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RESEARCH ARTICLE

Stability Analysis of SVEITR Model for Cholera Control with Treatment and Vaccination Using Laplace Adomian Decomposition Method

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Abstract:
This study presents a mathematical analysis of the SVEITR model, which incorporates susceptible, vaccinated, exposed, infected, treatment, and recovered populations to evaluate the dynamics of cholera spread. By integrating treatment and vaccination rates into the model, we aim to understand their impact on disease transmission and immunity. Our findings reveal that combining rapid treatment and vaccination significantly reduces the spread of cholera, highlighting the importance of these interventions in public health strategies. The model demonstrates that timely and widespread implementation of vaccination and treatment can effectively control outbreaks and mitigate the disease's impact. Through a numerical simulation of Laplace decomposition method the result reveal that treatment rate reduces the emanation of the disease and vaccination plays a vital role in curbing aftermath effect of wide-spread of the disease. Hence, the need for robust healthcare policies that prioritize these measures to achieve substantial progress in managing and eventually eradicating cholera, particularly in vulnerable regions. The SVEITR model provides a valuable framework for policymakers and healthcare professionals to devise efficient strategies for cholera control, contributing to improved public health outcomes.

Keywords: Cholera Control, Treatment Efficacy, Vaccination, Stability Analysis, Laplace Adomian Decomposition Method

1. Introduction

Cholera, a highly infectious disease caused by the bacterium Vibrio cholerae, which remains a public health challenge, particularly in regions with inadequate water and sanitation infrastructure. Characterized by severe diarrhea and dehydration, cholera can lead to death within hours if untreated. The disease primarily spreads through the consumption of contaminated water and food, making its control closely linked to the quality of water supply, sanitation, and hygiene practices in [1, 2]. Despite advancements in medical science and public health strategies, cholera outbreaks continue to pose substantial health risks, particularly in developing countries. The focus of contemporary

cholera research includes treatment efficacy, vaccination, water and environmental cleanliness, regional enlightenment, regular hand washing, proper waste disposal, and public awareness [3]. These components are critical in developing a comprehensive approach to control and prevent the disease effectively. Effective treatment of cholera involves prompt rehydration, which can be lifesaving. Oral rehydration salts (ORS) are the cornerstone of treatment for most patients, while intravenous fluids are necessary for severe cases. Antibiotics can also reduce the duration of diarrhea and the volume of rehydration fluids needed. Research into optimizing these treatments is ongoing, aiming to enhance their efficacy and accessibility, especially in resource-limited settings by [4–6]. The goal is to ensure that treatment protocols are both efficient and adaptable to various healthcare infrastructures, thus reducing mortality and morbidity associated with cholera. Vaccination is a crucial tool in cholera prevention [7]. Oral cholera vaccines (OCVs) have proven effective in providing immunity and reducing the incidence of the disease as in [8]. Integrating vaccination into public health strategies, particularly in high-risk areas, can prevent outbreaks and provide long-term protection. Ongoing research focuses on improving the efficacy and duration of vaccine-induced immunity, as well as logistics to enhance vaccine distribution and administration in endemic regions [9–11]. The success of vaccination campaigns depends on the timely and widespread coverage, particularly before and during outbreaks [12]. Clean water and proper sanitation are fundamental in preventing cholera transmission. Contaminated water sources are the primary vectors for the bacterium, highlighting the need for robust water treatment and safe water storage practices by [13–16]. Efforts to improve water quality through filtration, chlorination, and ensuring safe water access are critical. Additionally, environmental cleanliness, including the maintenance of clean living conditions and proper sanitation facilities, is essential as [17–19]. Public health initiatives must focus on infrastructure development and community education to promote sustainable practices that ensure water and environmental cleanliness. Educating communities about cholera prevention and control is vital. Regional enlightenment campaigns can significantly impact public health by raising awareness about the disease, its transmission, and preventive measures by [20, 21]. These campaigns should focus on informing individuals about the importance of using safe water, practicing good hygiene, and recognizing the symptoms of cholera for prompt treatment. Tailored educational programs that consider local customs and practices can enhance community engagement and compliance with preventive measures [22]. Hand washing with soap and clean water is one of the simplest yet most effective ways to prevent the spread of cholera. Regular hand washing, particularly before eating and after using the toilet, can significantly reduce the transmission of the bacterium [23, 24]. Public health campaigns must emphasize the importance of this practice and ensure that communities have access to soap and clean water. Installing hand washing stations in public places and schools can also promote this essential hygiene practice. Proper waste disposal is crucial in preventing cholera outbreaks. Improperly disposed of human waste can contaminate water sources, facilitating the spread of Vibrio cholerae [25, 26]. Implementing effective waste management systems, including the use of latrines and sewage treatment facilities, is vital. Public health initiatives should focus on constructing and maintaining these facilities and educating communities about the importance of proper waste disposal in [27]. Safe disposal practices help break the transmission cycle and reduce the risk of outbreaks. Awareness campaigns play a critical role in cholera prevention. Informing the public about the disease, its symptoms, and the importance of seeking immediate treatment can save lives [28–31]. Awareness efforts should also highlight the preventive measures individuals can take to protect themselves and their communities. Utilizing various media platforms, including radio, television, social media, and community outreach programs, can effectively disseminate information and reach a broad audience [32–34]. However, controlling cholera requires a multifaceted approach that includes effective treatment, vaccination, water and environmental cleanliness, regional enlightenment, regular hand washing, proper waste disposal, and public awareness [35]. By addressing these areas, public health initiatives can significantly reduce the incidence and impact of cholera,

ultimately aiming for its eradication. Continued research and investment in these strategies are essential to overcoming the persistent threat posed by cholera, particularly in vulnerable regions.

2. Materials and Method

2.1. Model Formulation

The total population $N(t)$ is distinctly divided into six sub-compartments of population sizes of are Susceptible $S(t)$, Vaccinated $V(t)$, Exposed $E(t)$, Infected $I(t)$, Hospitalized/Treatment $T(t)$ and Recovered population $R(t)$, The rate of migration π or inflow into the population resulting to the spread of cholera is β as human population are exposed through contaminated water and the hygienic measure put into practice to avoid ingestion and reduce the contact rate of the disease. Logistic coverage of public awareness of infected individual at a rate of δ where an exposed individuals are subjected to contracting cholera at k and rate of recovery denoted with γ, the treatment rate of the hospitalized individuals ϕ_2. More than 75% of contamination risk of vibro-cholerae resulting to spread of the disease and ϕ_1 vaccination rate of susceptible individuals, with ε representing the vaccine efficacy and shedding rate of infected human population coupled with natural mortality rate for human and vibro-cholerae are φ and μ.The above parameters can be demonstrated with schematic flow of figure 1 and a system of nonlinear differential equations in equation 1 below respectively.

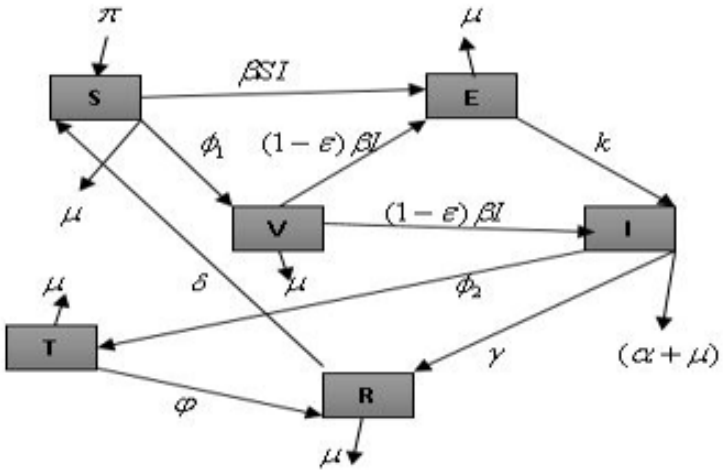

Figure 2.1: Schematic diagram illustrating the model formulation

$$\begin{aligned}
\frac{dS}{dt} &= \pi - \beta SI - (\phi_1 + \mu)S + \delta R \\
\frac{dV}{dt} &= \phi_1 S - (1-\varepsilon)\beta IV - \mu V \\
\frac{dE}{dt} &= (1-\varepsilon)\beta IV + \beta SI - (k+\mu)E \\
\frac{dI}{dt} &= kE - (\alpha + \mu + \gamma + \phi_2)I \\
\frac{dT}{dt} &= \phi_2 I - (\varphi + \mu)T \\
\frac{dR}{dt} &= \varphi T + \gamma I - (\delta + \mu)R
\end{aligned} \tag{2.1}$$

Subjected to the initial condition $S(0) = s_0, V(0) = v_0, E(0) = e_0, I(0) = i_0, T(0) = t_0, R(0) = r_0 \geq 0$

Table 2.1: Parameters Description, Values and References

Variable	Description
$S(t)$	Susceptible (Vulnerable) population
$V(t)$	Vaccinated population
$E(t)$	Exposed population
$I(t)$	Infected population
$T(t)$	Treatment (Hospitalized) population
$R(t)$	Recovered population

