Adıyaman University Journal of Science
https://dergipark.org.tr/en/pub/adyujsci
DergiPark AKADEMİK
ISSN 2147-1630
e-ISSN 2146-586X

# Quantitative Modeling of Treatment and Vaccination Effects on the Dynamics of Cholera Transmission

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**Received:** 03.11.2024 **Accepted:** 03.06.2025 **Published:** 30.06.2025

## Abstract

This study presents a mathematical analysis of the SVEITR model, which incorporates susceptible (s), vaccinated (v), exposed (e), infected (i), treated (t), and recovered (r) 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 the development of immunity. Our findings reveal that combining rapid treatment and vaccination significantly reduces the spread of cholera disease, 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 the Laplace Adomian Decomposition Method, the results reveal that treatment rate reduces the spread of the disease, and vaccination plays a vital role in curbing the aftermath of widespread disease. Hence, there is a 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 offers a valuable framework for policymakers and healthcare professionals to develop effective strategies for cholera control, contributing to improved public health outcomes.

* Corresponding Author DOI: 10.37094/adyujsci.1578202

***Keywords:*** Cholera control; Treatment efficacy; Vaccination rate; Stability analysis; Quantitative analysis; Simulation.

**Kolera Bulaşımının Dinamikleri Üzerinde Tedavi ve Aşılama Etkilerinin Kantitatif Modellemesi**

**Öz**

Bu çalışma, kolera yayılımının dinamiklerini değerlendirmek amacıyla duyarlı (s), aşılı (v), maruz kalmış (e), enfekte (i), tedavi edilen (t) ve iyileşen (r) popülasyonları içeren SVEITR modelinin matematiksel analizini sunmaktadır. Modele tedavi ve aşılama oranlarının entegre edilmesiyle, bu önlemlerin hastalığın bulaşması ve bağışıklık üzerindeki etkilerini anlamayı amaçlıyoruz. Bulgularımız, hızlı tedavi ve aşının birleştirilmesinin kolera hastalığının yayılmasını önemli ölçüde azalttığını ortaya koyarak, bu müdahalelerin halk sağlığı stratejilerindeki önemini vurgulamaktadır. Model, aşılama ve tedavinin zamanında ve yaygın olarak uygulanmasının salgınları etkili bir şekilde kontrol edebileceğini ve hastalığın etkilerini azaltabileceğini göstermektedir. Laplace-Adomian Ayrıştırma Yöntemi ile yapılan sayısal simülasyon sonucunda, tedavi oranının hastalığın yayılımını azalttığı ve aşının hastalığın yaygın etkilerini azaltmada hayati bir rol oynadığı ortaya çıkmıştır. Bu nedenle, bu önlemleri önceliklendiren sağlam sağlık politikalarına duyulan ihtiyaç, özellikle savunmasız bölgelerde koleranın yönetilmesi ve nihayetinde ortadan kaldırılması için önemli ilerlemeler sağlamak açısından gereklidir. SVEITR modeli, politika yapıcılar ve sağlık uzmanları için kolera kontrolüne yönelik etkili stratejiler geliştirmede değerli bir çerçeve sunarak, halk sağlığı sonuçlarının iyileştirilmesine katkıda bulunmaktadır.

***Anahtar Kelimeler*****:** Kolera kontrolü; Tedavi etkinliği; Aşılama oranı; Kararlılık analizi; Kantitatif analiz; Simülasyon.

## 1. Introduction

Cholera is 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, as in [1-2]. 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 [3]. Despite advancements in medical sciences and public health strategies, cholera outbreaks continue to pose a substantial health risk 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 community awareness by [4-5]. These components of sensitization and treatment are crucial in developing a comprehensive approach to control and prevent the disease effectively [6]. Effective treatment of cholera involves prompt rehydration, which can be lifesaving as discussed in [7]. Oral rehydration salts (ORS) are the cornerstone of treatment for most patients, while intravenous fluids are necessary for severe cases [8]. Antibiotics can also reduce the diarrhea and the volume of rehydration fluids needed can be increased instantaneously with researching into optimizing the treatment ongoing in endemic African regions such as Sudan, Ethiopia, Nigeria, Niger and Somalia, aiming to enhance their efficacy and accessibility to healthy water supply and hygienic environmental settings as in [9-10].

The goal is to ensure that treatment protocols are both efficient and adaptable to various healthcare infrastructures, thus reducing mortality and morbidity rates associated with cholera disease [11-12]. Oral cholera vaccines (OCVs) have proven effective in providing immunity and reducing the incidence of the disease as well [13]. The integration of vaccination into public health strategies, especially in high-risk areas, can prevent outbreaks and provide long-term protection [14]. Recently, researchers have focused on improving the efficacy and duration of vaccine-induced immunity, as well as logistics to enhance vaccine distribution and administration to endemic regions, where the success of vaccination campaigns depends on the timely and widespread coverage, particularly before and during cholera outbreaks, as in [15]. Clean water and proper sanitation are fundamental in preventing cholera transmission, where contaminated water sources are the primary vectors for the bacterium, highlighting the need for robust water treatment and safe water storage practices by [16].

Additionally, environmental cleanliness, including the maintenance of clean living conditions and proper sanitation facilities, is essential in [17-18]. 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, as in [19]. 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 [20]. Hand washing technique with soap, disinfectants, and clean water is one of the simplest and 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 [21]. 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 [22]. 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, as well as educating communities about the importance of proper waste disposal [23]. Safe disposal practices help break the transmission cycle and reduce the risk of outbreaks. Awareness campaigns play a crucial role in cholera prevention, informing the public about the deadly disease, its symptoms, and the importance of seeking immediate treatment can save lives [24]. Awareness efforts should also highlight the preventive measures individuals can take to protect themselves and their communities, as in [25]. Utilizing various media platforms, including radio, television, social media platforms, and community outreach programs, can effectively disseminate information and reach a broad audience [26].

However, this research focuses on controlling the spread of cholera, which requires a multifaceted approach. This involves developing a mathematical model that incorporates both effective treatment and vaccination rates, along with various control measures, including water and environmental cleanliness, regional education, regular hand washing, proper waste disposal, and public awareness. By addressing these areas, public health initiatives can significantly reduce the incidence and impact of cholera, ultimately aiming for its eradication. Proper and continuous investment in these strategies is essential to overcoming the persistent threat posed by cholera, particularly in vulnerable regions.

## 2. Materials and Methods

### 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/treated T(t) and recovered population R(t), The rate of migration $\pi$ or inflow into the population resulting to the spread of cholera is $\beta$ 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 individuals at a rate of $\delta$, where exposed individuals are subjected to contracting cholera at $k$, and the rate of recovery is denoted with $\gamma$

The treatment rate of the hospitalized individuals $\phi_2$. More than 75% of contamination risk of vibro-cholerae results in the spread of the disease and $\phi_1$ vaccination rate of susceptible individuals, with $\varepsilon$ representing the vaccine efficacy and shedding rate of infected human population, coupled with natural mortality rate for human and vibro-cholerae are $\varphi$ and $\mu$. The above parameters can be demonstrated with schematic flow in Fig. 1 and a system of nonlinear differential equations in Eqn. (1) below, respectively.

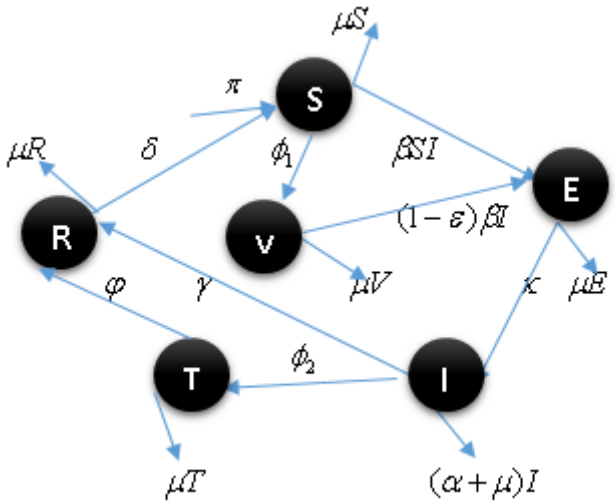


**Figure 1:** Schematic diagram of the model.

### 2.2. Model Analysis

From the schematic diagram above, an equation of the model is obtained in Eqn. (1) below:

$$\frac{dS}{dt} = \pi - \beta SI - (\phi_1 + \mu)S + \delta R$$

$$\frac{dV}{dt} = \phi_1 S - (1-\varepsilon)\beta IV - \mu V$$

$$\frac{dE}{dt} = (1-\varepsilon)\beta IV + \beta SI - (k+\mu)E$$

$$\frac{dI}{dt} = kE - (\alpha + \mu + \gamma + \phi_2)I$$

$$\frac{dT}{dt} = \phi_2 I - (\varphi + \mu)T$$

$$\frac{dR}{dt} = \varphi T + \gamma I - (\delta + \mu)R \qquad (1)$$

Subjected to the initial condition

$$S(0)=s_0, V(0)=v_0, E(0)=e_0, I(0)=r_0, T(0)=t_0, R(0)=r_0 \geq 0. \quad (2)$$

Parameters of the model solution, descriptions, and references are displayed in Table 1 and Table 2 with values that are time-dependent for cholera spread.

