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Modelling Cholera Transmission Dynamics in a Gender-Hygiene Driven Incidence Rate

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Abstract: This study develops and analyzes a non-linear deterministic mathematical model for cholera transmission that explicitly incorporates a gender-hygiene-driven incidence rate. Recognizing that social roles often determine exposure risks, particularly in communities where women are primarily responsible for water collection, childcare, and food handling, the model incorporates gender-disaggregated compartments for susceptible (S_m (t) and S_f (t)), infected (I_m (t) and I_f (t)), and recovered individuals (R_m (t) and R_f (t)), alongside an environmental reservoir representing Vibrio cholerae concentration (B(t)). The model extends the classical SIRB framework of [23], by introducing gender-specific hygiene compliance rates into the infection incidence term, reflecting gendered differences in exposure and hygiene practices. Analytical results include the derivation of the basic reproduction number (R_0), examination of equilibrium points, and proof of the positivity and boundedness of solutions. Stability analyses were conducted for both the disease-free and endemic equilibria, while sensitivity analysis identified the most influential parameters governing cholera spread. Numerical simulations were performed using parameter values from relevant literature to explore the effects of varying hygiene compliance, bacterial decay rate, and environmental sanitation on disease prevalence. Results reveal that gender-based differences in hygiene significantly affect cholera transmission. In particular, higher hygiene adherence among both men and women markedly reduces the reproduction number and infection prevalence, while poor hygiene and persistent environmental contamination sustain outbreaks. The study concludes that effective cholera control requires integrating behavioral and structural interventions—especially those that promote hygiene education among women and improve environmental sanitation. Overall, this work provides a more realistic and socially responsive framework for modeling cholera dynamics, bridging the gap between mathematical theory and public-health practice. It contributes to the growing field of gender-aware epidemiological modeling and offers practical insights for designing sustainable, behavior-driven cholera control strategies.

Keywords: *Choleraa Transmission, Gender-Hygiene Incidence Rate, Reproduction Number.*

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I. INTRODUCTION

Cholera remains one of the most persistent and devastating waterborne diseases affecting humanity, particularly in low- and middle-income countries where clean water and adequate sanitation are not guaranteed. The disease, caused by Vibrio cholerae, manifests through acute watery diarrhea, vomiting, dehydration, and in severe cases, death if untreated. The World Health Organization [1] estimated between 1.3 and 4.0 million cholera cases annually, with up to 143,000 fatalities worldwide, emphasizing its continued relevance as a global public-health challenge. Regions most affected include sub-Saharan Africa, South and Southeast Asia, and some parts of South America, where environmental sanitation, safe drinking water, and hygiene infrastructure are deficient [2]. Cholera outbreaks often follow natural disasters, floods, or displacement crises that compromise water systems and sanitation services. These environmental and socioeconomic vulnerabilities not only enable the persistence of V. cholerae but also exacerbate the health inequalities underlying the disease's transmission and impact.

Historically, cholera's epidemiological trajectory has evolved through seven pandemics, beginning in the early nineteenth century and continuing with recurring outbreaks across Asia and Africa [3], [4]. Mathematical models have played a central role in understanding these epidemiological trends by elucidating the relationships between human behavior, environmental factors, and pathogen dynamics ([5], [6]). Early models such as that of [7] considered simple deterministic approaches to capture epidemic dynamics, while subsequent works introduced environmental compartments to model bacterial persistence in aquatic reservoirs [8]. These frameworks established that cholera transmission is not merely a function of biological infection

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but also depends on social and ecological factors influencing human–environment interactions.

Despite extensive modeling efforts, most cholera models have assumed homogeneity within human populations, neglecting gender-based variations in exposure, hygiene practices, and behavioral responses. However, evidence from epidemiological and social studies has shown that gender is a significant determinant of infection risk. Women and girls, due to their traditional domestic roles, are often more exposed to cholera pathogens through caregiving for sick relatives, cleaning contaminated spaces, fetching untreated water, and preparing food ([9], [10]). This asymmetry implies that hygiene behavior is not uniform but socially structured, thereby influencing disease transmission dynamics in gender-differentiated ways. Incorporating gender and hygiene factors into mathematical models thus provides a more realistic framework for understanding cholera epidemiology and formulating equitable intervention strategies.

The application of mathematical models to disease control has expanded to include behavioral and environmental parameters that influence epidemic outcomes. For instance, [11] demonstrated that multiple control measures such as vaccination, treatment, and sanitation are more effective than single interventions. [12] developed and analyzed a reaction–convection–diffusion cholera model, demonstrating mathematically that the disease dynamics exhibit global stability and uniform persistence under specific biological and environmental conditions, thereby providing a rigorous framework for understanding spatial-temporal behavior in cholera epidemics, while [13] highlighted the role of awareness and behavioral responses in modifying cholera spread. More recently, [14] and [15] emphasized the integration of vaccination and hygiene education as effective long-term strategies. Yet, as [16] and [17] observed, most models treat population behavior as a uniform parameter, omitting the heterogeneity introduced by gendered hygiene practices—an omission that this study seeks to address.