Parameter	Description	Values	References
N	Total population	80,000	[11, 29]
ϕ	Recovery rate of hospitalized individuals	0.001	[2]
ε	Vaccination efficacy	0.5	[9]
φ_2	Treatment rate of hospitalized individuals	0.2	[1, 3, 16]
φ	Vaccination rate of infected individuals	0.03	[3, 5]
μ	Natural death	1.0	[4, 25, 30]
δ	Immunity waning rate	0.0016	[26, 32]
π	Recruitment rate	0.113	[6, 23]
β	Rate of cholera transmission	1.0126	[13]
α	Disease induced death rate	0.33182	[31]
γ	Natural recovery rate	0.16524	[18, 21]
k	Progression rate between exposed and infected class	0.25533	[10]

2.2. Existence and Uniqueness of Model Solution

The system (2.1), which desribes an epidemic disease within a human population, should have parameters than are nonnegative. To ensure that the system of differential equations in (2.1) is both mathematically and epidemiologically wel-posed, it is essential to demonstrate that the state variables in the model are nonnegative. System (2.1) is well-posed when system starts. Nonnegative initial condition $S(0) = s_0, V(0) = v_0, E(0) = e_0, I(0) = i_0, T(0) = t_0, R(0) = r_0 \geq 0$; In the case, the solutions of system (2.1) will persist in being nonnegative throughout their evolution, $t > 0$ and that these positive solutions are bounded. We thu apply the following theorems.

Theorem 2.1. *Let (x, y) be distinct points of normed linear space $(X, ||\ldots||)$ over $\mathcal{R}$. Then the map of $p : [0, 1] \subseteq \mathcal{R} \to (X, ||\ldots||)$ such that $p(\lambda) = \lambda x + (1 - \lambda)y$ is continuous on $[0, 1]$.*

Proof

Let $\lambda_0 \in [0, 1]$ then $p(\lambda_0) = \lambda_0 x + (1 - \lambda_0)y$ for any $\lambda_0 \in [0, 1]$

$$||p(\lambda) - p(\lambda_0)|| = ||(\lambda - \lambda_0)x + (\lambda - \lambda_0)y|| \leq |\lambda - \lambda_0|(||x|| + ||y||)$$

If $\varepsilon > 0$ is given, let $\delta = \frac{\varepsilon}{||x||+||y||}$. Jika $|\lambda - \lambda_0| < \delta$, then the $||p(\lambda) - p(\lambda_0)|| < \varepsilon$. Therefore, p is continuous at λ_0. Since λ_0 is arbitrary point in $[0, 1]$. Then p is continuous on $[0, 1]$. Let X be a linear space over $\mathcal{R}$. If (x, y) are distinct points of X, the set $\lambda x + (1 - \lambda)y$ lies in $0 \leq \lambda \leq 1$. Hence, the solutions of system (2.1) are bounded if we consider the total population

$$N(t) = S(t) + V(t) + E(t) + I(t) + T(t) + R(t) \tag{2.2}$$

The variation in the total population concerning time is given by:

$$\frac{dN(t)}{dt} = \frac{d}{dt}(S(t) + V(t) + E(t) + I(t) + T(t) + R(t)) \tag{2.3}$$

Such that $\frac{dN(t)}{dt} = \pi - \mu(S + V + E + I + T + R) - \alpha I \to \frac{dN(t)}{dt} \leq \pi - \mu N$. When no outbreak of cholera, $\delta = 0$. Thus, substituting (2.3) to (2.4) as time progressively increases yields:

$$\lim_{t\to\infty} N(t) \leq \lim_{t\to\infty} \left[\frac{\pi}{\mu} + \left(N(0) - \frac{\pi}{\mu}\right)e^{-\mu}\right] = \frac{\pi}{\mu} \tag{2.4}$$

If so $N(0) \leq \frac{\pi N}{\mu}$, as $N(t) \leq \frac{\pi N}{\mu}$. This is a positive invariant set under the flow described by (2.2) so that no solution path leaves through any boundary $\mathcal{R}_+^6$. However, it is sufficient to consider the dynamics of the model in the domain $\mathcal{R}_+^6$. In this region the model can be considered has been mathematically and epidemiologically well-posed.

2.3. Positivity and Boundedness of Invariance Region

This shows that the total population $N(t)$, and the subpopulation $S(t), E(t), I(t), T(t), R(t)$ of the model are bounded and is a unique solution. Hence, its applicability to study physical systems is feasible.

Theorem 2.2. *Suppose $X = x_0$ is a space of consecutive real number and which are defined as*

$$L(x,y) = \big(\sum_{i=1}^{n} |x_i|^{\Omega}\big)^{\frac{1}{\Omega}} \quad \Omega \geq 1 \tag{2.5}$$

X with the metric is called ξ_n^{Ω} space. If $\sum^{\infty} |x|^{\Omega} < \Omega$ or absolutely convergent and $L(x,y) = \big(\sum_{i=1}^{\infty} |x_i - y_i|^{\Omega}\big)^{\frac{1}{\Omega}}$, then X with this metric is called an ξ^{Ω} space.

Proof
It can be checked that for each n:

$$0 \leq x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2 \leq (|x_1| + |x_2| + |x_3| + \ldots + |x_n|)^2 \tag{2.6}$$

This will result to;

$$x_1^2 + x_2^2 \leq (|x_1| + |x_2)^2 \tag{2.7}$$

Therefore,

$$0 \leq (x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2)^{\frac{1}{2}} \leq |x_1| + |x_2| + |x_3| + \ldots + |x_n|, \tag{2.8}$$

These sequences x_n is monotone increasing and bounded above, it therefore converges. Thus $\sum_{n=1}^{\infty} x_n$ converges absolutely, if $x - n \in \xi^1$, then $x_n \in \xi^2$ where $\xi^1 \leq \xi^2$. In case of ξ^1 denote the set of all sequences of x_n of real numbers such that $\sum_{n=1}^{\infty} x_n$ is convergent absolutely. i.e $\sum_{n=1}^{\infty} |x_n| < \infty$ where as ξ^2 denote the set of all sequence x_n of real numbers such that $\sum_{n=1}^{\infty} x_n^2 < \infty$ converges. From the proceeding $x_n \in \xi^1 \Leftrightarrow x_n \in \xi^2$ i.e. $\xi^1 \subseteq \xi^2$. Further, if $x_n = \frac{1}{n^{\frac{3}{4}}}$, then $\sum_{n=1}^{\infty} |x_n|$ diverges and thus $x_n \notin \xi^1$. But $\sum_{n=1}^{\infty} x_n^2 = \sum_{n=1}^{\infty} \frac{1}{n^{\frac{3}{4}}}$ converges, implying that $x_n \in \xi^2$. We conclude that $\xi^1 \subseteq \xi^2$ and thus $\xi^1 \neq \xi^2$. If (x_n, y_n) are sequences of real numbers, then;

$$\sum_{n=1}^{\infty} (x_i - y_i)^2 \leq \sum_{n=1}^{\infty} x_i^2 + \sum_{n=1}^{\infty} y_i^2 + 2\big[\sum_{n=1}^{\infty} x_i^2\big]^{\frac{1}{2}} \big[\sum_{n=1}^{\infty} y_i^2\big]^{\frac{1}{2}}$$

Therefore if $\sum_{n=1}^{\infty} x_i^2 < \infty$ and $\sum_{n=1}^{\infty} y_i^2 < \infty$ then $\sum_{n=1}^{\infty} (x_i - y_i)^2 < \infty$ for all n. The monotone increasing sequence $\big[\sum_{n=1}^{\infty} (x_i - y_i)^2\big]$ is the bounded above and hence converges i.e. $\sum_{n=1}^{\infty} (x_i - y_i)^2 < \infty$. Thus $(x_i - y_i)^2 \in \xi^2$ if $(x_n, y_n) \in \xi^2$.
Given that the $S(0) = s_0 > 0, V(0) = v_0 > 0, E(0) = e_0 > 0, I(0) = i_0 > 0, T(0) = t_0 > 0, R(0) = r_0 > 0$, and $t > 0$, then the solutions $S(t), V(t), E(t), I(t), T(t), R(t)$ of the system (2.1) will always be nonnegative. Let:

$$\Psi = \big\{S(t), V(t), E(t), I(t), T(t), R(t) \in \mathcal{R}_+^6 : N(t) \leq \frac{\pi}{\mu}\big\} \tag{2.9}$$