**Table 1:** Description of model state variables.

| **Variables** | **Description** |
|---|---|
| S(t) | Susceptible population |
| V(t) | Vaccinated population |
| E(t) | Exposed population |
| I(t) | Infected population |
| T(t) | Treated population |
| R(t) | Recovered population |

**Table 2:** Description of model parameter, values and references

| **Parameter** | **Description** | **Values** | **References** |
|---|---|---|---|
| $N$ | Total population | 80,000 | [10] |
| $\varphi$ | Recovery rate of hospitalized individuals | 0.001 | [11] |
| $\varepsilon$ | Vaccination efficacy | 0.5 | [1] |
| $\phi_2$ | Treatment rate of hospitalized individuals | 0.2 | [18] |
| $\phi_1$ | Vaccination rate of infected individuals | 0.03 | [2] |
| $\mu$ | Natural death | 1.0 | [5] |
| $\delta$ | İmmunity waning rate | 0.0016 | [20] |
| $\pi$ | Recruitment rate | 0.113 | [3] |
| $\beta$ | Rate of cholera transmission | 1.0126 | [14] |
| $\alpha$ | Disease induced death rate | 0.33182 | [21] |
| $\gamma$ | Natural recovery rate | 0.16524 | [17] |
| $k$ | Progression rate between exposed and infected class of individuals | 0.25533 | [9,16] |

### 2.3. Existence and Uniqueness of Model

The system of Eqn. (1) above, which describes an epidemic disease within a human population, should have parameters that are non-negative. To ensure that the system of differential Eqn. (1) is both mathematically and epidemiologically well-posed, it is essential to demonstrate that the state variables in the model are nonnegative. Therefore, the system of Eqn. (1) is bounded by an initial condition of $S(0)=s_0,\ V(0)=v_0,\ E(0)=e_0,\ I(0)=i_0,\ T(0)=t_0,\ R(0)=r_0$, in which the solution will persist in being non-negative throughout their evolution, i.e., $t>0$ and that these positive solutions are bounded in this region. We thus apply the theorem (1) below.

**Theorem 2.3.1.** Let $(x,y)$ be distinct points of normed linear space $\left(X,\|\cdots\|\right)$ over $\Re$. Then the map of $p:[0,1]\subseteq\Re\to\left(X,\|\cdots\|\right)$ 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 $\lambda_0\in[0,1]$,

$$\|p(\lambda)-p(\lambda_0)\|=\|(\lambda-\lambda_0)x+(\lambda-\lambda_0)y\|\le|\lambda-\lambda_0|\left(\|x\|+\|y\|\right) \tag{3}$$

If $\varepsilon>0$ is given, let $\delta=\dfrac{\varepsilon}{\|x\|+\|y\|}$.If $|\lambda-\lambda_0|<\delta$, then the $\|p(\lambda)-p(\lambda_0)\|<\varepsilon$.

Therefore, p is continuous at $\lambda_0$. Sınce $\lambda_0$ is an arbitrary point in [0,1]. then p is continuous on [0,1]. Let X be a linear space over $\Re$. If x,y are distinct points of X, the set $\lambda x+(1-\lambda)y$ lies in $0\le\lambda\le1$.

Hence, the solutions of system of Eqn. (1) are bounded if we consider the total population

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

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

$$\frac{dN(t)}{dt}=\frac{dS(t)}{dt}+\frac{dV(t)}{dt}+\frac{dE(t)}{dt}+\frac{dI(t)}{dt}+\frac{dT(t)}{dt}+\frac{dR(t)}{dt} \tag{4}$$

Such that $\dfrac{dN(t)}{dt}=\pi-\mu(S+V+E+I+T+R)-\alpha I\ \ \to\ \ \dfrac{dN(t)}{dt}\le\pi-\mu N$. When there is no outbreak of cholera, $\delta=0$.Thus, substituting Eqn. (1) into Eqn. (4) as time progressively yields:

$$\lim_{t\to\infty} N(t) \le \lim_{t\to\infty}\left[\frac{\pi}{\mu}+\left(N(0)-\frac{\pi}{\mu}\right)e^{-\mu t}\right]=\frac{\pi}{\mu} \tag{5}$$

If so $N(0) \le \frac{\pi N}{\mu}$, as $N(t) \le \frac{\pi N}{\mu}$. This is a positive invariant set under the flow described by Eqn. (2) so that no solution path leaves through any boundary $\Re_+^6$. However, it is sufficient to consider the dynamics of the model in the domain $\Re_+^6$. In this region, the model can be considered mathematically and epidemiologically well-posed.

### 2.4. Positivity and Boundedness of Model

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

**Theorem 2.4.1.** Suppose $X = x_0$ is a space of consecutive real numbers, and that are defined as

$$L(x,y)=\left(\sum_{i=1}^{n}\left|x_i\right|^{\Omega}\right)^{\frac{1}{\Omega}} \qquad \Omega \ge 1 \tag{6}$$

X with the metric is called $\xi_n^{\Omega}$ space. If $\sum_{i=1}^{N}|x|^i < \infty$ or absolutely convergent and $L(x,y)=\left(\sum_{i=1}^{\infty}\left|x_i-y_i\right|^{\Omega}\right)^{\frac{1}{\Omega}}$, then X with this metric is called an $\xi^{\Omega}$ space.

**Proof.** It can be checked that for each $n$:

$$0 \le x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2 \le (|x_1| + |x_2| + |x_3| + \ldots + |x_n|)^2 \tag{7}$$

This will result to;

$$x_1^2 + x_2^2 \le \left(|x_1| + |x_2|\right)^2 \tag{8}$$

Therefore,

$$0 \le \left(x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2\right)^{\frac{1}{2}} \le \left|x_1\right| + \left|x_2\right| + \left|x_3\right| + \ldots + \left|x_n\right|,$$

If $\sum_{n=1}^{\infty}|x_n|$ converges, that is $\sum_{n=1}^{\infty}|x_n|$is absolutely convergent, then

$$0 \le \left(x_1^2+x_2^2+x_3^2+...+x_n^2\right)^{\frac{1}{2}} \le |x_1|+|x_2|+|x_3|+...+|x_n| = \sum_{n=1}^{\infty}|x_n| < \infty \tag{9}$$

Therefore,

$$0 \le x_n = x_1^2 + x_2^2 + x_3^2 + ... + x_n^2 \le \left[\sum_{n=1}^{\infty}|x_n|\right] < \infty \tag{10}$$

The sequence $x_n$ is monotone increasing and bounded above, it therefore converges. Thus $\sum_{n=1}^{\infty}x_n$ converges absolutely, i. E if $x_n \in \xi^1$, then $x_n \in \xi^2$ where $\xi^1 \le \xi^2$. In case of $\xi^1$ 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 $\xi^2$ 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 $\xi^1 \ne \xi^2$. If $(x_n, y_n)$ are sequences of real numbers, then;

$$\sum_{n=1}^{\infty}(x_i - y_i)^2 \le \sum_{n=1}^{\infty}{x_i}^2 + \sum_{n=1}^{\infty}{y_i}^2 + 2\left[\sum_{n=1}^{\infty}{x_i}^2\right]^{\frac{1}{2}}\left[\sum_{n=1}^{\infty}{y_i}^2\right]^{\frac{1}{2}} \tag{11}$$

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 $\left[\sum_{n=1}^{\infty}(x_i - y_i)^2\right]$ is then 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 $(x_n, y_n) \in \xi^2$ as in [18]. 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 solution of S(t), V(t), E(t), I(t), T(t), R(t) of the Eqn. (1) will always be nonnegative.

$$\text{Let: } \Psi = \left\{ \left(S(t), V(t), E(t), I(t), T(t), R(t)\right) \in \Re_+^6 : N(t) \le \frac{\pi}{\mu} \right\} \tag{12}$$

