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Modeling Cholera Transmission Dynamics and Control Strategies in Human, Animal and Environmental Systems

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Abstract

Cholera, an acute waterborne diarrheal disease caused by *Vibrio cholerae*, continues to pose a significant public health challenge globally, particularly in regions characterized by inadequate access to safe drinking water, poor sanitation infrastructure, and limited healthcare resources. Despite extensive public health interventions aimed at reducing transmission, recurrent outbreaks persist, highlighting the need for more comprehensive approaches to understanding the complex epidemiological mechanisms underlying disease spread. In this study, an enhanced cholera transmission model is developed by incorporating fish as an ecological vector within the disease transmission cycle, thereby extending conventional cholera models that primarily focus on human and environmental compartments. The model is formulated within a One Health framework, integrating interactions among human populations, aquatic reservoirs, and fish to capture the interconnected dynamics of disease transmission across human, animal, and environmental systems. The qualitative and quantitative properties of the model were investigated using standard mathematical epidemiological techniques. The next-generation matrix approach was employed to derive the basic reproduction number $(\Re_0)$, which serves as a threshold indicator for disease persistence and outbreak potential. Analytical results demonstrate that the disease-free equilibrium remains stable when $\Re_0 < 1$, leading to eventual disease elimination, whereas endemic persistence and recurrent outbreaks occur when $\Re_0 > 1$. Sensitivity analysis identified direct human-to-human transmission, exposure to contaminated water sources, and fish-mediated ecological interactions as the most influential parameters governing disease propagation. Numerical simulations further assessed the effectiveness of various control measures and revealed that integrated intervention strategies combining treatment programs with sustained public health awareness campaigns significantly reduce disease prevalence. These findings underscore the importance of incorporating environmental and animal reservoirs into cholera transmission models and provide valuable insights for designing effective, multidisciplinary, and evidence-based cholera control policies within a One Health framework.

Keywords

Impact,
Public health campaign,
Stability,
Transmission
Vibrio cholerae.

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1.0 INTRODUCTION

Cholera, an acute diarrheal illness caused by the bacterium *Vibrio cholerae*, remains a persistent and severe public health threat, especially in regions with limited access to clean water and adequate sanitation [24]. Africa is experiencing its worst cholera outbreak in 25years, with 300,000 cases with 7000 deaths, with 2.3% case fatality rate [17]. Traditional models of cholera transmission have primarily focused on two primary routes: direct human-to-human contact and indirect transmission through contaminated water sources, where the bacterium can persist and proliferate [7,12].

However, a growing body of ecological and epidemiological evidence suggests that this classic dichotomy is incomplete. *V. cholerae* is a natural inhabitant of aquatic ecosystems, and many studies have now confirmed that it can colonize a wide range of aquatic inhabitants (copepods, fish, water birds, etc.) [11,5]. Fish can act as mobile reservoirs, harboring the bacteria in their intestines, gills, and other tissues, often asymptomatically [19,3]. Transmission to humans occurs through the handling, preparation, and consumption of undercooked or raw fish, a dietary staple in many cholera-endemic regions [10,13]. This zoonotic pathway represents a critical, yet often overlooked, component of the cholera transmission cycle.

Recognizing this complexity necessitates a more holistic approach to disease modeling. The "One Health" paradigm, which integrates human, animal, and environmental health, provides an ideal framework for understanding such interconnected systems [25].

To address this gap, this study develops a mathematical model that conceptualizes cholera transmission through a One Health lens.

This research intends to formulate a model that incorporates zoonotic transmission of *V. cholerae* from fish to humans and analyze the model to identify critical parameters driving transmission, thereby informing targeted and effective multi-sectoral control strategies. Which in turn contribute to a more nuanced understanding of cholera dynamics and support the development of comprehensive intervention policies that extend beyond the human domain.

2.0 MATERIALS AND METHODS

2.1 Model Formulation

The mathematical model for cholera transmission is developed by extending the model of Dere and Oladapo (2024). Our model incorporated the aquatic animal (fish) population, which accounts for the zoonotic transmission. The work of [3,19] reported that fish and other aquatic organisms are reservoirs and vectors of the bacteria, which can be transmitted through contact and consumption of raw and undercooked seafood. Looking at the current health issues, which are always in line with one health initiative, to look at the disease in tandem with human-environment-animal.

Our model incorporates treatment and public health campaigns. The model contains eight 8 variables, which are susceptible, exposed, infected, recovered human population, fish population, which comprises susceptible, infected and recovered populations and the concentration of *Vibrio cholerae* in water. The

susceptible population is generated either through birth or through immigration. They acquire infection through consumption, coming in contact with an infected human or animal population, and move to the exposed class at the rate $(1-C_e)\left(\frac{\beta_{eh}B}{K+B}+\beta_{hh}I_h+\beta_{fh}I_f\right)$ and the fish population get infected at the rate $\left(\frac{\beta_{ef}B}{K+B}(1-C_e)+\beta_{ff}I_f\right)$. We assume since the bacteria kills the cells line in fishes and it cause illness in fish; it can lead to death.

Table 1: Variables of Cholera Disease Model

Variables	Description
$S_h(t)$	Susceptible Human population at time t,
$E_h(t)$	Exposed human population at time t,
$I_h(t)$	Infected human population at time t,
$R_h(t)$	Recovered human population at time t,
$S_f(t)$	Susceptible fish population at time t,
$I_f(t)$	Infected fish population at time t,
$R_f(t)$	Recovered fish population at time t,
$B(t)$	Bacteria *(Vibrio cholerae)* population at time t,

Table 2: Parameters of Cholera Disease Model

Parameters	Description	Value	Reference
Π_h	Recruitment rate due to birth and immigration in human	0.00913	[12]
Π_f	Recruitment rate due to birth and immigration in fish	0.2881394595	[21]
β_{eh}	Rate of exposure of susceptible human to contaminated water by bacteria (*Vibrio cholera*)	0.75	[23]
β_{hh}	Rate of contact susceptible human with infected human	0.2143	[2]
β_{ef}	Rate of exposure of fish to contaminated water by bacteria (*Vibrio cholera*)	0.9433743078	[21]
β_{ff}	Rate of contact with infected fish with susceptible fish	0.9433743078	[21]
β_{fh}	Rate of contact susceptible human with infected fish	0.00714285	[11]
τ_1	Recovery rate due to adherence to preventive measures in the exposed human compartment	0.003	[14]
τ_f	Recovery rate due to the treatment of infected aquatic animals	0.05	Estimated
τ_2	Recovery rate due to the treatment of infected human compartment	0.2	[15]

