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MATHEMATICAL MODELING OF CHOLERA TRANSMISSION IN EPIDEMIC AND ENDEMIC SETTINGS WITH CONTROL STRATEGIES

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Abstract. One of the most serious health issues in the world today is cholera, particularly in poorer nations with poor access to clean water. We have examined the distribution of cholera in both endemic and epidemic settings in this article. We develop the mathematical model of the dynamics of cholera transmission known as $SEIRH$ with controls. The time-dependent control mechanisms (vaccine, water purification, and sanitation) that govern the disease's transmission and management were incorporated into the model. It was possible to acquire the potential key measure R_0, a threshold value used to forecast the prognosis of a disease. The stability of the endemic disease equilibrium point (EEP) and the cholera-free equilibrium point ($DFEP$) was examined. If R_0 is less than 1, then $DFEP$ is locally asymptotically stable (LAS), and EEP is globally asymptotically stable (GAS) when R_0 is greater than 1. The impact of control measures on virus spread was investigated, and the optimal control value that minimizes the objective function was also investigated using Pontryagin's maximum principle. The model simulation shows that the methods used have a positive impact on public health by lowering morbidity and mortality. Cholera incidence can be considerably decreased by effective prevention and control measures, which will lower rates of morbidity and mortality.

1. Introduction

Cholera is a serious diarrheal disease caused by the bacterium Vibrio cholerae [1]. According to the WHO and CDC, the disease is spread through contaminated water or food and occurs in areas with poor sanitation and limited access to clean water. Cholera can cause severe acute watery diarrhea and rapid dehydration due to electrolyte imbalance, which can lead to life-threatening complications if not treated quickly. The review in [2] explored and identified the factors contributing

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to the re-emergence of cholera in Africa and the response strategies used to contain the problem, and collected the limited data needed to inform disease control and eradication policies. They reviewed 65 valid articles reporting risk factors for recurrent cholera outbreaks, endemicity and response strategies and found that trans-boundary migration, ecological reservoirs, socioeconomic factors, climate change and political instability are factors contributing to persistent and recurrent outbreaks of cholera in Africa. The review also identified specific response strategies and modeling approaches to help prevent and limit the impact of these outbreaks. Wolff et al. [3] described the theory of cholera risk and protection, developed inclusion criteria, searched for and selected studies, assessed the quality of the evidence, and established associations between seven hypothesized protective factors and eight factors. We conducted a systematic review. Exposure to water, sanitation and hygiene (WASH) to promote (risk factors) or stop (protective factors) the spread of cholera. To better understand the complex epidemiology of cholera, several mathematical models have been formulated and published. For example, the model shown in [7] specifically considers the concentration of Vibrio cholerae in the water supply in a typical SIR kinetic model. The research carried out in [6] examined the maintenance of disease-free and endemic balance to study the epidemiology of cholera complex epidemic and endemic diseases. This study demonstrates the real-world application of the model by examining a recent cholera outbreak in Zimbabwe. We also present numerical simulation results to verify the analytical predictions. Therefore, our goal is to determine how to suppress the spread of the disease using control measures (vaccination, water purification, sanitation) as a function of time. In addition, Pontryagin's maximum principle is used to investigate the impact of control measures on virus spread, how control levels reduce spread, and the optimal control value that minimizes the objective function. Recent studies have shown that immunity can be lost over weeks to months [15], [19].

1.1. **Literature Review.** In developing countries, diarrheal diseases are a leading cause of infant mortality due to lack of access to clean drinking water and sanitation [21]. Lemos-Paiao et al, in [20] proposed and analyzed the susceptible-infectious-isolated-recovered ($SIQR$) model under the assumption that infected individuals remain isolated during the treatment period. The susceptible-infected-recovered (SIR) epidemiological model proposed by [15] considers two classes of bacterial concentrations (highly infected and low infected) and two classes of infectious individuals (asymptomatic and symptomatic). Here, the authors use optimal control theory, parameter sensitivity analysis, and numerical modeling to compare the cost-effective trade-off of multiple intervention methods for two endemic populations. Wang and Modnak [22] also consider an SIR-type model using Vibrio cholerae concentrations in the environment. The model includes three control measures: vaccination, medical care and water purification. Balance point stability analysis is performed when the control is given a constant value. They also study a general cholera model with time-dependent control, prove the existence of a solution to the optimal control problem, and derive the necessary optimality conditions based on Pontryagin's maximum principle. In a

SIR type model, the authors [13] incorporate therapy, immunization, quarantine, and public health education campaigns as control methods. The concentration classes of the bacteria are also considered by the model. They analyze the potential community benefits of these measures by comparing reproductive rates resulting from education, vaccination, and treatment and combined reproductive rates with baseline reproductive rates. The Lyapunov functional technique is used to analyze equilibrium stability. The main indicators of cholera prevalence and reinfection are examined in this article along with stability analyses and preventative measures like "water purification and sanitation merged as a single control" and "vaccination" strategies that use Pontryagin's maximum principle method to lower infection rates and population exposure.

1.2. **Assumptions of the Model.**

(1) People of all ages living in areas with limited access to clean and safe water are susceptible to cholera.
(2) People who live in unsanitary areas are prone to cholera.
(3) People who drink contaminated beverages, ice or bottled water are equally susceptible to cholera.
(4) In cholera endemic region, everyone is susceptible.
(5) vaccinated infected individuals after recovery will not get infected for at least two years.