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

$$\left|\frac{df_1}{dS}\right| = \left|(\beta + \phi_1 + \mu + \delta)\right| < \infty, \quad \left|\frac{df_1}{dV}\right| = |0| < \infty, \quad \left|\frac{df_1}{dE}\right| = |0| < \infty, \quad \left|\frac{df_1}{dI}\right| = |\alpha| < \infty,$$

$$\left|\frac{df_1}{dT}\right| = |\alpha| < \infty$$

$$\left|\frac{df_1}{dR}\right| = |\delta| < \infty, \left|\frac{df_2}{dS}\right| = |\phi_1| < \infty, \left|\frac{df_2}{dV}\right| = \left|(1-\varepsilon)\beta + \mu\right| < \infty, \left|\frac{df_2}{dE}\right| = |0| < \infty,$$

$$\left|\frac{df_2}{dI}\right| = \left|(1-\varepsilon)\beta\right| < \infty \left|\frac{df_2}{dT}\right| = |0| < \infty, \quad \left|\frac{df_2}{dR}\right| = |0| < \infty, \quad \left|\frac{df_3}{dS}\right| = |\beta| < \infty,$$

$$\left|\frac{df_3}{dV}\right| = \left|(1-\varepsilon)\beta\right| < \infty, \quad \left|\frac{df_3}{dE}\right| = \left|(k+\mu)\right| < \infty, \quad \left|\frac{df_3}{dI}\right| = \left|(1-\varepsilon)\beta\right| < \infty, \left|\frac{df_3}{dT}\right| = |0| < \infty,$$

$$\left|\frac{df_3}{dR}\right| = |0| < \infty, \left|\frac{df_4}{dS}\right| = |0| < \infty, \left|\frac{df_4}{dV}\right| = |0| < \infty \left|\frac{df_4}{dE}\right| = |k| < \infty,$$

$$\left|\frac{df_4}{dI}\right| = \left|(\alpha + \gamma + \mu + \phi_2)\right| < \infty, \quad \left|\frac{df_4}{dT}\right| = |0| < \infty, \quad \left|\frac{df_4}{dR}\right| = |0| < \infty, \quad \left|\frac{df_5}{dS}\right| = |0| < \infty,$$

$$\left|\frac{df_5}{dV}\right| = |0| < \infty, \left|\frac{df_5}{dE}\right| = |0| < \infty, \left|\frac{df_5}{dI}\right| = |\phi_2| < \infty, \left|\frac{df_5}{dT}\right| = \left|(\varphi + \mu)\right| < \infty, \left|\frac{df_5}{dR}\right| = |\mu| < \infty,$$

$$\left|\frac{df_6}{dS}\right| = |0| < \infty, \quad \left|\frac{df_6}{dV}\right| = |0| < \infty, \quad \left|\frac{df_6}{dE}\right| = |0| < \infty, \quad \left|\frac{df_6}{dI}\right| = |\gamma| < \infty, \quad \left|\frac{df_6}{dT}\right| = |\varphi| < \infty,$$

$$\left|\frac{df_5}{dR}\right| = \left|(\mu + \delta)\right| < \infty \tag{13}$$

Eqn. (13) above confirms that system of Eqn. (1) is bounded, invariantly and attractively influential on the bounded region of $\Re_+^6$.

### 2.5. Disease Free Equilibrium

The cholera non-infected equilibrium state represents a scenario in which Eqn. (1) is entirely free from Vibrio cholerae spread as in [1]. Consequently, when the number of disease

population of infected, exposed individuals is at equilibrium, i.e I = E = 0. This context brings about the solution for the cholerae free equilibrium oint which is derived as follows in Eqn. (14)

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = \frac{dR}{dt} = 0 \tag{14}$$

At no outbreak of measles infection, the diseases class, at $t > 0$, from Eqn. (8),

$$(S_0, V_0, E_0, I_0, T_0, R_0) = \left( \frac{\pi}{(\phi_1 + \mu)}, \frac{\pi\phi_1}{(\phi_1 - \delta + \mu)(1-\varepsilon)\beta + \mu}, 0,0,0,0 \right) \tag{15}$$

### 2.6. Disease Endemic Equilibrium

Examining cholera endemicity spread, we focus on strategic interventions of treatment and vaccination of infected individuals with the aim of long-term eradication. The frequency of choleraon (S,V,E,I,T,R) at $t \neq 0$, stressing the dynamic aspect of it to gauge on the crucial role in its infectious disease and protection on the populace. Let the endemic nature of Eqn. (1) be $E_e = (S^*, V^*, E^*, I^*, T^*, R^*)$ at steady state, $I \neq 0$, hence the equation for its endemicity at equilibrium is obtained in Eqn. (16) below;

$$\begin{aligned}
S^* &= \frac{\pi(1-\varepsilon)^2[\phi_2 + \mu + \alpha] + \beta[\mu + \gamma + \varphi]}{[(\mu + \gamma + (1-\varepsilon)\phi_1) + \delta]\sqrt{\beta(\gamma + (1-\varepsilon) + (\mu + \gamma + \varphi)}}, \\
V^* &= \frac{(1-\varepsilon)\pi\sqrt{(\varphi + k + \mu)(\delta + \mu + \phi_1)}}{(\delta + \mu + \phi_2)[(\mu + \gamma + (1-\varepsilon)\beta]}, \; E^* = \frac{\beta(1-\varepsilon)(\phi_2 + \gamma + \mu)}{\sqrt{(\gamma + k + \mu)(\delta + \mu + \alpha)}} \\
I^* &= \frac{(1-\varepsilon)][\pi + (\mu + \gamma + \varphi)\sqrt{(k + \phi_1 + \mu)(\delta + \mu + \gamma)}]}{[\mu^2(\delta + \mu + \gamma)]}, \\
T^* &= \frac{(\mu + \gamma + k)}{(\delta + \mu + \alpha)[(\mu + k + \phi_2]} + \frac{\sqrt{(k + \varphi + \phi_1)(\delta + \mu + \delta)}}{(\gamma + \mu + k)} \\
R^* &= \frac{(\mu + \alpha + \phi_1)}{[\mu^2(\varphi + \mu) + (1-\varepsilon)]} + \sqrt{\frac{(\mu + \varphi + k) + \mu^2}{(k + \alpha)(\gamma + \mu + k)(\varepsilon + \mu + \delta)}}
\end{aligned} \tag{16}$$

### 2.7. Basic Reproduction Number

The basic reproduction number $R_{\bullet}$ measures cholera spread by calculating secondary infections using next-generation matrices for new infections and transitions. Let

$R_\bullet = \rho(G - \lambda I\,|)$ where $G = F \times V^{-1}$ and $\rho$ is the spectral radius of the matrix $|\,G - \lambda I\,|$. Thus, the $R_\bullet$ is obtained as it is defined that $F_i = \left(\frac{\partial f_i(x_i)}{\partial x_j}\right), V_i = \left(\frac{\partial \nu_i(x_i)}{\partial x_j}\right)$. Such that $f = \begin{pmatrix} \beta I S_0 + (1-\varepsilon)\beta I V_0 \\ 0 \end{pmatrix}$ and $\nu = \begin{pmatrix} (k+\mu)E \\ -kE + (\alpha+\mu+\gamma+\phi_2)I \end{pmatrix}$, the transmission matrix for the disease and its transition compartmentally for the disease spread is obtained below in Eqn. (17)

$$F = \begin{pmatrix} 0 & \frac{\pi[(1-\varepsilon)\beta + \mu + \phi_1]}{(\phi_1 - \delta + \mu)(1-\varepsilon)\beta + \mu} \\ 0 & 0 \end{pmatrix} V = \begin{pmatrix} (k+\varepsilon) & 0 \\ -k & (\alpha+\mu+\gamma+\phi_2) \end{pmatrix} \tag{17}$$

$$FV^{-1} = \frac{1}{(k+\mu)(\alpha+\mu+\gamma+\phi_2)} \begin{pmatrix} 0 & \frac{\pi[(1-\varepsilon)\beta + \mu + \phi_1]}{(\phi_1 + \mu)(1-\varepsilon)\beta + \mu} \\ 0 & 0 \end{pmatrix} \begin{pmatrix} (\alpha+\mu+\gamma+\phi_2) & 0 \\ -k & (k+\mu) \end{pmatrix}.$$

Resolving the above matrix respectively with their eigenvalues being independent factors of the leading variables, the basic reproduction number for Eqn. (17) is obtained in Eqn. (18) as discussed in [10]

$$R_\bullet = \frac{\pi[(1-\varepsilon)\beta + \mu + \phi_1]}{[(\phi_1 - \delta + \mu)(1-\varepsilon)\beta + \mu](\alpha+\mu+\gamma+\phi_2)} \tag{18}$$

### 2.8. Quantitative Analysis of $R_*$

Here, we conduct a quantitative analysis of $R_\varepsilon$ to assess its metric progression concerning each intervention method. By excluding the values of intervention parameters, we assess Eqn. (18) using the baseline values provided in Table 1, subsequently resulting in Eqns. (19)-(22). The outcomes of these calculations are presented in Table 3.

$$R_\bullet = \frac{\pi[(1-0.028726)\beta + 0.0087363 + 0.5263532\phi_1]}{[(1.27364 - 09377847 + 0.38736\alpha)(1-0.7653\varepsilon)\beta + 0.872625](0.9972 + 0.5243436\phi_2)}$$

$$R_\varepsilon = f(\phi_1)\big|_{\delta=0,\phi_2=0} = \text{-}1.7363526\phi_1 + 1.459137886\phi_2 \tag{19}$$

$$R_\varepsilon = f(\varphi)\big|_{\alpha=0,\rho_1=0} = 0.005728262500 + 0.0000133\varphi + 0.0039257856\alpha \tag{20}$$

$$R_{\varepsilon} = f(\phi_2)\big|_{c=0,\phi_2=0} = 0.093762(0.09756 + 0.54\phi_1) + 0.137747\phi_2 + 0.018\phi \tag{21}$$

$$R_{\varepsilon} = f(\varepsilon)\big|_{\rho_1=0,\tau=0} = 1.38947804 - 1.9276424\varepsilon \tag{22}$$