δ_h	Rate of Infection-induced death in humans	0.012	[20]
δ_f	Rate of Infection-induced death in fish	0.2881394595	[21]
K	Bacteria load (concentration)	10^6	[18]
μ_h	Natural death rate in human population	0.025	[8]
μ_f	Natural death rate in fish population	0.2881394595	[21]
α	Rate of loss of temporally acquired immunity due to treatment from the recovered compartment	0.003	[15]
ω	Contribution rate of each exposed person to the bacteria (*Vibrio cholera)* population	10	[15]
a	Contribution rate of each infected person to the bacteria (*Vibrio cholera)* population	10	[15]
σ	Death rate of bacteria due to environmental	0.33	[22]
ϕ	Progression rate from exposed to infected human compartment	0.054	[4]
x	Contribution rate of each infected fish to the bacteria (*Vibrio cholera)* population	0.1	Estimated
f	Growth rate of the bacteria	0.028	[8]
z	Fishing rate	0.02	low-scale fishing rate (Estimated)
C_e	Public health campaign	0.39	[2]

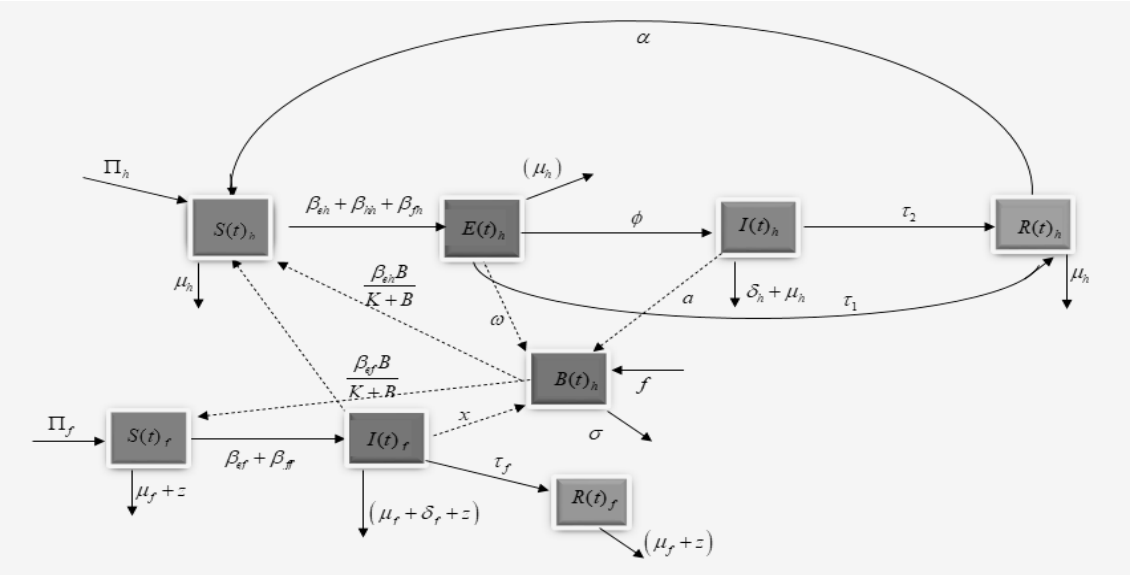

Figure 1: Schematic Diagram of Control-Free Cholera Disease Dynamics.

2.2 Cholera Disease Model Equations

The system of ordinary differential equations (ODEs) which describes the dynamics of cholera transmission across the three populations, deduced from the assumptions:

$$\left.\begin{aligned}
\frac{dS_h(t)}{dt} &= \Pi_h + \alpha R_h - \left(\lambda_h + \mu_h\right) S_h \\
\frac{dE_h(t)}{dt} &= \lambda_h S_h - \left(\phi + \tau_1 + \mu_h\right) E_h \\
\frac{dI_h(t)}{dt} &= \phi E_h - \left(\tau_2 + \mu_h + \delta_h\right) I_h \\
\frac{dR_h(t)}{dt} &= \tau_1 E_h + \tau_2 I_h - \left(\alpha + \mu_h\right) R_h \\
\frac{dS_f(t)}{dt} &= \Pi_f - \left(\lambda_f + \mu_f + z\right) S_f \\
\frac{dI_f(t)}{dt} &= \lambda_f S_f - \left(\tau_f + \mu_f + \delta_f + z\right) I_f \\
\frac{dR_f(t)}{dt} &= \tau_f I_f - \left(\mu_f + z\right) R_f \\
\frac{dB(t)}{dt} &= \omega E_h + a I_h + x I_f - \left(\sigma - f\right) B
\end{aligned}\right\} \quad (1)$$

Where;

$$\lambda_h = \left(1 - C_e\right)\left(\frac{\beta_{eh} B}{K + B} + \beta_{hh} I_h + \beta_{fh} I_f\right) \quad (2)$$

$$\lambda_f = \left(\frac{\beta_{ef} B}{K + B}\left(1 - C_e\right) + \beta_{ff} I_f\right) \quad (3)$$

2.3 MODEL ANALYSIS

Invariant Region

Theorem 1: The solutions of the proposed Cholera model are feasible for all $t > 0$, if they enter and remain in the invariant region Ω, which is given by:

$$\Omega = \left\{\begin{aligned}
&\left(S_h, E_h, I_h, R_h, S_f, I_f, R_f B\right) : S_h(t) > 0, E_h(t) > 0, I_h(t) > 0, R_h(t) > 0, S_f(t) > 0, I_f(t) > 0, R_f(t) > 0, B(t) > 0, \\
&N_h(t) \le \frac{\Pi_h}{\mu_h}, N_f(t) \le \frac{\Pi_f}{\mu_f + z} \text{ and } B(t) \le \frac{1}{\left(\sigma - f\right)}\left(\omega \frac{\Pi_h}{\mu_h} + a \frac{\Pi_h}{\mu_h} + x \frac{\Pi_f}{\left(\mu_f + z\right)}\right)
\end{aligned}\right\} \quad (4)$$

