and analyses a deterministic compartmental model for cholera disease dynamics, incorporating logistic growth of *Vibrio cholerae*. The model is formulated as a system of ordinary differential equations stratified into susceptible, exposed, infectious, vaccinated, and recovered human compartments, with a compartment for the bacteria concentration. The positivity of solutions is established to confirm the mathematical well-posedness of the model. The effective reproduction number R_e is computed using the next generation matrix operator, and the cholera-free equilibrium is shown to be locally asymptotically stable whenever $R_e < 1$. Normalised forward sensitivity analysis is performed to quantify the relative influence of model parameters on R_e. Parameters associated with vaccination, recovery rate, and bacteria death rate yield negative sensitivity indices, indicating that their increase drives R_e below unity. Conversely, parameters associated with transmission probability, human recruitment, and bacteria shedding rate yield positive sensitivity indices, such that their reduction is effective in curtailing transmission.

Keywords: Mathematical model, Cholera, Effective reproduction number, Stability, Sensitivity analysis

1. Introduction

Cholera is a waterborne infectious disease caused by the bacterium V. cholerae as depicted. These bacteria natural habitat are warm, salty marine surroundings, such as salt and brackish water [1]. Cholera is mainly spread through the consumption of contaminated water or food. Often times, environment with poor sanitation and polluted water sources with feces (human waste) makes the disease proliferate [2]. The disease transmission dynamics requires several contacts between human beings and the environment and the causative agent responsible for the proliferation of the disease in both routes [3]. According to WHO, it was reported that there are about 1.3 – 4 million cases of cholera and nearly 21,000 to 143,000 deaths recorded globally every year [4]. Mathematical modelling can be defined as the method of creating a simplified illustration of real-life scenarios which can be interpreted by variables, parameters and functions. Mathematical modelling plays a crucial role in guiding our decision-making processes for facilitating and implementing intervention strategies aimed at the effective control and curbing of infectious

diseases, social vices and ecological problems [5-9]. Many researches over the years formulated and analysed the transmission dynamics of cholera through mathematical modelling studies.

Okolo *et al.* [10] designed a deterministic model of cholera infection dynamics by integrating four control intervention policies: public health education, vaccination of susceptible persons, treatment of infected individuals, and water sanitation. Hidayati *et al.* [11] developed a Susceptible-Infected-Recovered model integrating the birth rate to regulate the susceptibility by vaccination. The model was formulated to understand the dynamic spread of cholera disease without vaccination. Maliki and Chibuike [12] formulated and examined a $SIR-B$ mathematical model to regulate the transmission of cholera without natural recovery. The model integrated key parameters such as treatment, water hygiene, and sanitation efforts, and to study its stability properties, a system of nonlinear ordinary differential equation was introduced. A comprehensive $SIR-B$ epidemiological model for cholera transmission dynamics was developed by [13], focusing on the impact of immigration and the relevance of the intervention measures. The model presented two equilibrium points: disease-free equilibrium (DFE) and endemic equilibrium (EE), both of which depended on the basic reproductive number, R_o, for their local stabilities.

Demir [14] designed a modified Susceptible-Infected-Recovered epidemic diffusion model for cholera. The model integrated migration dynamics among susceptible humans within a metapopulation structure of two remote cities. In [15], formulated a mathematical model for cholera dynamic spread considering a vaccination strategy was formulated. It is a $SVIR-B_{hi}B_{li}$ model showcasing the hyper-infectious and less infectious bacteria. In [16], a deterministic mathematical model featuring direct and indirect transmission routes was designed to analyse the cholera transmission dynamics using the Holling functional response of type III. Issam *et al.* [17] focused on developing control strategies for a discrete mathematical model of cholera transmission. The model was categorised into four compartments namely susceptible, infected, treated, and recovered populations. Hameed *et al.* [18] developed the logistic and Gompertz epidemic models to examine the spread and control of cholera pandemic in Iraq. These models were developed to consider critical epidemic factors, such as the number of infections, intrinsic growth rate, and carrying capacity. A within-host cholera mathematical model was proposed by considering vaccine efficacy in [19]. The developed model was well-posed as it was revealed to be both positive and bounded.