**Table 3:** Standalone metric of vaccination and general awareness on $R_{\varepsilon}$.

| **A** | | | | | | **B** | | | | | **C** | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| s/n | $\phi_1$ | $\beta$ | $\phi_2$ | $\varepsilon$ | $R_{\varepsilon}$ | s/n | $\phi_1$ | $\beta$ | $\phi_2$ | $\varepsilon$ | s/n | $\phi_1$ | $\beta$ | $\phi_2$ | $\varepsilon$ |
| 1 | 0 | 0 | 0 | 0 | 1.4591378 | 0 | 0 | 0 | 0 | 1.45913788 | 0 | 0 | 0 | 0 | 1.45913788 |
| 2 | 0.2 | 0 | 0 | 0 | 1.1964930 | 0 | 0.2 | 0 | 0 | 1.16731030 | 0 | 0 | 0.2 | 0 | 0.25434513 |
| 3 | 0.4 | 0 | 0 | 0 | 0.93384824 | 0 | 0.4 | 0 | 0 | 0.87548273 | 0 | 0 | 0.4 | 0 | 0.20246410 |
| 4 | 0.6 | 0 | 0 | 0 | 0.67120342 | 0 | 0.6 | 0 | 0 | 0.58365515 | 0 | 0 | 0.6 | 0 | 0.18416303 |

### 2.9. Local Stability for Disease Free Equilibrium

**Theorem 2.9.1.** The disease-free state of the model is locally asymptotically stable if the threshold of the disease spread $R_* < 1$ and unstable whenever it is of the form $R_* > 1$.

**Proof.** The disease-free equilibrium is obtained using the Jacobian matrix approach from next generation matrix of the resulting eigenvalues of respective parameters as of the system of Eqn. (1) and evaluated at the disease free state using the linearization method thus;

$$J_{(E_1)} = \begin{pmatrix} -(\beta+\mu+\phi_1+\delta) & 0 & 0 & \beta & 0 & 0 \\ \phi_1 & -[(1-\varepsilon)\beta+\mu] & 0 & (1-\varepsilon)\beta & 0 & 0 \\ \beta & (1-\varepsilon)\beta & -(k+\mu) & (1-\varepsilon)\beta & 0 & 0 \\ 0 & 0 & k & -(\alpha+\mu+\gamma+\phi_2) & 0 & 0 \\ 0 & 0 & 0 & \phi_2 & -(\varphi+\mu) & 0 \\ 0 & 0 & 0 & \gamma & \varphi & -(\delta+\mu) \end{pmatrix} \tag{23}$$

Computing for the eigenvalues from the characteristic equation as $\left|J_{E_1} - \lambda_i I\right| = 0$, we then have

$$\begin{vmatrix} a-\lambda & 0 & 0 & \beta & 0 & 0 \\ \phi_1 & b-\lambda & 0 & (1-\varepsilon)\beta & 0 & 0 \\ \beta & (1-\varepsilon)\beta & c-\lambda & (1-\varepsilon)\beta & 0 & 0 \\ 0 & 0 & k & d-\lambda & 0 & 0 \\ 0 & 0 & 0 & \phi_2 & e-\lambda & 0 \\ 0 & 0 & 0 & \gamma & \varphi & f-\lambda \end{vmatrix} = 0$$

$a = -(\beta + \mu + \phi_1 + \delta), \quad b = -[(1-\varepsilon)\beta + \mu], \quad c = -(k + \mu), \quad d = -(\alpha + \mu + \gamma + \phi_2),$ $e = -(\varphi + \mu), \ f = -(\delta + \mu)$ as obtained from Eqn. (23) the respective eigen vales are deduced in Eqn. (24) below:

$$\lambda = -(v+\mu), \lambda = -(\alpha_2 + \mu), \lambda = -\mu, \begin{vmatrix} -(\alpha+\mu+\gamma+\phi_2) - \lambda & 0 \\ \phi_2 & -(\varphi+\mu) - \lambda \end{vmatrix}, \lambda_3 = -(\alpha+\mu+\gamma+\phi_2), \lambda = -(\varphi+\mu) \Bigg\} \tag{24}$$

Respective eigenvalues are negatively invariant in the region $\Re_+^6$, indicating a biological implication that there will be a decrease in the spread over time if necessary control measures as indicated are strictly adhered to. Hence it is asymptomatically stable $\forall \lambda_n < 0, n = 1,2 \ldots 6, t > 0$.

### 3. Regional Resilience for Persistence Equilibrium State

**Theorem 3.1.** The local stability of the persistent equilibrium of the model is ensured that if $R_\bullet < 1$, invariantly and otherwise, it is unstable if $R_\bullet > 1$is in the modification of the model equation as discussed in Eqn. (1).

**Proof.** Suppose

$$S = x + S^*, V = y + V^*, E = z + E^*, I = a + I^*, T = b + T^*, R = c + R^* \tag{25}$$

Linearizing Eqn. (1), is then obtained as from Eqn. (26) for respective sensitive parameters for each state variable that are

$$\begin{aligned}
\frac{dS}{dt} &= \pi - \beta(x + S^*)(a + I^*) - (\phi_1 + \mu)(x + S^*) + \delta(x + R^*) \\
\frac{dV}{dt} &= \phi_1(x + S^*) - (1-\varepsilon)\beta(a + I^*)(y + V^*) - \mu(y + V^*) \\
\frac{dE}{dt} &= (1-\varepsilon)\beta(a + I^*)(y + V^*) + \beta(x + S^*)(a + I^*) - (k + \mu)(z + E^*) \\
\frac{dI}{dt} &= k(z + E^*) - (\alpha + \mu + \gamma + \phi_2)(a + I^*) \\
\frac{dT}{dt} &= \phi_2(a + I^*) - (\varphi + \mu)(b + T^*)
\end{aligned} \tag{26}$$

$$\frac{dR}{dt} = \varphi(b + T^*) + \gamma(a + I^*) - (\delta + \mu)(c + R^*)$$

Linearizing Eqn. (25), it is then obtained that each

$$\frac{dx}{dt} = -\beta ax - (\phi_1 + \mu)x - \delta x + \text{higher order} + \text{non - linear terms...}$$

$$\frac{dy}{dt} = \phi_1 x - (1-\varepsilon)\beta ay - \mu y + \text{higher order} + \text{non - linear terms...}$$

$$\frac{dz}{dt} = (1-\varepsilon)\beta ay + \beta ax - (k + \mu)z + \text{higher order} + \text{non - linear terms...} \quad (27)$$

$$\frac{da}{dt} = kz - (\alpha + \mu + \gamma + \phi_2)a + \text{higher order} + \text{non - linear terms...}$$

$$\frac{db}{dt} = \phi_2 a - (\varphi + \mu)b + \text{higher order} + \text{non - linear terms...}$$

$$\frac{dc}{dt} = \varphi b + a\gamma - (\delta + \mu) + \text{higher order} + \text{non - linear terms...}$$

The characteristic equation obtained from its Jacobian matrix is;

$$\begin{vmatrix} A-\lambda & 0 & 0 & -\beta ax & 0 & 0 \\ \phi_1 x & -[(1-\varepsilon)\beta ay + \mu)]y - \lambda & 0 & (1-\varepsilon)\beta ay & 0 & 0 \\ \beta ax & (1-\varepsilon)\beta ay & B-\lambda & \beta ax & 0 & 0 \\ 0 & 0 & kz & C-\lambda & 0 & 0 \\ 0 & 0 & 0 & a\phi_2 & -(\varphi+\mu)-\lambda & 0 \\ 0 & 0 & 0 & a\gamma & \varphi b & (\delta+\mu)c \end{vmatrix} = 0 \quad (28)$$

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

$$\lambda^6 - \big(x + (a + y) + (c + b)\big)\lambda^5 + \big((x + c)(y + s) + az + by\big)(1 + b)\lambda^4 - \big(ab(c + z) + bc(a + y)\big)(1 + z)\lambda^3$$

$$+ y\big(cz(a + b) + xb(a + y)\big)\lambda^2 - (a + c)(x + y)\lambda + abcxyz = 0. \quad (29)$$

With the invariance of the eigen values it is said to be locally asymptotically stable because all are of negative results.