Proof:

the total rate of change in the human population with respect to time in the absence of disease induced death is obtained as:

$$\frac{dN_h}{dt} \le \Pi_h - \mu_h N_h(t) \quad (5)$$

integrate equation (5) using the integrating factor, we obtained

$$N_h(t) \le \frac{\Pi_h}{\mu_h}, \text{ as } t \to \infty \quad (6)$$

In similar manner the total rate of change in the fish population in absence of disease induced death;

$$\frac{dN_f}{dt} \le \Pi_f - \left(\mu_f + z\right) N_f(t) \tag{7}$$

Integrate equation Eq. (7) using integrating factor method.

$$N_f(t) \le \frac{\Pi_f}{\left(\mu_f + z\right)}, \text{ as t} \to \infty \tag{8}$$

For the bacteria population,

Since $N_f(t) \le \frac{\Pi_f}{\left(\mu_f + z\right)}$ and $N_h(t) \le \frac{\Pi_h}{\mu_h}$, it implies that

$$I_f(t) \le \frac{\Pi_f}{\left(\mu_f + z\right)}, I_h(t) \le \frac{\Pi_h}{\mu_h}, \text{ and } E_h(t) \le \frac{\Pi_h}{\mu_h} \tag{9}$$

Substituting for Eq. (9) into the eighth equation in Eq. (1)

$$\frac{dB(t)}{dt} \le \omega \frac{\Pi_h}{\mu_h} + a\frac{\Pi_h}{\mu_h} + x\frac{\Pi_f}{\left(\mu_f + z\right)} - \left(\sigma - f\right)B \tag{10}$$

Integrate Eq. (10) using integrating factor method;

$$B(t) \le \frac{1}{\left(\sigma - f\right)}\left(\omega\frac{\Pi_h}{\mu_h} + a\frac{\Pi_h}{\mu_h} + x\frac{\Pi_f}{\mu_f + z}\right), \text{ as t} \to \infty, \text{ where } f < \sigma$$

Then,

$$N_h(t) \le \frac{\Pi_h}{\mu_h}, N_f(t) \le \frac{\Pi_f}{\left(\mu_f + z\right)} \text{and } B(t) \le \frac{1}{\left(\sigma - f\right)}\left(\omega\frac{\Pi_h}{\mu_h} + a\frac{\Pi_h}{\mu_h} + x\frac{\Pi_f}{\left(\mu_f + z\right)}\right).$$

Thus, this shows that the model is well posed and mathematically meaningful in Ω [1], which means the solutions of the model starts and remain in Ω, for all $t > 0$.

Positivity of Solution

Theorem 2: The solutions of Cholera disease model Eq. (1) given by the set $\left\{S_h, E_h, I_h, R_h, S_f, I_f, R_f, B\right\}$ with non-negative initial conditions $S_h(0)$, $E_h(0)$, $I_h(0)$, $R_h(0)$, $S_f(0)$, $I_f(0)$, $R_f(0)$, $B(0)$ remain non-negative for all time $t > 0$.

Proof:

Then from the first equation of model system Eq. (1) we have

$$\frac{dS_h(t)}{dt} = \Pi_h + \alpha R_h - \left[\frac{\beta_{eh}B}{K+B}\left(1-C_e\right) + \beta_{hh}\left(1-C_e\right)I_h + \beta_{fh}\left(1-C_e\right)I_f + \mu_h\right]S_h$$

Let $S_h = 0$, then

$$\frac{dS_h(t)}{dt} = \Pi_h + \alpha R_h > 0 \tag{11}$$

Hence, $S_h(t) > 0$ for all $t > 0$.

In the same process, the remaining state variables can be shown that $E_h, I_h, R_h, S_f, I_f, R_f, B > 0$.

Hence, all the solutions are positive for all non-negative initial conditions.

Cholera-Free Equilibrium Point (CFE)

The Cholera-Free Equilibrium (CFE) is the state where the population is free of the infection. In the absence of infection, , $E_h = I_h = R_h = B = I_f = R_f = 0$, then the system Eq. (1) has a point referred to be Cholera-Free Equilibrium (E_o). Let, $E_0 = \{S_{h0}, E_{h0}, I_{h0}, R_{h0}, S_{f0}, I_{f0}, R_{f0}, B_0\}$ be our CFE.

We obtained the cholera-free equilibrium by equating the right hand of the equation Eq. (1) to zero and solve simultaneously;

$$E_0 = \left(\frac{\Pi_h}{\mu_h}, 0, 0, 0, \frac{\Pi_f}{\mu_f + z}, 0, 0, 0 \right) \tag{12}$$

Basic Reproduction Number

The Basic Reproduction Number $\mathfrak{R}_0$, represents the average number of new infections that a single infected individual is expected to cause in a susceptible population [15].

We obtained the matrices using the Next Generation Matrix approach.

$$F_j = \begin{bmatrix} 0 & \frac{\beta_{hh} C \Pi_h}{\mu_h} & \frac{\beta_{fh} C \Pi_h}{\mu_h} & \frac{\beta_{eh} \Pi_h}{K \mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_{ff} \Pi_h}{\mu_f + z} & \frac{\beta_{ef} \Pi_h}{K(\mu_f + z)} \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{13}$$

$$V^{-1}{}_j = \begin{pmatrix} \frac{1}{l_1} & 0 & 0 & 0 \\ \frac{\phi}{l_1 l_2} & \frac{1}{l_2} & 0 & 0 \\ 0 & 0 & \frac{1}{l_3} & 0 \\ \frac{a\phi + \omega l_2}{l_1 l_2 l_4} & \frac{a}{l_2 l_4} & \frac{x}{l_3 l_4} & \frac{1}{l_4} \end{pmatrix} \tag{14}$$

Now, the product of Equation Eq. (13) and equation Eq. (14) gives

$$FV^{-1} = \begin{pmatrix} A_1 & A_2 & A_3 & A_4 \\ 0 & 0 & 0 & 0 \\ A_5 & A_6 & A_7 & A_8 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \tag{15}$$

