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Mathematical Modeling and Stability Analysis of Cholera Transmission Dynamics with Environmental Sanitation and Treatment Interventions

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ABSTRACT

Cholera remains a persistent public health challenge despite significant advancements in medicine and hygiene. Understanding its transmission dynamics is essential for effective control and eradication. This study develops a compartmental mathematical model to analyze the spread of Vibrio cholerae, incorporating key control measures such as treatment, natural recovery, reinfection, and environmental sanitation. The model is formulated as a system of nonlinear differential equations, representing different population compartments. A key component of the analysis is the derivation of the basic reproduction number (R_0) ,which serves as a threshold indicator for disease persistence. Stability analysis reveals that: If $(R_0 < 1)$, the disease-free equilibrium is globally asymptotically stable, indicating eventual cholera eradication. If $(R_0 > 1)$ an endemic equilibrium exists, signifying sustained cholera transmission within the population. Sensitivity analysis identifies the most influential parameters affecting (R_0), highlighting that increasing treatment rates and improving sanitation significantly reduce disease spread. The fourth order Runge–Kutta numerical scheme is implemented in MAPLE 21 to generate the numerical solutions, which demonstrate that, timely treatment, and environmental sanitation accelerates the reduction of R_0, moving the system toward the disease-free state.

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Introduction

Cholera is an infectious disease transmitted through contaminated water, primarily caused by the bacterium Vibrio cholerae World Health Organizations (2021) & Mo'tassem et al. [1]. The hallmark symptoms of this illness include severe diarrhea and dehydration, as noted by the World Health Organization (2022) & Capass et al. [2]. Individuals typically contract cholera by consuming water or beverages that are tainted, as well as through the ingestion of food contaminated with the bacteria. The rapid onset of cholera can significantly diminish life expectancy, particularly in situations where the outbreak is not identified promptly and adequate medical care is not provided. The urgency of early detection and intervention is critical in mitigating the disease's impact on affected populations Agbomola & Loyinmi [3].

Cholera tends to spread rapidly in regions characterized by inadequate food safety, poor hygiene, lack of clean water, and overcrowding [4]. The incubation period for the disease ranges from two to five days, during which time it can significantly increase the likelihood of outbreaks [5,6]. Despite various measures taken to control the transmission of cholera, the disease persists in causing both epidemic and pandemic outbreaks, thereby remaining a significant global public health threat [7]. Efforts to mitigate the impact of cholera have been numerous; however, the disease continues to pose challenges to public health systems worldwide Idowu et al. [8,9]. The ongoing prevalence of cholera highlights the need for sustained interventions and improved infrastructure in vulnerable communities to effectively combat this persistent health crisis.

The study of cholera transmission dynamics, particularly in relation to environmental sanitation and treatment interventions, has received limited attention in terms of interaction strategies. Building upon the work of Martins *et al*. this research extends the existing model by incorporating an exposed phase of infection, along with treatment, natural recovery, reinfection, and sanitation measures to provide a more comprehensive understanding of transmission and control mechanisms [10].

The paper is structured as follows: Section 2 details the materials and methods used in formulating the model, Section 3 discusses the theoretical framework and calculations, Section 4 presents the results, Section 5 provides an in-depth discussion of the findings, and Section 6 concludes the study.

Material and Methods

The model focuses on five compartmental models to acquire insight into the effect of campaign and treatment rate on the transmission of diarrhea disease in a community. The model includes the Susceptible individuals $S(t)$,Exposed individuals $E(t)$, Infected individuals, $I(t)$ Recovered individuals $R(t)$ and the concentration of Vibrio Cholera $V(t)$. The overall populace $N(t)$ is as follows:

$$N(t) = S + E + I + R \qquad (1)$$

The susceptible population increases by birth at a constant rate Ω and all individuals in the recovered class returns to the susceptible due to natural recovery of exposed individuals at a rate β ,where $(\alpha_I I + \alpha_V V)$ is the effective contact rate and the force of infection. The population of the susceptible decreases due to the natural death at the rate μ.

$$\frac{dS}{dt} = \Omega - (\alpha_I I + \alpha_V V)S - \mu S + \gamma R + \beta E \qquad (2)$$

The exposed individuals are the ones that carry the bacteria but there are not capable of infecting the susceptible class. it increases by new infected susceptible individuals who acquired Cholera with effective contact rate or force of infection $(\alpha_I I + \alpha_V V)$. The population decreases due the rate at which exposed individuals progresses infected class at the rate *K,* they also reduce back to the suspectable class due to natural recovery at a rate β and through natural death rate μ.

$$\frac{dE}{dt} = (\alpha_I I + \alpha_V V)S - (\kappa + \beta + \mu)E \qquad (3)$$

The infected individuals increase by the progression of the exposed individuals who began to show case signs and symptoms of Cholera at a rate *k,* alongside Re-infection at the rate ϕ and decreases due to the treatment rate θ they also reduce as a result of human contribution to cholera concentration σ , natural death rate and induced disease death rate ω.

$$\frac{dI}{dt} = \kappa E - (\sigma + \mu + \omega + \theta)I + \phi R \qquad (4)$$

The recovered individuals grow by the number treated individuals at the rate θ The population reduces due to natural death rate μ, recovered individuals who become re-infected at the rate ϕ and also recovered individuals returning to the susceptible compartment due to loss of immunity at a rate γ .

$$\frac{dR}{dt} = \theta I - (\gamma + \mu + \phi)R \qquad (5)$$

The concentration of *Vibrio Cholera* increases at the rate of human contribution to cholera concentration and the growth of *Vibrio Cholera* in the environment at the rate *g* and decreases at the rate of disinfection in the environment and the decay rate of Vibrio Cholera η and δ respectively.

$$\frac{dV}{dt} = \sigma I - (\eta + \delta - g)V \qquad (6)$$

The concentration of *Vibrio Cholera* increases at the rate of human contribution to cholera concentration σ and the growth of *Vibrio Cholera* in the environment at the rate and decreases at the rate of disinfection in the environment and the decay rate of Vibrio Cholera η and δ respectively.

$$\frac{dV}{dt} = \sigma I - (\eta + \delta - g)V \qquad (7)$$

Table 1: Description of Variables

Variables	Description
$N(t)$	Total human population at time t
$S(t)$	Susceptible at time t
$E(t)$	Exposed at time t
$I(t)$	Infected at time t
$R(t)$	Recovered at time t
$V(t)$	Concentration of V.C in contaminating the environment at time t

