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RESEARCH ARTICLE

Analysing Cholera-Measles Epidemics of a Fractional-Order Model with Preventive Strategies Using Laplace Adomian Decomposition Method

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Abstract:
This study provides an in-depth examination of cholera-measles epidemics through a fractional-order mathematical model that integrates essential preventive measure of treatment rate and vaccination efficacy. By employing fractional calculus, the model captures the memory and hereditary properties of disease transmission dynamics, offering a more realistic representation than classical integer-order models. This consists of multiple compartments representing the progression of each disease, with control measures such as treatment, vaccination, water sanitation and public health awareness integrated into the system. Sensitivity analysis further identifies crucial parameters influencing disease dynamics, guiding resource allocation for optimal control. Using a numerical iteration of Laplace Adomian Decomposition method on the model to see the effect of treatment rate on measles and vaccination efficacy on cholera transmission as these changes reduces the spread of the diseases. The results reveal that fractional-order model not only enhance the accuracy of epidemic forecasting but also demonstrate the effectiveness of timely and combined preventive strategies in reducing infection rates. The findings indicate the relevance of fractional modeling and provides valuable insights for informing strategic planning efforts to cease cholera-measles transmission.

Keywords: Fractional-order, Cholera-measles, Treatment rate, Vaccine efficacy, Laplace adomian decomposition method.

1. Introduction

Cholera-measles remain persistent public health challenges, particularly in regions with limited access to clean water, vaccination programs and adequate healthcare infrastructure. Despite numerous efforts to control their spread, these diseases continue to pose significant threats due to their high transmissibility and periodic outbreak patterns as discussed in [1]. Understanding the complex dynamics of their transmission is crucial for developing effective intervention strategies by [2]. An investigation into the dynamics of cholera-measles epidemics through the application of a fractional-order mathematical model [3]. Unlike classical integer-order models, fractional-order models incorporate memory and hereditary characteristics, which are essential in capturing the long-term effects

of infection and control measures [4]. By utilizing fractional calculus, the model offers a more accurate and realistic research for analysing epidemic behaviour over time.

The model is structured into multiple compartments that represent the different stages of disease progression for both cholera-measles [4]. It integrates preventive strategies in the like of treatment, vaccination, water sanitation and public health awareness, thereby reflecting real world intervention scenarios [5, 6]. Numerical simulations are employed to examine how variations in these control measures influence disease spread and to assess the model's predictive capabilities. Furthermore, sensitivity analysis is conducted to identify prominent parameters of the model that significantly affect disease transmission and its control outcomes [7]. These analysis helps to prioritize resources and optimize intervention efforts [8, 9]. The results demonstrate that fractional-order models not only improve the precision of epidemic forecasting but also highlight the effectiveness of timely, combined preventive actions.

However, the results from this research provide valuable insights into the application of fractional modeling in epidemiology. It offers a scientific basis for strategic planning and resource allocation aimed at mitigating the impact of cholera-measles outbreak [9–13]. By supporting strategic decision-making in disease control, this research contributes to the development of more robust and responsive disease control programs.

2. Materials and Method

2.1. Model Formulation

The model formulation is divided into population of susceptible $S(t)$, vaccinated $V(t)$, exposed $E(t)$, infected $I(t)$ and recovered $R(t)$. The population of exposed and infected are sub-divided into two (E_1, E_2) for cholera and (I_1, I_2) for cholera and measles respectively. The level of individual migrating into the population at Π and transmission rate of cholera-measles disease between the two or more population is at the rate of α. Cholera transmission rate is at β while the environmental bacterial capacity of the disease multiplicative effect is at a rate c and enlightenment through educational program initiatives on the rapid spread and how deadly cholera-measles is at a rate of β_1 and β_2. Prevention on the spread with a waning rate η and regular treatment of cholera-measles disease with environmental exposure is at rate of τ. An infected individual are subjected to recover at a rate of r and ρ. More so, set of bacteria exposed individual from the susceptible population through water treatment, hygienic practices and vaccination from birth goes to the recovered population through ω and also with an induced rate δ_1 and δ_2 in the disease progression when immunity level is high. Respective individuals across the sub-population are subjected to mortality rate by μ.

Here, the dynamics of the model is modified to be of fractional order derivatives which display a realistic behaviour of the effect of each parameter in the model. Thus, this is shown in System 2.1 and Figure 2.1 below,

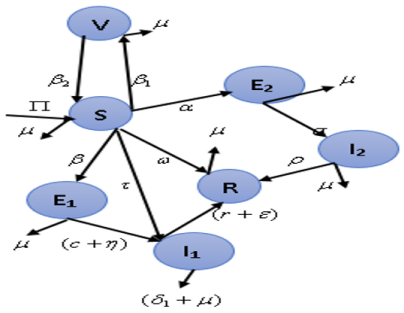

Figure 2.1: Schematic diagram of the model

$$
\begin{aligned}
D^{\eta_1} S(t) &= \Pi - \beta_1 S - \beta S I_1 - \alpha S I_2 - (\tau + \omega) S + \beta_2 V - \mu S \\
D^{\eta_2} V(t) &= \beta_1 S - (\beta_2 + \mu) V \\
D^{\eta_3} E_1(t) &= \beta S I_1 - (c + \eta + \mu) E_1 \\
D^{\eta_4} E_2(t) &= \alpha S I_2 - (\sigma + \mu) E_2 \\
D^{\eta_5} I_1(t) &= \tau S + (c + \eta) E_1 - (\varepsilon + \delta_1 + r + \mu) I_1 \\
D^{\eta_6} I_2(t) &= \sigma E_2 - (\delta_2 + \rho + \mu) I_2 \\
D^{\eta_7} R(t) &= \omega S + (r + \varepsilon) I_1 + \rho I_2 - \mu R
\end{aligned} \tag{2.1}
$$

Subjected to the initial condition

$$
S(0) = s_0, V(0) = v_0, E_1(0) = e_0, E_2(0) = e_0, I_1(0) = i_0, I_2(0) = i_0, R(0) = r_0 \geq 0 \tag{2.2}
$$

Table 2.1: Parameters description, values and references

Variable	**Description**		
$S(t)$	Susceptible population		
$V(t)$	Vaccinated population against measles		
$E_1(t)$	Exposed population to cholera		
$E_2(t)$	Exposed population to measles		
$I_1(t)$	Infected population with cholera		
$I_2(t)$	Infected population with measles		
$R(t)$	Recovered from either or both disease		
Parameter	**Description**	**Values**	**References**
N	Total population	120,000	[10]
Π	Recruitment rate	1.023	[3]
β	Cholera transmission rate	0.13786	[1]
α	Measles contact rate	0.005	[11]
β_1	Measles vaccination rate	1.087	[1, 4]
β_2	Vaccine waning rate	1.00	[2, 9]
τ	Cholera infection from environment	1.42	[6]
ω	Health education rate	0.8137	[7, 13]
c	Environmental bacterial contribution	0.126	[5]
η	Cholera immunity waning rate	0.382	[12]
σ	Progression rate from exposed measles to infected	0.181	[10]
ε	Cholera treatment rate	0.2582	[13]
r	Cholera recovery rate	0.1622	[3]
ρ	Measles recovery rate	0.118	[1]
δ_1	Cholera induce-death rate	0.0163	[2, 7]
δ_2	Measles induce-death rate	0.9828	[11]
μ	Natural death rate	1.00	[2]

2.2. Definition of Preliminaries

Some fundamental definitions and properties in fractional calculus are given in this section.

Definition 2.1. According to [2], a real function $\varphi(t), t > 0$, is said to be in the space $C_\mu, \mu \in R$ if there exist a real number $m > \mu$ such that $\varphi(t) = t^m \varphi_1(t)$ where $\varphi_1(t) \in C(0, \infty)$ and it is said to be in the space C_μ^n, if and only if $\varphi^{(n)} \in C_\mu^n, n \in N$.

Definition 2.2. The Riemann-Liouville fractional integration of order $\eta \geq 0$ of a positive real function $\varphi(t) \in C_\mu, \mu \geq -1, t > 0$ is defined as equation 2.3 as defined in [12].

$$I^\eta \varphi(t) = \frac{1}{\Gamma(\eta)} \int_0^t (t-x)^{\eta-1} \varphi(x) dx \text{ such that } I^0 \varphi(t) = \varphi(t) \tag{2.3}$$

The following properties hold for fractional integral operator I^η for $\varphi(t) \in C_\mu, \mu \geq -1, \eta, \alpha \geq 0$ and $\beta \geq -1$:

1. $I^\eta I^\alpha \varphi(t) = I^{\eta+\alpha} \varphi(t)$
2. $I^\eta I^\alpha \varphi(t) = I^\alpha I^\beta \varphi(t)$
3. $I^\eta I^\alpha \varphi(t) = \frac{\Gamma(\beta+1)}{\Gamma(\eta+\beta+1)} t^{\eta+\beta}$

Definition 2.3. The Caputo Fractional derivatives of a positive real function $\varphi(t)$ given as $D^\eta \varphi(t)$ is given in equation 2.4 as defined in [4]

$$D^\eta \varphi(t) = \frac{1}{\Gamma(n-\eta)} \int_0^t (t-x)^{\eta-1} \varphi^{(n)}(x) dx \; for \; n-1 < \eta \leq n, n \in N, t > 0, \varphi \in \frac{n}{c-1} \tag{2.4}$$

The following property holds for fractional integration of the Caputo Fractional derivatives, for $n-1 < \eta \leq n, n \in N, t > 0, \varphi \in \frac{n}{c-1}$, then

$$I^\eta D^\eta \varphi(t) = \varphi(t) - \sum_{k=0}^{n-1} \varphi^{(k)}(0) \frac{t^k}{k!} \tag{2.5}$$

2.3. Existence and Uniqueness of Model Solution

The System 2.1, which describes an epidemic disease within a human population, should have parameters that are nonnegative. To ensure that the system of differential equations in 2.1 is both mathematically and epidemiologically well-posed, it is essential to demonstrate that the state variables in the model are nonnegative. System 2.1 is well-posed when the system starts with non-negative initial conditions $S(0) = s_0, V(0) = v_0, E_1(0) = e_0, E_2(0) = e_0, I_1(0) = i_0, I_2(0) = i_0, R(0) = r_0$; In that 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 thus apply the following theorems.