In [20], a $SVIR-B$ mathematical model of cholera transmission considering medication and environmental hygienic effort was designed and analysed. The model was examined to categorise the equilibrium points, sensitivity, numerical simulation, and the effective reproduction number. The rise of antibiotic resistance has become a major concern in cholera treatment, especially with a drop in pharmaceutical companies. Mushanyu *et al.* [21] developed a multidrug-resistant (MDR) cholera model with stage-switching of two antibiotics to mitigate the resistance. Brhane *et al.* [22] formulated a mathematical model that addresses the dynamics of *Vibrio cholerae* within a certain population, integrating key factors like bacteria growth, hygiene, treatment, and vaccination rates.

The model was tested using real data of cholera case, and sensitivity analysis highlighted parameters influencing the disease spread. Mgumba and Opoku [23] extended an existing $SIR-B$ model to understand the impact of Rural Community Information Dissemination Centers (RCIDCs) on cholera transmission. A mathematical analysis was executed to ascertain the positivity, boundedness and reproduction number R_o of the model.

The model presented in this work consists of six compartments in humans, namely susceptible, exposed, infectious, vaccinated, recovered populations, and the concentration of *Vibrio cholerae*. The organisation of the other aspects of the study is structured as follows: Section 2 presents the mathematical formulation of the cholera model. Section 3 provides the model analysis, including the derivation of the effective reproduction number R_e, local stability of the cholera-free equilibrium, and normalised forward sensitivity analysis. Section 4 presents the results and discussion, and Section 5 concludes the study.

2. Model Formulation

Mathematical model for cholera transmission is formulated using the human and bacteria dynamics. The total human population at time t, denoted by $N(t)$, is sub-grouped into five different compartments, namely the susceptible humans, denoted by $S(t)$; exposed humans, denoted by $E(t)$; infectious humans at time t, denoted by $I(t)$; vaccinated humans, denoted by $V(t)$; and recovered humans, denoted by $R(t)$. The bacteria dynamics illustrate the concentration of *Vibrio cholerae* in the environment at time t, denoted by $B(t)$. As a consequence of the foregoing assumptions and combination of all the equations, a non-linear system of ordinary differential equations describing the transmission dynamics of cholera is given in (1).

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - (\beta I + \beta B)S - \nu S - \mu S + \theta R, \\
\frac{dE}{dt} &= (\beta I + \beta B)S - \alpha E - \mu E, \\
\frac{dI}{dt} &= \alpha E - \tau I - \delta I - \mu I, \\
\frac{dV}{dt} &= \nu S - \mu V, \\
\frac{dR}{dt} &= \tau I - \theta R - \mu R, \\
\frac{dB}{dt} &= \varepsilon I + rB\left(1 - \frac{B}{c}\right) - \sigma B.
\end{aligned} \tag{1}$$

The descriptions of variables and parameters of the model are given in Table 1 and Table 2, respectively.

Table 1: The description of variables of the cholera model (1)

Variable	Description
$S(t)$	Susceptible human population
$E(t)$	Exposed human population
$I(t)$	Infectious human population
$V(t)$	Vaccinated human population
$R(t)$	Recovered human population
$B(t)$	Concentration of *Vibrio cholerae*

Table 2: The description of parameters of the cholera model (1)

Parameter	Description
Λ	Recruitment rate
β	Contact rate
ν	Per capita vaccination rate
μ	Per capita natural death rate
θ	Per capita loss of immunity rate
α	Per capita progression rate from exposed to infected class
τ	Per capita recovery rate
δ	Per capita induced death rate by the infection
ε	Shedding rate of *Vibrio cholerae* by humans to the environment
r	Per capita intrinsic growth rate of the bacteria
c	Carrying capacity of bacteria
σ	Per capita natural death rate of *Vibrio cholerae*

2.1. Positivity of Solutions

It is needed to establish that the model is epidemiologically and mathematically well-posed by showing that its solutions are nonnegative. Hence, the need to prove that all state variables of cholera are nonnegative for all time t.