Table 2: Description of Parameters and Values

Parameter	Description	Value	Source
Ω	Recruitment rate	1	Assumed
β	Natural recovery of exposed individuals	0.4	Assumed
θ	Treatment rate	0.5	Varied
ϕ		0.04	Adedapo et al. [11]
κ	Progression rate	0.3	Assumed
μ	Natural death rate	0.2	Adedapo et al. [11]
g	Growth rate of Vibrio Cholera	0.04	Assumed
C_V	The density of V.C in the eco-system	0.01	Assumed
C_h	The density of infection among humans	0.02	Assumed
δ	The decay rate of Vibrio Cholera	0.3	Umar et al. [12]
ω	Disease induces death rate	0.2	Martins et al. [10]
η	The rate of disinfection in the environment	0.4	Umar et al. [12]
σ	The rate at which infectious humans contaminate the ecosystem	0.02	Assumed
γ	Rate of loss of immune by recovered individuals	0.5	Martins et al. [10]
a_I	A positive constant providing adjustment to the rate of infection	0.4	Varied
α_V	A positive constant providing adjustment to the rate of Vibrio ingestion in the ecosystem.	0.01	Assumed
	Basic reproduction number	0.7252	Estimated
$N(0)$	Total human population	3,050	Estimated
$S(0)$	Susceptible class	1,000	Estimated
$E(0)$	Exposed class	800	Estimated
$I(0)$	Infected class	500	Estimated
$R(0)$	Recovered class	700	Estimated
$V(0)$	Concentration of V.C in contaminating the environment	50	Estimated

$$\frac{dS}{dt} = \Omega - (\alpha_I I + \alpha_V V)S - \mu S + \gamma R + \beta E \qquad (1)$$

$$\frac{dE}{dt} = (\alpha_I I + \alpha_V V)S - (\kappa + \beta + \mu)E \qquad (2)$$

$$\frac{dI}{dt} = \kappa E - (\sigma + \mu + \omega + \theta)I + \phi R \qquad (3)$$

$$\frac{dR}{dt} = \theta I - (\gamma + \mu + \phi)R \qquad (4)$$

$$\frac{dV}{dt} = \sigma I - (\eta + \delta - g)V \qquad (5)$$

Where,

$$\alpha_I = \frac{C_h}{1 + \alpha_T} \text{ and } \alpha_V = \frac{C_v}{V + P}$$

Were,

$B_1 = (\kappa + \beta + \mu)$ $B_2 = (\sigma + \mu + \omega + \theta)$ $B_3 = (\gamma + \mu + \phi)$ and $B_4 = (\eta + \delta - g)$

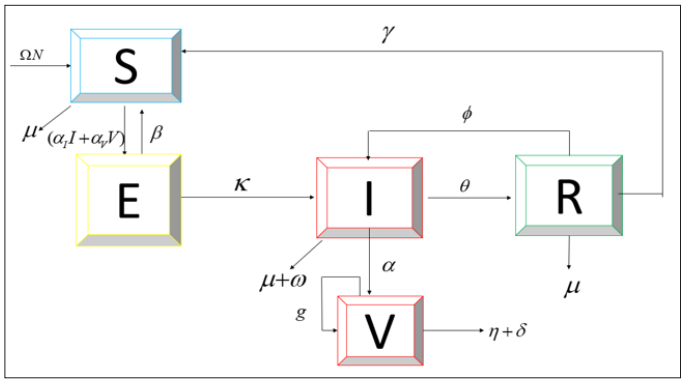

Figure 1: Flow Diagram of Seirv Epidemic Model

Analysis of Model Equation for Existence of Solution
Existence and Uniqueness of Solution

Theorem 3.1.1: (As proposed by Derrick and Grossman 1976) Let

$$x_1 = f_1(x_1, x_2, \ldots, x_n, t), x_1(t_0) = x_{10}$$

$$x_2 = f_2(x_1, x_2, \ldots, x_n, t), x_2(t_0) = x_{20}$$

:

$$x_n = f_n(x_1, x_2, \ldots, x_n, t), x_n(t_0) = x_{n0}$$

Let D denote the region in $[(n+1)$ dimensional space one dimension for t and n dimension for the vector $x]$

Then there is a constant $\delta > 0$ such that there exists a unique continuous vector solution.

$$\underline{x} = [x_1(t), x_2(t), \ldots, x_n(t)]$$

$$\frac{dS}{dt} = \Omega - (\alpha_I I + \alpha_V V)S - \mu S + \gamma R + \beta E$$

$$\frac{dE}{dt} = (\alpha_I I + \alpha_V V)S - (\kappa + \beta + \mu)E$$

$$\frac{dI}{dt} = \kappa E - (\sigma + \mu + \omega + \theta)I + \phi R$$

$$\frac{dR}{dt} = \theta I - (\gamma + \mu + \phi)R$$

$$\frac{dV}{dt} = \sigma I - (\eta + \delta - g)V$$

Where $B_1 = (\kappa + \beta + \mu)$ $B_2 = (\sigma + \mu + \omega + \theta)$ $B_3 = (\gamma + \mu + \phi)$ and $B_4 = (\eta + \delta - g)$

$D = \{(S, E, I, R, V) / S - S_0 / \le a, / E - E_0 / \le b, / I - I_0 / \le c, / R - R_0 / \le d, / V - V_0 / \le e\}$

Then the Equation has a Unique Solution
Proof

$$\left.\frac{df_1}{dS}\right|_{0,0,0,0} = |-(\alpha_I I + \alpha_V V) - \mu| = (\alpha_I I + \alpha_V V) + \mu < \infty$$

$$\left.\frac{df_1}{dE}\right|_{0,0,0,0} = |\beta| = \beta < \infty$$

$$\left.\frac{df_1}{dI}\right|_{0,0,0,0} = |-(\alpha_I)S| = (\alpha_I)S < \infty$$

$$\left.\frac{df_1}{dR}\right|_{0,0,0,0} = |\gamma| = \gamma < \infty$$

$$\left.\frac{df_1}{dV}\right|_{0,0,0,0} = |-(\alpha_V)S| = (\alpha_V)S < \infty$$

$$\left.\frac{df_2}{dS}\right|_{0,0,0,0} = |(\alpha_I I + \alpha_V V)| = (\alpha_I I + \alpha_V V) < \infty$$

$$\left.\frac{df_2}{dE}\right|_{0,0,0,0} = |-(\kappa + \beta + \mu)| = \kappa + \beta + \mu < \infty$$

$$\left.\frac{df_2}{dI}\right|_{0,0,0,0} = |-(\alpha_I)S| = (\alpha_I)S < \infty$$

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

$$\left.\frac{df_2}{dV}\right|_{0,0,0,0} = |-(\alpha_V)S| = (\alpha_V)S < \infty$$

$$\left.\frac{df_3}{dS}\right|_{0,0,0,0} = 0 < \infty$$

$$\left.\frac{df_3}{dE}\right|_{0,0,0,0} = |\kappa| = \kappa < \infty$$

$$\left.\frac{df_3}{dI}\right|_{0,0,0,0} = |-(\sigma + \mu + \omega + \theta)| = \sigma + \mu + \omega + \theta < \infty$$

$$\left.\frac{df_3}{dR}\right|_{0,0,0,0} = |\phi| = \phi < \infty$$

$$\left.\frac{df_3}{dV}\right|_{0,0,0,0} = 0 < \infty$$

$$\left.\frac{df_4}{dS}\right|_{0,0,0,0} = 0 < \infty$$

$$\left.\frac{df_4}{dE}\right|_{0,0,0,0} = 0 < \infty$$

$$\left.\frac{df_4}{dI}\right|_{0,0,0,0} = |\theta| = \theta < \infty$$

$$\left.\frac{df_4}{dR}\right|_{0,0,0,0} = |-(\gamma+\mu+\phi)| = \gamma+\mu+\phi < \infty$$

$$\left.\frac{df_4}{dV}\right|_{0,0,0,0} = 0 < \infty$$

$$\left.\frac{df_5}{dS}\right|_{0,0,0,0} = 0 < \infty$$

$$\left.\frac{df_5}{dE}\right|_{0,0,0,0} = 0 < \infty$$

$$\left.\frac{df_5}{dI}\right|_{0,0,0,0} = |\sigma| = \sigma < \infty$$

$$\left.\frac{df_5}{dR}\right|_{0,0,0,0} = 0 < \infty$$

$$\left.\frac{df_5}{dV}\right|_{0,0,0,0} = |-(\eta+\delta-g)| = \eta+\delta-g < \infty$$