Theorem 2.1. *Let (x, y) be distinct points of normed linear space $(X, \|\cdots\|)$ over $\Re$. Then the map of $p : [0,1] \subseteq \Re \to (X, \|\cdots\|)$, 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 $\lambda_0 \in [0,1]$,

$$\|p(\lambda) - p(\lambda_0)\| = \|(\lambda - \lambda_0)x + (\lambda - \lambda_0)y\| \leq |\lambda - \lambda_0|(\|x\| + \|y\|) \tag{2.6}$$

If $\varepsilon > 0$ is given, let $\delta = \frac{\varepsilon}{\|x\|+\|y\|}$. If $|\lambda - \lambda_0| < \delta$, then the $\|p(\lambda) - p(\lambda_0)\| < \varepsilon$. Therefore, p is continuous at λ_0. Since λ_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 $0 \leq \lambda \leq 1$

Hence, the solutions of system 2.1 are bounded if we consider the total population

$$N(t) = S(t) + V(t) + E_1(t) + E_2(t) + I_1(t) + I_2(t) + R(t) \tag{2.7}$$

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

$$\frac{dN(t)}{dt} = \frac{d}{dt}(S(t) + V(t) + E_1(t) + E_2(t) + I_1(t) + I_2(t) + R(t))$$

Such that $\frac{dN(t)}{dt} = \Pi - \mu(S(t) + V(t) + E_1(t) + E_2(t) + I_1(t) + I_2(t) + R(t)) - \alpha I$ When no outbreak of cholera, $\delta = 0$. Thus, substituting 2.4 into 2.5 as time progressively increases yields:

$$\lim_{t\to\infty} N(t) \leq \lim_{t\to\infty} \left[\frac{\Pi}{\mu} + \left(N(0) - \frac{\Pi}{\mu}\right) e^{-\mu t}\right] = \frac{\Pi}{\mu} \tag{2.8}$$

If so $N(0) \leq \frac{\Pi}{\mu}$, as $N(t) \leq \frac{\Pi}{\mu}$. This is a positive invariant set under the flow described by 2.3 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 has been mathematically and epidemiologically well-posed. □

2.4. 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*

$$L(x, y) = \left(\sum_{i=1}^{n} |x_i|^{\Omega}\right)^{\frac{1}{\Omega}} \qquad \Omega \geq 1 \tag{2.9}$$

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

Proof. It can be checked that for each n:

$$0 \leq x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2 \leq (|x_1| + |x_2| + |x_3| + \ldots + |x_n|)^2$$

This will result to;

$$x_1^2 + x_2^2 \leq (|x_1| + |x_2|)^2$$

Therefore,

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

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

$$0 \leq (x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2)^{\frac{1}{2}} \leq |x_1| + |x_2| + |x_3| + \ldots + |x_n| = \sum_{n=1}^{\infty} |x_n| < \infty \tag{2.10}$$

Therefore,

$$0 \leq x_n = x_1^2 + x_2^2 + x_3^2 + \ldots + x_n^2 \leq \left[\sum_{n=1}^{\infty} |x_n|\right] < \infty$$

These sequences x_n is monotone increasing and bounded above, it therefore converges. Thus $\sum_{n=1}^{\infty} x_n$ converges absolutely, if $x_n \in \zeta^1$, then $x_n \in \zeta^2$ where $\zeta^1 \leq \zeta^2$. In case of ζ^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 $\sum_{n=1}^{\infty} |x_n| < \infty$ whereas ζ^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 $x_n \in \zeta^1 \leftrightarrow x_n \in \zeta^2$ i.e. $\zeta^1 \subseteq \zeta^2$. Further, if $x_n = \frac{1}{n^{\frac{3}{4}}}$, then $\sum_{n=1}^{\infty} |x_n|$ diverges and thus $x_n \notin \zeta^1$. But $\sum_{n=1}^{\infty} x_n^2 = \sum_{n=1}^{\infty} \frac{1}{n^{\frac{3}{4}}}$ converges, implying that $x_n \in \zeta^2$. We conclude that $\zeta^1 \subseteq \zeta^2$ and thus $\zeta^1 \neq \zeta^2$. If (x_n, y_n) are sequences of real numbers, then;

$$\sum_{n=1}^{\infty}(x_i - y_i)^2 \leq \sum_{n=1}^{\infty} x_i^2 + \sum_{n=1}^{\infty} y_i^2 + 2\left[\sum_{n=1}^{\infty} x_i^2\right]^{\frac{1}{2}}\left[\sum_{n=1}^{\infty} y_i^2\right]^{\frac{1}{2}} \tag{2.11}$$

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 $(x_i - y_i)^2 \in \zeta^2$ if $(x_n, y_n) \in \zeta^2$.

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:

$$\Psi = \left\{(S(t), V(t), E(t), I(t), T(t), R(t)) \in \Re_+^6 : N(t) \leq \frac{\pi}{\mu}\right\} \tag{2.12}$$

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

$$\left|\frac{df_1}{dS}\right| = |(\beta + \beta_1 + \alpha + \mu + \tau + \omega + \delta)| < \infty, \left|\frac{df_1}{dV}\right| = |\beta_2| < \infty, \left|\frac{df_1}{dE_1}\right| = |0| < \infty, \left|\frac{df_1}{dE_2}\right| = |0| < \infty,$$

$$\left|\frac{df_1}{dI_1}\right| = |\beta| < \infty, \left|\frac{df_1}{dI_2}\right| = |\alpha| < \infty, \left|\frac{df_1}{dR}\right| = |0| < \infty$$

$$\left|\frac{df_2}{dS}\right| = |\beta_1| < \infty, \left|\frac{df_2}{dV}\right| = |\beta_2 + \mu| < \infty, \left|\frac{df_2}{dE_1}\right| = |0| < \infty, \left|\frac{df_2}{dE_2}\right| = |0| < \infty, \left|\frac{df_2}{dI_1}\right| = |0| < \infty,$$

$$\left|\frac{df_2}{dI_2}\right| = |0| < \infty, \left|\frac{df_2}{dR}\right| = |0| < \infty$$

$$\left|\frac{df_3}{dS}\right| = |\beta| < \infty, \left|\frac{df_3}{dV}\right| = |0| < \infty, \left|\frac{df_3}{dE_1}\right| = |c + \eta + \mu| < \infty, \left|\frac{df_3}{dE_2}\right| = |0| < \infty, \left|\frac{df_3}{dI_1}\right| = |\beta| < \infty,$$

$$\left|\frac{df_3}{dI_2}\right| = |0| < \infty, \left|\frac{df_3}{dR}\right| = |0| < \infty \tag{2.13}$$

$$\left|\frac{df_4}{dS}\right| = |\alpha| < \infty, \left|\frac{df_4}{dV}\right| = |0| < \infty, \left|\frac{df_4}{dE_1}\right| = |0| < \infty, \left|\frac{df_4}{dE_2}\right| = |\sigma + \mu| < \infty, \left|\frac{df_4}{dI_1}\right| = |0| < \infty,$$

$$\left|\frac{df_4}{dI_2}\right| = |\alpha| < \infty, \left|\frac{df_4}{dR}\right| = |0| < \infty$$

$$\left|\frac{df_5}{dS}\right| = |\tau| < \infty, \left|\frac{df_5}{dV}\right| = |0| < \infty, \left|\frac{df_5}{dE_1}\right| = |c + \eta| < \infty, \left|\frac{df_5}{dE_2}\right| = |0| < \infty,$$

$$\left|\frac{df_5}{dI_1}\right| = |\varepsilon + \delta_1 + r + \mu| < \infty, \left|\frac{df_5}{dI_2}\right| = |0| < \infty, \left|\frac{df_5}{dR}\right| = |0| < \infty$$

$$\left|\frac{df_6}{dS}\right| = |0| < \infty, \left|\frac{df_6}{dV}\right| = |0| < \infty, \left|\frac{df_6}{dE_1}\right| = |0| < \infty, \left|\frac{df_6}{dE_2}\right| = |\sigma| < \infty, \left|\frac{df_6}{dI_1}\right| = |0| < \infty,$$

$$\left|\frac{df_6}{dI_2}\right| = |\delta_2 + \rho + \mu| < \infty, \left|\frac{df_6}{dR}\right| = |0| < \infty$$

$$\left|\frac{df_4}{dS}\right| = |\omega| < \infty, \left|\frac{df_4}{dV}\right| = |0| < \infty, \left|\frac{df_4}{dE_1}\right| = |0| < \infty, \left|\frac{df_4}{dE_2}\right| = |\sigma + \mu| < \infty, \left|\frac{df_4}{dI_1}\right| = |r + \varepsilon| < \infty,$$

$$\left|\frac{df_4}{dI_2}\right| = |\rho| < \infty, \left|\frac{df_4}{dR}\right| = |\mu| < \infty$$

Equation 2.13 confirms that model equation 2.1 is bounded, invariantly and attractively influential on the bounded region of $\Re_+^7$ representing a physical problem. □

2.5. Disease Free Equilibrium

The cholera-measles-non-infected equilibrium state represents a situation where by the disease model is entirely free from vibro-cholerae and measles spread. Consequently, it follows that the number of exposed $(E_1 \& E_2)$ and infected $(I_1 \& I_2)$ population in equation 2.1, i.e. $E_1 = E_2 = I_1 = I_2 = 0$. The solution for the cholera-measles-free equilibrium point can be derived as follows: Let

$$N = S^* + V^* + E_1^* + E_2^* + I_1^* + I_2^* + R = 0 \tag{2.14}$$

When no outbreak of cholera-measles infection was observed, infectious population at $t = 0$, from 2.1, equation 2.15 below is the result obtained when no cholera-measles outbreak is observed.