If $f_i, i = 1, 2, \ldots, 6$ where f is a constant. Then

$$\begin{aligned}
\left|\frac{df_1}{dS}\right| &= |(\beta+\phi_1+\mu+\delta)<\infty, \left|\frac{df_1}{dV}\right| = |0|<\infty, \left|\frac{df_1}{dE}\right| = |0|<\infty, \left|\frac{df_1}{dI}\right| = |\alpha|<\infty, \\
&\quad \left|\frac{df_1}{dT}\right| = |\alpha|<\infty, \left|\frac{df_1}{dR}\right| = |\delta|<\infty \\
\left|\frac{df_2}{dS}\right| &= |\phi_1|<\infty, \left|\frac{df_2}{dV}\right| = |(1-\varepsilon)\beta+\mu|<\infty, \left|\frac{df_2}{dE}\right| = |0|<\infty, \left|\frac{df_2}{dI}\right| = |(1-\varepsilon)\beta|<\infty, \\
&\quad \left|\frac{df_2}{dT}\right| = |0|<\infty, \left|\frac{df_2}{dR}\right| = |0|<\infty \\
\left|\frac{df_3}{dS}\right| &= |\beta|<\infty, \left|\frac{df_3}{dV}\right| = |(1-\varepsilon)\beta|<\infty, \left|\frac{df_3}{dE}\right| = |(k+\mu)|<\infty, \left|\frac{df_3}{dI}\right| = |(1-\varepsilon)\beta|<\infty, \\
&\quad \left|\frac{df_3}{dT}\right| = |0|<\infty, \left|\frac{df_3}{dR}\right| = |0|<\infty \\
\left|\frac{df_4}{dS}\right| &= |0|<\infty, \left|\frac{df_4}{dV}\right| = |0|<\infty, \left|\frac{df_4}{dE}\right| = |k|<\infty, \left|\frac{df_4}{dI}\right| = |(\alpha+\gamma+\mu+\phi_2)|<\infty, \\
&\quad \left|\frac{df_4}{dT}\right| = |0|<\infty, \left|\frac{df_4}{dR}\right| = |0|<\infty \\
\left|\frac{df_5}{dS}\right| &= |0|<\infty, \left|\frac{df_5}{dV}\right| = |0|<\infty, \left|\frac{df_5}{dE}\right| = |0|<\infty, \left|\frac{df_5}{dI}\right| = |\phi_2|<\infty, \\
&\quad \left|\frac{df_5}{dT}\right| = |(\varphi+\mu)|<\infty, \left|\frac{df_5}{dR}\right| = |\mu|<\infty \\
\left|\frac{df_6}{dS}\right| &= |0|<\infty, \left|\frac{df_6}{dV}\right| = |0|<\infty, \left|\frac{df_6}{dE}\right| = |0|<\infty, \left|\frac{df_6}{dI}\right| = |\varphi|<\infty, \\
&\quad \left|\frac{df_6}{dT}\right| = |\varphi|<\infty, \left|\frac{df_6}{dR}\right| = |0(\mu+\delta)|<\infty
\end{aligned} \tag{2.10}$$

Equation (2.10) confirms that system (2.1) is bounded, invariantly and attractively influential on the bounded region of $\mathcal{R}_+^6$.

2.4. Model Disease Free Equilibrium

The cholera-non-infected equilibrium state represents a scenario in which the system is entirely free from vibro-cholerae spread. Consequently, when the number of infected individuals (I), it follows that the numbers of exposed (E), treated and recovered (R), i.e. $I = E = 0$. In this context, the solution for the cholera-free equilibrium point can be derived as follows:

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = \frac{dR}{dt} = 0 \tag{2.11}$$

At no outbreak of cholera infection, the diseases class, at $t > 0$, from (2.11)

$$\begin{aligned}
(S, V, E, I, T, R) &= \left(S_0 = \frac{\pi}{(\phi_1+\mu)}, V_0 = \frac{\pi\phi}{(\phi_1-\delta+\mu)(1-\varepsilon)\beta+\mu}, \right. \\
&\quad \left. E_0 = 0, I_0 = 0, T_0 = 0, R_0 = 0\right)
\end{aligned} \tag{2.12}$$

2.5. Endemic Equilibrium Point

We examine cholera endemicity in a specified population, focusing on strategic interventions like awareness, immunization, and customized educational programs, with the aim of long-term elim-

ination strategies. The frequency of cholera on (S_c, S_a, E, I, H, R) at $t \neq 0$, stressing the dynamic aspect of it to gauge the critical role in its infectious diseases and protect the populace. Let $E_e = (S_c, S_a, E, I, H, R^*)$ at steady state $I \neq 0$. Examine the equation system in (2.1). The points of equilibrium are:

$$\begin{aligned}
S^* &= \frac{(\phi_1+\mu)\pi(1-\varepsilon)^2[(\alpha+\mu+\gamma+\phi_2)]}{[(\mu+\phi_2+(1-\varepsilon))k+(1-\varepsilon)]\sqrt{(\varphi+\mu)\gamma(\gamma+(1-\varepsilon))k(\mu+\gamma+\beta)}} \\
V^* &= \frac{(1-\varepsilon)\pi\sqrt{(\alpha+\mu+\varphi_2)}}{(1-\varepsilon)k+(\delta+\mu+\phi_1)[(\mu+\alpha+\delta)]} \\
E^* &= \frac{[\pi(\mu=\alpha)+(\mu+\gamma+\phi_2)\sqrt{(k+\varphi+\mu)(\delta+\mu+\gamma)}]}{[\mu^2(\phi_1+\mu)+(1-\varepsilon)]} \\
I^* &= \frac{(1-\varepsilon)k(\mu+\gamma+(\varphi+\phi_1))}{(\beta+\mu+\varphi)[(\mu+\alpha+(1-\varepsilon)\gamma)]} + \frac{\sqrt{(k+\phi_2+\mu)(\delta+\mu+\phi_1)}}{(1+\alpha)^{-1}(\gamma+\mu+k)} \\
T^* &= \frac{(\mu+\gamma+(1-\varepsilon))}{(\delta+\mu+\varphi)[(\mu+\phi_2+(1-\varepsilon)\phi_1)]} + \frac{\sqrt{(k+\mu)(\delta+\mu+\varphi)}}{(1+\alpha)^{-1}(\gamma+\mu+k)} \\
R^* &= \frac{(1+\alpha)}{[\mu^2+(\varphi+\mu)+(1-\varepsilon)]} + \sqrt{\frac{(\mu+(1-\varepsilon)k+\pi)+\beta k^2}{(\beta+\alpha)(\gamma+\mu+k)(\varepsilon+\mu+\delta)}}
\end{aligned}$$

2.6. Basic Reproduction Number

The basic reproduction number, denoted as R_*. It is necessary to quantify the probability of new cholera infections resulting from a single carrier or sick person in a population without previous illnesses. We use the next-generation approach to create the system described in System (1), focusing on the infectious classes. The F and V matrices, which represent the rates of new infections and transitions into and out of the infected compartment, respectively, are computed as part of this methodology. These matrices are obtained using a complex derivation from the equations in system (2.1), $R_* = \rho(G - \lambda I)$ taking $G = F \times V^{-1}$ and ρ is the spectral radius of the matrix $|G - \lambda I|$. From the system of equation (2.1) it is obtained for matrix F and V:

$$F_i = \left(\frac{\partial f_i(x_i)}{\partial x_j}\right) \quad V_i = \left(\frac{\partial v_i(x_i)}{\partial x_j}\right)$$

And

$$f = \begin{pmatrix} \beta I S_0 + (1-\varepsilon)\beta I V_0 \\ 0 \end{pmatrix} \quad \text{and} \quad v = \begin{pmatrix} (k+\mu)E \\ -kE + (\alpha+\mu+\gamma+\phi_2)I \end{pmatrix}$$

Then,

$$\begin{aligned}
F &= \begin{pmatrix} 0 & \frac{\pi[(1-\varepsilon)\beta+\mu+\phi_1]}{(\phi_1-\delta+\mu)(1-\varepsilon)\beta+\mu} \\ 0 & 0 \end{pmatrix} \\
V &= \begin{pmatrix} (k+\varepsilon) & 0 \\ -k & (\alpha+\mu+\gamma+\phi_2) \end{pmatrix} \\
FV^{-1} &= \frac{1}{(k+\mu)(\alpha+\mu+\gamma+\phi_2)} \begin{pmatrix} 0 & \frac{\pi[(1-\varepsilon)\beta+\mu+\phi_1]}{(\phi_1+\mu)(1-\varepsilon)\beta+\mu} \\ 0 & 0 \end{pmatrix} \begin{pmatrix} (\alpha+\mu+\gamma+\phi_2) & 0 \\ -k & (k+\mu) \end{pmatrix} \\
R_* &= \frac{\pi[(1-\varepsilon)\beta+\mu+\phi_1]}{[(\phi_1-\delta+\mu)(1-\varepsilon)\beta+\mu](\alpha+\mu+\gamma+\phi_2)}
\end{aligned} \tag{2.13}$$