Therefore, $\left|\frac{df_i}{dx_j}\right| i, j = 1,2$ are continuous and bounded, then the

model has a unique solution.

Hence, the problem has a unique solution and the model is mathematically and epidemiologically well posed.

Theorem 3.1.2 (Positivity and Boundedness of Solution)

Let the initial data $\mathrm{S}(0),\ \mathrm{E}(0),\ \mathrm{I}(0), \mathrm{R}(0)$ be non-negative, then the solution of the model are non-negative for all $\mathrm{t} > 0$

Proof
Consider the biological feasibility region $D = \{(S,E,I,R) \in \mathbb{R}^4_+ N \le \frac{\Omega}{\mu}\}$

It will be shown that D is positive invariance (i.e. all solutions remain in D for all time $t > 0$)

The rate of change of total population

$$\frac{dN}{dt} = \Omega - \mu(S+E+I+R)$$

Where $\mathrm{N} = \mathrm{S}+\mathrm{E}+\mathrm{I}+\mathrm{R}$

$$\frac{dN}{dt} = \Omega - \mu N \Rightarrow \frac{dN}{dt} \le \Omega - \mu N$$

Using integrating factor

$$N(t) \le e^{-\int \mu dt}\left[\int \Omega e^{\int \mu dt}\, dt\right]$$

$$N(t) \le e^{\ \mu t}[— e^{\mu t} + C]$$

$$N(t) \le \frac{\Omega}{\mu} + Ce^{-\mu t} \text{ at } \mathrm{t} = 0$$

$$N(0) \le \frac{\Omega}{\mu} + C$$

$$N(0) - \frac{\Omega}{\mu} \le C$$

Therefore, $N(t) \le \frac{\Omega}{\mu} + e^{-\mu t}(N(0) - \frac{\Omega}{\mu})$

If $N(0) - \frac{\Omega}{\mu}$

As $t \to \infty, N \to \frac{\Omega}{\mu}$ Showing that

$$0 \le N < \frac{\Omega}{\mu} \text{ and } N \le \frac{\Omega}{\mu}$$

Hence, $D = \{(S,E,I,R) \in \mathbb{R}^4_+ : N \le \frac{\Omega}{\mu}\}$

Therefore, all solutions of the model with initial conditions in *D* remain in the region for $\mathrm{t} > 0$, this implies that *D* is positively invariant. In this region, the model can be considered as been epidemiologically and mathematically well posed.

Disease free Equilibrium
At disease free equilibrium, when there is no infection. That is $\mathrm{E} = \mathrm{I} = \mathrm{R} = 0$ and at equilibrium point, the normalized model is obtained by setting.

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$$

Hence, the disease-free equilibrium is given by

$$E_0 = \left(\frac{\Omega}{\mu}, 0, 0, 0, 0\right) \qquad (6)$$

The Endemic Equilibrium Point
The endemic equilibrium points are:

$$\mathrm{S} = \frac{-\mathrm{B}_1\mathrm{B}_4(\phi\theta - \mathrm{B}_2\mathrm{B}_3)}{(\kappa\mathrm{B}_3(\sigma\alpha_{\mathrm{V}} + \mathrm{B}_4\alpha_I)} \qquad (7)$$

$$\mathrm{E} = \frac{(\Omega\kappa\sigma\mathrm{B}_3\alpha_{\mathrm{V}} + \Omega\kappa\mathrm{B}_3\mathrm{B}_4\alpha_I + \mu\phi\theta\mathrm{B}_1\mathrm{B}_4 - \mu\mathrm{B}_1\mathrm{B}_2\mathrm{B}_3\mathrm{B}_4)(\phi\theta - \mathrm{B}_2\mathrm{B}_3)}{((-\beta\phi\sigma\theta\alpha_{\mathrm{V}} - \beta\phi\theta\mathrm{B}_4\alpha_1 + \beta\sigma\mathrm{B}_2\mathrm{B}_3\alpha_{\mathrm{V}} + \beta\mathrm{B}_2\mathrm{B}_3\mathrm{B}_4\alpha_1 + \gamma\kappa\sigma\theta\alpha\mathrm{V} + \gamma\kappa\theta\mathrm{B}_4\alpha_1 + \phi\sigma\theta\mathrm{B}_1\alpha_{\mathrm{V}} + \phi\theta\mathrm{B}_1\mathrm{B}_4\alpha_1 - \sigma\mathrm{B}_1\mathrm{B}_2\mathrm{B}_3\alpha_{\mathrm{V}} - \mathrm{B}_1\mathrm{B}_2\mathrm{B}_3\mathrm{B}_4\alpha_1)\kappa\mathrm{B}_3)} \qquad (8)$$

$$I = \frac{-(\Omega\kappa\sigma\mathrm{B}_3\alpha_{\mathrm{V}} + \Omega\kappa\mathrm{B}_3\mathrm{B}_4\alpha_I + \mu\phi\theta\mathrm{B}_1\mathrm{B}_4 - \mu\mathrm{B}_1\mathrm{B}_2\mathrm{B}_3\mathrm{B}_4)}{(-\beta\phi\sigma\theta\alpha_1 - \beta\phi\theta\mathrm{B}_4\alpha_1 + \beta\sigma\mathrm{B}_2\mathrm{B}_3\alpha_{\mathrm{V}} + \beta\mathrm{B}_2\mathrm{B}_3\mathrm{B}_4\alpha_1 + \gamma\kappa\sigma\theta\alpha_{\mathrm{V}} + \gamma\kappa\theta\mathrm{B}_4\alpha_V + \phi\sigma\theta\mathrm{B}_1\alpha_{\mathrm{V}} + \phi\theta\mathrm{B}_1\mathrm{B}_4\alpha_I - \sigma\mathrm{B}_1\mathrm{B}_2\mathrm{B}_3\alpha_{\mathrm{V}} - \mathrm{B}_1\mathrm{B}_2\mathrm{B}_3\mathrm{B}_4\alpha_1)} \qquad (9)$$