$$\left(S_0 = \frac{\Pi + \mu(\delta_2 + \mu + \rho) + \sigma(\delta_2 + \rho + \mu)}{\alpha\sigma}, V_0 = \frac{\beta_1[\mu(\delta_2 + \mu + \rho) + \sigma(\delta_2 + \rho + \mu)]}{\alpha\sigma(\beta_2 + \mu)}, 0, 0, 0, 0, R_0 = \frac{\omega S_0}{\mu}\right) \tag{2.15}$$

2.6. Endemic Equilibrium Point

We examine cholera endemicity in a population of exposed and infected, focusing on strategic interventions in the like of awareness rate and vaccination, with the aim of long-term elimination mechanism. Measles being a water borne receptor of pathogens can aid persistency with water logged regions, to curb the emanation of this outbreak in endemic regions over time, the frequency of cholera-measles on $(S, V, E_1, E_2, I_1, I_2, R)$ at $t \neq 0$, stressing the dynamic aspect of it to gauge the crucial role in its infectious diseases and protection against the total population. Let $e = (S^*, V^*, E_1^*, E_2^*, I_1^*, I_2^*, R^*)$ at steady state when $E_1, E_2, I_1, I_2 \neq 0$. Examine the system In equation (2.1). The endemic equilibrium point for respective populations are:

$$
\begin{aligned}
S^* &= \frac{\Pi + 2\beta_1(\delta_1+\mu) + \alpha(c+\eta)[(\tau+\omega+r+\mu)]}{\sqrt{\sigma(c+\eta+\mu) + \delta_2(\gamma+(\sigma+\rho+\varepsilon))^2 - (\mu+\tau+\beta_2)}} \\
V^* &= \frac{(r+\varepsilon)\pi\sqrt{(\alpha+\mu+\beta_2)}}{(\varepsilon+c+\eta)\alpha + (\delta+\mu+\beta_1)[(\mu+\alpha+\delta)]} \\
E_1^* &= \frac{[(\mu+\alpha+\beta)^2 + (\mu+c+\beta_2)\sqrt{(\sigma+\delta_2+\mu)(\delta_2+\rho+\omega)}]}{[\mu^2(\delta_1+\mu+\beta) + (\varepsilon+r+\eta)]}, \\
E_2^* &= \frac{(\varepsilon+c+\eta)^2\beta + (\mu+\omega+(\beta_1+\beta_2))}{(\beta+\mu+\alpha) - (\rho+\mu)^3[(\mu+\alpha+\sigma) - (c+r+\eta)]}, \\
I_1^* &= \frac{\sqrt{(\varepsilon+\delta_1+r+\mu)(\delta_2+\mu+\delta_1)}}{(1+\alpha)^{-1}(\sigma+\mu+\omega)} \\
I_2^* &= \frac{(\mu+\rho+\varepsilon)}{(\alpha+\mu+\beta)[(\mu+\delta_2+\varepsilon+\delta_1)]} + \frac{\sqrt{(c+\mu)(\delta_2+\mu+\rho)}}{(1+\alpha)^{-1}(\rho+\mu+\omega)} \\
R^* &= \frac{(\alpha+\rho+\tau)}{[\mu^2(\beta+\mu) - (\varepsilon+c+\eta)]} + \sqrt{\frac{(\mu+\varepsilon+\alpha+\pi)] + \beta_2\delta_1^2}{(\beta+\alpha)(\gamma+\mu+\omega)(\varepsilon+\mu+\delta_1)}}
\end{aligned}
\tag{2.16}
$$

2.7. Basic Reproduction Number

The basic reproduction number, denoted as R_*. It is necessary to quantify the probability of new cholera-measles infections resulting from a single carrier or sick person in a population without prior symptoms. Using the next-generation matrix approach to create the system described in equation 2.1, focusing on the infectious classes of (E_1, E_2, I_1, I_2). 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 $G = FV^{-1}$ 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:

$$
F_i = \left(\frac{\partial f_i(x_i)}{\partial x_j}\right) \quad V_i = \left(\frac{\partial v_i(x_i)}{\partial x_j}\right) \tag{2.17}
$$

$$
f = \begin{pmatrix} \beta_1+\beta+\alpha+(\tau+\omega)+\beta_2+\mu)S_0 + \beta_2 V_0 \\ 0 \\ 0 \end{pmatrix}, v = \begin{pmatrix} (c+\eta+\mu)E_1 \\ -\alpha S I_2 + (\sigma+\mu)E_2 \\ -rS - (c+\eta)E_1 + (\varepsilon+\delta_1+r+\mu)I_1 \\ -\sigma E_2 + (\delta_2+\rho+\mu)I_2 \end{pmatrix}
$$

Then, this is obtained as

$$
\mathrm{F} = \begin{pmatrix} \dfrac{\Pi+\mu(\delta_2+\mu+\rho)+\sigma(\delta_2+\rho+\mu)+\beta_1[\mu(\delta_2+\mu+\rho)+\sigma(\delta_2+\rho+\mu)]}{\alpha\sigma(\beta_2+\mu)} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}
$$

$$
V = \begin{pmatrix} (c+\eta+\mu) & 0 & 0 & 0 \\ 0 & (\sigma+\mu) & 0 & -\alpha \\ -(c+\eta) & 0 & (\varepsilon+\delta_1+r+\mu) & 0 \\ 0 & -\sigma & 0 & (\delta_2+\rho+\mu) \end{pmatrix} \tag{2.18}
$$

$$FV^{-1} = \frac{1}{abcd}\begin{pmatrix} \frac{\Pi+\mu(\delta_2+\mu+\rho)+\sigma(\delta_2+\rho+\mu)+\beta_1[\mu(\delta_2+\mu+\rho)+\sigma(\delta_2+\rho+\mu)]}{\alpha\sigma(\beta_2+\mu)} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

Considering the respective values of $a = (c+\eta+\mu)$, $b = (\sigma+\mu)$, $c = (\varepsilon+\delta_1+r+\mu)$, $d = (\delta_2+\rho+\mu)$

$$R_\bullet = \frac{\Pi+\mu(\delta_2+\mu+\rho)+\sigma(\delta_2+\rho+\mu)+\beta_1[\mu(\delta_2+\mu+\rho)+\sigma(\delta_2+\rho+\mu)]}{\alpha\sigma(\beta_2+\mu)(c+\eta+\mu)(\sigma+\mu)(\varepsilon+\delta_1+r+\mu)(\delta_2+\rho+\mu)} \tag{2.19}$$

2.8. Quantitative Analysis of R_ϵ

Here, a quantitative analysis of R_ϵ was conducted to assess its metric progression concerning each intervention method. By excluding the values of intervention parameters, we assess equation 2.19 using the baseline values provided in Table 2.1, yielding equation 2.20, subsequently resulting in equation 2.21 through 2.26. The outcomes of these calculations are presented in Table 2.2.

$$R_\bullet = \frac{\Pi[(1-0.04223)\beta_1+1.9837+2.823\beta_1]-(0.1\delta_1+1.27c)}{[(2.877\eta-1.97362\omega+0.38736\alpha)(1-0.7653\delta_2)\beta_2](0.9972+0.5243436\rho)} \tag{2.20}$$

$$R_\varepsilon = f(\beta_1)\Big|_{\substack{\delta=0\\ \tau=0\\ \phi_2=0}} = 0.7173\beta_1+2.9366\beta_2-0.0832\alpha-1.226\beta \tag{2.21}$$

$$R_\varepsilon = f(\eta)\Big|_{\substack{\alpha=0\\ \rho_1=0}} = 1.340\eta+0.5593c+0.2830\sigma \tag{2.22}$$

$$R_\varepsilon = f(\omega)\Big|_{\substack{c=0\\ \phi_2=0\\ \tau=0}} = \frac{1.529\beta_2(0.7452\alpha+0.54\delta_1)}{1.826334\delta_1+1.9423\rho} \tag{2.23}$$

$$R_\varepsilon = f(\delta_1)\Big|_{\substack{\rho_1=0\\ \tau=0\\ \rho_2=0}} = 1.32353\mu-1.0731\omega+2.97833\varepsilon \tag{2.24}$$

$$R_\varepsilon = f(\beta_2)\Big|_{\substack{c=0\\ \phi_2=0\\ \tau=0}} = \frac{1.529\beta_2(0.7452\alpha+0.54\delta_1)}{1.826334\delta_1+1.9423\rho} \tag{2.25}$$

$$R_\varepsilon = f(\sigma)\Big|_{\substack{\alpha=0\\ \rho_1=0}} = 0.883\alpha+1.6289\rho-0.04811\beta \tag{2.26}$$

Table 2.2: Standalone metric of vaccination and general treatment on R_ε

A						B					C				
s/n	β_1	β	β_2	η	R_ε	s/n	δ_1	σ	β_2	ρ	s/n	β_2	β	β	ε
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

Analysis of the above table reveals that utilizing vaccination and education independently at 65% efficacy effectively reduces disease transmission. However, even at 98% efficacy, treatment fails to significantly impact the reproduction number due to untreated individuals in the community. Hence, with 100% public awareness through educational programs in achieving R_ε of zero, is a level attainable through vaccination and antibiotic scheme.