2. Materials and Methods

2.1. **Model Formulation.** The population under study at time t is defined as $N(t)$. It is classified into five subsections: Susceptible (S) population, Exposed (E) population, Infectious (I) population, Recovered (R) and Vibro cholera population $H(t)$. Therefore, cholera model in this study is a combined system of human populations and the environmental component $H(t)$ and total population of species is classified into: The human population N_h and $N_b(t)$ are given as:

$$N_h(t) = S(t) + E(t) + I(t) + R(t).$$

$$N_b(t) = H(t).$$

In figure 1, the susceptible people (S), will move to the exposed compartment (E) updating the number of persons in the exposed class $\lambda = \alpha_1\left(\frac{H}{\kappa+H} + I\right)$, Out of this exposed ones, $\alpha_2 E$ individuals are moved from E compartment to the infectious class (I) and $\alpha_3 I$ are moved out of the infectious compartment to the recovery group. The class H is the *Vibrio Cholerae* population concentration in the aquatic environment at a time t. The infectious individuals shed *Vibrios cholerae* at a rate ρ and by asexual reproduction, *Vibrio cholerae* bacteria reproduce at the rate a. The population of the pathogenic bacteria is reduced by proper sanitation and or by infectious class compliance to hygiene principles at a rate τ. It is noted that an individual must consume at least the concentration, κ, of Vibrios cholera equivalent to an amount that increases the possibility of being infected to about 50% if they are to contact the infection. Overall, the human

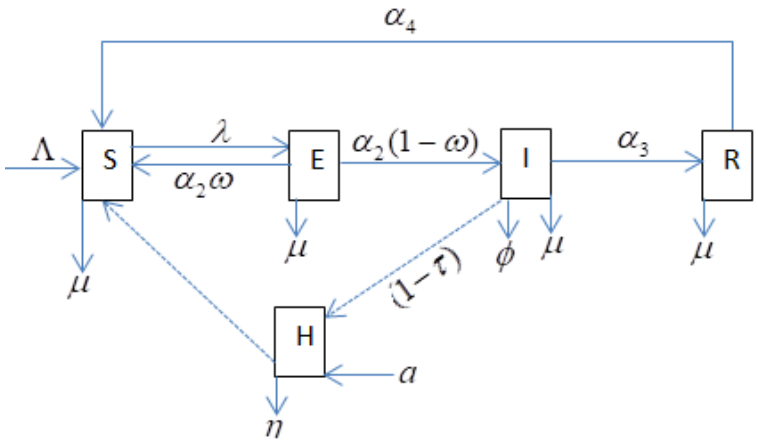

FIGURE 1. Model Diagram

populations is reduced by uniform natural death at a rate μ and the bacteria dies naturally at the rate η.

TABLE 1. Meaning of parameters

Parameters	Description
α_0	Recruitment rate of human.
μ	Natural death rate
α_1	Exposure rate to contaminated water.
ω	Exposed rate among individuals who avoid contracting the virus due to a strong immunity
α_2	Exposure rate of individuals to the cholera outbreak.
α_3	Rate of recovery of infected persons
η	Death rate induced of vibrio cholera.
ϕ	Disease induced death rate of humans
ρ	Contribution of infected persons to the number of vibriola cholera in water environment.
κ	Concentration of vibrio cholera in water bodies pave the way for cholera transmission.
τ	Infectious class compliance to hygienic principles.
α_4	The rate at which individual susceptible rather than fully recovering
a	Birth rate of Vibrio cholera.

2.2. **Model Equation.** Given the dynamics described in Figure 1, the following system of ordinary differential equations, with non-negative initial conditions,

describes the dynamics of Vibrio cholera bacteria:

$$\left.\begin{array}{rcl} \frac{dS}{dt} &=& \alpha_0 - \lambda S + \alpha_2\omega E - \mu S + \alpha_4 R,\ S(0) = S_0 > 0, \\ \frac{dE}{dt} &=& \lambda S - (\alpha_2 + \mu)E,\ E(0) = E_0 \geqslant 0, \\ \frac{dI}{dt} &=& \alpha_2(1-\omega)E - (\alpha_3 + \phi + \mu)I,\ I(0) = I_0 \geqslant 0, \\ \frac{dR}{dt} &=& \alpha_3 I - (\alpha_4 + \mu)R,\ R(0) = R_0 \geqslant 0, \\ \frac{dH}{dt} &=& \rho(1-\tau)I - (\eta - a)H,\ H(0) = H_0 \geqslant 0. \end{array}\right\} \tag{2.1}$$

3. Existence and Uniqueness

Theorem 3.1. *: Let $R^5_+ \in \Omega$ denote the region of feasibility for the model equation (2.1). If the model equations (2.1) is continuous, then the existence and uniqueness of the state variables $(S(t), E(t), I(t), R(t), H(t)) \in \Omega$ exist for all $t \geq 0$.*

Proof. From model equation (2.1)

$$\begin{array}{rcl} \frac{dS}{dt} &=& \alpha_0 - \lambda S + \alpha_2\omega E - \mu S + \alpha_4 R. \\ \frac{dS}{dt} &=& \alpha_0 - \alpha_1\left(\frac{H}{\kappa + H} + I\right)S + \alpha_2\omega E - \mu S + \alpha_4 R. \\ w(t,S) = \frac{dw}{ds} &=& -\alpha_1\left(\frac{H}{\kappa + H} + I\right) - \mu \end{array}$$