$$R = \frac{-\theta(\Omega\kappa\sigma B_3\alpha_V + \Omega\kappa B_3B_4\alpha_I + \mu\phi\theta B_1B_4 - \mu B_1B_2B_3B_4)}{((-\beta\phi\sigma\theta\alpha_V - \beta\phi\theta B_4\alpha_I + \beta\sigma B_2B_3\alpha_V + \beta B_2B_3B_4\alpha_I + \gamma\kappa\sigma\theta\alpha_V + \gamma\kappa\theta B_4\alpha_V + \phi\sigma\theta B_1\alpha_V + \phi\theta B_1B_4\alpha_I - \sigma B_1B_2B_3\alpha_V - B_1B_2B_3B_4\alpha_V)B_3)} \quad (10)$$

$$V = \frac{-\sigma(\Omega\kappa\sigma B_3\alpha_V + \Omega\kappa B_3B_4\alpha_1 + \mu\phi\theta B_1B_4 - \mu B_1B_2B_3B_4)}{((-\beta\phi\sigma\theta\alpha_V - \beta\sigma\theta B_4\alpha_1 + \beta\sigma B_2B_3\alpha_V + \beta B_2B_3B_4\alpha_I + \gamma\kappa\sigma\theta\alpha_V + \gamma\kappa\theta B_4\alpha_1 + \phi\sigma\theta B_1\alpha_V + \phi\theta B_1B_4\alpha_I - \sigma B_1B_2B_3\alpha_V - B_1B_2B_3B_4\alpha_1)B_4)} \quad (11)$$

The Basic Reproduction Number (R_0)

The basic reproduction number is the average number of secondary infections generated from a single infectious source found only in the susceptible class. Following Driessche and Watmough and apply the next generation approach, where the matrices F; (for the new infective terms) and V; (for the transition terms) are respectively given by [13].

$$F = \left(\frac{\partial F(E_0)}{\partial x_j}\right)\left(\frac{\partial V(E_0)}{\partial x_J}\right) \quad (12)$$

$$F = \begin{bmatrix} (\alpha_I I + \alpha_V V)S \\ 0 \\ 0 \end{bmatrix} \quad V = \begin{bmatrix} (\kappa+\beta+\mu)E \\ -\kappa E + (\sigma+\mu+\omega+\theta)I + \phi R \\ -\sigma I + (\eta+\delta-g)V \end{bmatrix}$$

$$F = \begin{bmatrix} 0 & \frac{\alpha_I\Omega}{\mu} & \frac{\alpha_V\Omega}{\mu} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad V = \begin{bmatrix} \kappa+\beta+\mu & 0 & 0 \\ -\kappa & \sigma+\mu+\omega+\theta & 0 \\ 0 & -\sigma & \eta+\delta-g \end{bmatrix}$$

$$R_0 = \rho(FV^{-1})$$

Were,

$$F = \begin{bmatrix} 0 & \frac{\alpha_I\Omega}{\mu} & \frac{\alpha_V\Omega}{\mu} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

And $V^{-1} = \begin{bmatrix} \frac{1}{\kappa+\beta+\mu} & 0 & 0 \\ \frac{\kappa}{(\kappa+\beta+\mu)(\sigma+\mu+\omega+\theta)} & \frac{1}{\sigma+\mu+\omega+\theta} & 0 \\ \frac{\kappa\sigma}{(\kappa+\beta+\mu)(\sigma+\mu+\omega+\theta)(\eta+\delta-g)} & \frac{\sigma}{(\sigma+\mu+\omega+\theta)(\eta+\delta-g)} & \frac{1}{(\eta+\delta-g)} \end{bmatrix}$

$$FV^{-1} = \left[\left[\frac{\alpha_I\Omega\kappa}{\mu(\kappa+\beta+\mu)(\sigma+\mu+\omega+\theta)} + \frac{\alpha_V\Omega\kappa\sigma}{\mu(\kappa+\beta+\mu)(\sigma+\mu+\omega+\theta)(\eta+\delta-g)}, \right.\right.$$

$$\left.\left.\frac{\alpha_I\Omega}{\sigma+\mu+\omega+\theta} + \frac{\alpha_V\Omega\sigma}{(\sigma+\mu+\omega+\theta)(\eta+\delta-g)}, \frac{\alpha_V\Omega}{(\eta+\delta-g)}\right],\right.$$

$$[0,0,0],$$

$$\left.[0,0,0]\right]$$

The spectral radius for FV^{-1} gives the effective Basic reproduction number denoted by which gives

$$R_0 = \frac{\Omega\kappa(\alpha_I(\eta+\delta-g)+\alpha_V\sigma)}{\mu(\kappa+\beta+\mu)(\sigma+\mu+\omega+\theta)(\eta+\delta-g)} \quad (13)$$

Local Stability of Disease-Free Equilibrium (LDFE)

Theorem 3.1.3 The Disease-Free equilibrium of model equation is locally asymptotically stable whenever the basic reproduction number R_0 is less than one $(R_0<1)$ and when R_0 is greater than one $(R_0>1)$ it is locally asymptomatically unstable.

Proof: The Jacobian Matrix of the System will be Computed at

$$E_0 = (S_0, E_0, I_0, R_0, V_0) = \left(\frac{\Omega}{\mu}, 0, 0, 0, 0\right)$$

$$J = \begin{bmatrix} -\mu & \beta & \frac{-\alpha_I\Omega}{\mu} & \gamma & \frac{-\alpha_V\Omega}{\mu} \\ 0 & -(\kappa+\beta+\mu) & \frac{\alpha_I\Omega}{\mu} & 0 & \frac{\alpha_V\Omega}{\mu} \\ 0 & \kappa & -(\sigma+\mu+\omega+\theta) & \phi & 0 \\ 0 & 0 & \theta & -(\gamma+\mu+\phi) & 0 \\ 0 & 0 & \sigma & 0 & -(\eta+\delta-g) \end{bmatrix} \quad (14)$$

Then the characteristics equation is obtained as $|J-\lambda I|=0$ where λ is the eigenvalues

$$\lambda I = \begin{bmatrix} \lambda & 0 & 0 & 0 & 0 \\ 0 & \lambda & 0 & 0 & 0 \\ 0 & 0 & \lambda & 0 & 0 \\ 0 & 0 & 0 & \lambda & 0 \\ 0 & 0 & 0 & 0 & \lambda \end{bmatrix} \quad (15)$$

Hence $|J-\lambda I|=0$ implies that:

$$|J-\lambda I| = \begin{bmatrix} -\mu-\lambda_1 & \beta & \frac{-\alpha_I\Omega}{\mu} & \gamma & \frac{-\alpha_V\Omega}{\mu} \\ 0 & -(\kappa+\beta+\mu)-\lambda_2 & \frac{\alpha_I\Omega}{\mu} & 0 & \frac{\alpha_V\Omega}{\mu} \\ 0 & \kappa & -(\sigma+\mu+\omega+\theta)-\lambda_3 & \phi & 0 \\ 0 & 0 & \theta & -(\gamma+\mu+\phi)-\lambda_4 & 0 \\ 0 & 0 & \sigma & 0 & -(\eta+\delta-g)-\lambda_5 \end{bmatrix} \quad (16)$$