2.9. Local Stability of Disease Free State

We examined the local stability of the disease-free state for cholera-measles by analysing the minimal recurrence rate impact. When the recurrence rate $R_* < 1$, 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 disease free-state in order to determine the disease-free equilibrium at zero time.

$$J_{(E_1)} = \begin{pmatrix} -(\beta+\beta_1+\alpha+\tau+\omega+\mu) & \beta_2 & 0 & 0 & 0 & 0 & 0 \\ \beta_1 & -(\beta_2+\mu) & 0 & 0 & 0 & 0 & 0 \\ \beta & 0 & -(c+\eta+\mu) & 0 & 0 & 0 & 0 \\ \alpha & 0 & 0 & -(\sigma+\mu) & 0 & 0 & 0 \\ \tau & 0 & (c+\eta) & 0 & -(\varepsilon+\delta_1+r+\mu) & 0 & 0 \\ 0 & 0 & 0 & \sigma & 0 & -(\delta_2+\rho+\mu) & 0 \\ \omega & 0 & 0 & 0 & (r+\varepsilon) & \rho & -\mu \end{pmatrix}$$

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

$$\begin{vmatrix} -(\beta+\beta_1+\alpha+\tau+\omega+\mu)-\lambda & \beta_2 & 0 & 0 & 0 & 0 & 0 \\ \beta_1 & -(\beta_2+\mu)-\lambda & 0 & 0 & 0 & 0 & 0 \\ \beta & 0 & -(c+\eta+\mu)-\lambda & 0 & 0 & 0 & 0 \\ \alpha & 0 & 0 & -(\sigma+\mu)-\lambda & 0 & 0 & 0 \\ \tau & 0 & (c+\eta) & 0 & -(\varepsilon+\delta_1+r+\mu)-\lambda & 0 & 0 \\ 0 & 0 & 0 & \sigma & 0 & -(\delta_2+\rho+\mu)-\lambda & 0 \\ \omega & 0 & 0 & 0 & (r+\varepsilon) & \rho & -\mu-\lambda \end{vmatrix} = 0$$

as obtained from the Jacobian matrix result for respective eigen values:

$$\lambda = -(c+\eta+\mu),\ \lambda = -(\sigma+\mu),\ \lambda = -\mu,\ \begin{vmatrix} -(\varepsilon+\mu+\delta_1+r)-\lambda & 0 \\ \phi_2 & -(\delta_2+\rho+\mu)-\lambda \end{vmatrix},\ \lambda_3 = -(\beta_2+\mu+\alpha+\omega),\ \lambda = -(\delta_2+\rho+\mu)$$

Respective eigenvalues are negatively invariant in the region $\Re_+^7$, 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. Hence the system of 2.1 is asymptotically stable $\forall\, \lambda_n < 0, n = 1, 2 \ldots 7,\ t > 0$. □

2.10. Local Stability of Endemic Equilibrium

Theorem 2.4. *The suggested model of cholera-measles disease has a locally asymptotically stable regional resilience if the recurrence rate $R_* < 1$ and unstable whenever $R_* > 1$.*

Proof. Suppose,

$$S = x + S^*, V = y + V^*, E_1 = z + E^*, E_2 = a + E^*, I_1 = b + I^*, I_2 = c + I^*, R = d + R^* \tag{2.27}$$

Linearizing equation 2.1, is then obtained as

$$\begin{aligned} D^{n_1}S(t) &= \Pi - \beta_1(x+S^*) - \beta(x+S^*)(b+I_1^*) - \alpha(x+S^*)(c+I_2^*) - (\tau+\omega)(x+S^*) + \\ &\quad \beta_2(y+V^*) - \mu(x+S^*) \\ D^{n_2}V(t) &= \beta_1(x+S^*) - (\beta_2+\mu)(y+S^*) \\ D^{n_3}E_1(t) &= \beta(x+S^*)(b+I_1^*) - (c+\eta+\mu)(z+E_1^*) \\ D^{n_4}E_2(t) &= \alpha(x+S^*)(c+I_2^*) - (\sigma+\mu)(a+E_2^*) \\ D^{n_5}I_1(t) &= \tau(x+S^*) + (c+\eta)(z+E_1^*) - (\varepsilon+\delta_1+r+\mu)(b+I_1^*) \\ D^{n_6}I_2(t) &= \sigma(a+E_2^*) - (\delta_2+\rho+\mu)(c+I_2^*) \\ D^{n_7}R(t) &= \omega(z+S^*) + (r+\varepsilon)(b+I_1^*)\, I_1^* + \rho(c+I_2^*) - \mu(d+R^*) \end{aligned} \tag{2.28}$$

Linearizing equation 2.28, is then obtained as

$$
\begin{aligned}
D^{n_1}S(t) &= -\beta_1 x - \beta b x - \alpha c x - (\tau+\omega)x + \beta_2 y - \mu x + \text{higher order} + \text{non-linear terms}\ldots\\
D^{n_2}V(t) &= \beta_1 x - (\beta_2+\mu)y + \text{higher order} + \text{non-linear terms}\ldots\\
D^{n_3}E_1(t) &= \beta b x - (c+\eta+\mu)z + \text{higher order} + \text{non-linear terms}\ldots\\
D^{n_4}E_2(t) &= \alpha c x - (\sigma+\mu)a + \text{higher order} + \text{non-linear terms}\ldots\\
D^{n_5}I_1(t) &= \tau x + (c+\eta)z - (\varepsilon+\delta_1+r+\mu)b + \text{higher order} + \text{non-linear terms}\ldots\\
D^{n_6}I_2(t) &= \sigma b - (\delta_2+\rho+\mu)d + \text{higher order} + \text{non-linear terms}\ldots\\
D^{n_7}R(t) &= \omega x + (r+\varepsilon)b + \rho c - \mu d + \text{higher order} + \text{non-linear terms}\ldots
\end{aligned}
$$

The characteristic equation obtained from its Jacobian matrix is;

$$
J_{(EE)} = \begin{pmatrix}
-(\beta+\beta_1+\alpha+\tau+\omega+\mu) & \beta_2 & 0 & 0 & 0 & 0 & 0\\
\beta_1 & -(\beta_2+\mu) & 0 & 0 & 0 & 0 & 0\\
\beta & 0 & -(c+\eta+\mu) & 0 & 0 & 0 & 0\\
\alpha & 0 & 0 & -(\sigma+\mu) & 0 & 0 & 0\\
\tau & 0 & (c+\eta) & 0 & -(\varepsilon+\delta_1+r+\mu) & 0 & 0\\
0 & 0 & 0 & \sigma & 0 & -(\delta_2+\rho+\mu) & 0\\
\omega & 0 & 0 & 0 & (r+\varepsilon) & \rho & -\mu
\end{pmatrix}
$$

Denoting that $A = -(\beta+\beta_1+\alpha+\tau+\omega+\mu)$, $B = -(\beta_2+\mu)$, $C = -(c+\eta+\mu)$, $D = -(\sigma+\mu)$, $E = -(\varepsilon+\delta_1+r+\mu)$, $F = -(\delta_2+\rho+\mu)$, $G = -\mu$ the resulting eigenvalue of the above matrix is obtained as;

$$
\begin{vmatrix}
A-\lambda & \beta_2 & 0 & 0 & 0 & 0 & 0\\
\beta_1 & B-\lambda & 0 & 0 & 0 & 0 & 0\\
\beta & 0 & C-\lambda & 0 & 0 & 0 & 0\\
\alpha & 0 & 0 & D-\lambda & 0 & 0 & 0\\
\tau & 0 & (c+\eta) & 0 & E-\lambda & 0 & 0\\
0 & 0 & 0 & \sigma & 0 & F-\lambda & 0\\
\omega & 0 & 0 & 0 & (r+\varepsilon) & p & G-\lambda
\end{vmatrix} = 0
$$

$$
\begin{aligned}
&\lambda^7 - (x + b(a+y) + c(d+e) + (c+b))\lambda^6 + ((x+c)(y+s) + az + by)(1+b)\lambda^5\\
&- (ab(c+z) + bc(a+y))(1+z)\lambda^4 + y(cz(a+b) + xb(a+y))\lambda^3\\
&- [(a+c)(x+y) + a(e+f) + bcdf]\lambda^2 + (-(a+c)(x+y)\lambda + xyzabcd = 0
\end{aligned}
$$

with the invariance of the eigen-values it is said to be locally asymptotically stable.

3. Global Stability of Disease Free equilibrium

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

$$
\Gamma(t, S, V, E_1, E_2, I_1, I_2, R) = C_1 I_1 + C_2 I_2 + C_3 I_3 + C_4 I_4 \tag{3.29}
$$

$$
\begin{aligned}
D^{n_3}E_1(t) &= \beta S I_1 - (c+\eta+\mu)E_1\\
D^{n_4}E_2(t) &= \alpha S I_2 - (\sigma+\mu)E_2\\
D^{n_5}I_1(t) &= \tau S + (c+\eta)E_1 - (\varepsilon+\delta_1+r+\mu)I_1\\
D^{n_6}I_2(t) &= \sigma E_2 - (\delta_2+\rho+\mu)I_2
\end{aligned}
$$

$$\begin{aligned}\frac{d\Phi}{dt} &= C_1 I_1^{\bullet} + C_2 I_2^{\bullet} + C_3 I_3^{\bullet} + C_4 I_4^{\bullet} \\ &= C_1(\beta S I_1 - (c+\eta+\mu)E_1) + C_2(\alpha S I_2 - (\sigma+\mu)E_2) + C_3(\tau S + (c+\eta)E_1 - (\varepsilon+\delta_1+r+\mu)I_1) \\ &\quad C_4(\sigma E_2 - (\delta_2+\rho+\mu)I_2) \\ &= C_2\alpha I_1 - C_1(\sigma+\mu)I_1 + C_1(\sigma+\rho+\delta_1)\beta I_2 + C_1\beta I_2 S_0 - C_2(\alpha+\beta_2+\mu)I_2 + C_3\delta_2 I_2 - C_3(\eta+ \\ &\quad \omega+\mu)I_3 \\ &\leq (C_2\Pi - C_1(\sigma+\beta+\mu))I_1 + (C_1(\varepsilon+\delta_1+r)\beta V_0 + C_1\beta S_0 - C_2(c+\alpha+\eta+\beta_2+\mu) + C_3\delta_2)I_2 - \\ &\quad C_3(\delta_2+\mu)I_3\end{aligned}$$