Theorem 1: Let $S(0)\geq 0, E(0)\geq 0, I(0)\geq 0, V(0)\geq 0, R(0)\geq 0, B(0)\geq 0,$ then the solution set $\{S(t), E(t), I(t), V(t), R(t), B(t)\}$ of the system (1) is non-negative for all time $t>0$.

Proof:

From the first equation of the system (1) given by

$$\frac{dS}{dt} = \Lambda - (\beta I + \beta B)S - \nu S - \mu S + \theta R \,.$$

Then, the following differential inequality is obtained

$$\frac{dS}{dt} \geq -(\lambda + \nu + \mu)S \,. \tag{2}$$

where $\lambda = \beta(I + B)$.

Solving (2) using the method of integrating factor gives

$$S(t) \geq S(0)\left[\exp\left(-\left(\lambda(\omega) + (\nu + \mu)t\right)\right)\right] > 0 \text{, for all time } t>0. \tag{3}$$

The remaining compartments of the cholera model (1) are given as follows.

$$E(t) \geq E(0)\left[\exp\left(-(\alpha + \mu)t\right)\right] > 0 \text{, for all time } t>0.$$

$$I(t) \geq I(0)\left[\exp\left(-(\tau + \delta + \mu)t\right)\right] > 0 \text{, for all time } t>0.$$

$$V(t) \geq V(0)\left[\exp\left(-(\mu)t\right)\right] > 0 \text{, for all time } t>0. \tag{4}$$

$$R(t) \geq R(0)\left[\exp\left(-(\theta + \mu)t\right)\right] > 0 \text{, for all time } t>0.$$

$$B(t) \geq \frac{1}{\left(\frac{\sigma c + rB(0)}{\sigma cB(0)}\right)\exp(\sigma t) - \frac{r}{\sigma c}} \geq 0 \text{, for all time } t=0.$$

Hence, the solution set $\{S(t), E(t), I(t), V(t), R(t), B(t)\}$ of the cholera model (1) is non-negative for all time $t>0$. This concludes the proof.

3. Model Analysis

It is imperative to state that all the associated parameters of the cholera model are non-negative since the model addresses the interaction between human (host) and *Vibrio cholerae* (bacteria) concentration.

3.1 Effective reproduction number for the cholera model

Effective reproduction number is defined as the average number of new cases an index individual or a primarily infected person can produce during its state of infectiousness in a mixed population, that is, a susceptible and vaccinated environment [24,25]. Here, the effective reproduction number, usually designated by R_e, which is a pertinent notion in the study of epidemiological models will

be calculated using the celebrated next generation operator method and notations in [26]. In order to do this, the infection related compartments of the model are considered as presented hereunder

$$\left.\begin{aligned} \frac{dE}{dt} &= (\beta I + \beta B)S - \alpha E - \mu E, \\ \frac{dI}{dt} &= \alpha E - \tau I - \delta I - \mu I, \\ \frac{dB}{dt} &= \varepsilon I + rB\left(1 - \frac{B}{c}\right) - \sigma B, \end{aligned}\right\} \tag{5}$$

The transmission matrix f of the model equation (5) is presented as follows

$$f = \begin{pmatrix} \beta SI + \beta SB \\ 0 \\ 0 \end{pmatrix}. \tag{6}$$

Then, the Jacobian matrix of f evaluated at disease-free is given by

$$F = \begin{pmatrix} 0 & \frac{\beta\Lambda}{\nu+\mu} & \frac{\beta\Lambda}{\nu+\mu} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}. \tag{7}$$

Also, matrix of the transfer of infection is given by

$$v = \begin{pmatrix} (\alpha+\mu)E \\ (\tau+\delta+\mu)I - \alpha E \\ \sigma B - \varepsilon I - rB\left(1 - \frac{B}{c}\right) \end{pmatrix}. \tag{8}$$

Further, the Jacobian matrix of (8) evaluated at disease-free is obtained as

$$V = \begin{pmatrix} k_1 & 0 & 0 \\ -\alpha & k_2 & 0 \\ 0 & -\varepsilon & k_3 \end{pmatrix}, \tag{9}$$

where $k_1 = (\alpha+\mu)$, $k_2 = (\tau+\delta+\mu)$, $k_3 = (\sigma - r)$.