Where:

$$A_1=\left(\frac{\beta_{hh}\Pi_h\phi C}{\mu_h l_1 l_2}+\frac{\beta_{eh}\Pi_h\left(a\phi+\omega l_2\right)C}{K\mu_h l_1 l_2 l_4}\right), A_2=\left(\frac{\beta_{hh}\Pi_h\phi C}{\mu_h l_1 l_2}+\frac{\beta_{eh}\Pi_h\left(a\phi+\omega l_2\right)C}{K\mu_h l_1 l_2 l_4}\right), A_3=\left(\frac{\beta_{fh}\Pi_h C}{\mu_h l_3}+\frac{\beta_{eh}x\Pi_h C}{K\mu_h l_3 l_4}\right), A_4=\frac{\beta_{eh}\Pi_h}{K\mu_h l_4},$$

$$A_5=\frac{\beta_{ef}\Pi_f\left(a\phi+\omega l_2\right)}{K\left(\mu_f+z\right)l_1 l_2 l_4}, A_6=\frac{a\beta_{ef}\Pi_f}{K\left(\mu_f+z\right)l_2 l_4}, A_7=\frac{\beta_{ff}\Pi_f}{\left(\mu_f+z\right)l_3}+\frac{\beta_{ef}\Pi_f x}{K\left(\mu_f+z\right)l_3 l_4}, A_8=\frac{\beta_{ef}\Pi_f}{K\left(\mu_f+z\right)l_4}$$

Next, obtaining the eigenvalues of FV^{-1}, given the characteristic equation $\left|FV^{-1}-\lambda I\right|=0$.

The rows with only diagonal elements to be non-zero reduces our matrix to;

$$\begin{vmatrix} A_1-\lambda & A_3 \\ A_5 & A_7-\lambda \end{vmatrix}=0$$

$$\lambda^2-\left(A_1+A_7\right)\lambda+A_1A_7-A_5A_3=0$$

Since the basic reproduction number is the spectral radius of FV^{-1}, therefore $\Re_0=dom\left\{\lambda_i\right\}$.

$$\Re_0=\frac{\left(A_1+A_7\right)+\sqrt{\left(A_1+A_7\right)^2-4\left(A_1A_7-A_5A_3\right)}}{2} \tag{16}$$

Cholera Endemic Equilibrium Point

Theorem 3

The endemic equilibrium points of the Cholera model Eq. (1) is stable if $R>1$ and unstable if $R<1$

Proof

We set the RHS of the model equation to zero and solve for the variables; Let the endemic equilibrium point be denoted by E_0^{**} such that $E_0^{**}=\left(S_h^{**}, E_h^{**}, I_h^{**}, R_h^{**}, S_f^{**}, I_f^{**}, R_f^{**}, B^{**}\right)$ and set $S_h\neq 0, E_h\neq 0, I_h\neq 0, R_h\neq 0, S_f\neq 0, I_f\neq 0, R_f\neq 0, B\neq 0$. Then, at steady states

$$\frac{dS_h^*}{dt}=\frac{dE_h^*}{dt}=\frac{dI_h^*}{dt}=\frac{dR_h^*}{dt}=\frac{dS_f^*}{dt}=\frac{dI_f^*}{dt}=\frac{dR_f^*}{dt}=\frac{dB^*}{dt}=0 \tag{17}$$

The endemic equilibrium point Eq. (1), is obtained as;

$$E_0^{**}=\left\{\begin{array}{l} S_h^{**}=\dfrac{l_5 l_1 l_2 \Pi_h}{m} \\ E_h^{**}=\dfrac{\Pi_h l_5 l_2 \lambda_h^{**}}{m} \\ I_h^{**}=\dfrac{\phi \Pi_h l_5 \lambda_h^{**}}{m} \\ R_h^{**}=\dfrac{\left(\tau_1 l_2 \lambda_h^{**}+\tau_2 \phi \lambda_h^{**}\right)\Pi_h}{m} \\ S_f^{**}=\dfrac{\Pi_f}{\left(\lambda_f^{**}+l_6\right)} \\ I_f^{**}=\dfrac{\lambda_f^{**}\Pi_f}{l_3\left(\lambda_f^{**}+l_6\right)} \\ R_f^{**}=\dfrac{\tau_f \lambda_f^{**}\Pi_f}{l_3 l_6\left(\lambda_f^{**}+l_6\right)} \\ B^{**}=\dfrac{l_3\left(\lambda_a^{**}+l_6\right)\left(\left(\omega\zeta_h l_5 l_2 \lambda_h^{**}+a\phi\zeta_h l_5 \lambda_h^{**}\right)+mx\lambda_f^{**}\Pi_f\right)}{m l_4 l_3\left(\lambda_f^{**}+l_6\right)} \end{array}\right\} \quad (18)$$

Where;

$$\left.\begin{array}{l} l_1=\left(\phi+\tau_1+\mu_h\right), l_2=\left(\tau_2+\mu_h+\delta_h\right), l_3=\left(\tau_f+\mu_f+\delta_f+z\right), l_4=\left(\sigma-f\right), C=1-C_e \\ l_5=\alpha+\mu_h, l_6=\mu_f+z, m=l_5 l_1 l_2\left(\lambda_h^{**}+\mu_h\right)-\alpha\left(\tau_1 l_2 \lambda_h^{**}+\tau_2 \phi \lambda_h^{**}\right) \end{array}\right\} \quad (19)$$

Local stability of cholera-free equilibrium (CFE) point

Theorem 4: The cholera-free equilibrium points of the model Eq. (1) is locally asymptotically stable (LAS) when $\Re_0 < 1$ and unstable otherwise.