2.7. Quantitative Analysis of R_ε

Performing a quantitative analysis for the basic reproduction number R_0 using initial values lies in its ability to assess the potential spread of a cholera disease as an infectious disease within a population. This analysis helps in understanding the epidemiological dynamics and effectiveness of control measures of vaccination and treatment in the control of its spread where if $R_0 > 1$, the disease can spread in the population and the disease will eventually die out. Initial values number of susceptible individuals, infection rates, recovery rates provide the starting conditions for the model. These values are critical for accurately estimating R_0 and predicting the early-stage growth of the infection. Applying differential equations to describe disease transmission. Application and interpretation of this helps in public health decision-making by determining the required vaccination coverage or intervention strategies to control an outbreak. Provides insight into the potential severity of an epidemic and guides resource allocation. Here, we conduct a quantitative analysis of R_ε to assess its metric progression concerning each intervention method. By excluding the values of intervention parameters, we assess equation (2.13) using the baseline values provided in Table 1, yielding equation (2.14), subsequently resulting in equations (2.15) through (2.18). The outcomes of these calculations are presented in Table 2.

$$R_{\cdot} = \frac{\pi[(1-0.028762)\beta + 0.0087363 + 0.5263532\phi_1]}{[(1.27364 - 0.9377847 + 0.38736\alpha)(1-0.7653\varepsilon)\beta + 0.872625](0.9972 + 0.5243436\phi_2)} \tag{2.14}$$

$$R_\varepsilon = f(\phi)\big|_{\delta=0,\tau=0,\phi_2} = -1.7363526\phi_1 + 1.459137886\phi_2 \tag{2.15}$$

$$R_\varepsilon = f(\varphi)\big|_{\alpha=0,\rho_1=0,\rho_2=0} = \frac{0.005728262500}{0.0000133\varphi + 0.0039257856\alpha} \tag{2.16}$$

$$R_\varepsilon = f(\phi_2)\big|_{c=0,\phi_2=0,\tau=0} = \frac{0.093762(0.09756 + 0.54\phi_1)}{0.137747\phi_2 + 0.018\phi_1} \tag{2.17}$$

$$R_\varepsilon = f(\varepsilon)\big|_{\rho_1=0,\tau=0,\rho_2=0} = 1.38947804 - 1.9276424\varepsilon \tag{2.18}$$

Table 2.2: Standalone Metric of Vaccination and General Treatment R_ε

s/n	φ	β	φ_2	ε	R_ε	s/n	φ_1	β	φ_2	R_ε
1	0	0	0	0	1.4591378	0	0	0	0	1.45913788
2	0.2	0	0	0	1.1964930	0	0.2	0	0	1.16731030
3	0.4	0	0	0	0.93384824	0	0.4	0	0	0.87548273
4	0.6	0	0	0	0.67120342	0	0.6	0	0	0.58365515

s/n	φ_1	β	φ_2	R_ε
0	0	0	0	1.45913788
0	0	0.2	0	0.25434513
0	0	0.4	0	0.20246410
0	0	0.6	0	0.18416303

Analysis of the above table reveals that utilizing vaccination and treatment independently at 40% to 60% efficacy effectively reduces disease transmission. However, even at 90% efficacy, treatment fails to significantly impact the reproduction number due to untreated individuals in the community. Hence, with 100% public awareness and sensitization which treatment and vaccination are prioritized and achieving $R_\varepsilon = 1$ is a level attainable through vaccination and treatment campaign.

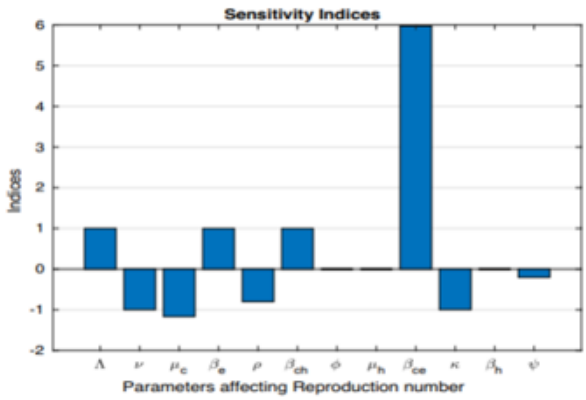

Figure 2.2: Sensitivity indices as it affects the basic reproduction number

2.8. Local Stability of Disease Free State

We examined the local stability of the disease-free state for cholera by analysing the minimal recurrence rate impact. When the recurrence rate R_*, the disease declines, to determine stability using a Jacobian matrix and a characteristic equation.

Theorem 2.3. *The disease-free state of the model is locally asymptotically stable* $R_* < 1$, *otherwise* $R_* > 1$

Proof
The linearization method is used to construct the Jacobian matrix of the system of (2.1) and assess it at the illness free-state in order to determine the disease-free equilibrium.

$$J_{E_1} = \begin{pmatrix} -(\beta+\mu+\phi_1+\delta) & 0 & 0 & \beta & 0 & 0 \\ \phi_1 & -[(1-\varepsilon)\beta+\mu] & 0 & (1-\varepsilon)\beta & 0 & 0 \\ \beta & (1-\varepsilon)\beta & -(k+\mu) & (1-\varepsilon)\beta & 0 & 0 \\ 0 & 0 & k & -(\alpha+\mu+\gamma+\phi_2) & 0 & 0 \\ 0 & 0 & 0 & \phi_2 & -(\varphi+\mu) & 0 \\ 0 & 0 & 0 & \gamma & \varphi & -(\delta+\mu) \end{pmatrix} \tag{2.19}$$

Computing for the eigenvalues, $|J_{E_1} - \lambda_i I| = 0$

$$\begin{vmatrix} -(\beta+\mu+\phi_1+\delta) & 0 & 0 & \beta & 0 & 0 \\ \phi_1 & -[(1-\varepsilon)\beta+\mu] & 0 & (1-\varepsilon)\beta & 0 & 0 \\ \beta & (1-\varepsilon)\beta & -(k+\mu) & (1-\varepsilon)\beta & 0 & 0 \\ 0 & 0 & k & -(\alpha+\mu+\gamma+\phi_2) & 0 & 0 \\ 0 & 0 & 0 & \phi_2 & -(\varphi+\mu) & 0 \\ 0 & 0 & 0 & \gamma & \varphi & -(\delta+\mu) \end{vmatrix} = 0$$

as obtained:

$$\begin{aligned} \lambda &= -(v+\mu), \\ \lambda &= -(\alpha_2+\mu), \\ \lambda &= -\mu \begin{vmatrix} -(\alpha+\mu+\gamma+\phi_2)-\lambda & 0 \\ \phi_2 & -(\varphi+\mu)-\lambda \end{vmatrix}, \\ \lambda &= -(\alpha+\mu+\gamma+\phi_2), \\ \lambda &= -(\varphi+\mu) \end{aligned}$$

respective eigenvalues in invariant in the region $\mathcal{R}_+^6$ of the model, indicating a biological implication that there will be a decreases in the spread over time if necessary control measures as indicated are strictly adhere to. Where

$$\begin{aligned}
\lambda_1 &= -(\beta+\mu+\phi_1+\delta) \\
\lambda_2 &= -[(1-\varepsilon)\beta+\mu] \\
\lambda_3 &= -(k+\delta) \\
\lambda_4 &= -(\alpha+\mu+\phi_1+\gamma) \\
\lambda_5 &= -(\varphi+\mu) \\
\lambda_6 &= -(\mu+\delta)
\end{aligned} \tag{2.20}$$

Hence the system of the (2.20) obtained from (2.19) is asymptomatically stable $\forall \lambda_n < 0,\ n = 1,2,\ldots,6,\ t>0$

2.9. Local Stability of Endemic Equilibrium Point

Theorem 2.4. *The suggested model of cholera disease has a locally asymptotically stable in region resilience if the recurrence rate $R_* < 1$ and unstable whenever $R_* > 1$. If endemicity of cholera spread within the population is not curtailed*

Proof
Suppose,

$$S = x + S^*,\ V = y + V^*,\ E = z + E^*,\ I = \alpha + I^*,\ T = b + T^*,\ R = c + R^*$$

Linearizing equation (2.1), is then obtained as

$$\begin{aligned}
\frac{dS}{dt} &= \pi - \beta(x+S^*)(\alpha+I^*) - (\phi_1+\mu)(x+S^*) + \delta(x+R^*) \\
\frac{dV}{dt} &= \phi_1(x+S^*) - (1-\varepsilon)\beta(\alpha+I^*)(y+V^*) - \mu(y+V^*) \\
\frac{dE}{dt} &= (1-\varepsilon)\beta(\alpha+I^*)(y+V^*) + \beta(x+S^*)(\alpha+I^*) - (k+\mu)(z+E^*) \\
\frac{dI}{dt} &= k(z+E^*) - (\alpha+\mu+\gamma+\phi_2)(\alpha+I^*) \\
\frac{dT}{dt} &= \phi_2(\alpha+I^*) - (\varphi+\mu)(b+T^*) \\
\frac{dR}{dt} &= \varphi(b+T^*) + \gamma(\alpha+I^*) - (\delta+\mu)(c+R^*)
\end{aligned}$$