$S_0 = V_0 \neq 0$, $E_1 = E_2 = I_1 = I_2 = 0$ at equilibrium state of the disease spread,

$$S_0 = V_0 \neq 0, \quad C_1 = \frac{1}{(k+\mu)}, \quad C_2 = \left\{ \frac{(\beta_i+\mu+\delta_1)(\varepsilon+\eta+\sigma) + (\alpha+\beta+\omega)}{\rho(\delta_2+\mu+\delta_1+c)(\alpha+r++\mu)} \right\}$$

$$R_{\bullet} = \frac{\Pi + \mu(\delta_2+\mu+\rho) + \sigma(\delta_2+\rho+\mu) + \beta_1[\mu(\delta_2+\mu+\rho) + \sigma(\delta_2+\rho+\mu)]}{\alpha\sigma(\beta_2+\mu)(c+\eta+\mu)(\sigma+\mu)(\varepsilon+\delta_1+r+\mu)(\delta_2+\rho+\mu)}$$

$$\frac{d\Gamma}{dt} \leq C_1 \left(\frac{\Pi + \mu(\delta_2+\mu+\rho) + \sigma(\delta_2+\rho+\mu) + \beta_1[\mu(\delta_2+\mu+\rho) + \sigma(\delta_2+\rho+\mu)]}{\alpha\sigma(\beta_2+\mu)(c+\eta+\mu)(\sigma+\mu)(\varepsilon+\delta_1+r+\mu)(\delta_2+\rho+\mu)} - 1 \right) I_1$$

$$\frac{d\Gamma}{dt} \leq (R_0 - 1) \tag{3.30}$$

It is pertinent that when at $t \to \infty$ and $C_1, C_2 < 1$. Substituting into the model system of equation 2.19 reveals that, based on LaSalle's invariance principle $\frac{d\Gamma}{dt} = 0$, is globally asymptotically stable whenever $R_* > 1$. □

3.1. Global Stability of Endemic Equilibrium

Theorem 3.1. *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:

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

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

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

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^*). 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 (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 $\exists B(x,y) \in C^1$ 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 of Dulac's criterion. Let $X = (S, V, E_1, ., E_2, I_1, I_2, R)$ define the Dulac's function

$G = \dfrac{1}{SI}$. The following system of equation are obtained;

$$\begin{aligned}
G\big(D^{\eta_1}S^*(t)(SI)^{-1}\big) &= \Pi - \beta_1 S - \beta S I_1 - \alpha S I_2 - (\tau+\omega)S + \beta_2 V - \mu S \\
G\big(D^{\eta_1}V^*(t)(SI)^{-1}\big) &= \beta_1 S - (\beta_2+\mu)V \\
G\big(D^{\eta_1}E_1^*(t)(SI)^{-1}\big) &= \beta S I_1 - (c+\eta+\mu)E_1 \\
G\big(D^{\eta_1}E_2^*(t)(SI)^{-1}\big) &= \alpha S I_2 - (\sigma+\mu)E_2 \\
G\big(D^{\eta_1}I_1^*(t)(SI)^{-1}\big) &= \tau S + (c+\eta)E_1 - (\varepsilon+\delta_1+r+\mu)I_1 \\
G\big(D^{\eta_1}I_2^*(t)(SI)^{-1}\big) &= \sigma E_2 - (\delta_2+\rho+\mu)I_2 \\
G\big(D^{\eta_1}R^*(t)(SI)^{-1}\big) &= \omega S + (r+\varepsilon)I_1 + \rho I_2 - \mu R
\end{aligned} \tag{3.31}$$

$$\begin{aligned}
G\frac{dS}{dt} &= \frac{1}{SI}\{\Pi - \beta_1 S - \beta S I_1 - \alpha S I_2 - (\tau+\omega)S + \beta_2 V - \mu S\} \\
G\frac{dV}{dt} &= \frac{1}{SI}\{\beta_1 S - (\beta_2+\mu)V\} \\
G\frac{dE_1}{dt} &= \frac{1}{SI}\{\beta S I_1 - (c+\eta+\mu)E_1\} \\
G\frac{dE_2}{dt} &= \frac{1}{SI}\{\alpha S I_2 - (\sigma+\mu)E_2\} \\
G\frac{dI_1}{dt} &= \frac{1}{SI}\{\tau S + (c+\eta)E_1 - (\varepsilon+\delta_1+r+\mu)I_1\} \\
G\frac{dI_2}{dt} &= \frac{1}{SI}\{\sigma E_2 - (\delta_2+\rho+\mu)I_2\} \\
G\frac{dI_2}{dt} &= \frac{1}{SI}\{\sigma E_2 - (\delta_2+\rho+\mu)I_2\}
\end{aligned} \tag{3.32}$$

The above system of equations results to; At $t > 0$ orbital resolution of the system of equations is given by $\dfrac{d(GX)}{dt}$ as obtained below.

$$\frac{d(GX)}{dt} = \frac{\partial}{\partial S}\left(G\frac{dS}{dt}\right) + \frac{\partial}{\partial V}\left(G\frac{dV}{dt}\right) + \frac{\partial}{\partial E_1}\left(G\frac{dE_1}{dt}\right) + \frac{\partial}{\partial E_2}\left(G\frac{dE_2}{dt}\right) + \frac{\partial}{\partial I_1}\left(G\frac{dI_1}{dt}\right) + \frac{\partial}{\partial I_2}\left(G\frac{dI_2}{dt}\right) + \frac{\partial}{\partial R}\left(G\frac{dR}{dt}\right)$$

$$\frac{d(GX)}{dt} = \frac{\partial}{\partial S}\left(G\frac{dS}{dt}\right) + \frac{\partial}{\partial V}\left(G\frac{dV}{dt}\right) + \frac{\partial}{\partial E_1}\left(G\frac{dE_1}{dt}\right) + \frac{\partial}{\partial E_2}\left(G\frac{dE_2}{dt}\right) + \frac{\partial}{\partial I_1}\left(G\frac{dI_1}{dt}\right) + \frac{\partial}{\partial I_2}\left(G\frac{dI_2}{dt}\right) + \frac{\partial}{\partial R}\left(G\frac{dR}{dt}\right)$$

$$\begin{aligned}
\frac{d(GX)}{dt} = {} & \frac{\partial}{\partial S}\{\Pi - \beta_1 S - \beta S I_1 - \alpha S I_2 - (\tau+\omega)S + \beta_2 V - \mu S\} + \frac{\partial}{\partial V}\{\beta_1 S - (\beta_2+\mu)V\} \\
& + \frac{\partial}{\partial E_1}\{\beta S I_1 - (c+\eta+\mu)E_1\} + \frac{\partial}{\partial E_2}\{\alpha S I_2 - (\sigma+\mu)E_2\} \\
& + \frac{\partial}{\partial I_1}\{\tau S + (c+\eta)E_1 - (\varepsilon+\delta_1+r+\mu)I_1\} + \frac{\partial}{\partial I_2}\{\sigma E_2 - (\delta_2+\rho+\mu)I_2\} \\
& + \frac{\partial}{\partial R}\{\omega S + (r+\varepsilon)I_1 + \rho I_2 - \mu R\}
\end{aligned}$$

$$\frac{d(GX)}{dt} = -\left\{\frac{\Pi(\tau+c+\eta+\mu)+[(\rho+\rho+\beta)+(\omega+\delta_1+\mu)]}{SI}+\frac{(\delta_2+\rho+\alpha)-(\rho+\beta+\mu)}{I} +\frac{(\rho+\beta+\omega+\mu)}{I}+\frac{(\rho+\sigma+\beta_2)+(\varepsilon+\mu+r)}{SI}+\frac{(\beta+\delta_1+\mu)}{SI}\right\}$$

$$\frac{d(GX)}{dt} = -\left\{\frac{\Pi(\rho+\omega+\beta)-2(\sigma+\tau+\mu)+\sigma^2[c+r(\delta_1-\rho-2\mu)-\delta_2(\rho+\beta_1+\mu)]+(\beta+\delta_2+\mu)}{SI}\right\} < 0$$

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 quite obvious that in allocation of resources for the control of the cholera-measles spread, vaccination will help to eradicate the rapid spread of measles with time. □

3.2. 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. As a result of this investigation, the normalized forward sensitivity index, as obtained

$$\frac{\partial R_*}{\partial \beta_1} = \frac{\partial R_*}{\partial \beta_1}\times\frac{\beta_1}{R_*} = 1.0132, \quad \frac{\partial R_0}{\partial \varepsilon} = \frac{\partial R_0}{\partial \varepsilon}\times\frac{\varepsilon}{R_0} = 0.3251, \quad \frac{\partial R_*}{\partial \Pi} = \frac{\partial R_*}{\partial \Pi}\times\frac{\Pi}{R_*} = 1.1972$$
$$\frac{\partial R_*}{\partial \delta_1} = \frac{\partial R_*}{\partial \delta_1}\times\frac{\delta_1}{R_*} = 0.0930, \quad \frac{\partial R_*}{\partial \mu} = \frac{\partial R_*}{\partial \mu}\times\frac{\mu}{R_*} = -1.0991, \quad \frac{\partial R_*}{\partial \beta_2} = \frac{\partial R_*}{\partial \beta_2}\times\frac{\beta_2}{R_*} = 0.1850$$
$$\frac{\partial R_*}{\partial \delta_2} = \frac{\partial R_*}{\partial \delta_2}\times\frac{\delta_2}{R_*} = 1.1087, \quad \frac{\partial R_*}{\partial \eta} = \frac{\partial R_*}{\partial \eta}\times\frac{\eta}{R_*} = 1.2778, \quad \frac{\partial R_*}{\partial r} = \frac{\partial R_*}{\partial r}\times\frac{r}{R_*} = 1.5643$$
$$\frac{\partial R_*}{\partial \rho} = \frac{\partial R_*}{\partial \rho}\times\frac{\rho}{R_*} = 1.3381, \quad \frac{\partial R_*}{\partial \sigma} = \frac{\partial R_*}{\partial \sigma}\times\frac{\sigma}{R_*} = 1.2778, \quad \frac{\partial R_*}{\partial c} = \frac{\partial R_*}{\partial c}\times\frac{c}{R_*} = -1.9613$$
$$\frac{\partial R_*}{\partial \alpha} = \frac{\partial R_*}{\partial \alpha}\times\frac{\alpha}{R_*} = -1.7635, \quad \frac{\partial R_*}{\partial \omega} = \frac{\partial R_*}{\partial \omega}\times\frac{\omega}{R_*} = -1.7635$$

Table 3.3: Sensitivity analysis and parameter indices

Parameters	**Sensitivity indices**
β_1	$+$
ε	$+$
Π	$+$
δ_1	$+$
μ	$-$
β_2	$+$
δ_2	$+$
η	$+$
r	$+$
ω	$-$
ρ	$+$
σ	$+$
c	$-$
α	$-$

Table 3.3, shows that the sensitivity indices of are positively invariant in $\mathfrak{R}_6^+$ the sensitivity indices 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 3.3 above, we can concluded that parameter r is the most sensitive to the basic reproduction number of the cholera disease. Particularly, increasing the value of ε will result in a 78.05% increase in R_*, while increasing the value of α will lead to a 42.73% decrease in R_*.