Proof:

Using the Jacobian matrix to prove the local stability of the cholera free equilibrium point

The Jacobian matrix evaluated at E_0 becomes

$$J(E_0)=\begin{pmatrix} -\mu_h & 0 & -\frac{\beta_{hh}\Pi_h}{\mu_h} & \alpha & 0 & -\frac{\beta_{fh}\Pi_h}{\mu_h} & 0 & -\frac{\beta_{eh}\Pi_h}{K\mu_h} \\ 0 & -l_1 & \frac{\beta_{hh}\Pi_h}{\mu_h} & 0 & 0 & \frac{\beta_{fh}\Pi_h}{\mu_h} & 0 & \frac{\beta_{eh}\Pi_h}{K\mu_h} \\ 0 & \phi & -l_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & \tau_1 & \tau_2 & -l_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -l_6 & -\frac{\beta_{ff}\Pi_f}{l_6} & 0 & -\frac{\beta_{ef}\Pi_f}{Kl_6} \\ 0 & 0 & 0 & 0 & 0 & \frac{\beta_{ff}\Pi_f}{l_6}-l_3 & 0 & \frac{\beta_{ef}\Pi_f}{Kl_6} \\ 0 & 0 & 0 & 0 & 0 & \tau_f & -l_6 & 0 \\ 0 & \omega & a & 0 & 0 & x & 0 & -l_4 \end{pmatrix} \quad (20)$$

The matrix Eq. (20) can be reduced, since the first, fifth and seventh column consist of only the diagonal element; the negative eigenvalues obtained are; $\lambda=-\mu_h,-l_6 twice$. Furter simplification reveals fourth, column will give us $\lambda=-l_5$

$$J(E_0)=\begin{bmatrix} -l_1 & \frac{\beta_{hh}\Pi_h}{\mu_h} & \frac{\beta_{fh}\Pi_h}{\mu_h} & \frac{\beta_{eh}\Pi_h}{K\mu_h} \\ \phi & -l_2 & 0 & 0 \\ 0 & 0 & \frac{\beta_{ff}\Pi_f}{l_6}-l_3 & \frac{\beta_{ef}\Pi_f}{Kl_6} \\ \omega & a & x & -l_4 \end{bmatrix} \quad (21)$$

The characteristic polynomial of the matrix Eq. (21) is

$$A_0\lambda^4+A_1\lambda^3+A_2\lambda^2+A_3\lambda+A_4 \quad (22)$$

Where;

$A_0=1>0$

$$A_1=\left(\frac{l_1l_6+l_2l_6+l_3l_6+l_4l_6-\beta_{ff}\Pi_f}{l_6}\right)>0$$

Whenever; $l_1l_6+l_2l_6+l_3l_6+l_4l_6>\beta_{ff}\Pi_f$

$$A_2=\begin{pmatrix} Kl_1l_2l_6\mu_h+Kl_1l_3l_6\mu_h+Kl_1l_4l_6\mu_h \\ +Kl_2l_3l_6\mu_h+Kl_2l_4l_6\mu_h+Kl_3l_4l_6\mu_h \\ -K\Pi_f l_1\mu_h\beta_{ff}-K\Pi_f l_2\mu_h\beta_{ff} \\ -K\Pi_f l_4\mu_h\beta_{ff}-K\Pi_f\beta_{hh}l_6\phi \\ -x\beta_{ef}\Pi_f\mu_h-\omega\beta_{eh}\Pi_h l_6 \end{pmatrix}>0$$

Whenever; $\begin{pmatrix} Kl_1l_2l_6\mu_h+Kl_1l_3l_6\mu_h+Kl_1l_4l_6\mu_h \\ +Kl_2l_3l_6\mu_h+Kl_2l_4l_6\mu_h+Kl_3l_4l_6\mu_h \end{pmatrix}>\begin{pmatrix} -K\Pi_f l_1\mu_h\beta_{ff}-K\Pi_f l_2\mu_h\beta_{ff} \\ -K\Pi_f l_4\mu_h\beta_{ff}-K\Pi_f\beta_{hh}l_6\phi \\ -x\beta_{ef}\Pi_f\mu_h-\omega\beta_{eh}\Pi_h l_6 \end{pmatrix}$

$$A_3=\begin{pmatrix} K\Pi_h\Pi_f\beta_{hh}\beta_{ff}\phi+Kl_1l_2l_3l_6\mu_h+Kl_1l_2l_4l_6\mu_h+Kl_1l_3l_4l_6\mu_h+Kl_2l_3l_4l_6\mu_h \\ +\Pi_f\Pi_h\omega\beta_{eh}\beta_{ff}-\Pi_f l_1\mu_h x\beta_{ef}-\Pi_f l_2\mu_h x\beta_{ef}-\Pi_h al_6\phi\beta_{eh}-\Pi_h l_2l_6\omega\beta_{eh} \\ -\Pi_h l_3l_6\omega\beta_{eh}-K\Pi_f l_1l_2\beta_{ff}\mu_h-K\Pi_f l_1l_4\beta_{ff}\mu_h-K\Pi_f l_2l_4\beta_{ff}\mu_h \\ -K\Pi_h l_3l_6\beta_{hh}\phi-K\Pi_h l_4l_6\beta_{hh}\phi-\Pi_f\Pi_h\omega\beta_{ef}\beta_{fh} \end{pmatrix}>0$$

Whenever;

$$\begin{pmatrix} K\Pi_h\Pi_f\beta_{hh}\beta_{ff}\phi+Kl_1l_2l_3l_6\mu_h+Kl_1l_2l_4l_6\mu_h \\ +Kl_1l_3l_4l_6\mu_h+Kl_2l_3l_4l_6\mu_h+\Pi_f\Pi_h\omega\beta_{eh}\beta_{ff} \end{pmatrix}>\begin{pmatrix} -\Pi_f l_1\mu_h x\beta_{ef}-\Pi_f l_2\mu_h x\beta_{ef}-\Pi_h al_6\phi\beta_{eh} \\ -\Pi_h l_2l_6\omega\beta_{eh}-\Pi_h l_3l_6\omega\beta_{eh}-K\Pi_f l_1l_2\beta_{ff}\mu_h \\ -K\Pi_f l_1l_4\beta_{ff}\mu_h-K\Pi_f l_2l_4\beta_{ff}\mu_h \\ -K\Pi_h l_3l_6\beta_{hh}\phi-K\Pi_h l_4l_6\beta_{hh}\phi-\Pi_f\Pi_h\omega\beta_{ef}\beta_{fh} \end{pmatrix}$$

$$A_1=\begin{pmatrix} K\Pi_h\Pi_f\beta_{hh}\beta_{ff}\phi+Kl_1l_2l_3l_6\mu_h+Kl_1l_2l_4l_6\mu_h+Kl_1l_3l_4l_6\mu_h+Kl_2l_3l_4l_6\mu_h \\ +\Pi_f\Pi_h\omega\beta_{eh}\beta_{ff}-\Pi_f l_1\mu_h x\beta_{ef}-\Pi_f l_2\mu_h x\beta_{ef}-\Pi_h al_6\phi\beta_{eh}-\Pi_h l_2l_6\omega\beta_{eh} \\ -\Pi_h l_3l_6\omega\beta_{eh}-K\Pi_f l_1l_2\beta_{ff}\mu_h-K\Pi_f l_1l_4\beta_{ff}\mu_h-K\Pi_f l_2l_4\beta_{ff}\mu_h \\ -K\Pi_h l_3l_6\beta_{hh}\phi-K\Pi_h l_4l_6\beta_{hh}\phi-\Pi_f\Pi_h\omega\beta_{ef}\beta_{fh} \end{pmatrix}>0$$