Linearizing equation (2.12), is then obtained as

$$\begin{aligned}
\frac{dx}{dt} &= -\beta\alpha x - (\alpha_1+\mu)x - \delta x + \text{higher order} + \text{non-linear terms}\ldots \\
\frac{dy}{dt} &= \phi_1 x - (1-\varepsilon)\beta\alpha y - \mu y + \text{higher order} + \text{non-linear terms}\ldots \\
\frac{dz}{dt} &= (1-\varepsilon)\beta\alpha y + \beta\alpha x - (k+\mu)z + \text{higher order} + \text{non-linear terms}\ldots \\
\frac{da}{dt} &= kz - (\alpha+\mu+\gamma+\phi_2)\alpha + \text{higher order} + \text{non-linear terms}\ldots \\
\frac{db}{dt} &= \phi_2 a - (\varphi+\mu)b + \text{higher order} + \text{non-linear terms}\ldots \\
\frac{dc}{dt} &= \varphi b + a\gamma - (\delta+\mu) + \text{higher order} + \text{non-linear terms}\ldots
\end{aligned}$$

The characteristic equation obtained from its Jacobian matrix is;

$$\begin{vmatrix} A-\lambda & 0 & 07-\beta ax & 0 & 0 & \\ \phi_1 x-[(1-\varepsilon)\beta ay+\mu]y-\lambda & 0 & (1-\varepsilon)\beta ay & 0 & 0 & \\ \beta ax & (1-\varepsilon)\beta ay & B-\lambda & \beta ax & 0 & 0 \\ 0 & 0 & kz & C-\lambda & 0 & 0 \\ 0 & 0 & 0 & a\phi_2 & -(\varphi+\mu)-\lambda & 0 \\ 0 & 0 & 0 & a\gamma & \varphi b & (\delta+\mu)c \end{vmatrix} = 0$$

Denoting that $A=-(\beta a+\mu+\phi_1)x,\ B=-[\beta ax-(k+\mu)z],\ C=-(\alpha+\mu+\gamma+\phi_2)$ the resulting eigenvalue of the above matrix is obtained as;

$$\lambda^6-(x(a+y)+(c+b))\lambda^5+((x+c)(y+s)+az+by)(1+b)\lambda^4-(ab(c+z)+bc(a+y))$$
$$(1+z)\lambda^3+y(cz(a+b)+xb(a+y))\lambda^2-(a+c)(x+y)\lambda+abcxyz=0$$

With the invariance of the eigen-values it is said to be locally assymptotically stable.

2.10. Global Stability of Disease Free Equilibrium

Using the Lyapunov method and Lyapunov's function approach, we determine the global asymptotic stabillity of the model for equation (2.1) at the disease free equilibrium.

$$\Phi(t,S,V,E,I,T,R)=C_1I_1+C_2I_2+C_3I_3$$

$$\begin{aligned}
\frac{d\Phi}{dt} &= C_1I^*+C_2I^*+C_3I^* \\
&= C_1((1-\varepsilon)\beta I_2V+\beta I_2S-(k+\mu)I_1)+C_2(kI_1-(\alpha+\gamma+\phi_2+\mu)I_2)+C_2(\phi_2I_2-(\varphi_2+\mu)I_3) \\
&= C_2kI_2-C_1(k+\mu)I_1+C_1(1-\varepsilon)\eta I_2V+C_1\beta I_2S-C_2(\alpha+\gamma+\phi_2+\mu)I_2+C_3\phi_2I_2 \\
&\quad -C_3(\varphi_2+\mu)I_3 \\
&\leq C_2k-C_1(k+\mu))I_1+(C_1(1-\varepsilon)\beta V_0)+C_1\beta S_0-C_2(\alpha+\gamma+\phi_2+\mu)+C_3\phi_2)I_2 \\
&\quad -C_3(\varphi_2+\mu)I_3
\end{aligned}$$

$$\begin{aligned}
S_0 &= \frac{\pi}{(\phi_1+\mu)}, V_0=\frac{\pi\phi_1}{(\phi_1-\delta+\mu)(1-\varepsilon)\beta+\mu}, E_0=0, I_0=0, T_0=0, R_0 \\
S_0 &= \frac{\pi}{(\phi_1+\mu)}, V_0=\frac{\pi\phi_1}{(\phi_1-\delta+\mu)(1-\varepsilon)\beta+\mu}, C_1=\frac{1}{(k+\mu)}, C_2=\left(\frac{(\phi_2+\mu-\delta)(1-\varepsilon)_\alpha+\beta}{\pi(\phi_2+\mu+\delta+\gamma)(\alpha+\mu)}\right) \\
\frac{d\Phi}{dt} &\leq C_1\left(\frac{\pi(1-\varepsilon)\beta+\pi\mu+\pi\phi_1}{[(\phi_1-\delta+\mu)(1-\varepsilon)]}-\frac{(k+\mu)}{(k+\mu)}\right)I_1 \\
&\quad -\left(\frac{\pi(\gamma+\mu+\delta)(1-\varepsilon)\beta+\mu}{(\phi_1+\mu)(\gamma+\mu+\delta)(1-\varepsilon)}-\frac{\pi(\gamma+\mu+\delta)(1-\varepsilon)\beta+\mu}{(\phi_1+\mu)(\gamma+\mu+\delta)(1-\varepsilon)}\right)I_2 \\
\frac{d\Phi}{dt} &\leq \psi(R_0-1)
\end{aligned}$$

It is pertinent to note that when at $t\to\infty$ and $C_1<1$. Substituting into model system of equation (18) reveal that, based on LaSalle's invariance principle $\frac{d\Phi}{dt}=0$, is globally asymptotically stable whenever $R_*>1$.

2.11. Global Stability of Endemic Equilibrium

Theorem 2.5. *The Dulac criterion is a method used in dynamical systems to determine the absence of periodic orbits in a given region of the phase plane, which can be extended to analyze the global stability of an equilibrium point.*

Proof
For a dynamical system described by the differential equation:

$$\frac{dx}{dt} = f(x,y) \Leftrightarrow \frac{dy}{dt} = g(x,y) \tag{2.21}$$

The Dulac criterion states that if there exists a continuously differentiable function $B(x,y)$ (called the Dulac function) ssuch that the expression:

$$\frac{\partial}{\partial x}(B(x,y)f(x,y)) + \frac{\partial}{\partial x}(B(x,y)g(x,y)) \tag{2.22}$$

is either strictly positive or strictly negative throughout a simply connected region D of the phase plane, then there are no closed trajectories (periodic orbits) contained within D.
TO apply this to determine the global stability of an endemic equilibrium (x^*, y^*). Also define the Dulac function $B(x,y)$ and the expression $\frac{\partial}{\partial x}(B(x,y)f(x,y)) + \frac{\partial}{\partial x}(B(x,y)g(x,y))$ as $B(x,y)g(x,y)$ This shows that this expression is of one sign 9either strictly positive or strictly negative) in the region of interest. If such a Dulac function $B(x,y)$ can be found, the system has no periodic orbits in that region, suggesting the global stability of the endemic equilibrium if no other attractors exist. Hence, if $\exists B(x,y) \in C^1$ such that $\frac{\partial}{\partial x}(B(x,y)f(x,y)) + \frac{\partial}{\partial x}(B(x,y)g(x,y)) \neq 0$ in D. Then there are no closed trajectories in D. This criterion is useful in proving the global stability of the endemic equilibrium when combined with other stability analysis techniques.
We employ this concept Dulac's criterion Let $X = (S, V, E, I, T, R)$ define the Dulac's function $G = \frac{1}{SI}$ The following system of equation are obtained;

$$\begin{aligned}
G\frac{dS}{dt} &= \frac{1}{SI}\{\pi - \beta SI - (\phi_1 + \mu)S + \delta R\} && (2.23)\\
G\frac{dV}{dt} &= \frac{1}{SI}\{\phi_1 S - (1-\varepsilon)\beta IV - \mu V\} && (2.24)\\
G\frac{dE}{dt} &= \frac{1}{SI}\{(1-\varepsilon)\beta IV + \beta SI - (k+\mu)E\} && (2.25)\\
G\frac{dI}{dt} &= \frac{1}{SI}\{kE - (\alpha + \mu + \gamma + \phi_2)I\} && (2.26)\\
G\frac{dT}{dt} &= \frac{1}{SI}\{\phi - 2I - (\varphi + \mu)T\} && (2.27)\\
G\frac{dR}{dt} &= \frac{1}{SI}\{\varphi T + \gamma I - (\delta + \mu)R\} && (2.28)
\end{aligned}$$

The above system of equations results to;

$$\begin{aligned}
G\frac{dS}{dt} &= \left\{\frac{\pi}{SI} - \beta - \frac{(\phi_1 + \mu)}{I} + \frac{\delta R}{SI}\right\}\\
G\frac{dV}{dt} &= \left\{\frac{\phi_1}{I} - \frac{(1-\varepsilon)\beta}{S} - \frac{\mu}{S}\right\}\\
G\frac{dE}{dt} &= \left\{\frac{(1-\varepsilon)\beta}{S} + \beta - \frac{(k+\mu)E}{S}\right\}\\
G\frac{dI}{dt} &= \left\{\frac{kE}{SI} - \frac{(\alpha + \mu + \gamma + \phi_2)}{I}\right\}
\end{aligned}$$

$$
\begin{aligned}
G\frac{dT}{dt} &= \left\{\frac{\phi_2}{S} - \frac{(\varphi+\mu)T}{SI}\right\} \\
G\frac{dR}{dt} &= \left\{\frac{\varphi T}{SI} + \frac{\gamma}{S} - \frac{(\delta+\mu)R}{SI}\right\}
\end{aligned}
$$