3.3. Numerical Simulation

Conducting a numerical simulation on the cholera-measles mathematical model, the following iterative scheme of Laplace adomian decomposition method for the model equation is considered. 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.

$$\begin{aligned}
L\left[\frac{dS}{dt}\right] &= L\left[\Pi + \beta_2 V\right] - L\left[\beta_1 S + \beta S I_1 + \alpha S I_2 + (\tau+\omega)S + \mu S\right] \\
L\left[\frac{dV}{dt}\right] &= L\left[\beta_1 S\right] - L\left[(\beta_2+\mu)V\right] \\
L\left[\frac{dE_1}{dt}\right] &= L\left[\beta S I_1\right] - L\left[(c+\eta+\mu)E_1\right] \\
L\left[\frac{dE_2}{dt}\right] &= L\left[\alpha S I_2\right] - L\left[(\sigma+\mu)E_2\right] \\
L\left[\frac{dI_1}{dt}\right] &= L\left[\tau S + (c+\eta)E_1\right] - L\left[(\varepsilon+\delta_1+r+\mu)I_1\right] \\
L\left[\frac{dI_2}{dt}\right] &= L\left[\sigma E_2\right] - L\left[(\delta_2+\rho+\mu)I_2\right] \\
L\left[\frac{dR}{dt}\right] &= L\left[\omega S + (r+\varepsilon)I_1 + \rho I_2\right] - L\left[\mu R\right]
\end{aligned} \tag{3.33}$$

Substituting from 2.2 into 3.33 to yield

$$\begin{aligned}
mL[S(t)] &= S(0) + L\left[\Pi + \beta_2 V\right] - L\left[\beta_1 S + \beta S I_1 + \alpha S I_2 + (\tau+\omega)S + \mu S\right] \\
mL[V(t)] &= V(0) + L\left[\beta_1 S\right] - L\left[(\beta_2+\mu)V\right] \\
mL[E_1(t)] &= E_1(0) + L\left[\beta S I_1\right] - L\left[(c+\eta+\mu)E_1\right] \\
mL[E_2(t)] &= E_2(0) + L\left[\alpha S I_2\right] - L\left[(\sigma+\mu)E_2\right] \\
mL[I_1(t)] &= I_1(0) + L\left[\tau S + (c+\eta)E_1\right] - L\left[(\varepsilon+\delta_1+r+\mu)I_1\right] \\
mL[I_2(t)] &= I_2(0) + L\left[\sigma E_2\right] - L\left[(\delta_2+\rho+\mu)I_2\right] \\
mL[R(t)] &= R(0) + L\left[\omega S + (r+\varepsilon)I_1 + \rho I_2\right] - L\left[\mu R\right]
\end{aligned} \tag{3.34}$$

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

$$
\begin{aligned}
L[S(t)] &= \frac{s_0}{m} + \frac{\Pi}{m^2} + \frac{1}{m}L\left[\Pi + \beta_2 V\right] - L\left[\beta_1 S + \beta S I_1 + \alpha S I_2 + (\tau+\omega)S + \mu S\right] \\
L[V(t)] &= \frac{v_0}{m} + \frac{1}{m}L\left[\beta_1 S\right] - L\left[(\beta_2+\mu)V\right] \\
L[E_1(t)] &= \frac{e_1}{m} + \frac{1}{m}L\left[\beta S I_1\right] - L\left[(c+\eta+\mu)E_1\right] \\
L[E_2(t)] &= \frac{e_2}{m} + \frac{1}{m}L\left[\alpha S I_2\right] - L\left[(\sigma+\mu)E_2\right] \\
L[I_1(t)] &= \frac{i_1}{m} + \frac{1}{m}L\left[\tau S + (c+\eta)E_1\right] - L\left[(\varepsilon+\delta_1+r+\mu)I_1\right] \\
L[I_2(t)] &= \frac{i_2}{m} + \frac{1}{m}L\left[\sigma E_2\right] - L\left[(\delta_2+\rho+\mu)I_2\right] \\
L[R(t)] &= \frac{r_0}{m} + \frac{1}{m}L\left[\omega S + (r+\varepsilon)I_1 + \rho I_2\right] - L\left[\mu R\right]
\end{aligned}
\tag{3.35}
$$

Where $S(0) = s_0, V(0) = v_0, E(0) = e_0, I(0) = i_0, R(0) = r_0$. Letting the non-linear terms in the above iteration and substitutes by taking the inverse Laplace transform of both sides,

$$
\begin{aligned}
S(t) &= s_0 + \Pi t + L^{-1}\left(\frac{1}{m}[\beta_2 V] - L[\beta_1 S + \beta S I_1 + \alpha S I_2 + (\tau+\omega)S + \mu S]\right) \\
V(t) &= v_0 + L^{-1}\left(\frac{1}{m}L[\beta_1 S] - L[(\beta_2+\mu)V]\right) \\
E_1(t) &= e_0 + L^{-1}\left(\frac{1}{m} + L[\beta S I_1] - L[(c+\eta+\mu)E_1]\right) \\
E_2(t) &= i_0 + L^{-1}\left(\frac{1}{m} + L[\alpha S I_2] - L[(\sigma+\mu)E_2]\right) \\
I_1(t) &= i_0 + L^{-1}\left(\frac{1}{m} + L[\tau S + (c+\eta)E_1] - L[(\varepsilon+\delta_1+r+\mu)I_1]\right) \\
I_2(t) &= r_0 + L^{-1}\left(\frac{1}{m}L[\sigma E_2] - L[(\delta_2+\rho+\mu)I_2]\right) \\
R(t) &= r_0 + L^{-1}\left(\frac{1}{m}L[\omega S + (r+\varepsilon)I_1 + \rho I_2] - L[\mu R]\right)
\end{aligned}
\tag{3.36}
$$

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

$$\sum_{k=0}^{\infty} S_n(t) = s_0 + \Pi t + L^{-1}\left(\frac{1}{m}\left[\beta_2 \sum_{k=0}^{\infty} V_n\right] - L\left[\beta_1 \sum_{k=0}^{\infty} S_n + \beta \sum_{k=0}^{\infty} SI_n + \alpha \sum_{k=0}^{\infty} SI_n + [(\tau+\omega+\mu)] \sum_{k=0}^{\infty} S_n\right]\right)$$
$$\sum_{k=0}^{\infty} V_n(t) = v_0 + L^{-1}\left(\frac{1}{m} L\left[\beta_1 \sum_{k=0}^{\infty} S_n\right] - L\left[(\beta_2+\mu) \sum_{k=0}^{\infty} V_n\right]\right)$$
$$\sum_{k=0}^{\infty} E_1(t) = e_0 + L^{-1}\left(\frac{1}{m} + L\left[\beta \sum_{k=0}^{\infty} SI_n\right] - L\left[(c+\eta+\mu) \sum_{k=0}^{\infty} E_1\right]\right)$$
$$\sum_{k=0}^{\infty} E_2(t) = i_0 + L^{-1}\left(\frac{1}{m} + L\left[\alpha \sum_{k=0}^{\infty} SI_2\right] - L\left[(\sigma+\mu) \sum_{k=0}^{\infty} E_2\right]\right)$$
$$\sum_{k=0}^{\infty} I_1(t) = i_0 + L^{-1}\left(\frac{1}{m} + L\left[\tau \sum_{k=0}^{\infty} S_n + (c+\eta) \sum_{k=0}^{\infty} E_1\right] - L\left[(\varepsilon+\delta_1+r+\mu) \sum_{k=0}^{\infty} I_1\right]\right)$$
$$\sum_{k=0}^{\infty} I_2(t) = r_0 + L^{-1}\left(\frac{1}{m} L\left[\sigma \sum_{k=0}^{\infty} E_2\right] - L\left[(\delta_2+\rho+\mu) \sum_{k=0}^{\infty} I_2\right]\right)$$
$$\sum_{k=0}^{\infty} R(t) = r_0 + L^{-1}\left(\frac{1}{m} L\left[\omega \sum_{k=0}^{\infty} S + (r+\varepsilon) \sum_{k=0}^{\infty} I_1 + \rho \sum_{k=0}^{\infty} I_2\right] - L\left[\mu \sum_{k=0}^{\infty} R\right]\right)$$