Whenever;

$$\begin{pmatrix} K\Pi_h\Pi_f\beta_{hh}\beta_{ff}\phi+Kl_1l_2l_3l_6\mu_h+Kl_1l_2l_4l_6\mu_h \\ +Kl_1l_3l_4l_6\mu_h+Kl_2l_3l_4l_6\mu_h+\Pi_f\Pi_h\omega\beta_{eh}\beta_{ff} \end{pmatrix}>\begin{pmatrix} -\Pi_f l_1\mu_h x\beta_{ef}-\Pi_f l_2\mu_h x\beta_{ef}-\Pi_h al_6\phi\beta_{eh} \\ -\Pi_h l_2l_6\omega\beta_{eh}-\Pi_h l_3l_6\omega\beta_{eh}-K\Pi_f l_1l_2\beta_{ff}\mu_h \\ -K\Pi_f l_1l_4\beta_{ff}\mu_h-K\Pi_f l_2l_4\beta_{ff}\mu_h-K\Pi_h l_3l_6\beta_{hh}\phi \\ -K\Pi_h l_4l_6\beta_{hh}\phi-\Pi_f\Pi_h\omega\beta_{ef}\beta_{fh} \end{pmatrix}$$

With the above condition, all the coefficient of the eigenvalues are positive, which is necessary condition for all roots to have a negative real part. Then by Descartes' rule of sign, it follows that all the eigenvalues are negative, real and distinct, in the absence of change of signs. Therefore, the diseases free equilibrium is locally asymptotically stable whenever $\Re_0<1$.

Global Stability of Cholera-Free Equilibrium (CFE) point

Theorem 5: The cholera-free equilibrium of the system Eq. (1) is globally asymptotically stable if the $\Re_0<1$.

Proof:

In order to proof the theorem, then the model Eq. (1), can be restructured in the form

$$\left.\begin{aligned} \frac{dX_1}{dt}&=F\left(X_1,\,X_2\right) \\ \frac{dX_2}{dt}&=G\left(X_1,\,X_2\right),\;G\left(X_1,\,0\right)=0 \end{aligned}\right\} \tag{23}$$

Where, $X_1\in R_+^4$ and $X_2\in R_+^4$. X_1 Component represents the uninfected class while X_2 represents the infected class, in essence

$X_1=\left(S_h,R_h,S_f,R_f\right)$ and $X_2=\left(E_h,I_h,I_f,B\right)$

Let, the CFE of the model Eq. (1) be given by $E_0=\left(X_1^*,0\right)$. Then the following properties must be satisfied in order to establish the global asymptotic stability of the model Eq. (1)

H_1 : for $\frac{dX_1}{dt}=F\left(X_1^*,0\right)$, X_1^* is globally asymptotically stable

$H_2 : G(X_1, X_2) = AX_2 - \hat{G}(X_1, X_2), \hat{G}(X_1, X_2) \geq 0$ for $(X_1, X_2) \in \Omega$

$A = \dfrac{\partial G}{\partial X_2}$ Is a matrix evaluated at $\left(X_1^*, 0\right)$,

Where, the off-diagonal elements are non-negative.

$F(X_1, X_2)$ And $G(X_1, X_2)$ are obtained from Eq. (1) as follows

$$F\left(X_1, X_2\right) = \begin{pmatrix} \Pi_h + \alpha R_h - \left[\dfrac{\beta_{eh} B}{K+B}\left(1-C_e\right) + \beta_{hh}\left(1-C_e\right) I_h + \beta_{fh}\left(1-C_e\right) I_f + \mu_h\right] S_h \\ \tau_1 E_h + \tau_2 I_h - \left(\alpha + \mu_h\right) R_h \\ \Pi_a - \left(\dfrac{\beta_{ef} B}{K+B}\left(1-C_e\right) + \beta_{ff} I_f + \mu_f + z\right) S_a \\ \tau_f I_f - \left(\mu_f + z\right) R_f \end{pmatrix} \tag{24}$$

$$G\left(X_1, X_2\right) = \begin{pmatrix} \left[\dfrac{\beta_{eh} B}{K+B}\left(1-C_e\right) + \beta_{hh}\left(1-C_e\right) I_h + \beta_{fh}\left(1-C_e\right) I_f\right] S_h - \left(\phi + \tau_1 + \mu_h\right) E_h \\ \phi E_h - \left(\tau_2 + \mu_h + \delta_h\right) I_h \\ \left(\dfrac{\beta_{ef} B}{K+B}\left(1-C_e\right) + \beta_{ff} I_f\right) S_a - \left(\tau_f + \mu_f + \delta_f + z\right) I_f \\ \omega E_h + a I_h + x I_a - (\sigma - f) B \end{pmatrix} \tag{25}$$

$\dfrac{dX_1}{dt} = F(X_1\, 0)$ Implies that

$$\left.\begin{aligned} \frac{dS_h}{dt} &= \Pi_h - \mu_h S_h \\ \frac{dR_h}{dt} &= 0 \\ \frac{dS_f}{dt} &= \Pi_f - \left(\mu_f + z\right) S_f \\ \frac{dR_f}{dt} &= 0 \end{aligned}\right\} \tag{26}$$

Hence, $S_h(t), R_h(t), S_f(t), I_f(t) \to \left(\dfrac{\Pi_h}{\mu_h}, 0, \dfrac{\Pi_f}{\left(\mu_f + z\right)}, 0\right)$ irrespective of the initial value of the variables as $t \to \infty$. Therefore, X_1^* is globally asymptotically stable satisfying H_1