Therefore, the inverse of the transition matrix is obtained as presented below

$$V^{-1} = \frac{1}{|V|} \times AdjV.$$

Then,

$$V^{-1}=\begin{pmatrix} \frac{1}{k_1} & 0 & 0 \\ \frac{\alpha}{k_1k_2} & \frac{1}{k_2} & 0 \\ \frac{\alpha\varepsilon}{k_1k_2k_3} & \frac{\varepsilon}{k_2k_3} & \frac{1}{k_3} \end{pmatrix}. \tag{10}$$

Obtaining the product of FV^{-1}

$$FV^{-1}=\begin{pmatrix} \frac{\beta\Lambda\alpha(k_3+\varepsilon)}{k_1k_2k_3(\nu+\mu)} & \frac{\beta\Lambda k_3+\beta\Lambda\varepsilon(\nu+\mu)}{k_2k_3(\nu+\mu)} & \frac{\beta\Lambda}{k_3(\nu+\mu)} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}. \tag{11}$$

Next, obtain the eigenvalues of FV^{-1}, using the characteristic equation $\left|FV^{-1}-\lambda I\right|=0$.

Then, it follows that

$$FV^{-1}=\begin{vmatrix} \frac{\beta\Lambda\alpha(k_3+\varepsilon)}{k_1k_2k_3(\nu+\mu)}-\lambda & \frac{\beta\Lambda k_3+\beta\Lambda\varepsilon(\nu+\mu)}{k_2k_3(\nu+\mu)} & \frac{\beta\Lambda}{k_3(\nu+\mu)} \\ 0 & 0-\lambda & 0 \\ 0 & 0 & 0-\lambda \end{vmatrix}=0. \tag{12}$$

Further simplification of (1) gives

$$\lambda_1=0,\lambda_2=0,\lambda_3=\frac{\beta\Lambda\alpha(k_3+\varepsilon)}{k_1k_2k_3(\nu+\mu)}. \tag{13}$$

The maximum eigenvalue of the characteristic equation is therefore, given by

$$\lambda_3=\frac{\beta\Lambda\alpha(k_3+\varepsilon)}{k_1k_2k_3(\nu+\mu)}. \tag{14}$$

Since effective reproduction number is the spectral radius of FV^{-1}, therefore the effective reproduction number of the cholera model (1) is given by $R_e=\max\{\lambda_i\}$.

Therefore,

$$R_e=\frac{\beta\Lambda\alpha(k_3+\varepsilon)}{k_1k_2k_3(\nu+\mu)}. \tag{15}$$

3.2. Local stability of cholera-free equilibrium

Theorem 2: The cholera-free equilibrium, $\mathcal{E}_0$, of the cholera model (1) is locally asymptotically stable (LAS) in D if R_e, given in (15) is below unity and unstable otherwise.

Proof:

In order to determine the locally asymptotically stability of the cholera-free equilibrium of the cholera model (1), the following system of differential equations is considered

$$\left.\begin{aligned} f_1 &= \frac{dS}{dt} = \Lambda - (\beta I + \beta B)S - \nu S - \mu S + \theta R, \\ f_2 &= \frac{dE}{dt} = (\beta I + \beta B)S - \alpha E - \mu E, \\ f_3 &= \frac{dI}{dt} = \alpha E - \tau I - \delta I - \mu I, \\ f_4 &= \frac{dV}{dt} = \nu S - \mu V, \\ f_5 &= \frac{dR}{dt} = \tau I - \theta R - \mu R, \\ f_6 &= \frac{dB}{dt} = \varepsilon I + rB\left(1 - \frac{B}{c}\right) - \sigma B. \end{aligned}\right\} \tag{16}$$