Where $w(t,s)$ and its derivatives is continuous. The application of Cauchy-Lipschitz condition is employed to show existence and uniqueness of the model equation.

$$\begin{array}{rcl} \mid w(t,s_1) - w(t,s_2) \mid &=& \mid -\alpha_1\left(\frac{H}{\kappa + H} + I\right)s_1 - \mu s_1 + \alpha_1\left(\frac{H}{\kappa + H} + I\right)s_2 + \mu s_2 \mid \\ &=& \mid -\alpha_1\left(\frac{H}{\kappa + H} + I\right) - \mu \mid\mid s_1 - s_2 \mid \\ &\leq& \mid -1 \mid\mid \alpha_1\left(\frac{H}{\kappa + H} + I\right) + \mu \mid\mid s_1 - s_2 \mid \\ &\leq& \mid \alpha_1\left(\frac{H}{\kappa + H} + I\right) + \mu \mid\mid s_1 - s_2 \mid \\ &\leq& L \mid s_1 - s_2 \mid \end{array}$$

Where $L = \alpha_1\left(\frac{H}{\kappa+H} + I\right) + \mu$ is the Lipschitz constant and following the same argument, $w(t,E), w(t,I), w(t,R), w(t,H)$ and their derivatives are continuous respectively. Therefore, the model equations (2.1) all satisfied the Lipschitz condition; hence there exist a unique solution to the all state variables $(S(t), E(t), I(t), R(t), H(t))$ for all $t \geq 0$ □

3.1. Basic Reproduction Number, R_0.

The reproduction number, R_0 of the model at ε_0, is calculated by the application of next-generation matrix [5]. E, I and H denote the infected classes, then the values of F and V represented the

new infection terms and the transmission terms given as:
Writing the model in matrix form:

$$\frac{dX}{dt} = f(x) - v(x),$$

Where

$$f(x) = \begin{pmatrix} (\lambda)S \\ 0 \\ 0 \end{pmatrix}, \; v(x) = \begin{pmatrix} (\alpha_2+\mu)E \\ -\alpha_2(1-\omega)E + (\alpha_3+\phi+\mu)I \\ -\rho(1-\tau)I + (\eta-a)H \end{pmatrix}.$$

The Jacobian of $f(x)$ and $v(x)$ at the disease-free equilibrium point $\varepsilon_0 = \left(\frac{alpha_0}{\mu}, 0, 0, 0, 0\right)$ and $|V| = (\alpha_2+\mu)(\alpha_3+\phi+\mu)(\eta-a)$.
On evaluating the dominant eigenvalue $\rho(FV^{-1})$ of the matrix FV^{-1}, the reproduction number (R_0) is the spectral radius of the matrix FV^{-1} given as:
Therefore

$$R_0 = \frac{\alpha_0\alpha_1\alpha_2(1-\omega)[\kappa(\eta-a)+\rho(1-\tau)]}{\kappa\mu(\alpha_2+\mu)(\alpha_3+\phi+\mu)(\eta-a)}. \tag{3.1}$$

4. Stability analysis

Here, the application of Stability analysis used to determine the behavior of the model over time.

4.1. **Local Stability of Cholera Free Equilibrium Point.**

Theorem 4.1. *The disease free equilibrium (DFE) Point ϵ^0 is locally asymptotically stable (LAS) whenever $R_0 < 1$ but unstable whenever $R_0 > 1$.*

Proof. To analyze the stability at (DFE), we find the Jacobian matrix J of the model equation 2.1 evaluated at ϵ^0.

$$(J_{\epsilon^0} - Iy) =$$

$$\begin{pmatrix} -(\mu)-\lambda & \alpha_2\omega & 0 & \alpha_4 & 0 \\ 0 & -(\alpha_2+\mu)-\lambda & 0 & 0 & 0 \\ 0 & \alpha_2(1-\omega) & -(\alpha_3+\phi+\mu)-\lambda & 0 & 0 \\ 0 & 0 & \alpha_3 & -(\alpha_4+\mu)-\lambda & 0 \\ 0 & 0 & \rho(1-\tau) & 0 & (a-\eta)-\lambda \end{pmatrix} = 0$$

$(-(\mu)-y)(-(\alpha_2+\mu)-y)(-(\alpha_3+\phi+\mu)-y)(-(\alpha_4+\mu)-y)((a-\eta)-y) = 0$
Solving this polynomial, the eigenvalues are given by

$$\begin{aligned} \lambda_1 &= -\mu \\ \lambda_2 &= -(\alpha_2+\mu) \\ \lambda_3 &= -(\alpha_3+\phi+\mu) \\ \lambda_4 &= -(\alpha_4+\mu) \\ \lambda_5 &= -(\eta-a) \end{aligned}$$

If $R_0 < 1$ and the eigenvalues are all non positive, hence ϵ^0 is locally asymptotically stable otherwise unstable. □

4.2. **Global Stability of the cholera Equilibrium point.**

Theorem 4.2. *Suppose that $R_0 < 1$, then the cholera-free state which occurs at the point ε^0 for the model equation (2.1) is globally asymptomatic stable otherwise unstable at $R_0 > 1$.*