Furthermore, the matrix A is given as

$$A = \frac{\partial G}{\partial X_2}\left(X_1^*\, 0\right) = \begin{pmatrix} -l_1 & \beta_{hh} S_h(0) & \beta_{fh} S_h(0) & \dfrac{\beta_{eh}}{K} S_h(0) \\ \phi & -l_2 & 0 & 0 \\ 0 & 0 & \beta_{ff} S_f(0) - l_3 & \dfrac{\beta_{ef}}{K} S_f(0) \\ \omega & a & x & -l_4 \end{pmatrix} \begin{pmatrix} E_h \\ I_h \\ I_f \\ B \end{pmatrix} \tag{27}$$

$\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2)$; when B is extremely small

$$\hat{G}(X_1\ X_2) = \begin{pmatrix} \left(\frac{\beta_{eh}}{K}B + \beta_{hh}I_h + \beta_{fh}I_f\right)(S_h(0) - S_h) \\ 0 \\ \left(\frac{\beta_{ef}}{K}B + \beta_{ff}I_f\right)(S_f(0) - S_f) \\ 0 \end{pmatrix} \tag{28}$$

Since $0 \le S_h \le \frac{\Pi_h}{\mu_h}$ and $0 \le S_f \le \frac{\Pi_f}{(\mu_f + z)}$, it is obvious that $\hat{G}(X_1, X_2) \ge 0$. Thus H_2 holds.

Hence, the cholera-free equilibrium is globally asymptotically stable.

Sensitivity Analysis of Cholera Model

The sensitivity analysis is important in investigating the best way to minimize the effect and the transmission of cholera, through studying the factors responsible for its transmission and prevalence with respect to the parameters.

The normalized forward sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter [6]. The basic reproduction number's $(\Re_0)$ sensitivity indices, was computed using the formula:

$$\gamma_{\chi}^{\Re_0} = \frac{\partial \Re_0}{\partial p} \times \frac{p}{\Re_0} \tag{29}$$

Where:

$\Re_0$: Is the basic reproduction number

p : Is the parameter of interest

Solving for the parameters of interest, we obtained the results as presented in Table 3

Table 3: Baseline values for parameters: Sensitivity indices of the cholera model.

Parameters	Sensitivity	Parameters	Sensitivity
μ_f	-1.380937341	β_{ef}	$9.093164521*10^{(-7)}$
Π_f	0.9999994220	μ_h	$-7.658036492*10^{(-7)}$
β_{ff}	0.9999985120	β_{fh}	$5.781710152*10^{(-7)}$
δ_f	-0.4458435582	x	$3.311454372*10^{(-7)}$
z	-0.09585201163	a	$1.072931970*10^{(-7)}$
τ_f	-0.07736593228	f	$8.430981246*10^{(-8)}$
Π_h	$5.783521550*10^{(-7)}$	τ_2	$-9.067862465*10^{(-8)}$
ω	$4.708979201*10^{(-7)}$	τ_1	$-2.115178995*10^{(-8)}$
ϕ	$-2.732829744*10^{(-7)}$	δ_h	$-5.440717479*10^{(-9)}$
C_e	$-3.697628819*10^{(-7)}$	β_{hh}	$1.559804512*10^{(-10)}$

σ	$-9.936513611*10^{(-7)}$	β_{eh}	$2.509689243*10^{(-11)}$
K	$-9.093415510*10^{(-7)}$		

The sensitivity indices results presented in Table 3 showed that, those parameters with positive indices $\left(a,\omega,x,f,\beta_{hh},\beta_{ef},\beta_{ff},\beta_{fh},\beta_{eh},\Pi_h,\Pi_f\right)$ increase the $\Re_0$. This implies that the parameters have great impact of transmitting the disease in the population if it increases in value. Similarly, those parameters with negative sensitivity indices $\left(\tau_1,\tau_2,\delta_f,\delta_h,\mu_f,\mu_h,K,\sigma,z,C_e,\phi,\tau_f\right)$ decreases the $\Re_0$, which also implies that, the parameters have the ability of minimizing the disease in the population if their values are increased while others are kept constant.

Numerical Simulation

In this section, we performed numerical simulation on the system of equations (1) using the values in

Table **2** to explore the impact of different parameters on cholera dynamics, highlighting effective strategies for curtailing/controlling the spread of the disease. The results are presented in figures below;