The initial approximations of each class are given by; $S_0(t) = s_0$, $V_0(t) = v_0$, $E_1(t) = e_0$, $E_2(t) = e_0$, $I_1(t) = i_0$, $I_2(t) = i_0$, $R_0(t) = r_0$. Now, comparing the coefficients at $n = 1, 2, 3$. Using the recurrence relations obtained from the iterations. Compartmentally it is obtained that

$$\begin{aligned}
D^{\eta_1} S(t) &= \Pi - \frac{1}{2}\beta_1 s_0 i_0 - \frac{2}{3}\beta s_1 e_1 - \alpha s_1 s_2 - \frac{3}{5}(\tau+\omega) s_0 + \beta_2 v_0 - \mu s_0 \\
D^{\eta_1} V(t) &= \beta_1 s_0 - \frac{1}{3}(\beta_2+\mu) v_0 \\
D^{\eta_1} E_1(t) &= \frac{1}{2}\beta s_0 i_0 - \frac{2}{3}(c+\eta+\mu) e_0 E_1 \\
D^{\eta_1} E_2(t) &= \alpha s_0 i_0 - (\sigma+\mu) e_0 \\
D^{\eta_1} I_1(t) &= -\frac{1}{2}\tau s_0 r_0 + (c+\eta) e_1 i_1 - (\varepsilon+\delta_1+r+\mu) i_0 r_1 \\
D^{\eta_1} I_2^*(t) &= \sigma e_0 s_0 - \frac{1}{5}(\delta_2+\rho+\mu) i_0 \\
D^{\eta_1} R^*(t) &= \frac{1}{6}\omega s_0 + (r+\varepsilon) i_0 r_0 - \frac{1}{3}\rho i_0 e_1 - \mu v_0 r_0
\end{aligned} \tag{3.37}$$

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

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

$$
\begin{aligned}
&+\left(-\frac{1}{6}ai_0\pi\beta_1-\frac{1}{3}\beta_1\mu\pi-\frac{1}{6}\beta_1^2\pi+\frac{1}{6}\beta_2\pi\beta_1\right)t^3\\
E_{12}(t)&=\left(-\frac{1}{6}\alpha^2i^2\pi-\frac{1}{3}ai_0\pi\delta-\frac{2}{3}ai_0\pi\mu-\frac{1}{3}ai_0\pi\rho+\frac{1}{3}ae_0\pi\sigma_1-\frac{1}{6}\mu^2\pi-\frac{1}{6}ai_0\pi\beta_1\right)t^3\\
&+\left(-\frac{1}{2}\alpha^2i^2s_0-\frac{1}{2}\sigma is_0-\frac{2}{3}ais_0\mu_0-\frac{1}{2}ais_0\rho_0+\frac{1}{2}ais_0v_0-\mu^2ie_0\beta_1+\frac{1}{2}aie_0\sigma^2\right)t^2\\
E_{22}(t)&=\left(-\frac{1}{6}\alpha^2i^2\pi-\frac{1}{3}ai_0\pi\delta-\frac{2}{3}ai_0\pi\mu-\frac{1}{3}ai_0\pi\rho+\frac{1}{3}ae_0\pi\sigma_1-\frac{1}{6}\mu^2\pi-\frac{1}{6}ai_0\pi\beta_1\right)t^3\\
&+\left(-\frac{1}{2}\alpha^2i^2s_0-\frac{1}{2}\sigma is_0-\frac{2}{3}ais_0\mu_0-\frac{1}{2}ais_0\rho_0+\frac{1}{2}ais_0v_0-\mu^2ie_0\beta_1+\frac{1}{2}aie_0\sigma^2\right)t^2\\
I_{12}(t)&=-\frac{1}{6}\alpha^2i^2\theta+\left\{\begin{pmatrix}\frac{1}{2}\sigma ais_0+\frac{1}{2}\delta^2i_0+\delta\mu i_0-\frac{1}{2}\delta\sigma ie_0+\frac{1}{2}\mu^2i_0-\mu\rho i_0\\-\mu\sigma i_0+\frac{1}{2}\rho^2i_0-\frac{1}{2}\rho\sigma ie_0-\frac{1}{2}\sigma^2e_0\end{pmatrix}\right\}t^2\\
I_{22}(t)&=-\frac{1}{6}\alpha^2i^2\theta+\left\{\begin{pmatrix}\frac{1}{2}\sigma ais_0+\frac{1}{2}\delta^2i_0+\delta\mu i_0-\frac{1}{2}\delta\sigma ie_0+\frac{1}{2}\mu^2i_0-\mu\rho i_0\\-\mu\sigma i_0+\frac{1}{2}\rho^2i_0-\frac{1}{2}\rho\sigma ie_0-\frac{1}{2}\sigma^2e_0\end{pmatrix}\right\}t^2\\
R_2(t)&=\left(-\frac{1}{2}\delta\rho i_0+\frac{1}{2}\mu^2r_0-\mu\sigma i_0-\frac{1}{2}\rho^2i_0+\frac{1}{2}\phi 1\varphi e_0\right)t^2
\end{aligned}
\tag{3.38}
$$

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:

$$
\begin{aligned}
&S(t)=\sum_{k=0}^{3}s_k(t),\quad V(t)=\sum_{k=0}^{3}v_k(t),\quad E_1(t)=\sum_{k=0}^{3}e_k(t),\quad E_2(t)=\sum_{k=0}^{3}e_k(t),\\
&I_1(t)=\sum_{k=0}^{3}i_k(t),\quad I_2(t)=\sum_{k=0}^{3}e_k(t),\quad R(t)=\sum_{k=0}^{3}r_k(t)
\end{aligned}
\tag{3.39}
$$

Evaluating these series results using the corresponding variables and parameter values for the iterative terms gives

$$
\begin{aligned}
S(t)&=0.1739989120t^5-3.533740138t^4-138.3514794t^3+174.7274788t^2-89.34895283t+27.15,\\
V(t)&=4.671044382t^4+182.0996275t^3+109.1002246t^2-0.1960329605\times10^{-1}t^{1.42}-210.48795t\\
&\quad+120,\\
I_1(t)&=-0.4671030819\times10^{-2}t^5+3.193922301t^4+117.2743940t^3-93.96760837t^2+38.399597t\\
&\quad+23.09,\\
I_2(t)&=-0.2726645556\times10^{-1}t^4-50.75344213t^3+77.07799304t^2-83.59318206t+48.92383,\\
E_1(t)&=-0.1583478276t^5-5.522198199t^4-58.53997772t^3-61.60059124t^2-11.59646209t+65,\\
E_2(t)&=1.673648686t^4+70.48429300t^3+41.51033700t^2-0.440381512710^{-2}t^{1.42}-118.5445901t\\
&\quad+106,\\
R(t)&=0.4167563352\times10^{-2}t^5+1.670999701t^4-1342.400000t^3+4138.966974t^2-8344.357997t\\
&\quad+8381.93.
\end{aligned}
$$

3.4. Results and Discussion

Graphical illustration of the resulting iterations is shown in Figure 3.2 – Figure 3.7:

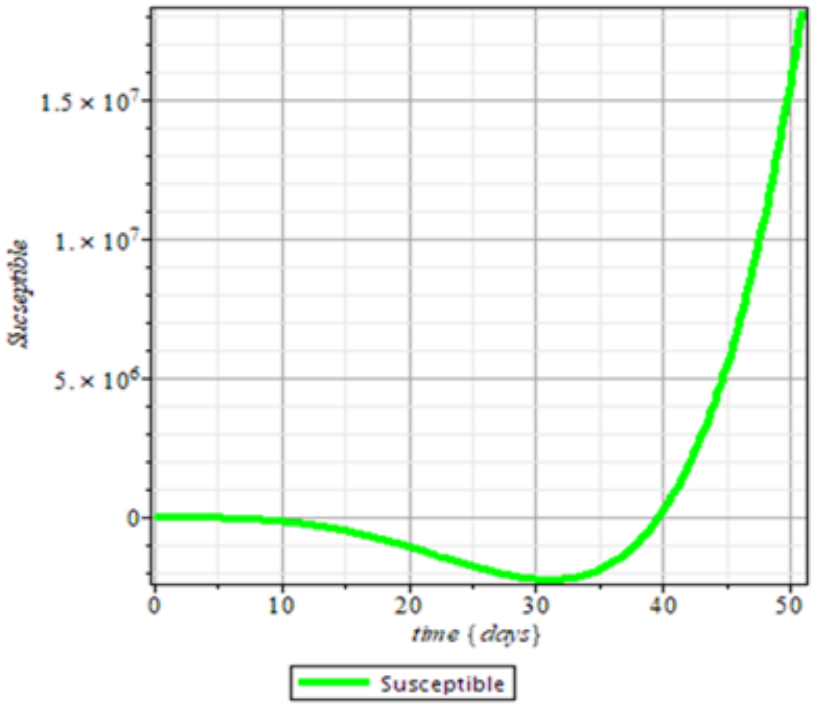

Figure 3.2: Time profile of the susceptible population. The susceptible class decreases rapidly at the early stage of the outbreak and then approaches a stable level as infection and recovery dynamics evolve over time.

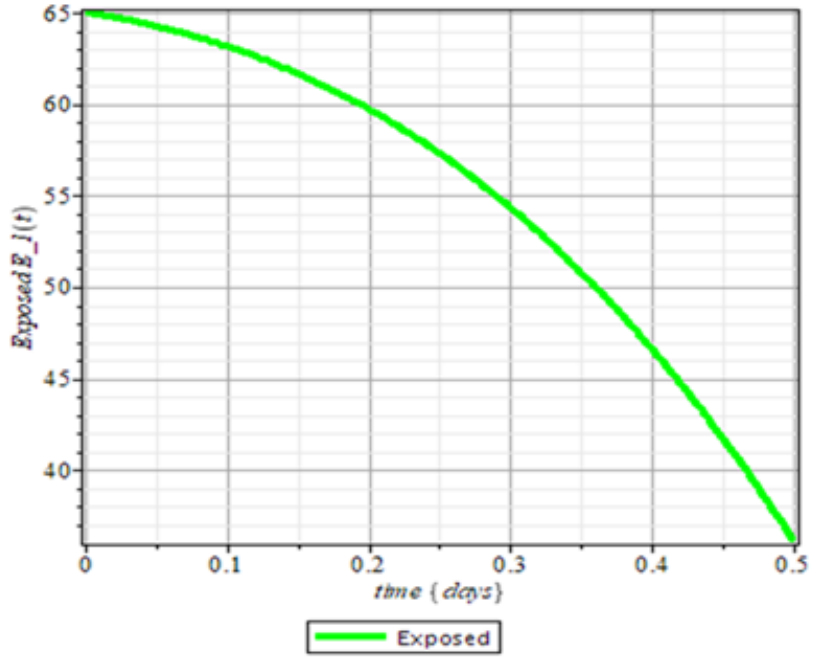

Figure 3.3: Time profile of the infectious population under public awareness intervention. The infectious population rises initially, reaches a peak, and then declines gradually as awareness reduces exposure and promotes control of transmission.