Proof. The global asymptomatic behavior of the model (2.1) is carried out by the application of a suitable Lyapunov function by considering the derivatives of those infectious compartments of the model equations, i.e $\frac{dE}{dt}, \frac{dI}{dt}, \frac{dH}{dt}$.
Setting, $\frac{dI}{dt} = 0 = \frac{dE}{dt}$

$$\frac{dI}{dt} = \alpha_2(1-\omega)E - (\alpha_3+\phi+\mu)I = 0$$

Then

$$E = \frac{(\alpha_3+\phi+\mu)I}{\alpha_2(1-\omega)} \tag{4.1}$$

$$\frac{dE}{dt} = \alpha_1\left(\frac{H}{\kappa+H}+I\right)S - (\alpha_2+\mu)E = 0$$

Then

$$E = \frac{\alpha_1\left(\frac{H}{\kappa+H}+I\right)S}{(\alpha_2+\mu)} \tag{4.2}$$

Equating equations (4.2)and (4.1), we have

$$I = \frac{\alpha_1\alpha_1(1-\omega)\frac{H}{\kappa+H}S}{(\alpha_2+\mu)(\alpha_3+\phi+\mu) - \alpha_1\alpha_2S(1-\omega)} \tag{4.3}$$

$$\frac{dH}{dt} = \rho(1-\tau)I - (\eta-a)H. \tag{4.4}$$

Substituting I in equation (4.3) into equation (4.4) at $\varepsilon^0 = \left(\frac{\alpha_0}{\mu}, 0, 0, 0, 0\right)$.

$$\frac{dH}{dt} = \frac{\rho(1-\tau)\alpha_1\alpha_2S(1-\omega)\frac{H}{\kappa+H}}{(\alpha_2+\mu)(\alpha_3+\phi+\mu) - \alpha_1\alpha_2S(1-\omega)} - (\eta-a)H$$

Setting $\frac{H}{\kappa+H} \leq \frac{H}{\kappa}$

$$\begin{aligned}
\frac{dH}{dt} &\leq \frac{\alpha_0\alpha_1\alpha_2\rho(1-\tau)(1-\omega)H}{\kappa\mu(\alpha_2+\mu)(\alpha_3+\phi+\mu) - \kappa\alpha_1\alpha_2(1-\omega)} - (\eta-a)H \\
&\leq \frac{\alpha_0\alpha_1\alpha_2\rho(1-\tau)(1-\omega)H - (\eta-a)H\kappa\mu(\alpha_2+\mu)(\alpha_3+\phi+\mu) + (\eta-a)\kappa\alpha_0\alpha_1\alpha_2(1-\omega)}{\kappa\mu(\alpha_2+\mu)(\alpha_3+\phi+\mu) - \kappa\alpha_0\alpha_1\alpha_2(1-\omega)} \\
&\leq \frac{H\alpha_0\alpha_1\alpha_2(1-\omega)\left[\rho(1-\tau)+\kappa(\eta-a)\right] - (\eta-a)H\kappa\mu(\alpha_2+\mu)(\alpha_3+\phi+\mu)}{\kappa\mu(\alpha_2+\mu)(\alpha_3+\phi+\mu) - \kappa\alpha_0\alpha_1\alpha_2(1-\omega)}
\end{aligned}$$

Putting R_0 from equation (3.1), then

$$\begin{aligned}\frac{dH}{dt} &\leq \frac{[R_0-1]\left(H(\eta-a)\kappa\mu(\alpha_2+\mu)(\alpha_3+\phi+\mu)\right)}{\kappa\mu(\alpha_2+\mu)(\alpha_3+\phi+\mu)-\kappa\alpha_0\alpha_1\alpha_2(1-\omega)}\\ \frac{dH}{dt} &< \frac{[R_0-1]\left(H(\eta-a)\kappa\mu(\alpha_2+\mu)(\alpha_3+\phi+\mu)\right)}{\kappa\mu(\alpha_2+\mu)(\alpha_3+\phi+\mu)}\end{aligned}$$

Therefore,

$$\frac{dH}{dt} = [R_0-1]\left((\eta-a)H\right)$$

Whenever $R_0 < 1$, $\frac{dH}{dt}$ turned out to be negative semi-definite while its negative definite when $R_0 > 1$. According to LaSalle's variance principle, this means that the no-cholera equilibrium is globally symptomatically stable. □

4.3. **Endemic Equilibrium.** Given the model system 2.1; there exist a unique endemic equilibrium point denoted by $\varepsilon^* = (S^*, E^*, I^*, R^*, H^*)$; representing the equilibrium point with all positive components, $(S^*, E^*, I^*, R^*, H^*) \neq (0,0,0,0,0)$. We therefore calculate the endemic equilibrium point by setting the five derivatives equal to zero and finding solutions of the equations give:

$$S^* = -\frac{\alpha_0(\alpha_2+\mu)(\alpha_3+\phi+\mu)(\alpha_4+\mu)}{\alpha_2\omega\lambda(\alpha_3+\phi+\mu)(\alpha_4+\mu)+\alpha_4\alpha_3(1-\omega)-\lambda+\mu)(\alpha_2+\mu)(\alpha_3+\phi+\mu)(\alpha_4+\mu)}$$

$$E^* = -\frac{\lambda\alpha_0(\alpha_3+\phi+\mu)(\alpha_4+\mu)}{\alpha_2\omega\lambda(\alpha_3+\phi+\mu)(\alpha_4+\mu)+\alpha_4\alpha_3(1-\omega)-\lambda+\mu)(\alpha_2+\mu)(\alpha_3+\phi+\mu)(\alpha_4+\mu)}$$