This is computed numerically by substituting the parameter values given in Table 2. i.e.

$$|J-\lambda I| = \begin{bmatrix} -0.2-\lambda_1 & 0.4 & -2 & 0.5 & -0.05 \\ 0 & -0.9-\lambda_2 & 2 & 0 & 0.05 \\ 0 & 0.3 & -0.92-\lambda_3 & 0.04 & 0 \\ 0 & 0 & 0.5 & -0.74-\lambda_4 & 0 \\ 0 & 0 & 0.02 & 0 & -0.66-\lambda_5 \end{bmatrix}$$

The eigenvalues are evaluated to be

$$\begin{bmatrix}\lambda_1\\ \lambda_2\\ \lambda_3\\ \lambda_4\\ \lambda_5\end{bmatrix}\begin{bmatrix}-0.200000000000000\\ -1.69514962037179\\ -0.118920505451593\\ -0.745428288259487\\ -0.660501585917130\end{bmatrix}$$

Hence, it is locally asymptotically stable since all the eigenvalues are all negative and have real roots.

Global Stability of Disease-Free Equilibrium (GSDFE)

Theorem 3.1.4. The disease-free equilibrium (DFE) is globally asymptotically stable if all trajectories of the system converge to the (DFE) as time approaches infinity, provided the basic reproduction number R_0 is less than one $(R_0<1)$ and when R_0 is greater than one $(R_0>1)$ it is Globally asymptomatically unstable.

Proof: Using Lyapunov Function for Equation (1to5)

$$R_0=\frac{\Omega\kappa(\alpha_I(\mu+\delta-g)+\alpha_V\sigma)}{\mu(\kappa+\beta+\mu)(\sigma+\mu+\omega+\theta)(\eta+\delta-g)} \tag{20}$$

$$\frac{R_0}{1}=\frac{\Omega\kappa(\alpha_I(\mu+\delta-g)+\alpha_V\sigma)}{\mu(\kappa+\beta+\mu)(\sigma+\mu+\omega+\theta)(\eta+\delta-g)}$$

$$\kappa(\alpha_I(\eta+\delta-g)+\alpha_V))=\frac{R_0\mu(\kappa+\beta+\mu)(\sigma+\mu+\omega+\theta)(\eta+\delta-g)}{\Omega}$$

Using infected compartment for DFE, Let L denote the Lyapunov function which is given by

$$L=AE+BI+CV \tag{21}$$

$$L'=AE'+BI'+CV' \tag{22}$$

$$L'=A[(\alpha_I I+\alpha_V V)S-(\kappa+\beta+\mu)E]+B[\kappa E-(\sigma+\mu+\omega+\theta)I]+C[\sigma I-(\eta+\delta-g)V]$$

$$E'=-A(\kappa+\beta+\mu)+B\kappa$$

$$I'=A\alpha_I-B(\sigma+\mu+\omega+\theta)+C\sigma$$

$$V'=A\alpha_V S-C(\eta+\delta-g)$$

$$A=(\eta+\delta-g)$$

$$B=\frac{\alpha_I S(\eta+\delta-g)+\alpha_V S\sigma}{\sigma+\mu+\omega+\theta}$$

$$C=\alpha_V S$$

$$L'=\left[\frac{\alpha_I\Omega(\eta+\delta-g)\kappa+\alpha_V\Omega\sigma\kappa}{\mu(\sigma+\mu+\omega+\theta)}-(\eta+\delta-g)(\kappa+\beta+\mu)\right]E \tag{23}$$

$$L'=\left[\frac{\Omega[R_0\mu(\kappa+\beta+\mu)(\sigma+\mu+\omega+\theta)(\eta+\delta-g)}{\Omega\mu(\sigma+\mu+\omega+\theta)}-(\eta+\delta-g)(\kappa+\beta+\mu)\right]E \tag{24}$$

$$L'=[R_0A-A]E$$

$$L'=(R_0-1)AE$$

$$L'<0$$

Implying that $R_0<0$

Local Stability of Endemic Equilibrium

Theorem 3.1.5: If $R_0>1$ the endemic equilibrium of the system is locally asymptotically stable.

Proof: Linearizing the Jacobian Matrix of System of E^*

$$J(E^*)=\begin{bmatrix}(-\alpha_I I^*-\alpha_V V^*)-\mu & \beta & -\alpha_I S^* & \gamma & -\alpha_V S^*\\ (\alpha_I I^*+\alpha_V V^*) & -B_1 & \alpha_I S^* & 0 & \alpha_V S^*\\ 0 & \kappa & -B_2 & \phi & 0\\ 0 & 0 & \theta & -B_3 & 0\\ 0 & 0 & \sigma & 0 & -B_4\end{bmatrix} \tag{26}$$

$$J(E^*)=\begin{bmatrix}(-\alpha_I I^*-\alpha_V V^*)-\mu-\lambda & \beta & -\alpha_I S^* & \gamma & -\alpha_V S^*\\ (\alpha_I I^*+\alpha_V V^*) & -B_1-\lambda & \alpha_I S^* & 0 & \alpha_V S^*\\ 0 & \kappa & -B_2-\lambda & \phi & 0\\ 0 & 0 & \theta & -B_3-\lambda & 0\\ 0 & 0 & \sigma & 0 & -B_4-\lambda\end{bmatrix} \tag{27}$$

$$\lambda^5+(\alpha_I I+\alpha_V V+B_1+B_2+B_3+B_4+\mu)\lambda^4+(\alpha_I I+\alpha_V V+B_1+B_2+B_3+\mu)B_4+(\alpha_I I+\alpha_V V+B_1+B_2+\mu)B_3$$
$$+(B_1 I+B_2 I-\beta I-S\kappa)\alpha_I+(V\alpha_V+\mu+B_2)B_1+(V\alpha_V+\mu)B_2-V\beta\alpha_V-\theta\phi)\lambda^3+((\alpha_1 I+\alpha_V V+B_1+B_2+\mu)B_3$$
$$+(B_1 I+B_2 I-\beta I-S\kappa)\alpha_I+(V\alpha_V+\mu+B_2)B_1+(V\alpha_V+\mu)B_2-V\beta\alpha_V-\theta\phi)B_4$$
$$+((B_1 I+B_2 I-\beta I-S\kappa)\alpha_I+(V\alpha_V+\mu+B_2)B_1+(V\alpha_V+\mu)B_2-V\beta\alpha_V)B_3$$
$$+(B_1B_2 I-B_2\beta I-\phi\theta I-S\mu\kappa)\alpha_I+((V\alpha_V+\mu)B_2-\theta\phi)B_1-V\beta B_2\alpha_V+(-S\kappa\sigma-V\phi\theta)\alpha_V-\mu\phi\theta)\lambda^2+$$
$$(((B_1 I+B_2 I-\beta I-S\kappa)\alpha_I+(V\alpha_V+\mu+B_2)B_1+(V\alpha_V+\mu)B_2-V\beta\alpha_V)B_3+$$
$$(B_1B_2 I-B_2\beta I-\phi\theta I-S\mu\kappa)\alpha_I+((V\alpha_V+\mu)B_2-\theta\phi)B_1-V\beta B_2\alpha_V-\phi\theta(V\alpha_V+\mu))B_4$$
$$+((B_1B_2 I-B_2\beta I-S\mu\kappa)\alpha_I+(V\alpha_V+\mu)B_2B_1-\alpha_V(S\kappa\sigma+V\beta B_2))B_3$$
$$-(-\beta\phi+\gamma\kappa+\phi B_1)\theta\alpha_I I-\phi\theta(V\alpha_V+\mu)B_1-\alpha_V(-\theta(\beta\phi-\gamma\kappa)V+S\mu\kappa\sigma)\lambda+(((B_1B_2 I-B_2\beta I-S\mu\kappa)\alpha_I$$
$$+((V\alpha_V+\mu)B_1-V\beta\alpha_V)B_2)B_3-((B_1\phi I-\phi\beta I+\gamma\kappa I)\alpha_I+\phi(V\alpha_V+\mu)B_1-\alpha_V V(\beta\phi-\gamma\kappa))\theta)B_4-S\kappa\mu\sigma B_3\alpha_V)$$