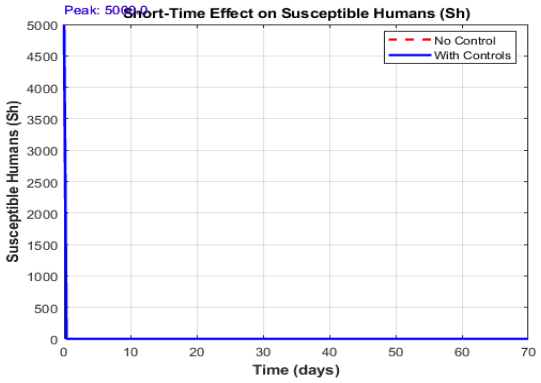

Figure 3.2: Susceptible Humans $\left(S_h\right)$

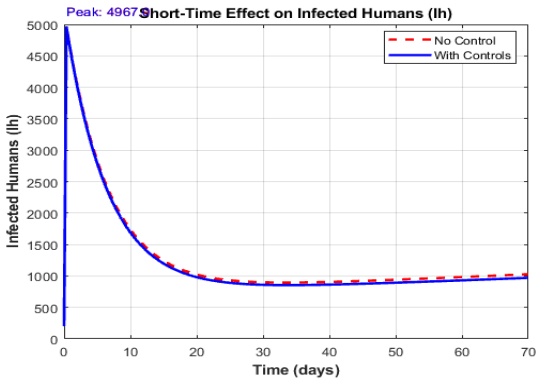

Figure 3.3: Infected Humans $\left(I_h\right)$

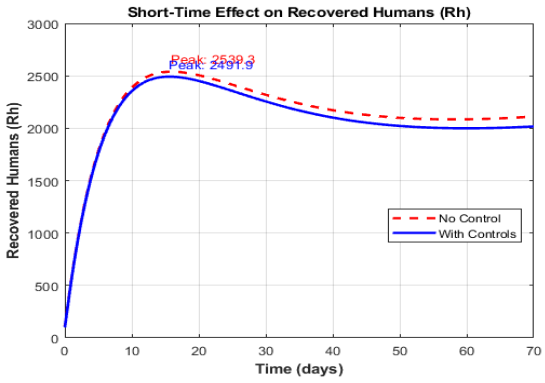

Figure 3.4: Recovered Humans $\left(R_h\right)$

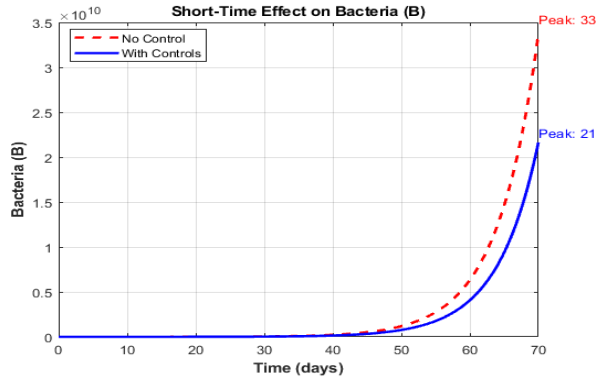

Figure 3.5: Bacteria Concentration $\left(B\right)$

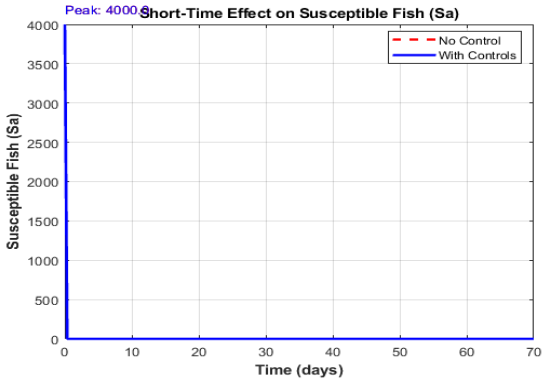

Figure 3.6: Susceptible Fish (S_a)

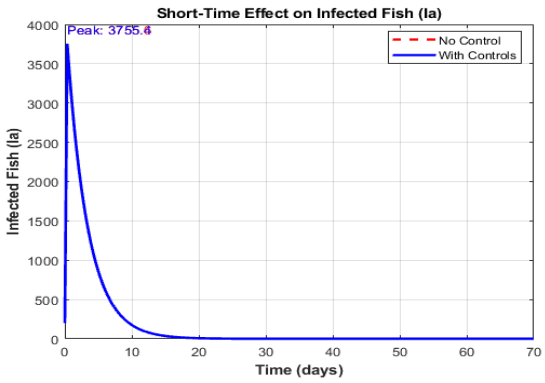

Figure 3.7: Infected Fish (I_a)

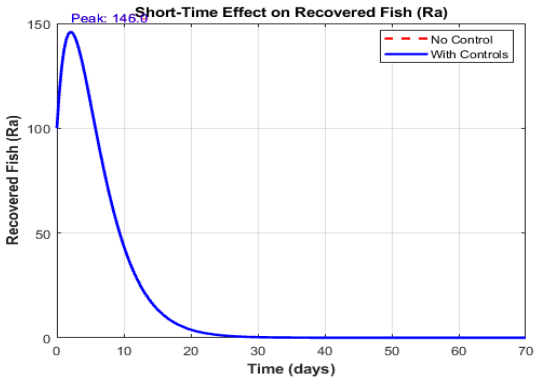

Figure 3.8: Recovered Fish (R_a)

3.0 RESULT DISCUSSION

The numerical simulations presented in Figure 3.2 - Figure 3.8 demonstrate the significant influence of transmission and control parameters on the dynamics of cholera spread within the human-fish-environment system. In the absence of interventions, susceptible human and fish populations decline rapidly due to increased transmission, while infected populations and environmental bacterial concentration rise sharply, leading to severe epidemic outbreaks. Conversely, the implementation of effective control measures, including vaccination, quarantine, public health awareness, sanitation improvement, and fish-to-human transmission prevention, substantially mitigates disease spread by reducing infection peaks, delaying outbreak progression, and preserving larger susceptible populations.

The results further show that intervention strategies enhance recovery dynamics in both humans and fish, indicating improved disease management and reduced infection pressure within the environment. Environmental bacterial concentration is notably suppressed under controlled conditions, thereby lowering the risk of sustained transmission between humans, aquatic animals, and contaminated water sources. These findings emphasize the critical role of integrated intervention strategies in minimizing epidemic severity.

Overall, the simulations validate the analytical results by confirming that transmission-related parameters intensify disease propagation, whereas control-related

parameters significantly reduce the effective reproduction potential. The outcomes strongly support the One Health approach, highlighting that cholera elimination requires coordinated human, animal, and environmental health interventions. Comprehensive control strategies targeting sanitation, vaccination, behavioural modification, treatment, and environmental management provide the most effective pathway toward reducing disease persistence and preventing large-scale outbreaks.

4.0 CONCLUSION

In this study, we meticulously developed a mathematical framework to explore the dynamics of Cholera transmission, incorporating treatment and public health campaign as means of control. We employ the nonlinear system of differential equations, where we determined the basic reproduction number $\Re_0$ of the Cholera model, demonstrating that the system achieves local asymptotic stability when $\Re_0 < 1$, global asymptotic stability when $\Re_0 < 1$, and maintains a stable endemic equilibrium when $\Re_0 > 1$. Through sensitivity analysis, we assessed the impact of various parameters on the basic reproduction number, noting that proper combination of control measures could drastically reduce the spread.

RECOMMENDATIONS

Based on the findings of this study, effective cholera control requires improved access to clean water, adequate treatment centres, enhanced sanitation and waste management systems, and stronger public health awareness campaigns on hygiene practices such as hand washing and safe food handling. Governments should invest in water treatment, drainage, flood control, and healthcare infrastructure, while supporting community-led sanitation initiatives and rapid outbreak response programs. Collectively, these measures will help reduce environmental contamination, limit disease transmission, and strengthen cholera prevention and control within the population.

Declaration of competing interests

The authors declare that they have no conflicting interest as regard to this work.

Authors consent

The submission of this article has been approved by all the listed authors as an original world and has not been previously published.

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