The Jacobian matrix of the system (16) evaluated at ε_0 is given by

$$J_{\varepsilon_0} = \begin{pmatrix} -(\nu+\mu) & 0 & \frac{-\beta\Lambda}{\nu+\mu} & 0 & \theta & \frac{-\beta\Lambda}{\nu+\mu} \\ 0 & -k_1 & \frac{\beta\Lambda}{\nu+\mu} & 0 & 0 & \frac{\beta\Lambda}{\nu+\mu} \\ 0 & \alpha & -k_2 & 0 & 0 & 0 \\ \nu & 0 & 0 & -\mu & 0 & 0 \\ 0 & 0 & \tau & 0 & -(\theta+\mu) & 0 \\ 0 & 0 & \varepsilon & 0 & 0 & -k_3 \end{pmatrix}. \tag{17}$$

Now, using the characteristic equation

$$|J|_{\varepsilon_0} - \lambda I| = 0 \tag{18}$$

Then, the resulting Jacobian matrix (17) becomes

$$J_{\varepsilon_0} = \begin{pmatrix} -(\nu+\mu)-\lambda & 0 & \frac{-\beta\Lambda}{\nu+\mu} & 0 & \theta & \frac{-\beta\Lambda}{\nu+\mu} \\ 0 & -k_1-\lambda & \frac{\beta\Lambda}{\nu+\mu} & 0 & 0 & \frac{\beta\Lambda}{\nu+\mu} \\ 0 & \alpha & -k_2-\lambda & 0 & 0 & 0 \\ \nu & 0 & 0 & -\mu-\lambda & 0 & 0 \\ 0 & 0 & \tau & 0 & -(\theta+\mu)-\lambda & 0 \\ 0 & 0 & \varepsilon & 0 & 0 & -k_3-\lambda \end{pmatrix} = 0. \tag{19}$$

Clearly, three of the eigenvalues of the resulting Jacobian matrix are obtained as

$$\left(-(\nu+\mu)-\lambda\right)\left(-\mu-\lambda\right)\left(-(\theta+\mu)-\lambda\right) = 0, \tag{20}$$

and the remaining eigenvalues of the Jacobian matrix is given by

$$\begin{vmatrix} -k_1-\lambda & \frac{\beta\Lambda}{\nu+\mu} & \frac{\beta\Lambda}{\nu+\mu} \\ \alpha & -k_2-\lambda & 0 \\ 0 & \varepsilon & -k_3-\lambda \end{vmatrix} = 0. \quad (21)$$

Further simplification of (21) gives

$$\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0. \quad (22)$$

Therefore, the polynomial $P(\lambda)$ of order three obtained in (22) is the solution of the characteristic equation (21), where

$$A_1 = k_1 + k_2 + k_3 > 0,$$

$$A_2 = k_1k_2 + k_1k_3 + k_2k_3 - \frac{\beta\Lambda\alpha}{\nu+\mu} > 0,$$

$$A_3 = k_1k_2k_3(1-R_e) > 0.$$

Clearly, it follows using Descarte's rule of signs [27] that the eigenvalues are negative, real and distinct, since all the coefficients of the polynomial are positive. Therefore, the cholera-free equilibrium is locally asymptotically stable whenever $R_e < 1$ in A_3. Hence, the proof.

3.3 Sensitivity Analysis

The influence of certain parameters of the cholera model relative to the effective reproduction number is examined through sensitivity analysis using the normalized forward sensitivity index. By definition, the normalized forward sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter [28]. The normalized forward sensitivity index of the effective reproduction number, R_e, relative to its parameter, p, is given by

$$\Gamma_p^{R_e} = \frac{\partial R_e}{\partial p} \times \frac{p}{R_e}, \quad (23)$$

Recall that,

$$R_e = \frac{\beta\Lambda\alpha(K_3+\varepsilon)}{k_1k_2k_3(\nu+\mu)},$$

with $k_1 = (\alpha+\mu), k_2 = (\tau+\delta+\mu), k_3 = (\sigma-r)$. Upon substitution of k_1, k_2 and k_3 into the effective reproduction number, it follows that

$$R_e = \frac{\beta\Lambda\alpha(\sigma-r+\varepsilon)}{(\alpha+\mu)(\tau+\delta+\mu)(\sigma-r)(\nu+\mu)}. \quad (24)$$

Particularly, the sensitivity indices of the effective reproduction number relative to its associated parameter are investigated using the parameter values in Table 3.