### 3.1. Global Stability of Disease-Free Equilibrium

We employ the Lyapunov's function approach to establish the global asymptotic stability of the model solution for Eqn. (1) and ta disease free equilibrium, utilizing the Lyapunov algorithm for the model state variables, this is deduced in Eqn. (30) thus;

$$\Phi\left(t,S,V,E,I,T,R\right)=C_1I_1+C_2I_2+C_3I_3 \tag{30}$$

$$\frac{d\Phi}{dt}=C_1I_1^{\bullet}+C_2I_2^{\bullet}+C_3I_3^{\bullet}$$

$$\begin{aligned}&=C_1\left((1-\varepsilon)\beta I_2V+\beta I_2S-(k+\mu)I_1\right)+C_2\left(kI_1-(\alpha+\gamma+\phi_2+\mu)I_2\right)\\&\quad+C_2\left(\phi_2I_2-(\varphi_2+\mu)I_3\right)\end{aligned}$$

$$\begin{aligned}&=C_2kI_1-C_1(k+\mu)I_1+C_1(1-\varepsilon)\beta I_2V+C_1\beta I_2S-C_2(\alpha+\gamma+\phi_2+\mu)I_2\\&\quad+C_3\phi_2I_2-C_3(\varphi_2+\mu)I_3\end{aligned}$$

$$\begin{aligned}&\le\left(C_2k-C_1(k+\mu)\right)I_1+\left(C_1(1-\varepsilon)\beta V_0+C_1\beta S_0-C_2(\alpha+\gamma+\phi_2+\mu)+C_3\phi_2\right)I_2\\&\quad-C_3(\varphi_2+\mu)I_3\end{aligned}$$

$$S_0=\frac{\pi}{(\phi_1+\mu)},V_0=\frac{\pi\phi_1}{(\phi_1-\delta+\mu)(1-\varepsilon)\beta+\mu},E_0=0,I_0=0,T_0=0,R_0=0 \tag{31}$$

$$S_0=S_0=\frac{\pi}{(\phi_1+\mu)},V_0=\frac{\pi\phi_1}{(\phi_1-\delta+\mu)(1-\varepsilon)\beta+\mu},C_1=\frac{1}{(k+\mu)},$$

$$C_2=\left(\frac{(\phi_i+\mu-\delta)(1-\varepsilon)+\alpha+\beta)}{\pi(\phi_2+\mu+\delta+\gamma)(\alpha+\mu)}\right)$$

$$\frac{d\Phi}{dt}\le C_1\left(\frac{\pi(1-\varepsilon)\beta+\pi\mu+\pi\phi_1}{[(\phi_1-\delta+\mu)(1-\varepsilon)}-\frac{(k+\mu)}{(k+\mu)}\right)I_1-\left(\frac{\pi(\gamma+\mu+\delta)(1-\varepsilon)\beta+\mu}{(\phi_1+\mu)(\gamma+\mu+\delta)(1-\varepsilon)}-\frac{\pi(\gamma+\mu+\delta)(1-\varepsilon)\beta+\mu}{(\phi_1+\mu)(\gamma+\mu+\delta)(1-\varepsilon)}\right)I_2$$

$$\frac{d\Phi}{dt}\le\psi(R_*-1) \tag{32}$$

It is pertinent to observe that when time of the disease spread is simultaneously increasing at $t\to\infty$ and the constant of $C_1<1$, substituting into the model Eqn. (31), it is revealed that,

based on Lasalle's invariance principle $\Phi^{\bullet} = 0$, is globally asymptotically stable whenever $R_* > 1$ as seen in Eqn. (32), because the basics reproduction number of the model system of Eqn. (1) is independent of the deiseal state eigen parameters.

### 3.2. Global Stability of Disease Endemic Equilibrium

**Theorem 3.2.1** 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 equations, i. e

$$\frac{dx}{dt} = f(x,y) \Leftrightarrow \frac{dy}{dt} = g(x,y) \tag{33}$$

The Dulac criterion states that if there exists a continuously differentiable function $B(x,y)$ (called the Dulac function) such that the expression in Eqn. (33) gives the equation below;

$$\frac{\partial}{\partial x}\big(B(x,y)f(x,y)\big) + \frac{\partial}{\partial x}\big(B(x,y)g(x,y)\big) \tag{34}$$

Which 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 entirely within $D$. To apply this to determine the global stability of an endemic equilibrium $(x^*, y^*)$ of a mathematical model, the endemic equilibrium point $(x^*, y^*)$, which is also defined by the Dulac function $B(x,y)$ gives the equation below

$$\frac{\partial}{\partial x}\big(B(x,y)f(x,y)\big) + \frac{\partial}{\partial x}\big(B(x,y)g(x,y)\big) \text{ as } B(x,y)g(x,y) \tag{35}$$

This indicates that the equation has a constant sign (either 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 $\exists B(x,y) \in C^1$ is such that

$$\frac{\partial}{\partial x}\big(B(x,y)f(x,y)\big) + \frac{\partial}{\partial x}\big(B(x,y)g(x,y)\big) \neq 0 \text{ in } D. \tag{36}$$

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 then employ this concept of Dulac's criterion. Let, $X = (S, V, E, I, T, R)$ define the Dulac's function, i. E if $G = \frac{1}{SI}$. The following system of equations are derived.

$$
\begin{aligned}
G\frac{dS}{dt} &= \frac{1}{SI}\{\pi - \beta SI - (\phi_1 + \mu)S + \delta R\} \\
G\frac{dV}{dt} &= \frac{1}{SI}\{\phi_1 S - (1-\varepsilon)\beta IV - \mu V\} \\
G\frac{dE}{dt} &= \frac{1}{SI}\{(1-\varepsilon)\beta IV + \beta SI - (k+\mu)E\} \\
G\frac{dI}{dt} &= \frac{1}{SI}\{kE - (\alpha + \mu + \gamma + \phi_2)I\} \\
G\frac{dT}{dt} &= \frac{1}{SI}\{\phi_2 I - (\varphi + \mu)T\} \\
G\frac{dR}{dt} &= \frac{1}{SI}\{\varphi T + \gamma I - (\delta + \mu)R\}
\end{aligned}
\tag{37}
$$

The above system of equations then results to;

$$
\begin{aligned}
G\frac{dS}{dt} &= \left\{\frac{\pi}{SI} - \beta - \frac{(\phi_1 + \mu)}{I} + \frac{\delta R}{SI}\right\} \\
G\frac{dV}{dt} &= \left\{\frac{\phi_1}{I} - \frac{(1-\varepsilon)\beta}{S} - \frac{\mu}{S}\right\} \\
G\frac{dE}{dt} &= \left\{\frac{(1-\varepsilon)\beta}{S} + \beta - \frac{(k+\mu)E}{S}\right\} \\
G\frac{dI}{dt} &= \left\{\frac{kE}{SI} - \frac{(\alpha + \mu + \gamma + \phi_2)}{I}\right\} \\
G\frac{dT}{dt} &= \left\{\frac{\phi_2}{S} - \frac{(\varphi + \mu)T}{SI}\right\} \\
G\frac{dR}{dt} &= \left\{\frac{\varphi T}{SI} + \frac{\gamma}{S} - \frac{(\delta + \mu)R}{SI}\right\}
\end{aligned}
\tag{38}
$$

At an initial time, $t > 0$ orbital resolution of the system of equations is given by $\frac{d(GX)}{dt}$ which are obtained below compartmentally for each state variable and resolved descriptively to have,

$$\frac{d(GX)}{dt} = \frac{\partial}{\partial S}\left\{G\frac{dS}{dt}\right\} + \frac{\partial}{\partial V}\left\{G\frac{dV}{dt}\right\} + \frac{\partial}{\partial E}\left\{G\frac{dE}{dt}\right\} + \frac{\partial}{\partial I}\left\{G\frac{dI}{dt}\right\} + \frac{\partial}{\partial T}\left\{G\frac{dT}{dt}\right\} + \frac{\partial}{\partial R}\left\{G\frac{dR}{dt}\right\}$$

$$\frac{d(GX)}{dt} = \frac{\partial}{\partial S}\left\{\frac{\pi}{SI} - \beta - \frac{(\phi_1+\mu)}{I} + \frac{\delta R}{SI}\right\} + \frac{\partial}{\partial V}\left\{\frac{\phi_1}{I} - \frac{(1-\varepsilon)\beta}{S} - \frac{\mu}{S}\right\} + \frac{\partial}{\partial E}\left\{\frac{(1-\varepsilon)\beta}{S} + \beta - \frac{(k+\mu)E}{S}\right\}$$

$$+ \frac{\partial}{\partial I}\left\{\frac{kE}{SI} - \frac{(\alpha+\mu+\gamma+\phi_2)}{I}\right\} + \frac{\partial}{\partial T}\left\{\frac{\phi_2}{S} - \frac{(\varphi+\mu)T}{SI}\right\} + \frac{\partial}{\partial R}\left\{\frac{\varphi T}{SI} + \frac{\gamma}{S} - \frac{(\delta+\mu)R}{SI}\right\} \quad (39)$$

$$\frac{d(GX)}{dt} = \left\{-\frac{[\pi+\beta+(\phi_1+\mu)+\delta]}{SI}\right\} + \frac{\partial}{\partial V}\left\{-\frac{\phi_1+(1-\varepsilon)\beta+\mu}{SI}\right\} + \frac{\partial}{\partial E}\left\{-\frac{(1-\varepsilon)\beta+\beta+(k+\mu)}{S}\right\}$$

$$+ \frac{\partial}{\partial I}\left\{-\frac{k+(\alpha+\mu+\gamma+\phi_2)}{SI}\right\} + \frac{\partial}{\partial T}\left\{-\frac{\phi_2+(\varphi+\mu)}{S}\right\} + \frac{\partial}{\partial R}\left\{-\frac{\varphi+\gamma+(\delta+\mu)}{SI}\right\}$$

$$\frac{d(GX)}{dt} = -\left\{\begin{array}{l}\frac{A(1-v)+[(1-\rho)+\rho\beta]+(m+\mu)}{SI} + \frac{m+(1-\rho)-(\rho\beta+\mu)}{I} \\ + \frac{(1-\rho)\beta+(\delta+\mu)}{I} + \frac{\delta+\rho\beta+((\gamma+\mu+d))}{SI} + \frac{Av+\gamma-\mu}{SI}\end{array}\right\} \quad (40)$$

$$\frac{d(GX)}{dt} = -\left\{\frac{[2(1-\rho)^2+3\rho\beta^2]+\sqrt{\gamma[2m^2+m(1-\rho)-v^2(\rho\beta+\mu)]}}{SI}\right\} < 0 \quad (41)$$