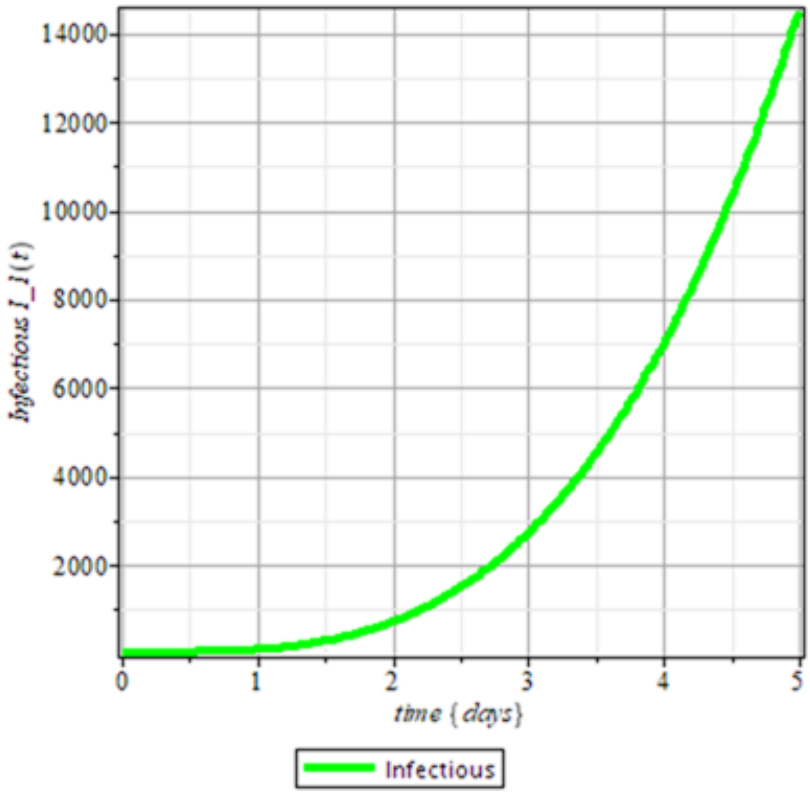

Figure 3.4: Time profile of the recovered population under water treatment intervention. The recovered class increases over time, indicating improved recovery and reduced transmission as access to clean water interrupts the infection cycle.

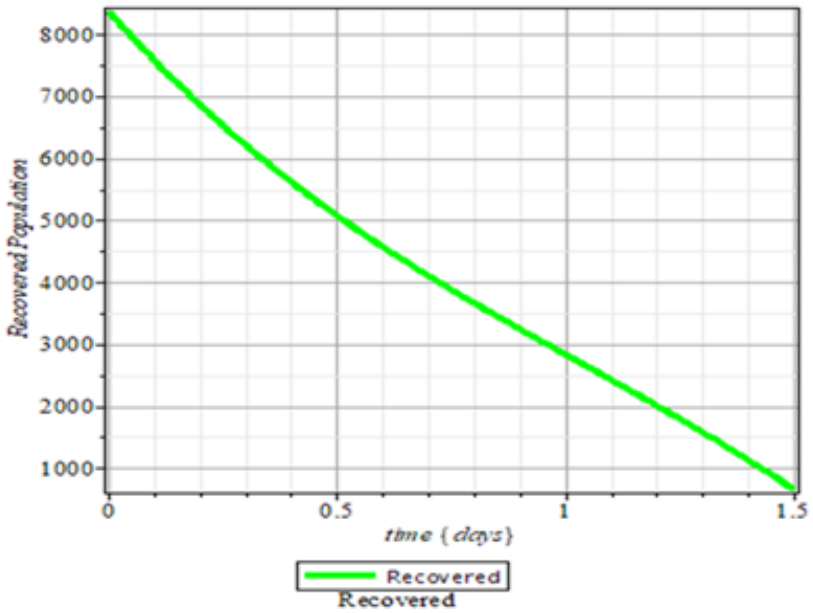

Figure 3.5: Time profile of environmental bacterial concentration. The bacterial concentration shows an initial dip followed by a sustained increase, reflecting the continuing contribution of infected individuals to environmental contamination despite control efforts.

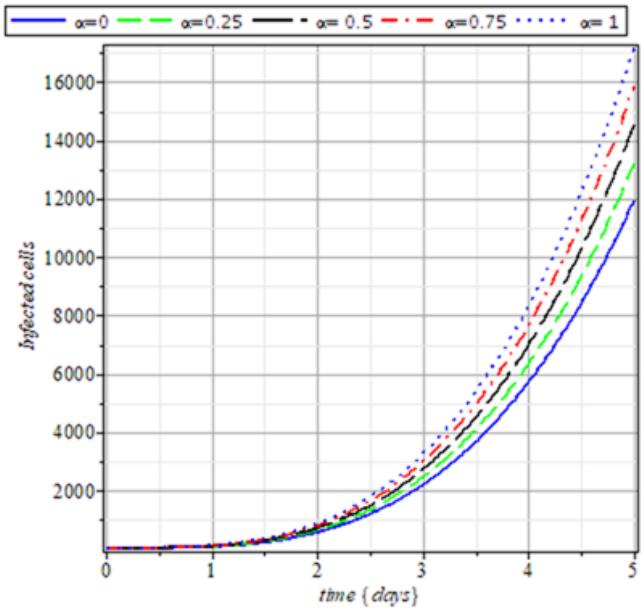

Figure 3.6: Effect of varying contact rate on the infectious population. Higher contact rates produce larger infection peaks and slower declines, whereas lower contact rates reduce the magnitude of infection and improve disease control.

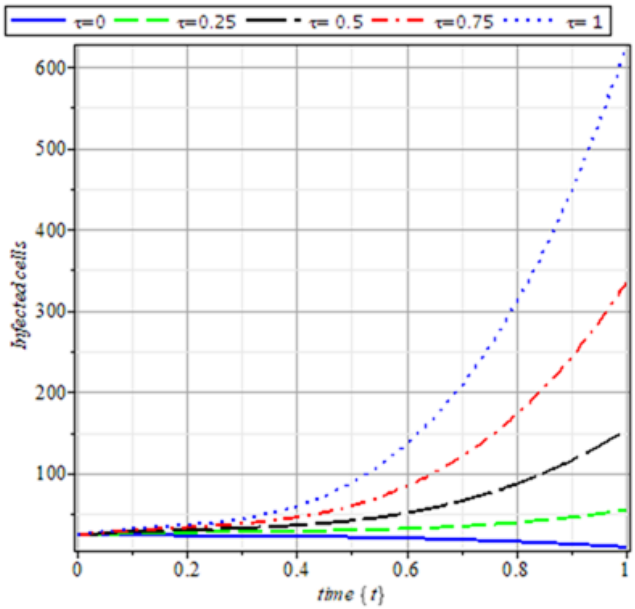

Figure 3.7: Effect of infectious contact with the environment on bacterial concentration. Increased infectious contact with the environment leads to higher bacterial concentration, while reduced contact lowers environmental contamination and supports outbreak control.

The simulation results reveal the dynamic effect of the intervention measures on the cholera–measles model. Figure 3.2 shows that the susceptible population declines sharply during the early phase of the outbreak before approaching a more stable level. This indicates that many susceptible individuals are rapidly moved into other compartments at the onset of transmission, but

the rate of depletion slows as intervention and recovery begin to moderate the epidemic process. Figure 3.3 illustrates the behaviour of the infectious population under public awareness measures. The infectious class rises to an early peak and then declines gradually with time. This suggests that public awareness contributes to behavioural changes such as reduced exposure, earlier response to symptoms, and better adherence to preventive measures, thereby lowering disease transmission in the later phase of the outbreak. Figure 3.4 shows the recovered population under water treatment intervention. After a slight initial decrease, the recovered class rises steadily, indicating that improved water quality supports recovery and reduces further exposure to infection. This emphasizes the importance of water treatment as a practical strategy for interrupting disease transmission and improving public health outcomes. Figure 3.5 presents the environmental bacterial concentration. The trajectory shows a brief initial reduction followed by a substantial increase over time, which implies that environmental contamination persists as infected individuals continue to shed pathogens into the environment. This means that treatment alone may not be sufficient unless it is combined with strong sanitation and hygiene measures.

Figure 3.6 compares the infectious population at different contact rates. The curves show that larger contact rates produce higher peaks and a slower decline in the number of infectious individuals, while smaller contact rates suppress the outbreak more effectively. This confirms that reducing effective contact remains one of the most important mechanisms for controlling disease spread.

Figure 3.7 describes the effect of infectious contact with the environment on bacterial concentration. As the level of environmental contact increases, bacterial concentration rises more rapidly and reaches a higher level. Conversely, reduced contact with contaminated environmental sources lowers bacterial build-up. This highlights the importance of sanitation enforcement, environmental protection, and reduced human–environment exposure during outbreaks.

4. Conclusion

In conclusion, the study demonstrates that combining rapid treatment and vaccination significantly controls the spread of cholera and emancipation of measles diseases. These interventions reduce infection rates and mitigate the diseases' impact. It is imperative for healthcare personnel to prioritize and adhere to these measures to control cholera outbreaks effectively. Prompt treatment and widespread vaccination should be integral components of public health strategies to combat this persistent and potentially devastating disease. By implementing these recommendations, we can achieve substantial progress in managing and eventually eradicating cholera and measles disease.

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