$$I^* = -\frac{\alpha_0\alpha_2\lambda(1-\omega)(\alpha_4+\mu)}{\alpha_2\omega\lambda(\alpha_3+\phi+\mu)(\alpha_4+\mu)+\alpha_4\alpha_3(1-\omega)-\lambda+\mu)(\alpha_2+\mu)(\alpha_3+\phi+\mu)(\alpha_4+\mu)}$$

$$R^* = -\frac{\alpha_0\alpha_2\alpha_3\lambda(1-\omega)}{\alpha_2\omega\lambda(\alpha_3+\phi+\mu)(\alpha_4+\mu)+\alpha_4\alpha_3(1-\omega)-\lambda+\mu)(\alpha_2+\mu)(\alpha_3+\phi+\mu)(\alpha_4+\mu)}$$

$$H^* = -\frac{\alpha_0\alpha_2\rho\lambda(1-\tau)(1-\omega)(\alpha_4+\mu)}{(a-\eta)[\alpha_2\omega\lambda(\alpha_3+\phi+\mu)(\alpha_4+\mu)+\alpha_4\alpha_3(1-\omega)-\lambda+\mu)(\alpha_2+\mu)(\alpha_3+\phi+\mu)(\alpha_4+\mu)]}$$

4.4. **Local Stability of Cholera endemic equilibrium.**

Theorem 4.3. *Whenever $R_0 > 1$, then the cholera endemic equilibrium (CEE) at ϵ^* is locally asymptotically stable (LAS).*

Proof. To analyze the stability at (DFE), we find the Jacobian matrix J of the model equation 2.1 evaluated at ϵ^*.

$$(J_{\epsilon^*} - I\lambda) = \begin{pmatrix} -(A+\mu)-\lambda & \alpha_2\omega & -\alpha_1 S^* & \alpha_4 & -B \\ A & -C-\lambda & \alpha_1 S^* & 0 & B \\ 0 & \alpha_2(1-\omega) & -D-\lambda & 0 & 0 \\ 0 & 0 & \alpha_3 & -E-\lambda & 0 \\ 0 & 0 & \rho(1-\tau) & 0 & F-\lambda \end{pmatrix} = 0$$

Where
$A = \alpha_1\left(\frac{H^*}{\kappa+H^*} + I^*\right), B = \left(\frac{\alpha_1\kappa S^*}{(\kappa+H^*)^2}\right), C = (\alpha_2+\mu), D = (\alpha_3+\phi+\mu), E = (\alpha_4+\mu), F = (a-\eta)$
The polynomial equation for $(J_{\epsilon^*} - I\lambda)$ i calculated thus

$$a_5\lambda^5 + a_4\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 \tag{4.5}$$

Where

$$a_5 = 1$$

$$a_4 = (A+\mu) + (\alpha_2+\mu) + (\alpha_3+\phi+\mu) + (\alpha_4+\mu) + (\eta - a)$$

$$\begin{aligned} a_3 &= (A+\mu)\Big((\alpha_4+\mu) + (\alpha_3+\phi+\mu) + (\eta-a)\Big) + (\alpha_4+\mu)\Big((\alpha_2+\mu) + (\alpha_3+\phi+\mu) + (\eta \\ &- a)\Big) + (\alpha_2+\mu)\Big((\alpha_3+\phi+\mu) + (\eta-a)\Big) + (\alpha_3+\phi+\mu)(\eta-a) + \alpha_1\alpha_2S^*(\omega-1) - \alpha_2\omega A \end{aligned}$$

$$\begin{aligned} a_2 &= (A+\mu)(\alpha_4+\mu)\Big((\alpha_3+\phi+\mu) + (\alpha_2+\mu) + (\eta-a)\Big) + (\alpha_3+\phi+\mu)(\alpha_4+\mu)\Big((\alpha_2+\mu) \\ &+ (\eta-a)\Big) + (\alpha_2+\mu)(\eta-a)\Big((\alpha_4+\mu) + (\alpha_3+\phi+\mu) + (A+\mu)\Big) + (A+\mu)(\alpha_3+\phi+\mu)(\eta \\ &- a) + \alpha_1\alpha_2(\omega-1)S^*\Big((A+\mu) + (\alpha_4+\mu) + (a-\eta)\Big)\frac{\alpha_1\alpha_3\rho\kappa S^*}{(\kappa+H^*)^2}(1-\omega)(1-\tau) - \alpha_2\omega A\Big((\alpha_3 \\ &+ \phi+\mu) + (\alpha_4+\mu) + (\eta-a)\Big) + \alpha_1\alpha_2(\omega-1)AS^* \end{aligned}$$