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

### 3.3. Sensitivity Analysis

The primary aim is to assess the sensitivity of the basic reproduction number by computing its derivatives concerning all relevant parameters. This analysis will result in the determination of the normalized forward sensitivity index which is denoted as $\Gamma_Z^{R_\bullet} = \frac{\partial R_\bullet}{\partial Z} \times \frac{Z}{R_\bullet}$. Here we investigate the influence of various factors that affects the dynamic progression of cholera disease dynamics. Hence, the respective equations depicting the influence on cholera spread is obtained in Eqn. (42) and its values are illustrated in Table 4 below.

$$\begin{aligned}
&\frac{\partial R_*}{\partial \beta} = \frac{\partial R_*}{\partial \beta} \times \frac{\beta}{R_*} = 1.002863633, \frac{\partial R_*}{\partial \phi_1} = \frac{\partial R_*}{\partial \phi_1} \times \frac{\phi_1}{R_*} = 0.001307654, \\
&\frac{\partial R_*}{\partial \phi_2} = \frac{\partial R_*}{\partial \phi_2} \times \frac{\phi_2}{R_*} = 1.1096546, \frac{\partial R_*}{\partial \mu} = \frac{\partial R_*}{\partial \mu} \times \frac{\mu}{R_*} = 0.15356728, \\
&\frac{\partial R_*}{\partial \varphi} = \frac{\partial R_*}{\partial \varphi} \times \frac{\varphi}{R_*} = 0.765438, \frac{\partial R_*}{\partial \pi} = \frac{\partial R_*}{\partial \pi} \times \frac{\pi}{R_*} = 0.564321
\end{aligned} \tag{42}$$

The above results generated descriptively are depicted in Table 3 below.

**Table 4:** Sensitivity analysis of parameter and indices.

| Parameters | Sensitivity indices |
|---|---|
| $\beta$ | 1.002863633 |
| $\phi_1$ | 0.001307654 |
| $\phi_2$ | 1.1096546 |
| $\mu$ | 0.15356728 |
| $\varphi$ | 0.765438 |
| $\pi$ | 0.564321 |

Table 4 above shows that the sensitivity indices are positively invariant in $\Re_6^+$. The sensitivity indices depend on the values of each parameter of $R_*$ and this brings about changes in the values that will affect the behavior of the threshold on the spread or vanity of cholera disease. Based on the Table 3, above, we can conclude that parameter $\varepsilon$ is the most sensitive to the basic reproduction number of the cholera disease. Particularly, increasing the value of $\varepsilon$ will result in a 80.86% increase in $R_*$, while increasing the value of $k$ will lead to a 91.52% decrease in $R_*$.

### 3.4. Numerical Simulation

Numerical simulation was conducted on the cholera model by creating the following iterative scheme of Laplace Adomian Decomposition Method for the model equation. The (LADM) was employed to computationally analyze the epidemic model. Also, Maple 18 software

facilitated the generation of iteration formulas for each compartment. These formulars were then iteratively solved which enables the numerical evaluation of the model's dynamics and providing insights into the epidemic's behavior and progression. Taking the (LADM) transforms from both sides of the system of Eqn. (1) to the form of Eqn. (43) below.

$$
\begin{aligned}
L\left[\frac{dS}{dt}\right] &= L[\pi] - L[\beta SI - (\phi_1 + \mu)S + \delta R] \\
L\left[\frac{dV}{dt}\right] &= L[\phi_1 S] - L[(1-\varepsilon)\beta IV - \mu V] \\
L\left[\frac{dE}{dt}\right] &= L[(1-\varepsilon)\beta IV] - L[\beta SI - (k+\mu)E] \\
L\left[\frac{dI}{dt}\right] &= L[kE] - L[(\alpha + \mu + \gamma + \phi_2)I] \\
L\left[\frac{dT}{dt}\right] &= L[\phi_2 I] - L[(\varphi + \mu)T] \\
L\left[\frac{dR}{dt}\right] &= L[\varphi T] - L[\gamma I - (\delta + \mu)R]
\end{aligned}
\tag{43}
$$

Substituting from Eqn. (1) into Eqn. (43) above to yield

$$
\begin{aligned}
mL[S(t)] &= S(0) + \pi + L[-\alpha[SI] - \pi - \beta SI - (\phi_1 + \mu)S + \delta R] \\
mL[V(t)] &= V(0) + L[\beta_1 S] - L[\phi_1 S - (1-\varepsilon)\beta IV - \mu V] \\
mL[E(t)] &= E(0) + L[\alpha SI] - L[(1-\varepsilon)\beta IV + \beta SI - (k+\mu)E] \\
mL[I(t)] &= I(0) + L[kE] - L[(\alpha + \mu + \gamma + \phi_2)I] \\
mL[T(t)] &= I(0) + L[\phi_2 I] - L[(\varphi + \mu)T] \\
mL[R(t)] &= R(0) + L[\varphi T] - L[\gamma I - (\delta + \mu)R]
\end{aligned}
\tag{44}
$$

Where at initial values of each state variables, $S(0) = s_0$, $V(0) = v_0$, $E(0) = e_0$, $I(0) = i_0$, $R(0) = r_0$, the resulting iteration from Eqn. (44) gives that in Eqn. (45) below

$$
\begin{aligned}
L[S(t)] &= \frac{s_0}{m} + \frac{\pi}{m^2} + \frac{1}{m} L[-\alpha[SI] - \beta SI - (\phi_1 + \mu)S + \delta R] \\
L[V(t)] &= \frac{v_0}{m} + \frac{1}{m} L[\phi_1 S] - L[(1-\varepsilon)\beta IV - \mu V]
\end{aligned}
$$

$$L[E(t)] = \frac{e_0}{m} + \frac{1}{m} + L[(1-\varepsilon)\beta I] - L[\beta SI - (k+\mu)E] \tag{45}$$

$$L[I(t)] = \frac{i_0}{m} + \frac{1}{m} + L[kE] - L[(\alpha + \mu + \gamma + \phi_2)I]$$

$$mL[T(t)] = \frac{r_0}{m} + \frac{1}{m} + L[\phi_2 I] - L[(\varphi + \mu)T]$$

$$mL[R(t)] = \frac{r_0}{m} + \frac{1}{m} + L[\varphi T] - L[\gamma I - (\delta + \mu)R]$$

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

$$S(t) = s_0 + \pi t + L^{-1}\left(\frac{1}{m}L[\pi - \beta SI - (\phi_1 + \mu)S + \delta R]\right)$$

$$V(t) = v_0 + L^{-1}\left(\frac{1}{m}L[\phi_1 S] - L[(1-\varepsilon)\beta IV - \mu V]\right)$$

$$E(t) = e_0 + L^{-1}\left(\frac{1}{m} + L[(1-\varepsilon)\beta] - L[\beta SI - (k+\mu)E]\right) \tag{46}$$

$$I(t) = i_0 + L^{-1}\left(\frac{1}{m} + L[kE] - L[(\alpha + \mu + \gamma + \phi_2)I]\right)$$

$$T(t) = i_0 + L^{-1}\left(\frac{1}{m} + L[\phi_2 I] - L[(\varphi + \mu)T]\right)$$

$$R(t) = r_0 + L^{-1}\left(\frac{1}{m}L[\varphi T] - L[\gamma I - (\delta + \mu)R]\right)$$