At $t > 0$ orbital resolution of the system of equations is given by $\frac{d(GX)}{dt}$ as obtained below.

$$
\begin{aligned}
\frac{d(GX)}{dt} &= \frac{\partial}{\partial S}\{G\frac{dS}{dt}\} + \frac{\partial}{\partial V}\{G\frac{dV}{dt}\} + \frac{\partial}{\partial E}\{G\frac{dE}{dt}\} + \frac{\partial}{\partial I}\{G\frac{dI}{dt}\} + \frac{\partial}{\partial T}\{G\frac{dT}{dt}\} + \frac{\partial}{\partial R}\{G\frac{dR}{dt}\} \\
\frac{d(GX)}{dt} &= \frac{\partial}{\partial S}\{\frac{\pi}{SI} - \beta - \frac{(\phi_1+\mu)}{I} + \frac{\delta R}{SI}\} + \frac{\partial}{\partial V}\{\frac{\phi_1}{I} - \frac{(1-\varepsilon)\beta}{S} + \frac{\mu}{S}\} + \frac{\partial}{\partial E}\{\frac{(1-\varepsilon)\beta}{S} \\
&\quad +\beta - \frac{(k+\mu)E}{S}\} \\
&\quad \frac{\partial}{\partial I}\{\frac{kE}{SI} - \frac{(\alpha+\mu+\gamma+\phi_2)}{I}\} + \frac{\partial}{\partial T}\{\frac{\phi_2}{S} - \frac{(\varphi+\mu)T}{SI}\} + \frac{\partial}{\partial R}\{\frac{\varphi T}{SI} + \frac{\gamma}{S} - \frac{(\delta+\mu)R}{S}\} \\
\frac{d(GX)}{dt} &= \{-\frac{[\pi+\beta+(\phi_1+\mu)+\delta]}{SI}\} + \frac{\partial}{\partial V}\{-\frac{\phi_1+(1-\varepsilon)\beta+\mu}{SI}\} + \frac{\partial}{\partial E}\{-\frac{(1-\varepsilon)\beta+\beta+(k+\mu)}{S}\} \\
&\quad \frac{\partial}{\partial I}\{-\frac{k+(\alpha+\mu+\gamma+\phi_1)}{SI}\} + \frac{\partial}{\partial T}\{-\frac{\phi_2+(\varphi+\mu)}{S}\} + \frac{\partial}{\partial R}\{-\frac{\varphi+\gamma+(\delta+\mu)}{SI}\} \\
\frac{d(GX)}{dt} &= -\left\{\frac{A(1-v)+[(1-\rho)+\rho\beta]+(m+\mu)}{SI} + \frac{m+(1-\rho)-(\rho\beta+\mu)}{I} + \frac{(1-\rho)\beta+(\delta+\mu)}{I}\right. \\
&\quad \left.+\frac{\delta+\rho\beta+(\gamma+\mu+d)}{SI} + \frac{Av+\gamma-\mu}{SI}\right\} \\
\frac{d(GX)}{dt} &= -\{\frac{A(1-v)+[(1-\rho)+\rho\beta]+\sigma(m+\mu)+\gamma[m+m(1-\rho)-v(\rho\beta+\mu)]+(1-\rho)\beta+(\delta+\mu)}{SI}\} < 0
\end{aligned}
$$

This implies that the system has no closed orbit. It therefore portray epidemiologically that, no existence of a periodic orbit which implies that there are fluctuations in the number of infective, which makes it pretty obvious that in allocation of resources for the control of the disease, vaccination will help to eradicate the rapid spread of cholera with time.

2.12. Sensitivity Analysis of R_*

The principal objective is to evaluate the recurrence rate's sensitivity by calculating its derivative with respect to all pertinent parameters. Sensitivity analysis value indices assess how treatment and vaccination impact the basic reproduction number R_* of cholera, identifying key factors that reduce transmission and guide effective control strategies to curb outbreaks. As a result of this investigation, the normalized forward sensitivity index, as obtained

$$
\begin{aligned}
\frac{\partial R_*}{\partial\beta}\frac{\partial R_*}{\partial\beta}\times\frac{\beta}{R_*} &= 1.002863633 & \frac{\partial R_0}{\partial\varepsilon}\frac{\partial R_0}{\partial\varepsilon}\times\frac{\varepsilon}{R_0} &= 0.00733623 \\
\frac{\partial R_*}{\partial\pi}\frac{\partial R_*}{\partial\pi}\times\frac{\pi}{R_*} &= 1.03267370 & \frac{\partial R_*}{\partial\phi_1}\frac{\partial R_*}{\partial\phi_1}\times\frac{\phi_1}{R_*} &= 0.001307654 \\
\frac{\partial R_*}{\partial\phi_2}\frac{\partial R_*}{\partial\phi_2}\times\frac{\phi_2}{R_*} &= 1.1096546 & \frac{\partial R_*}{\partial\mu}\frac{\partial R_*}{\partial\mu}\times\frac{\mu}{R_*} &= 0.15356728 \\
\frac{\partial R_*}{\partial\delta}\frac{\partial R_*}{\partial\delta}\times\frac{\delta}{R_*} &= 1.108763 & \frac{\partial R_*}{\partial\varphi}\frac{\partial R_*}{\partial\varphi}\times\frac{\varphi}{R_*} &= 0.765438 \\
\frac{\partial R_*}{\partial\phi_2}\frac{\partial R_*}{\partial\phi_2}\times\frac{\phi_2}{R_*} &= 0.564321
\end{aligned}
$$

Table (2.3) shows that the sensitivity indices of are positively invariant in $\mathcal{R}_6^+$ the sensitivity indices

Table 2.3: Sensitivity Analysis and Parameter Indices

Parameters	Sensitivity Indices
β	0.01206000
ε	1.03267370
α	0.18743076
δ	0.00001001
φ_1	1.00000000
φ_2	1.000201001

depend on the values of the each parameters of R_*, and this brings about changes in the values that will affect the behaviour of the threshold on the spread or vanity of cholera disease. Based on the Table 2.3 above, we can concluded that parameter ε is the most sensitive to the basic reproduction number of the cholera disease. Particularly, increasing the value of ε will result in a 80.86% increase in R_*, while increasing the value of will lead to a 91.52% decrease in R_*.

2.13. Numerical Simulation

We conducted numerical simulation on the mathematical model, we create the following iterative scheme of Laplace adomian decomposition method for the model equation. The Laplace adomian decomposition method was employed to computationally analyse the epidemic model. Maple software facilitated the generation of iteration formulas for each compartment. These formulas were then iteratively solved, enabling the numerical evaluation of the model's dynamics and providing insights into the epidemic's behaviour and progression. Taking the Laplace transform of both sides of the above equation.

$$\begin{aligned}
L[\frac{dS}{dt}] &= L[\pi] - L[\beta SI - (\phi_1 + \mu)S + \delta R] \\
L[\frac{dV}{dt}] &= L[\phi_1 S] - L[(1-\varepsilon)\beta IV - \mu V] \\
L[\frac{dE}{dt}] &= L[(1-\varepsilon)\beta IV] - L[\beta SI - (k+\mu)E] \\
L[\frac{dI}{dt}] &= L[kE] - L[(\alpha + \mu + \gamma + \phi_2)I] \\
L[\frac{dT}{dt}] &= L[\phi_2 I] - L[(\varphi + \mu)T] \\
L[\frac{dR}{dt}] &= L[\varphi T] - L[\gamma I - (\delta + \mu)R]
\end{aligned}$$

Substituting from (2.22) into (2.23) to yeild

$$\begin{aligned}
mL[S(t)] &= S(0) + \pi + L[-\alpha[SI] - \pi - \beta SI - (\phi_1 + \mu)S + \delta R] \\
mL[V(t)] &= V(0) + L[\beta_1 S] - L[\phi_1 S - (1-\varepsilon)\beta IV - \mu V] \\
mL[E(t)] &= E(0) + L[\alpha SI] - L[(1-\varepsilon)\beta IV + \beta SI - (k+\mu)E] \\
mL[I(t)] &= I(0) + L[kE] - L[(\alpha + \mu + \gamma + \phi_2)I] \\
mL[T(t)] &= I(0) + L[\phi_2 I] - L[(\varphi + \mu)T] \\
mL[R(t)] &= R(0) + L[\varphi T] - L[\gamma I - (\delta + \mu)R]
\end{aligned}$$