$$C_0=1$$

$$C_1=(\alpha_I I+\alpha_V V+B_1+B_2+B_3+B_4+\mu)$$

$$C_2=(\alpha_I I+\alpha_V V+B_1+B_2+B_3+\mu)B_4+(\alpha_I I+\alpha_V V+B_1+B_2+\mu)B_3$$
$$+(B_1 I+B_2 I-\beta I-S\kappa)\alpha_I+(V\alpha_V+\mu+B_2)B_1+(V\alpha_V+\mu)B_2-V\beta\alpha_V-\theta\phi)$$

$$C_3=(\alpha_1 I+\alpha_V V+B_1+B_2+\mu)B_3$$
$$+(B_1 I+B_2 I-\beta I-S\kappa)\alpha_I+(V\alpha_V+\mu+B_2)B_1+(V\alpha_V+\mu)B_2-V\beta\alpha_V-\theta\phi)B_4$$
$$+((B_1 I+B_2 I-\beta I-S\kappa)\alpha_I+(V\alpha_V+\mu+B_2)B_1+(V\alpha_V+\mu)B_2-V\beta\alpha_V)B_3$$
$$+(B_1B_2 I-B_2\beta I-\phi\theta I-S\mu\kappa)\alpha_I+((V\alpha_V+\mu)B_2-\theta\phi)B_1-V\beta B_2\alpha_V+(-S\kappa\sigma-V\phi\theta)\alpha_V-\mu\phi\theta$$

$$C_4=(B_1 I+B_2 I-\beta I-S\kappa)\alpha_I+(V\alpha_V+\mu+B_2)B_1+(V\alpha_V+\mu)B_2-V\beta\alpha_V)B_3+$$
$$(B_1B_2 I-B_2\beta I-\phi\theta I-S\mu\kappa)\alpha_I+((V\alpha_V+\mu)B_2-\theta\phi)B_1-V\beta B_2\alpha_V-\phi\theta(V\alpha_V+\mu))B_4$$
$$+((B_1B_2 I-B_2\beta I-S\mu\kappa)\alpha_I+(V\alpha_V+\mu)B_2B_1-\alpha_V(S\kappa\sigma+V\beta B_2))B_3$$
$$-(-\beta\phi+\gamma\kappa+\phi B_1)\theta\alpha_I I-\phi\theta(V\alpha_V+\mu)B_1-\alpha_V(-\theta(\beta\phi-\gamma\kappa)V+S\mu\kappa\sigma$$

$$C_5=((B_1B_2 I-B_2\beta I-S\mu\kappa)\alpha_I+((V\alpha_V+\mu)B_1-V\beta\alpha_V)B_2)B_3$$
$$-((B_1\phi I-\phi\beta I+\gamma\kappa I)\alpha_I+\phi(V\alpha_V+\mu)B_1-\alpha_V V(\beta\phi-\gamma\kappa))\theta)B_4-S\kappa\mu\sigma B_3\alpha_V$$

Where $\alpha_I=\frac{C_h}{1+\alpha_T}$, and $\alpha_V=\frac{C_V}{V+P}$

According to Hurwitz Criterion, when $R_0>1$ the endemic equilibrium E^* of system is locally asymptotically stable if $C_2C_1-C_3C_0>0$ and $C_4C_3C_2C_1-C_5C_1^2-C_4^2C_0>0$

Global Stability of Endemic Equilibrium

Theorem 3.1.6: Let En^* be the unique positive equilibrium point of the system (6), If $R_0>1$ then the endemic equilibrium En^* of the system is globally asymptotically stable.

Proof: Using the Lyapunov Function:

$$L(S^*E^*I^*R^*V^*)=\begin{cases}\left(S-S^*-S^*\ln\left(\frac{S^*}{S}\right)\right)+\left(E-E^*-E^*\ln\left(\frac{E^*}{E}\right)\right)+\left(I-I^*-I^*\ln\left(\frac{I^*}{I}\right)\right)\\ +\left(R-R^*-R^*\ln\left(\frac{R^*}{R}\right)\right)+\left(V-V^*-V^*\ln\left(\frac{V^*}{V}\right)\right)\end{cases}$$

The Derivative of Along the Solution of the System is Direct:

$$\frac{dL}{dt}=\left(\frac{S-S^*}{S}\right)\frac{dS}{dt}+\left(\frac{E-E^*}{E}\right)\frac{dE}{dt}+\left(\frac{I-I^*}{I}\right)\frac{dI}{dt}+\left(\frac{R-R^*}{R}\right)\frac{dR}{dt}+\left(\frac{V-V^*}{V}\right)\frac{dV}{dt}$$

$$\frac{dL}{dt}=\begin{cases}\left(\frac{S-S^*}{S}\right)[\Omega N-(\alpha_I I+\alpha_V V)S-\mu S+\gamma R+\beta E]\\ +\left(\frac{E-E^*}{E}\right)[(\alpha_I I+\alpha_V V)S-(\kappa+\beta+\mu)E]\\ +\left(\frac{I-I^*}{I}\right)[\kappa E-(\sigma+\mu+\omega+\theta)I+\phi R]\\ +\left(\frac{R-R^*}{R}\right)[\theta I-(\gamma+\mu+\phi)R]\\ +\left(\frac{V-V^*}{V}\right)[\sigma I-(\eta+\delta-g)V]\end{cases}$$

By Expansion:

$$\frac{dL}{dt}=\begin{cases}\Omega-(\alpha_I I+\alpha_V V)S-\mu S+\gamma R+\beta E-\frac{\Omega S^*}{S}+(\alpha_I I+\alpha_V V)S^*+\mu S^*-\frac{\gamma RS^*}{S}-\frac{\beta ES^*}{S}\\ +(\alpha_I I+\alpha_V V)S-(\kappa+\beta+\mu)E-\frac{(\alpha_I I+\alpha_V V)SE^*}{E}+(\kappa+\beta+\mu)E^*\\ +\kappa E-(\sigma+\mu+\omega+\theta)I+\phi R-\frac{\kappa EI^*}{I}+(\sigma+\mu+\omega+\theta)I^*-\frac{\phi RI^*}{I}\\ +\theta I-(\gamma+\mu+\phi)R-\frac{\theta IR^*}{R}+(\gamma+\mu+\phi)R^*\\ +\sigma I-(\eta+\delta-g)V-\frac{\sigma IV^*}{V}+(\eta+\delta-g)V^*\end{cases}$$