Subsequently, iteration results obtained from the above Eqn. (46) which is deduced from the general iterative series in Eqn. (47) below as,

$$\sum_{k=0}^{\infty} S_n(t) = s_0 + \pi t + L^{-1}\left(\frac{1}{m}L\left[-\alpha\sum_{k=0}^{\infty}\pi_n - \phi_1\sum_{k=0}^{\infty}S_n + \phi_2\sum_{k=0}^{\infty}V_n - \mu\sum_{k=0}^{\infty}S_n\right]\right)$$

$$\sum_{k=0}^{\infty} V_n(t) = v_0 + \pi t + L^{-1}\left(\frac{1}{m}L\left[-\sum_{k=0}^{\infty}((1-\varepsilon)\beta) - \beta_1\sum_{k=0}^{\infty}V_n + \beta_2\sum_{k=0}^{\infty}V_n - \mu\sum_{k=0}^{\infty}V_n\right]\right)$$

$$\sum_{k=0}^{\infty} E_n(t) = e_0 + L^{-1}\left(\frac{1}{m} + L\varphi\sum_{k=0}^{\infty}\varepsilon_n - L[\beta-(k+\mu)]\sum_{k=0}^{\infty}E_n\right)$$

$$\sum_{k=0}^{\infty} I_n(t) = i_0 + L^{-1}\left(\frac{1}{m} + L\delta\sum_{k=0}^{\infty}E_n - L[(\alpha+\mu+\gamma+\phi_2)]\sum_{k=0}^{\infty}I_n\right) \quad (47)$$

$$\sum_{k=0}^{\infty} T_n(t) = i_0 + L^{-1}\left(\frac{1}{m} + L\alpha\sum_{k=0}^{\infty}E_n - L[(\varphi+\mu)]\sum_{k=0}^{\infty}T_n\right)$$

$$\sum_{k=0}^{\infty} R_n(t) = i_0 + L^{-1}\left(\frac{1}{m} + L\phi_2\sum_{k=0}^{\infty}E_n - L[(\delta+\mu)]\sum_{k=0}^{\infty}R_n\right)$$

The initial approximations of each class are given by Eqn. (44). Comparing the coefficients at $n=1$, using the recurrence relations obtained from the iterations in Eqn. (47). Compartmentally, we obtained that

$$S_1(t) = (\pi i_0 s_0 - \mu s_0 - \beta s_0 + \phi_1 v_0)t + \left(-\frac{1}{2}\alpha i_0\varepsilon - \frac{1}{2}\mu\pi - \frac{1}{2}\delta\varphi_1\right)t^2$$

$$V_1(t) = \left(-\mu s_0 + \varphi i_1 s_0 - \beta v_0 + (1-\varepsilon)\varepsilon e_0\right)t + \frac{1}{2}\varepsilon\beta_1 t^2$$

$$E_1(t) = \left(\varphi i_0 s_0 - \mu e_0 - \delta e_0\right)t + \frac{1}{2}\alpha\pi i_1 t^2$$

$$I_1(t) = kE - (\alpha+\mu+\gamma+\phi_2)I\left(-\delta i_0 - \alpha + \mu + \gamma + \phi_2 e_0 i_0 + \sigma e_0\right)t$$

$$T_1(t) = \left(-\mu r_0 + \phi_2 s_0 v_0 + (\varphi+\mu)i_0\right)t$$

$$R_1(t) = \frac{1}{3}\varphi\phi_2\left(-\varphi + \mu r_0 + \frac{1}{2}(\delta+\mu)i_0\right)t \quad (48)$$

Further iterations are done to obtain successive iterative terms at n = 2 which gives Eqn. (49) below

$$S_2(t) = \left(\frac{1}{2}\alpha^2 i^2 s_0 + \frac{1}{2}\alpha i s_0 + \frac{1}{3}\alpha i s_0\mu_0 + \frac{1}{2}\alpha i s_0\rho_0 - \frac{1}{2}\alpha i s_0 e_0 + \frac{1}{2}\alpha i s_0\beta_1 - \frac{1}{2}\alpha i s_0\beta_2\right)t$$
$$+\left(\frac{1}{2}\mu^2 s_0 + \beta_1\mu s_0 + \beta_1\mu v_0 + \frac{1}{2}\beta^2 s_0 + \frac{1}{2}\beta_1\right)t^2 + \left(\frac{1}{6}\alpha^2 i^2\theta + \frac{1}{3}\alpha i_0\pi\delta + \frac{2}{3}\alpha i_0\pi\mu\right)t^3$$

$$V_2(t) = -\frac{1}{2}\left(\alpha i s_0\beta_1 + \frac{1}{2}\mu^2 v_0 - \beta_1\mu s_0 + \beta_2\mu s_0 - \frac{1}{2}\beta_1\beta_2 v_0 + \frac{1}{2}{\beta_2}^2 v_0\right)t^2$$
$$-\left(\frac{1}{6}\alpha i_0\pi\beta_1 - \frac{1}{3}\beta_1\mu\pi - \frac{1}{6}{\beta_1}^2\pi + \frac{1}{6}\beta_2\pi\beta_1\right)t^3$$

$$E_2(t) = \left(-\frac{1}{6}\alpha^2 i^2 - \frac{1}{3}\alpha i_0\delta - \frac{2}{3}\alpha i_0\mu - \frac{1}{3}\alpha i_0\rho + \frac{1}{3}\alpha e_0\sigma_1 - \frac{1}{6}\mu^2 - \frac{1}{6}\alpha i_0\pi\beta_1\right)t^3$$
$$-\left(\frac{1}{2}\alpha^2 i^2 s_0 - \sigma i s_0 - \frac{2}{3}\alpha i s_0\mu_0 - \frac{1}{3}\alpha i s_0\rho_0 - \mu^2 i e_0\beta_1 + \frac{1}{2}\alpha i e_0\sigma^2\right)t^2$$

$$I_2(t) = -\frac{1}{6}\alpha^2 + \left(\sigma\alpha i s_0 + \frac{1}{2}\delta^2 i_0 + \delta\mu i_0 - \delta\sigma i e_0 + \mu^2 i_0 - \mu\rho i_0\right)t - \left(\mu\sigma i_0 + \frac{1}{2}\rho^2 i_0\right)t^2$$

$$T_2(t) = \left(\frac{1}{2}\sigma\alpha i s_0 + \frac{1}{2}\delta^2 i_0 + \delta\mu i_0 - \frac{1}{2}\delta\sigma i e_0 + \frac{1}{2}\mu^2 i_0 - \mu\rho i_0\right)t$$
$$+\left(\mu\sigma i_0 + \frac{1}{2}\rho^2 i_0 - \frac{1}{2}\rho\sigma i e_0\right)t^2$$

$$R_2(t) = \left(-\frac{1}{2}\delta\rho i_0 + \frac{1}{2}\mu^2 r_0 - \mu\sigma i_0 - \frac{1}{2}\rho^2 i_0 + \frac{1}{2}\phi 1\varphi e_0\right)t^2$$

and so on. This can be further resolved untived until the desired number of iterations is obtained. Thus, the obtained raw solution to each model compartment is summarily expressed in some form as shown in Eqn. (49) below:

$$S(t) = \sum_{k=0}^{3} s_k(t), V(t) = \sum_{k=0}^{3} v_k(t), E(t) = \sum_{k=0}^{3} e_k(t), I(t) = \sum_{k=0}^{3} i_k(t), R(t) = \sum_{k=0}^{3} r_k(t) \quad (49)$$

Evaluating these series results using the corresponding variables and parameter values, gives:

$$S(t) = 500.012 - 30.4440t + 1.1315290300t^2 - 0.05075029853t^3$$
$$-3.509616000x10^{-13}t^5 - 5.179149070x10^{-7}t^4$$

$$V(t) = 120 - 1.5060t - 0.01591470000t^2 + 0.001033697580t^3$$
$$+ 9.015111000\times10^{-9}t^4$$

$$E(t) = 65 + 18.1785t - 1.171778775t^2 + 0.04929560765t^3$$
$$+ 5.087939775\times10^{-7}t^4$$

$$I(t) = 23.0.9 - 60t + 0.0292567500t^2 - 0.0008440367798t^3$$
$$- 4.378044000\times10^{-9}t^4 \quad (50)$$

$$T(t) = 23.0.9 - 60t + 0.0292567500t^2 - 0.0008440367798t^3 - 4.378044000\times10^{-9}t^4$$

$$R(t) = 14 - 0.0155t - 0.005054500000t^2 + 0.0001458242541t^3 + 2.473075000\times10^{-10}t^4$$

Hence, from the results of successive iterations in Eqn. (50), the comparison of control intervention effects on sub-populations in its graphical illustration is depicted as shown in Table 5 below.