Where $S(0) = s_0,\ V(0) = v_0,\ E(0) = e_0,\ I(0) = i_0,\ R(0) = r_0$

$$\begin{aligned}
L[S(t)] &= \frac{s_0}{m} + \frac{\pi}{m^2} + \frac{1}{m}L[-\alpha[SI] - \beta SI - (\phi_1 + \mu)S + \delta R] \\
L[V(t)] &= \frac{v_0}{m} + \frac{1}{m}L[\phi_1 S] - L[(1-\varepsilon)\beta IV - \mu V] \\
L[E(t)] &= \frac{e_0}{m} + \frac{1}{m} + L[(1-\varepsilon)\beta IV] - L[\beta SI - (k+\mu)E] \\
L[I(t)] &= \frac{i_0}{m} + \frac{1}{m} + L[kE] - L[(\alpha + \mu + \gamma + \phi_2)I] \\
mL[T(t)] &= \frac{r_0}{m} + \frac{1}{m} + L[\phi_2 I] - L[(\varphi + \mu)T] \\
mL[R(t)] &= \frac{r_0}{m} + \frac{1}{m} + L[\varphi T] - L[\gamma I - (\delta + \mu)R]
\end{aligned}$$

Letting the non-linear terms in the above iteration and substitutes by taking the inverse Laplace transform of both sides,

$$\begin{aligned}
S(t) &= s_0 + \pi + L^{-1}\big(\frac{1}{m}L[\pi - \beta SI - (\phi_1 + \mu)S + \delta R]\big) \\
V(t) &= v_0 + L^{-1}\big(\frac{1}{m}L[\phi S] - L[(1-\varepsilon)\beta IV - \mu V]\big) \\
E(t) &= e_0 + L^{-1}\big(\frac{1}{m} + L[(1-\varepsilon)\beta] - L[\beta SI - (k+\mu)E]\big) \\
I(t) &= i_0 + L^{-1}\big(\frac{1}{m}L[kE] - L[(\alpha + \mu + \gamma + \phi_2)I]\big) \\
T(t) &= i_0 + L^{-1}\big(\frac{1}{m} + L[\phi_2 I] - L[(\varphi + \mu)T]\big) \\
R(t) &= r_0 + L^{-1}\big(\frac{1}{m}L[\varphi T] - L[\gamma I - (\delta + \mu)R]\big)
\end{aligned}$$

Subsequently, iteration result obtained from the above equation of systems is deduced as;

$$\begin{aligned}
\sum_{k=0}^{\infty} S_n(t) &= s_0 + \pi t + L^{-1}\left(\frac{1}{m}L\left[- \alpha\sum_{k=0}^{\infty}\pi_n - \phi_1\sum_{k=0}^{\infty}S_n + \phi_2\sum_{k=0}^{\infty}V_0 - \mu\sum_{k=0}^{\infty}S_n\right]\right) \\
\sum_{k=0}^{\infty} V_n(t) &= v_0 + \pi t + L^{-1}\left(\frac{1}{m}L\left[- \sum_{k=0}^{\infty}((1-\varepsilon)\beta) - \beta_1\sum_{k=0}^{\infty}V_n + \beta_2\sum_{k=0}^{\infty}V_n - \mu\sum_{k=0}^{\infty}V_n\right]\right) \\
\sum_{k=0}^{\infty} E_n(t) &= e_0 + L^{-1}\left(\frac{1}{m} + L\varphi\sum_{k=0}^{\infty}\varepsilon_n - L[\beta - (k+\mu)]\sum_{k=0}^{\infty}E_n\right) \\
\sum_{k=0}^{\infty} I_n(t) &= i_0 + L^{-1}\left(\frac{1}{m} + L\delta\sum_{k=0}^{\infty}E_n - L[(\alpha + \mu + \gamma + \phi_2)]\sum_{k=0}^{\infty}I_n\right) \\
\sum_{k=0}^{\infty} T_n(t) &= i_0 + L^{-1}\left(\frac{1}{m}L\alpha\sum_{k=0}^{\infty}E_n - L[(\varphi + \mu)]\sum_{k-0}^{\infty}T_n\right) \\
\sum_{k=0}^{\infty} R_n(t) &= i_0 + L^{-1}\left(\frac{1}{m} + L\phi_2\sum_{k=0}^{\infty}E_n - L[(\delta + \mu)]\sum_{k=0}^{\infty}R_n\right)
\end{aligned}$$

The initial approximations of each class are given by; $S_0(t) = s_0 + \pi t,\ V_0(t) = v_0,\ E_0(t) = e_0,\ I_0(t) = i_0,\ T_0(t) = t_0,\ R_0(t) = r_0$. Now, comparing the coefficients $n = 1$. Using the recurrence relations

obtained from the iterations, Compartmentally it is obtained that

$$\begin{aligned}
S_1(t) &= (\pi i_0 s_0 - \mu s_0 - \beta s_0 + \phi_1 v_0)t + \Big(- \frac{1}{2}\alpha i_0 \varepsilon - \frac{1}{2}\mu\pi - \frac{1}{2}\delta\varphi_1 \Big)t^2 \\
V_1(t) &= (-\mu s_0 + \varphi_0 s_0 - \beta s_0 + (1-\varepsilon)s_0)t + \frac{1}{2}\varepsilon\beta_1 t^2 \\
E_1(t) &= (\varphi i_0 s_0 - \mu e_0 - \delta e_0)t + \frac{1}{2}\alpha\pi i_1 t^2 \\
I_1(t) &= kE - (\alpha + \mu + \gamma + \phi_2)I(-\delta i_0 - \alpha + \mu + \gamma + \phi_2 e_0 i_0 + \sigma e_0)t \\
T_1(t) &= (-\mu r_0 + \phi_2 s_0 v_0 + (\varphi + \mu)i_0)t \\
R_1(t) &= \frac{1}{3}\varphi\phi_2 \Big(- \varphi + \mu r_0 + \frac{1}{2}(\delta + \mu)i_0 \Big)t
\end{aligned}$$

Further iteration are done to obtain successive iterative terms at $n = 2$

$$\begin{aligned}
S_2(t) &= \Big(\frac{1}{2}\alpha^2 i^2 s_0 + \frac{1}{2}\alpha i s_0 + \frac{1}{3}\alpha i s_0\mu_0 + \frac{1}{2}\alpha i s_0\rho_0 - \frac{1}{2}\alpha i s_0 e_0 + \frac{1}{2}\alpha i s_0\beta_1 - \frac{1}{2}\alpha i s_0\beta_2 \\
&\quad + \frac{1}{2}\mu^2 s_0 + \beta_1\mu s_0 + \beta_1\mu v_0 + \frac{1}{2}\beta^2 s_0 + \frac{1}{2}\beta_1\beta_2 s_0 - \frac{1}{2}\beta_1\beta_2 v_0 - \frac{1}{2}\beta_2^2 v_0\Big)t^2 \\
&\quad + \Big(\frac{1}{6}\alpha^2 i^2\theta + \frac{1}{3}\alpha i_0\pi\delta + \frac{2}{3}\alpha i_0\pi\mu + \frac{1}{3}\alpha i_0\pi\rho - \frac{1}{3}\alpha e_0\theta\sigma + \frac{1}{3}\alpha i_0\pi\beta_1 + \frac{1}{6}\mu^2\theta \\
&\quad + \frac{1}{3}\beta_0\pi\mu + \frac{1}{6}\beta_1^2\pi + \frac{1}{6}\beta_2\pi\beta_1\Big)t^3 \\
V_2(t) &= \Big(- \frac{1}{2}\alpha i s_0\beta_1 + \frac{1}{2}\mu^2 v_0 - \beta_1\mu s_0 + \beta_2\mu s_0 - \frac{1}{2}\beta^2 s_0 + \frac{1}{2}\beta_1\beta_2 s_0 - \frac{1}{2}\beta_1\beta_2 v_0 + \frac{1}{2}\beta_2^2 v_0\Big)t^2 \\
&\quad + \Big(- \frac{1}{6}\alpha i_0\pi\beta_1 - \frac{1}{3}\beta_1\mu\pi - \frac{1}{6}\beta_1^2\pi + \frac{1}{6}\beta_2\pi\beta_1\Big)t^3
\end{aligned}$$