Sensitivity of parameter Λ : Taking the partial derivative of (24) with respect to the parameter Λ gives

$$\frac{\partial R_e}{\partial \Lambda} = \frac{\beta\alpha\left(\sigma - r + \varepsilon\right)}{\left(\alpha+\mu\right)\left(\tau+\delta+\mu\right)\left(\sigma-r\right)\left(\nu+\mu\right)}.$$

Then, the sensitivity index for the parameter Λ is obtained as follows

$$\Gamma_{\Lambda}^{R_e} = \frac{\partial R_e}{\partial \Lambda} \times \frac{\Lambda}{R_e} = +1.0000.$$

Sensitivity of parameter β : Taking the partial derivative of (24) with respect to the parameter β gives

$$\frac{\partial R_e}{\partial \beta} = \frac{\Lambda\alpha\left(\sigma - r + \varepsilon\right)}{\left(\alpha+\mu\right)\left(\tau+\delta+\mu\right)\left(\sigma-r\right)\left(\nu+\mu\right)}.$$

Then, the sensitivity index for the parameter β is obtained as follows

$$\Gamma_{\beta}^{R_e} = \frac{\partial R_e}{\partial \beta} \times \frac{\beta}{R_e} = +1.0000.$$

Sensitivity of parameter α : Taking the partial derivative of (24) with respect to the parameter α gives

$$\frac{\partial R_e}{\partial \alpha} = \frac{\beta\Lambda\left(\sigma - r + \varepsilon\right)\left(\mu\right)}{\left(\alpha+\mu\right)^2\left(\tau+\delta+\mu\right)\left(\sigma-r\right)\left(\nu+\mu\right)}.$$

Then, the sensitivity index for the parameter α is obtained as follows

$$\Gamma_{\alpha}^{R_e} = \frac{\partial R_e}{\partial \alpha} \times \frac{\alpha}{R_e} = \frac{\mu}{\left(\alpha+\mu\right)}.$$

Sensitivity of parameter ν : Taking the partial derivative of (24) with respect to the parameter ν gives

$$\frac{\partial R_e}{\partial \nu} = \frac{-\beta\Lambda\left(\sigma - r + \varepsilon\right)}{\left(\alpha+\mu\right)\left(\tau+\delta+\mu\right)\left(\sigma-r\right)\left(\nu+\mu\right)^2}.$$

Then, the sensitivity index for the parameter ν is obtained as follows

$$\Gamma_{\nu}^{R_e} = \frac{\partial R_e}{\partial \nu} \times \frac{\nu}{R_e} = \frac{-\nu}{\left(\nu+\mu\right)}$$

Sensitivity of parameter τ: Taking the partial derivative of (24) with respect to the parameter τ gives

$$\frac{\partial R_e}{\partial \tau} = \frac{-\beta\Lambda\alpha\left(\sigma - r + \varepsilon\right)}{\left(\alpha+\mu\right)\left(\tau+\delta+\mu\right)^2\left(\sigma-r\right)\left(\nu+\mu\right)}.$$

Then, the sensitivity index for the parameter τ is obtained as follows

$$\Gamma_{\tau}^{R_e} = \frac{\partial R_e}{\partial \tau} \times \frac{\tau}{R_e} = \frac{-\tau}{\left(\tau+\delta+\mu\right)}.$$

Sensitivity of parameter δ : Taking the partial derivative of (24) with respect to the parameter δ gives

$$\frac{\partial R_e}{\partial \delta} = \frac{-\beta\Lambda\alpha\left(\sigma - r + \varepsilon\right)}{\left(\alpha+\mu\right)\left(\tau+\delta+\mu\right)^2\left(\sigma-r\right)\left(\nu+\mu\right)}.$$