By Simplification:

$$\frac{dL}{dt}=\begin{cases}\Omega-\mu S-\frac{\Omega S^*}{S}+(\alpha_I I+\alpha_V V)S^*+\mu S^*-\frac{\gamma RS^*}{S}-\frac{\beta ES^*}{S}\\ -\mu E-\frac{(\alpha_I I+\alpha_V V)SE^*}{E}+(\kappa+\beta+\mu)E^*\\ -(\mu+\omega)I-\frac{\kappa EI^*}{I}+(\sigma+\mu+\omega+\theta)I^*-\frac{\phi RI^*}{I}\\ -\mu R-\frac{\theta IR^*}{R}+(\gamma+\mu+\phi)R^*\\ -(\eta+\delta-g)V-\frac{\sigma IV^*}{V}+(\eta+\delta-g)V^*\end{cases}$$

Suppose, $\frac{dL}{dt}=P-M$ (28)

Where P denotes the positive terms and M denotes the negative terms so that

$$P=\Omega N+(\alpha_I I+\alpha_V V)S^*+\mu S^*+(\kappa+\beta+\mu)E^*+(\sigma+\mu+\omega+\theta)I^*+(\gamma+\mu+\phi)R^*+(\eta+\delta-g)V^*$$

The largest invariant set is $\left\{(S^*,E^*,I^*,R^*,V^*)\in\theta:\frac{dL}{dt}=0\right\}$ a unit set of En^*

Where En^* is the endemic equilibrium signifying that the endemic is globally asymptotically stable.

Sensitivity Analysis

To evaluate the sensitivity of the basic reproduction number with respect to each of the following parameters $\Omega,\kappa,\alpha_I,\mu,\delta,g,\alpha_V,\sigma,\beta,\omega,\theta$ and η by calculating each value applying the derivative-based method, which reflects the bond between every parameter and the basic reproduction number R_0 Inserting the value of each parameter into equation and solving them using;

$$X_x^{R_0}=\frac{\partial R_0}{\partial x}\cdot\frac{x}{R_0} \quad (29)$$

Table 3: Parameter Sensitivity Index

Parameter	Sensitivity Expression	Sensitivity Value	Sensitivity Index
Ω	1	1	+
K	$\frac{\beta+\mu}{B_1}$	0.7	+
σ	$-\frac{\sigma((-\eta-\delta+g)\alpha_I+\alpha_V(\theta+\mu+\omega))}{((-\eta-\delta+g)\alpha_I-\alpha_V\sigma)B_2}$	-0.01	-
α_I	$\frac{\alpha_I B_4}{\alpha_I B_4+\alpha_V\sigma}$	0.99	+
α_V	$\frac{\sigma\alpha_V}{\alpha_I B_4+\alpha_V\sigma}$	0.008	+
η	$-\frac{\eta\alpha_V\sigma}{(-\eta-\delta+g)((-\eta-\delta+g)\alpha_I-\alpha_V\sigma)}$	-0.005	-
δ	$-\frac{\delta\alpha_V\sigma}{(-\eta-\delta+g)((-\eta-\delta+g)\alpha_I-\alpha_V\sigma)}$	-0.0003	-
g	$\frac{g\alpha_V\sigma}{(-\eta-\delta+g)((-\eta-\delta+g)\alpha_I-\alpha_V\sigma)}$	0.0005	+
μ	$\frac{-3\mu^2+(-2\beta-2\kappa-2\omega-2\sigma-2\theta)\mu-(\beta+\kappa)(\sigma+\omega+\theta)}{B_1B_2}$	-1	-
β	$-\frac{\beta}{B_1}$	-0.4	-
ω	$-\frac{\omega}{B_2}$	-0.2	-
θ	$-\frac{\theta}{B_2}$	-0.5	-

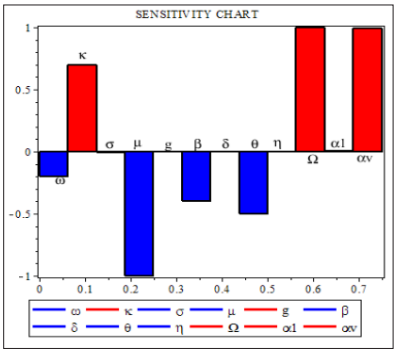

Figure 2: The Graph of Sensitivity Analysis

The parameter sensitivity index uses the derivative-based local method as described in Table 3 which displays that the parameter μ , κ , g , α_I , and α_V have direct relationship with the basic reproduction number R_0 and it can increase the rate at which cholera progresses in the population. While σ , ω , β , δ , θ , Ω, and η have inverse relationship with R_0, here this parameters decreases the rate at which cholera spreads. However, reducing effective contact rate between infected human and susceptible individuals as well as providing adequate treatment, practicing personal hygiene alongside disinfecting the environment regularly beginning from keeping their environment clean and also restricting infected individual direct access to public food and water this could significantly reduce the R_0 . The sensitivity analysis findings shows that disinfection of environment and treatment can completely lower the basic reproduction number; they effectively help in disease control [14,15].

Numerical Simulations

To evaluate the theoretical calculation of the model, the numerical simulation of the model (6) is carried out by differential transformation method using a set of parameter values given in Table 2.