$$\begin{aligned}
E_2(t) &= \Big(- \frac{1}{6}\alpha^2 i^2\pi - \frac{1}{3}\alpha i_0\pi\delta - \frac{2}{3}\alpha i_0\pi\mu - \frac{1}{3}\alpha i_0\pi\rho + \frac{1}{3}\alpha e_0\pi\sigma_1 - \frac{1}{6}\mu^2\pi - \frac{1}{6}\alpha i_0\pi\beta_1\Big)t^3 \\
&\quad + \Big(- \frac{1}{2}\alpha^2 i^2 s_0 - \frac{1}{2}\sigma i s_0 - \frac{2}{3}\alpha i s_0\mu_0 - \frac{1}{2}\alpha i s_0\rho_0 + \frac{1}{2}\alpha i s_0 v_9 - \mu^2 i e_0\beta_1 + \frac{1}{2}\alpha i e_0\sigma^2\Big)t^2 \\
I_2(t) &= -\frac{1}{6}\alpha^2 i^2\theta + \Big(\frac{1}{2}\sigma\alpha i s_0 + \frac{1}{2}\delta^2 i - 0 + \delta\mu i_0 - \frac{1}{2}\delta\sigma i e_0 + \frac{1}{2}\mu^2 i_0 - \mu\rho i_0 - \mu\sigma i_0 + \frac{1}{2}\rho^2 i_0 \\
&\quad - \frac{1}{2}\rho\sigma i e_0 - \frac{1}{2}\sigma^2 e_0\Big)t^2 \\
T_2(t) &= \frac{1}{6}\alpha^2 i^2\theta + \Big(\frac{1}{2}\sigma\alpha i s_0 + \frac{1}{2}\delta^2 i - 0 + \delta\mu i_0 - \frac{1}{2}\delta\sigma i e_0 + \frac{1}{2}\mu^2 i_0 - \mu\rho i_0 - \mu\sigma i_0 + \frac{1}{2}\rho^2 i_0 \\
&\quad - \frac{1}{2}\rho\sigma i e_0 - \frac{1}{2}\sigma^2 e_0\Big)t^2 \\
R_2(t) &= \Big(- \frac{1}{2}\delta\rho i_0 + \frac{1}{2}\mu^2 r_0 - \mu\sigma i_0 - \frac{1}{2}\rho^2 i_0 + \frac{1}{2}\phi_1\varphi e_0\Big)t^2
\end{aligned}$$

and so on. This can be further till desired number of iterations are obtained. Thus, the obtained raw solution to each model compartment is obtained as:

$$S(t) = \sum_{k=0}^{3} s_k(t),\ V(t) = \sum_{k=0}^{3} v_k(t),\ E(t) = \sum_{k=0}^{3} e_k(t),\ I(t) = \sum_{k=0}^{3} i_k(t),\ R(t) = \sum_{k=0}^{3} r_k(t)$$

Evaluating these series result using corresponding variables and parameter values,

$$\begin{aligned}
S(t) &= 500.012 - 30.4440t + 1.1315290300t^2 - 0.0507029853t^3 - 3.509616000 \times 10^{-13}t^5 \\
&\quad -5.179149070 \times 10^{-7}t^4 \\
V(t) &= 120 - 1.5060t - 0.01591470000t^2 + 0.001033697580t^3 + 9.015111000 \times 10^{-9}t^4 \\
E(t) &= 65 + 18.178t - 1.171778775t^2 + 0.04929560765t^3 + 5.087939775 \times 10^{-7}t^4 \\
&\quad +3.509616000 \times 10^{-13}t^5 \\
I(t) &= 23.09 - 60t + 0.0292567500t^2 - 0.0008440367798t^3 - 4.378044000 \times 10^{-9}t^4 \\
T(t) &= 23.09 - 60t + 0.0292567500t^2 - 0.0008440367798t^3 - 4.378044000 \times 10^{-9}t^4 \\
R(t) &= 14 - 0.0155t - 0.005054500000t^2 + 0.0001458242541t^3 + 2.437075000 \times 10^{-10}t^4
\end{aligned}$$

Hence from the results of successive iterations, comparison of control intervention effects on sub-populations in its graphical illustration depicts as;

Table 2.4: Comparison of parameters φ_1 and φ_2 values for ε at $\varepsilon = 0 \ldots 0.5$

Variables	**Description**	**At** $\varepsilon = 0.1, 0.25, 0.5$
$E(t)$	exposed population	0.5, 0.25, 0.125
$I(t)$	infected population	0.5, 0.25
$R(t)$	recovered population	0.125
φ	treatment intervention	0.1, 0.2, 0.5
φ_2	vaccination intervention	0, 0.1, . . ., 0.5
Time	$0 \leq t \leq 20$	

3. Result and Discussion

Graphical illustration of the resulting iterations is thus shown below:

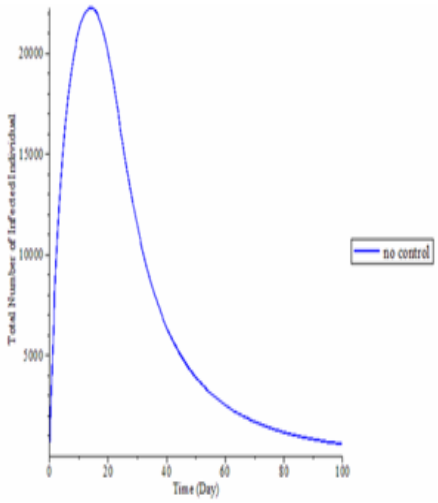

Figure 3.3: Total number of infected individuals without any control measure

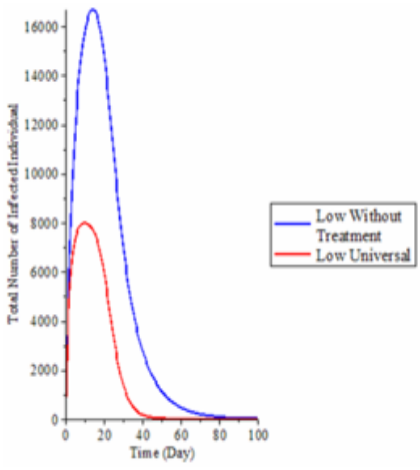

Figure 3.4: Adverse effect of low vaccination and treatment campaign to infected individuals

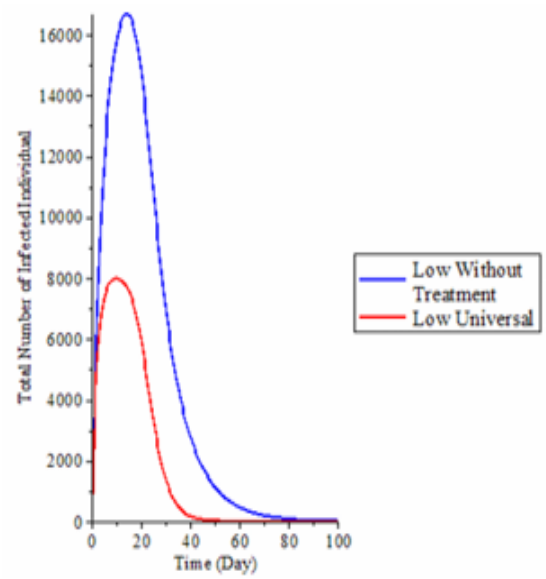

Figure 3.5: Comparison between moderate treatment and vaccination in infected cholera patients

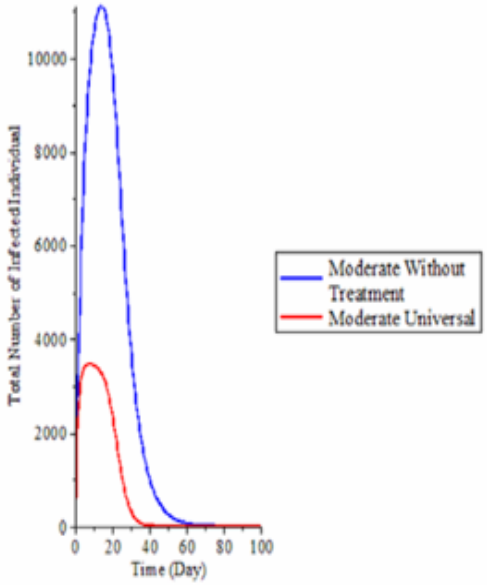

Figure 3.6: Comparison between control measures for infected individuals: moderate vaccination without treatment

From results obtained, Figure 3.3, depicts that the effect of treatment ϕ_2 and vaccination rate ϕ_1 on the population of infected individuals which is vital in the control of cholera disease as this brings about a fall in its absence and steep-slope in the spread of the cholera disease in the infected individuals. Figure 3.4 shows the adverse effect of low vaccination rate on infected individuals, as a fall in the vaccination rate increases the population of the susceptible and recovered population will leads to a drastic rise in the exposed population. Consequently, Figure 3.5 depicts similarly as Figure 3.4 from above that an increases in treatment rate of exposed individuals will lead to an increases in the population of non-diseases classes. Moreover, comparison of the control policies of treatment and vaccination with adequate measure will bring about drastic fall in infected individuals Figure 3.6 came with a rise in treatment and vaccination rate which increases brings about drastic fall in the wide spread of cholera disease in the disease population. However, infected individuals with moderate vaccination will recover rapidly haven been treated properly and individuals without proper treatment are not free of the deadly diseases as vaccination and treatment rate are vital as control measure to eradicating the wide spread of cholera from the population

4. Conclusion

Combining rapid and moderate treatment with vaccination to infected regions this will drastically reduce the spread of cholera. These interventions lower infection rates and mitigate the disease's impact. Healthcare personnel must prioritize and adhere to these measures for effective outbreak control. Prompt and moderate treatment, along with widespread vaccination, should be key components of public health strategies to combat this persistent disease. Implementing these recommendations will significantly aid in managing and ultimately eradicating cholera.

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Conflict of Interest

Authors declare that there is no conflict of interest.

Data Availability Statement

Data sets generated during this research are available from the corresponding author upon request

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