$$\begin{aligned} a_1 &= (\alpha_4+\mu)(\alpha_3+\phi+\mu)(A+\mu)\Big((\alpha_2+\mu) + (\eta-a)\Big) + (\alpha_2+\mu)(\alpha_4+\mu)\Big((\alpha_3+\phi+\mu)(\eta-a) \\ &+ (A+\mu)(\eta-a)\Big) + \alpha_1\alpha_2S^*(\omega-1)\Big((A+\mu)(\alpha_4+\mu) + (A+\mu)(a-\eta) + (\alpha_4+\mu)(a-\eta)\Big) \\ &+ \frac{\alpha_1\alpha_2\rho\kappa S^*(1-\omega)(1-\tau)}{(\kappa+H^*)^2}\Big((A+\mu) + (\alpha_4+\mu)\Big) + \alpha_2\omega A\Big((a-\eta)(\alpha_3+\phi+\mu) + (\alpha_4 \\ &+ \mu)(a-\eta) - (\alpha_3+\phi+\mu)(\alpha_4+\mu)\Big) + \alpha_1\alpha_2S^*(A)(\omega-1)\Big((\alpha_4+\mu) + (\eta-a)\Big) + \frac{\alpha_1\alpha_2\rho\kappa S^*}{(\kappa+H^*)^2} \\ &(\omega-1)(1-\tau)A + \alpha_3\alpha_2(1-\omega)A \end{aligned}$$

$$\begin{aligned} a_0 =& \alpha_2\alpha_3\alpha_4(1-\omega)(a-\eta)A + \frac{\rho\alpha_1\alpha_2\kappa S^*}{(\kappa+H^*)^2}A(1-\omega)(\alpha_4+\mu)(1-\tau) + (A+\mu)(\alpha_2+\mu)(\alpha_3+\phi+\mu) \\ &(\alpha_4+\mu)(\eta-A) + (A+\mu)(\alpha_4+\mu)(\eta-a)(1-\omega)\alpha_1\alpha_2S^* + \frac{\kappa\rho\alpha_1\alpha_2S^*}{(\kappa+H^*)^2}(A+\mu)(\alpha_4+\mu) \\ &(1-\omega)(\tau-1) + \alpha_2\omega(A)(\alpha_2+\phi+\mu)(\alpha_4+\mu)(\eta-a) + \alpha_1\alpha_2A(1-\omega)(\alpha_4+\mu)(\eta-a) \end{aligned}$$

$$H_n = \begin{bmatrix} a_1 & a_3 & a_3 & 0 & 0 \\ a_0 & a_2 & a_4 & 0 & 0 \\ 0 & a_2 & a_3 & a_5 & 0 \\ 0 & {}_{a_0} & a_2 & a_4 & 0 \\ 0 & 0 & a_1 & a_3 & a_5 \\ 0 & 0 & a_0 & a_2 & a_4 \\ . & . & . & . & . \end{bmatrix} \tag{4.6}$$

The equation (4.5) is asymptotically stable given that all the principal minors of (4.6) is positive and non-zero by application of Routh-Hurwitz stability criterion. Then on simplification, the outcome of the principal minors are: $\delta_1 = a_1$, $\delta_2 = a_1a_2 - a_0a_3$, $\delta_3 = a_1(a_2a_3 - a_1a_4)$, $\delta_4 = a_1a_2(a_3a_4 - a_2a_5) - a_1a_4(a_1a_4 - a_0a_5) - a_0a_3(a_3a_4 - a_2a_5) + a_0a_5(a_1a_4 - a_0a_5)$, $\delta_5 = a_5\delta_4$. If $\delta_5 > 0$, $\delta_4 > 0$, $\delta_3 > 0$, $\delta_2 > 0$, $\delta_1 > 0$ and $R_0 > 1$ then by of Routh-Hurwitz criterion, the endemic equilibrium ϵ^* of the system (2.1) is locally asymptotically stable. □

5. Optimal Control Problem

The model equation (2.1) is modified by introducing controls u and v which stands for "water treatment, sanitation, and hygiene practices" and "Vaccination strategies" .

$$\left.\begin{array}{rcl} \frac{dS}{dt} &=& \alpha_0 - u\lambda S + \alpha_2\omega v - \mu S + \alpha_4 R. \\ \frac{dE}{dt} &=& u\lambda S - (\alpha_2 + \mu)E. \\ \frac{dI}{dt} &=& \alpha_2 v(1-\omega)E - (\alpha_3 + \phi + \mu)I. \\ \frac{dR}{dt} &=& \alpha_3 I - (\alpha_4 + \mu)R. \\ \frac{dH}{dt} &=& \rho(1-\tau)I - (\eta - a)H. \end{array}\right\} \tag{5.1}$$

The optimal control problem is aimed at calculating the level of controls which limits the spread and transmission of cholera in endemic region. Here, we sort for the optimal values u and v^* of the control u, and v along time t such that the associated state trajectories S^*, E^*, I^*, R^* and H^* are solutions of the model (5.1) equations and $u^*(.)$, and $v^*(.)$ minimizes the objective functional given as

$$J(.) = \min_{u,v} \int_0^T I(t) - (u^2 + v^2)dt.$$

The objective function above contains the population of infectious with the severity of the side effects of the control strategies 'u and v'. Hence, setting $0 \leq u < U_{max}$ and $0 \leq v < U_{max}$. Appliction of Pontryagins maximum principle to the Hamiltonian was used to infer the conditions necessary for the optimal

control, where

$$\begin{aligned} L = I - (u^2 + v^2) &+ \lambda_S\{\alpha_0 - u\alpha_1\left(\frac{H}{\kappa + H} + I\right)S + \alpha_2\omega vE - \mu S + \alpha_4 R\} \\ &+ \lambda_E\{u\alpha_1\left(\frac{H}{\kappa + H} + I\right)S - (\alpha_2 + \mu)E\} \\ &+ \lambda_I\{\alpha_2 v(1-\omega)E - (\alpha_3 + \phi + \mu)I\} \\ &+ \lambda_R\{\alpha_3 I - (\alpha_4 + \mu)R\} \\ &+ \lambda_H\{\rho(1-\tau)I + (a - \eta)H\}. \end{aligned}$$