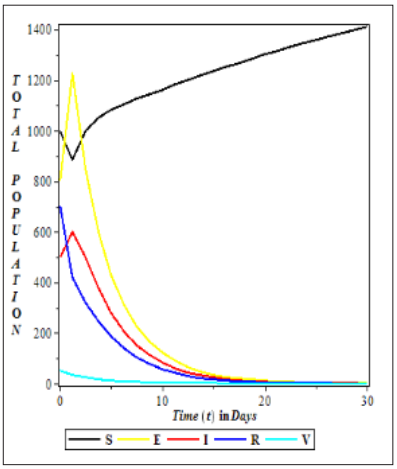

Figure 3: Total Population

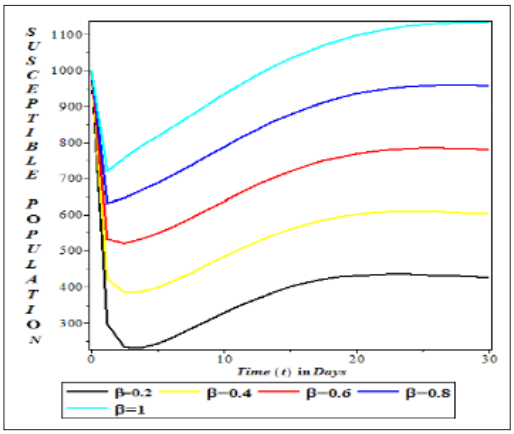

Figure 4: Susceptible Population for β = 0.2, 0.4, 0.6, 0.8 and 1

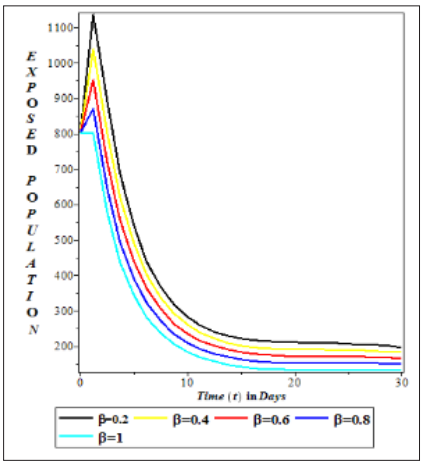

Figure 5: Population of Exposed Individuals for β= 0.2, 0.4, 0.6, 0.8 and 1

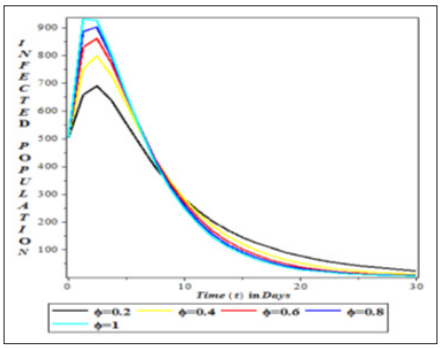

Figure 6: Population of Infected Individuals for ϕ= 0.2, 0.4, 0.6, 0.8 and 1

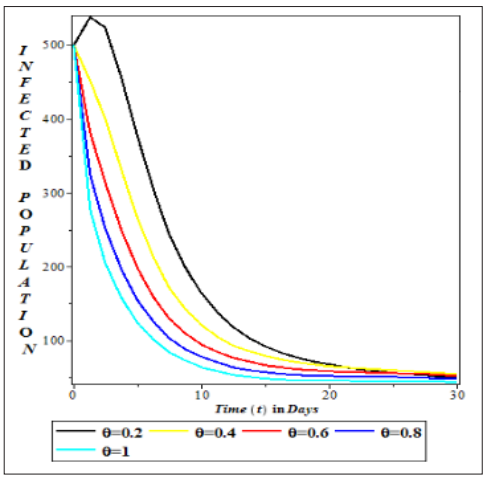

Figure 7: Population of Infected Individuals for θ= 0.2, 0.4, 0.6, 0.8 and 1

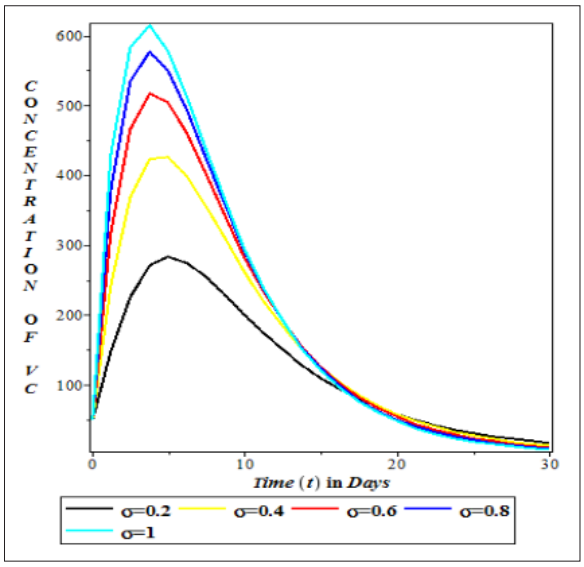

Figure 8: Population of Concentration of Vibrio Cholera for σ= 0.2, 0.4, 0.6, 0.8 and 1

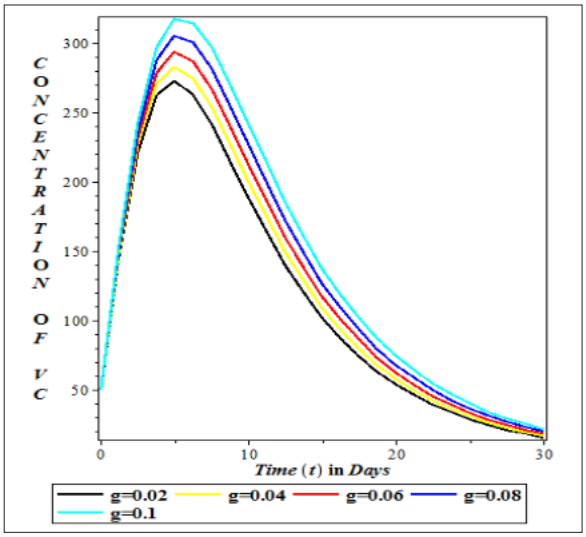

Figure 9: Population of Concentration of Vibrio Cholera for g= 0.2, 0.4, 0.6, 0.8 and 1

Discussion

Figure 3 highlights the significance of implementing control measures across the entire population. It demonstrates that both the exposed and infected groups would diminish as long as individuals comply with public health guidelines, while the number of susceptible individuals would rise as a result of their collective efforts. Figure 4 illustrates the effects of natural recovery on those who are susceptible. It clarifies that as the rate of natural recovery escalates, there is a corresponding increase in the susceptible population.

Figure 5 illustrates the significant impact that natural recovery has on individuals who are exposed to disease. It highlights the critical role of a robust immune system in combating illnesses. As individuals enhance their immune responses, the transmission rate of Vibrio cholera is likely to diminish, leading to a reduction in the basic reproduction number of the pathogen. This suggests that, over time, the disease could ultimately be eradicated.

Figure 6 illustrates how the rate of re-infection contributes to the growth of the infected compartment, subsequently enhancing the transmission of cholera within the population. This phenomenon positively affects the basic reproduction number. In contrast, Figure 6 highlights the critical role of treatment within the infected compartment, showing that as the treatment rate approaches 1, the spread of cholera diminishes and ultimately ceases.

Figure 7 highlights the necessity of decreasing the rate at which infectious individuals contribute to environmental contamination, as their presence significantly accelerates the spread of cholera. Therefore, isolating and treating this group is essential to mitigate the disease's advancement.

Figure 8 details the growth rate of Vibrio cholera within the population if stringent measures are not enacted. The data presented in the graph underscores the disease's capacity to cause fatalities within a brief timeframe if no preventive actions are taken.

Figure 9 emphasizes the critical need for environmental disinfection to avert the global dissemination of Vibrio cholera.

In conclusion; this study highlights the importance of public health interventions in controlling cholera. Adhering to treatment and sanitation measures reduces infections and enhances recovery, lowering the basic reproduction number and aiding disease eradication. Reinfection sustains transmission, emphasizing the need for continuous monitoring. Effective treatment and environmental sanitation play crucial roles in preventing cholera outbreaks. An integrated approach combining treatment, immunity enhancement, and sanitation is essential for long-term disease control and eradication.

Declaration

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