Then, the sensitivity index for the parameter δ is obtained as follows

$$\Gamma_{\delta}^{R_e}=\frac{\partial R_e}{\partial \delta}\times\frac{\delta}{R_e}=\frac{-\delta}{(\tau+\delta+\mu)}.$$

Sensitivity of parameter ε : Taking the partial derivative of (24) with respect to the parameter ε gives

$$\frac{\partial R_e}{\partial \varepsilon}=\frac{\beta\Lambda\alpha}{(\alpha+\mu)(\tau+\delta+\mu)(\sigma-r)(\nu+\mu)}.$$

Then, the sensitivity index for the parameter ε is obtained as follows

$$\Gamma_{\varepsilon}^{R_e}=\frac{\partial R_e}{\partial \varepsilon}\times\frac{\varepsilon}{R_e}=\frac{\varepsilon}{(\sigma-r+\varepsilon)}.$$

Sensitivity of parameter σ : Taking the partial derivative of (24) with respect to the parameter σ gives

$$\frac{\partial R_e}{\partial \sigma}=\frac{\beta\Lambda\varepsilon}{(\alpha+\mu)(\tau+\delta+\mu)(\sigma-r)^2(\nu+\mu)}.$$

Then, the sensitivity index for the parameter σ is obtained as follows

$$\Gamma_{\sigma}^{R_e}=\frac{\partial R_e}{\partial \sigma}\times\frac{\sigma}{R_e}=\frac{-\varepsilon\sigma}{(\sigma-r)(\sigma-r+\varepsilon)}.$$

Sensitivity of parameter r : Taking the partial derivative of (24) with respect to the parameter r gives

$$\frac{\partial R_e}{\partial r}=\frac{-\beta\Lambda\alpha\varepsilon}{(\alpha+\mu)(\tau+\delta+\mu)(\sigma-r)^2(\nu+\mu)}.$$

Then, the sensitivity index for the parameter r is obtained as follows

$$\Gamma_{r}^{R_e}=\frac{\partial R_e}{\partial r}\times\frac{r}{R_e}=\frac{-\varepsilon r}{(\sigma-r)(\sigma-r+\varepsilon)}.$$

Sensitivity of parameter μ : Taking the partial derivative of (24) with respect to the parameter μ gives

$$\frac{\partial R_e}{\partial r}=\frac{-\beta\Lambda\alpha(\sigma-r+\varepsilon)\left[(\tau+\delta+\mu)(\nu+\mu)+(\alpha+\mu)(\nu+\mu)+(\alpha+\mu)(\tau+\delta+\mu)\right]}{(\alpha+\mu)^2(\tau+\delta+\mu)^2(\sigma-r)(\nu+\mu)^2}.$$

Then, the sensitivity index for the parameter μ is obtained as follows

$$\Gamma_{\mu}^{R_e}=\frac{\partial R_e}{\partial \mu}\times\frac{\mu}{R_e},$$

$$=\frac{-\mu\left[(\tau+\delta+\mu)(\nu+\mu)+(\alpha+\mu)(\nu+\mu)+(\alpha+\mu)(\tau+\delta+\mu)\right]}{(\alpha+\mu)(\tau+\delta+\mu)(\nu+\mu)}.$$

4. Result and Discussion

A novel mathematical model focusing on the time-evolution of cholera dynamics with logistic growth rate based on six-dimensional system of ordinary differential equations was formulated. The feasible region D established the well-posedness of the model in Equation (1), following the positivity of solutions of the model. The threshold parameter known as effective reproduction number (R_e) for the cholera model was determined, employing the renowned next generation

matrix operator [26], as illustrated in Equation (15). This threshold parameter measures the spread potential of cholera in the populations of susceptible and vaccinated environment.

Table 3: Values of parameters of the cholera model.