Theorem 5.1. *Given the optimal controls u and v and solutions S^*, E^*, I^*, R^* and H^* of the control system that maximizes $J(u, v)$ there exist adjoint variables valid $\frac{\partial \lambda_j}{\partial t} = -\frac{\partial L}{\partial t}$. The transversality conditions*

$$\lambda_S(T) = \lambda_E(T) = \lambda_I(T) = \lambda_R(T) = \lambda_H(T) = 0.$$

The optimality condition is given by $\frac{\partial L(.)}{\partial u} = \frac{\partial L(.)}{\partial v} = 0$. Furthermore, the optimal control

$$\begin{aligned} u^* &= \min\left\{u_{\max}, \max(0, 0.5(\alpha_1 S(\lambda_E - \lambda_S)\left(\frac{H}{\kappa + H} + I\right)\right\}, \\ v^* &= \min\left\{u_{\max}, \max(0, 0.5((\lambda_S - \lambda_I)(\alpha_2\omega E) + \lambda_I\alpha_2 E)\right\}. \end{aligned}$$

Proof. Using the Hamiltonian, we obtain the adjoint variables $\lambda_S, \lambda_E, \lambda_I, \lambda_R$, and λ_H by solving the system:

$$\dot{\lambda}_S = -\frac{\partial L(.)}{\partial S}, \dot{\lambda}_E = -\frac{\partial L(.)}{\partial E}, \dot{\lambda}_I = -\frac{\partial L(.)}{\partial I}, \dot{\lambda}_R = -\frac{\partial L(.)}{\partial R}, \dot{\lambda}_H = -\frac{\partial L(.)}{\partial H}.$$

Thus,

$$\begin{aligned} \dot{\lambda}_S &= u\alpha_1\left(\frac{H}{\kappa + H} + I\right)(\lambda_S - \lambda_E) + \lambda_S\mu, \\ \dot{\lambda}_E &= (\lambda_I - \lambda_S)\alpha_2\omega v + \lambda_E(\alpha_2 + \mu) - \lambda_I\alpha_2 v, \\ \dot{\lambda}_I &= -1 + (\lambda_S - \lambda_E)u\alpha_1 S + \lambda_I\left((\alpha_3 + \phi + \mu) - \rho(1-\tau)\right) - \lambda_R\alpha_3, \\ \dot{\lambda}_R &= \lambda_R(\alpha_4 + \mu) - \lambda_S\alpha_4, \\ \dot{\lambda}_H &= (\lambda_S - \lambda_E)\left(u\alpha_1\frac{\kappa}{(\kappa + H)^2}\right)S + \lambda_H(\eta - a). \\ \lambda_S(T) &= \lambda_E(T) = \lambda_I(T) = \lambda_R(T) = \lambda_H(T) = 0. \end{aligned}$$

The optimal controls u^* and v^* are derived from the stationary conditions

$$\frac{\partial L(.)}{\partial u} = \frac{\partial L(.)}{\partial v} = 0.$$

For the optimization problem, the optimal control is characterized in compact form as:

$$\begin{aligned} u^* &= \min\left\{u_{\max}, \max(0, 0.5(\alpha_1 S(\lambda_E - \lambda_S)\left(\frac{H}{\kappa + H} + I\right)\right\}, \\ v^* &= \min\left\{u_{\max}, \max(0, 0.5((\lambda_S - \lambda_I)(\alpha_2 \omega E) + \lambda_I \alpha_2 E)\right\}. \end{aligned}$$

□

6. Result

7. Numerical Simulation

To simulate the state and adjoint equations, we therefore apply the numerical approach of the Forward-Backward Sweep method and MATLAB script written to implement this method through Runge-Kutta fourth order method. A MAT-

Table 2. Meaning of parameters

Parameters	values	Reference
α_0	9.13×10^{-3}	[11]
μ	0.025	[12]
α_1	0.02	[18]
ω	(00.9]	Assumed
α_2	0.054	Assumed
α_3	$0.2/day$	[13]
η	$0.33/day$	[14]
ϕ	$0.012/day$	[17]
ρ	0.0031	Assumed
κ	10^6(cell/ml)	[16]
τ	0.85	[14]
α_4	0.001096	[15]
a	0.028	[12]

LAB script was written to solve the system of ODEs using the ode45 solver, plots the results. The values of the parameters and initial conditions of the model are adjusted as needed to analyze the model at different control level and observe the epidemic scenarios.