**Table 5:** Comparison of parameters $\phi_1$ and $\phi_2$ values for $\varepsilon$ at $\varepsilon$ = 0, 0.2, .. 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 | 0.1, 0.25...0.5 |
| $\phi_1$ | treatmen tintervention | 0.1, 0.2,... 0.5 |
| $\phi_2$ | Vaccination intervention | 0.1, 0.2,... 0.5 |

## 4. Iterative Results

Graphical illustration of the resulting iterations is thus shown below:

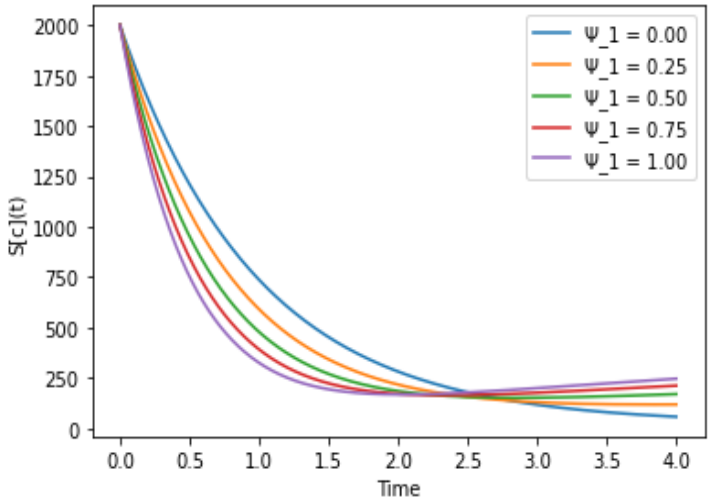


**Figure 2:** Effect of treatment rate on susceptible individuals.

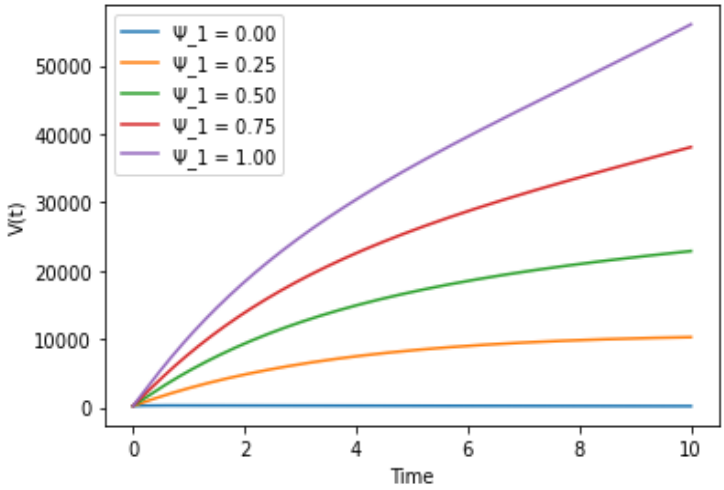


**Figure 3:** Adverse effect of treatment rate on the population of vaccinated individuals.

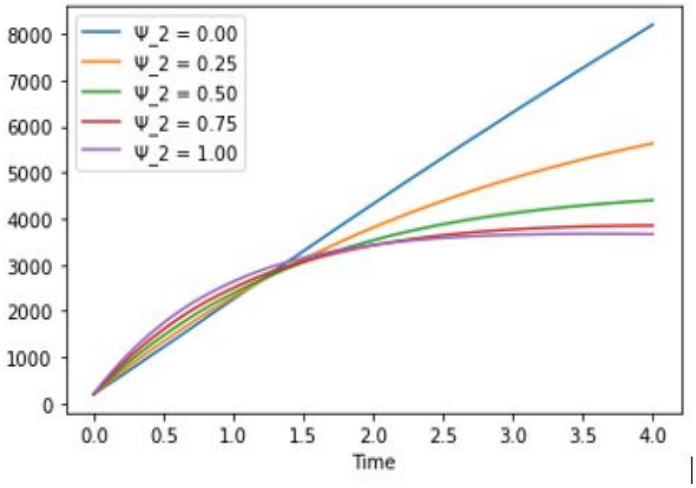


**Figure 4:** Adverse Increase of vaccination efficacy on individuals in the recovered population.

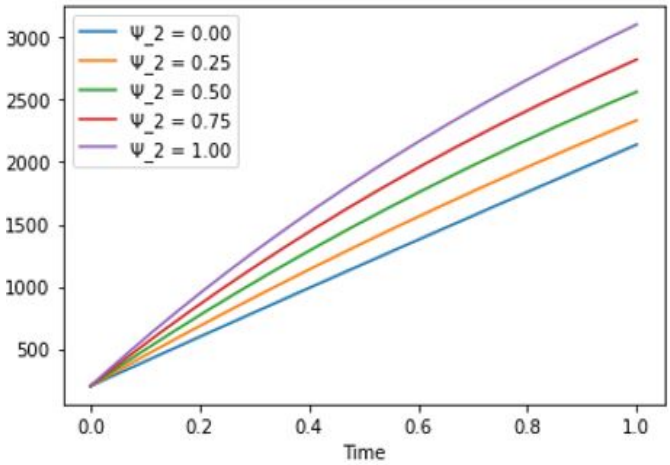


**Figure 5:** Increase in vaccination rate on exposed population.

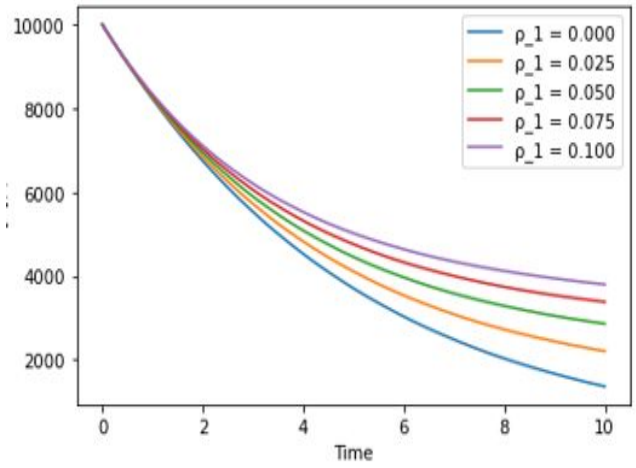


**Figure 6:** Effect of treatment rate on infected population.

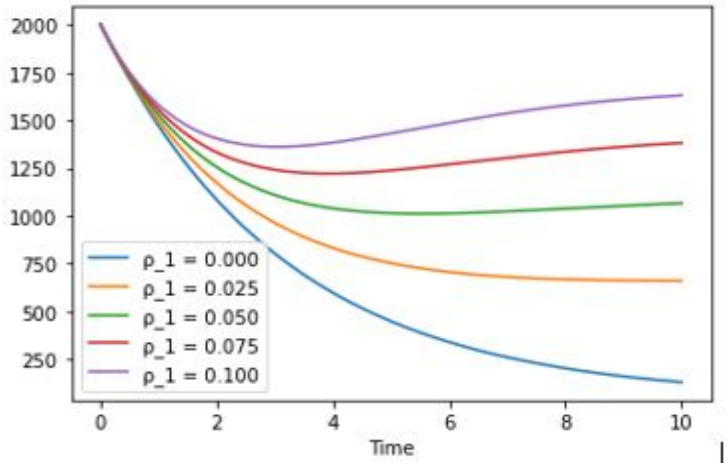


**Figure 7:** Effect of treatment rate on infected population.

### 5. Discussion of Graphical Results

From results obtained, Fig. 2 and Fig. 3, depicts that the effect of treatment $\phi_2$ and vaccination rate $\phi_1$ on the population of susceptible, infected, exposed and recovered is vital to the control of cholera disease as this brings about a rise in its efficacy and steep-slope in the spread of the viral disease in the disease compartments. In Fig. 4 shows the effect of vaccination rate on the population of the recovered individuals, as a rise the vaccination rate increases the population of the susceptible and rescored population which leads to a drastic fall in the exposed and infected population. Consequently, Fig. 5 and Fig. 6 depicts that an increases in treatment rate of exposed individuals will lead to an increase in the population of non-diseases classes. However, comparison of the control policies of on the infected population of Fig. 7 came with a rise in treatment and vaccination rate increases bring about drastic fall in the wide spread of cholera

disease in the disease population. Lastly, from the susceptible population of Fig. 8, the adverse effect of vaccination and treatment on this population brings about a rapid influx of non-disease class of individuals who reunite into the population free of the deadly diseases. This implies that, vaccination and treatment rate are vital control measures to eradicating the wide spread of cholera from the populace. The effect of vaccination and treatment brings about convergence to the spread of cholera disease in the exposed and infected population respectively.

## 6. Conclusion

The study explains that combining rapid treatment, and vaccination will significantly aid in the control of the spread of cholera. These interventions reduce infection rates and mitigate the disease's impact in endemic regions. It is imperative for healthcare personnel to prioritize and adhere to these measures to assist in controlling cholera outbreaks effectively. Prompt treatment and widespread vaccination should be an integral component of public health strategies to combat this persistent and potentially devastating disease. It is therefore recommended that adequate awareness, environmental sanitation in lieu of water treatment, and antibiotics can also be looked into in the future to study towards curtailing the spread of this disease. We can achieve substantial progress in managing and eventually controlling the spread of cholera.

## Acknowledgements

Authors appreciate the academic staffs of the Department of Mathematical Sciences, Osun State University, Osogbo, and anonymous reviewers who have improved this manuscript.

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