Parameter	Value	Source
Λ	10	[17, 29, 30]
β	0.00011	[31]
ν	0.2	[22]
μ	0.0000548	[29]
θ	0.00055	[31]
α	0.2	[4]
τ	0.2	[30]
δ	0.015	[29]
ε	50	[22]
r	0.2	[22]
c	$1\,0^{5}$	Assumed
σ	0.7	Assumed

Table 4: Sensitivity indices of parameter

Parameter	**Sensitivity Index**
Λ	+1.0000
β	+1.0000
ν	-0.9997
μ	-0.000802
α	+0.000274
τ	-0.9299
δ	-0.06975
ε	+0.9901
r	+0.39603
σ	-1.3861

The local asymptotic stability analysis for the cholera-free equilibrium point of the model was carried out using the linearized Jacobian matrix approach. As specified in Theorem 2, the cholera model (1) has a locally asymptotically stable cholera-free equilibrium. The consequence of this from the epidemiological viewpoint is that cholera can be managed in the population whenever

the initial sizes of the active cholera cases are in the basin of attraction of the cholera-free equilibrium such that $R_e<1$. In other words, this result implies that cholera disease can be eliminated in the population if the reproduction number is maintained below unity. This agrees with the results obtained in [20, 21]. By using the parameter values in Table 3, the sensitivity indices of the effective reproduction number, R_e, relative to its parameters in Table 4 inferred that there are parameters with positive and negative sensitivity indices. Parameters such as natural mortality rate for human, (μ), progression rate from exposed population to infectious population, (α), death due to cholera, (δ), recovery due to treatment, (τ), and the vaccination rate, (ν), are having negative sensitivity indices, while other parameters, including intrinsic growth rate, (r), recruitment rate, (Λ) and contact rate, (β) are with positive sensitivity indices, accordingly. It is worth noting that, increase (decrease) in the values of parameters with positive sensitivity indices led to a corresponding increase (decrease) in the value of the associated effective reproduction number. While increase (decrease) in the values of parameters with negative sensitivity indices led to a corresponding decrease (increase) in the value of the effective reproduction number. This result is in agreement with [3].

In particular, increasing β representing effective contact 100% for instance, will yield a corresponding rise in the value of the effective reproduction number by 100%. The practical implication of this from the epidemiological point of view, is that cholera will be more prevalent in the population as long as efforts are not made to address the factors contributing to the spread of the disease in the population. A 100% increase in recruitment rate, (Λ) will result in approximately a 99.01% increase in the effective reproduction number, while a 100% decrease will lead to an approximately 99.01% decrease. Epidemiologically, an increase in the recruitment of susceptible individuals into the population increases the number of people at risk of infection, thereby facilitating the transmission of cholera. Increasing the recovery rate due to treatment, γ, by 100% will result in approximately a 92.99% decrease in the effective reproduction number. Conversely, a reduction in the recovery rate will increase the effective reproduction number. From an epidemiological perspective, increasing the recovery rate through effective treatment and improved healthcare access reduces the infectious period of infected individuals, thereby limiting the transmission of cholera within the population.

5. Conclusion

A new mathematical model which is a six compartmental deterministic time-invariant system describing the time-evolution of cholera dynamics has been formulated and examined extensively. The autonomous system involves the human population and the bacteria concentration. The model stratified the total human population at time t, denoted by $N(t)$, into five mutually exclusive compartments, including susceptible population, $S(t)$, exposed population, $E(t)$, infectious population, $I(t)$, vaccinated population, $V(t)$, and recovered population, $R(t)$, respectively. While the concentration of *Vibrio cholerae,* $B(t)$, has one compartment.

The cholera model was first demonstrated to be mathematically well-posed and epidemiologically meaningful through the theory of positivity. The autonomous cholera model was revealed to have a locally asymptotically stable cholera-free equilibrium point when the effective reproduction number is less than unity. Furthermore, effects of some key epidemiological parameters of the model on the effective reproduction number were investigated using the normalized forward sensitivity index. Positive and negative sensitivity indices were obtained for the parameters, where it was shown that increase in the value of the parameters with positive sensitivity indices resulted in a corresponding increase in the value of the effective reproduction number. This, epidemiologically, indicates that cholera may become endemic in the population if efforts are not put in place to hamper the surge of the parameters that are capable of increasing the effective reproduction number. Further extension to the model formulation and analysis could be done by considering fractional order framework [33,34] in order to account for the memory effects inherent in the cholera transmission dynamics.

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