8. Discussion of Result

Cholera is a growing concern worldwide, especially in developing countries where access to clean water is limited. Looking at the graphs below, the cholera infection rate in figures 3, 4, 5, 6 and 7 denote the group with controls. The percentage increase in control measures shows a significant decrease in the number of exposed and the number of infected individuals, that is in figure 2, $u = 0$ and $v = 0$; figure 3, $u = 0.2(20\%)$ and $v = 0.3(30\%)$; figure 4, $u = 0.3(30\%)$ and $v = 0.4(40\%)$; figure 5, $u = 0.4(40\%)$ and $v = 0.6(60\%)$; figure 6, $u = 0.6(60\%)$ and $v = 0.75(75\%)$ and figure 7, $u = 0(0\%)$ and $v = 0.4(40\%)$. Figure (7) shows

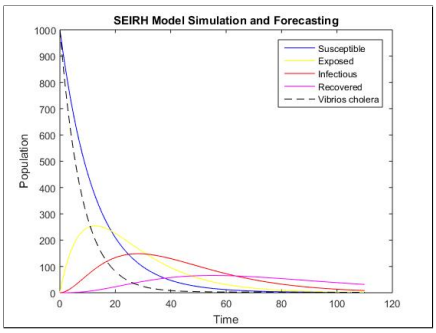

FIGURE 2. Graph of the model population without controls

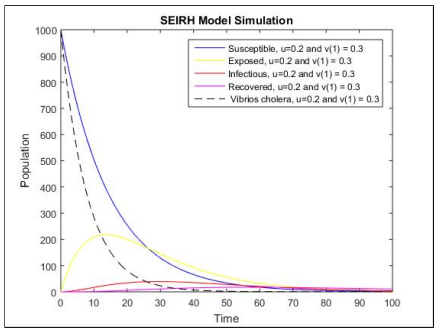

FIGURE 3. Graph of the model population with control

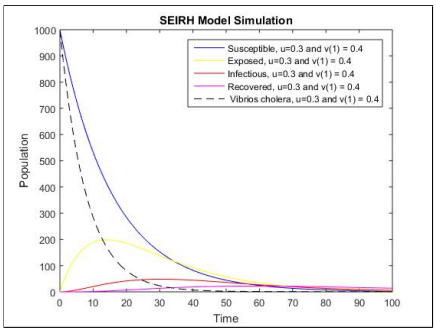

FIGURE 4. Graph of the model population with control

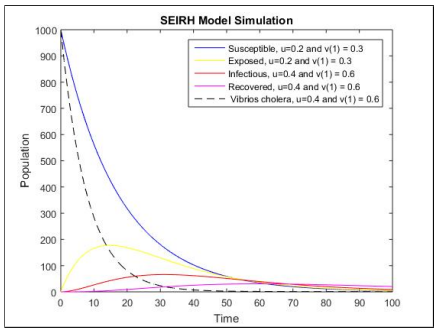

FIGURE 5. Graph of the model population with control

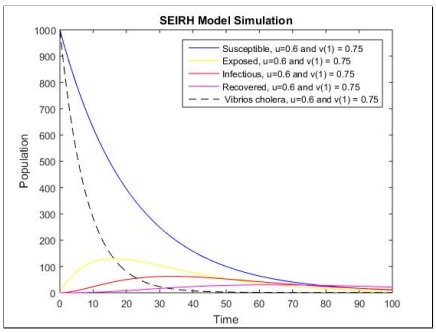

FIGURE 6. Graph of the model population with control

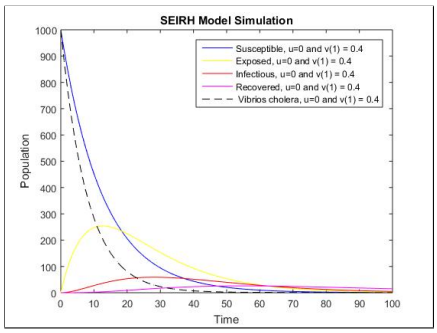

FIGURE 7. Graph of the model population with control

a significant reduction in the number of exposed and infected individuals compared to other indicators with minimal controls. Levels of exposed and infected individuals in the control plots are lower than in figure (2) with no controls at all. This highlights the importance of control measures in controlling the spread of cholera. The effectiveness of a single control strategy in figure 7, shows a significant reduction only in cholera patients. Figures 3, 4, 5 and 6 show the efficacy of the joint control measures in curtailing the spread of the infection in the population. Cholera cannot be eradicated or controlled without strict control measures. We have learned from this study and the results that educating the public about the various ways in which cholera can spread is one of the most crucial aspects in stopping its spread. If we respond to this work's call, we can eradicate or curtail cholera in our society; if not, cholera outbreaks will still occasionally occur in developing countries. Strictly speaking, it is evident that cholera can re-infect recovered people if they are exposed to the disease again. This occurs because cholera leaves the body without a lasting immunity.

9. Conclusion

The epidemiology and management of cholera in endemic and endemic locations are reviewed in this article. According to the $SEIRH$ data, fewer persons are exposed to or contract cholera when control measures are strengthened. This study demonstrates that the efficacy of cholera prevention tactics aligns with suggested preventative actions. In conclusion, studies have demonstrated that immunization, water filtration, and hygienic practices can effectively reduce cholera and lessen its impact. By boosting the immunity of those who have been vaccinated against cholera, vaccinations significantly reduce the disease's potential to spread, as do appropriate sanitation and water purification practices. In order to manage cholera epidemics and eventually eradicate the illness, a thorough strategy must be put in place. According to the model's findings, which indicate that sanitation and vaccination significantly lowers infection rates, and expanding access to clean water in cholera-prone areas is important.

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N. N. Ezieke: Project management, visualization, and resources.

Authors' Conflicts of interest. I hereby attest that the information I have disclosed is accurate and that I am not aware of any other circumstances involving actual, potential, or apparent conflicts of interest. S.N. Aloke on behalf of the authors.

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