A review of the literature reveals a growing interest in gender-sensitive approaches to public-health interventions, though their integration into quantitative models remains limited. Studies such as [18] in Uganda, [19] in South Africa and [20], [21] in Nigeria show that gender and environmental sanitation interact to shape disease exposure, but few have mathematically modeled this relationship. Traditional models fail to account for the differential impact of hygiene compliance between men and women, despite evidence that women's sanitation and water-handling behaviors significantly influence cholera transmission at the household level [22]. Thus, a gender-hygiene-driven modeling framework can bridge the gap between epidemiological modeling and social realities, offering insights into how behavioral interventions tailored to gender dynamics could enhance the effectiveness of public-health policies.

In light of these findings, this study formulates a deterministic cholera transmission model that incorporates gender-specific hygiene compliance rates into the force of infection. The model extends the work of [23] by subdividing the human population into male and female compartments and associating each with hygiene-dependent exposure rates. Through analytical derivations and numerical simulations, the study examines how gendered hygiene behaviors influence the basic reproduction number (Ro), equilibrium stability, and long-term disease persistence. The aim is to provide a more nuanced understanding of cholera dynamics that can inform gender-responsive health strategies, enhance policy formulation, and ultimately contribute to sustainable disease control.

II. MATERIAL AND METHODS

➢ *Assumptions of the Model*

Model assumptions play an important guideline in model building, hence in the formulation of the model, we are guided by the following assumptions:

- The human population is heterogeneous, composed of males and females with differing hygiene practices.
- Both groups acquire infection from contaminated water containing Vibrio cholerae.
- Recovered individuals gain temporary immunity but may relapse.
- Deaths occur naturally and from cholera-induced causes.
- Environmental sanitation and personal hygiene reduce bacterial concentration in water.
- Treatment confers recovery at gender-specific rates.
- Birth and immigration contribute to population inflow.

➢ *Mathematical Formulation and Description of the Model*

We extend the a Mathematical Model for the Control of Cholera Epidemic without Natural Recovery of [23] by modifying their model and considering a gender driven hygienic incidence rate for male and female respectively. The model consists of the following seven compartments; susceptible male $S_m(t)$, infected male $I_m(t)$, recovered male $R_m(t)$, susceptible female $S_f(t)$, infected female $I_f(t)$,, recovered female $R_f(t)$, total human population $N(t)$ and the concentration of *Vibrio cholerae* in water $B(t)$. The susceptible male class (S_m) population and susceptible female class (S_f) are generated either through birth or through immigration at the rate βN. Both susceptible male and susceptible female acquired infection through contaminated water at the rate $\frac{\alpha_m(1-C_{wm})}{K+B}$ and $\frac{\alpha_f(1-C_{wf})}{K+B}$ respectively.

Table 1 Variables and Parameters of the Model

Variables	Description
$S_m(t)$	Susceptible male human population at time t
$S_f(t)$	Susceptible female human population at time$t$

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$I_m(t)$	Infected male human population at time t
$I_f(t)$	Infected female human population at time t
$R_m(t)$	Recovered male human population at time t
$R_f(t)$	Recovered female human population at time t
$B(t)$	concentration of *Vibrio cholerae* population at time t
$N(t)$	Total population humans at time t
Parameter	**Description**
β	Birth rate of humans
μ	Death rate of humans
α_m	Rate of exposure of male to contaminated water
α_f	Rate of exposure of female to contaminated water
K	concentration of *Vibrio cholerae* in water
τ_m	recovery rate due to treatment in male
τ_f	recovery rate due to treatment in female
C_{ω_m}	Rate of compliance of male with water hygiene
C_{ω_f}	Rate of compliance of female with water hygiene
C_{s_m}	Rate of compliance of male with environmental sanitation
C_{s_f}	Rate of compliance of female with environmental sanitation
δ	Cholera induced death
ω_m	Loss rate of male immunity by recovered individuals (transfer rate)
ω_f	Loss rate of female immunity by recovered individuals (transfer rate)
e	Contribution of each infected person to the population of *Vibrio cholerae* in the aquatic environment.
ρ	Growth rate of Vibrio cholerae in the aquatic environment

The rate at which susceptible male and susceptible female are transferred to the infected male and infected female human population at time t are given as α_m and α_f respectively. The rate at which the infected male and infected female human population becomes infectious with the *Vibrio cholerae* population at time t is given by $e(1-C_{sm})$ and $e(1-C_{sf})$ respectively. Infected male and female are treated at the rate of τ_m and τ_f respectively. While ω_m ω_m and ω_f are the transfer rate at which the recover male class and the recover female class transferred back to the susceptible classes at time t respectively. Disease induced death rate is given by δ for both male and female.

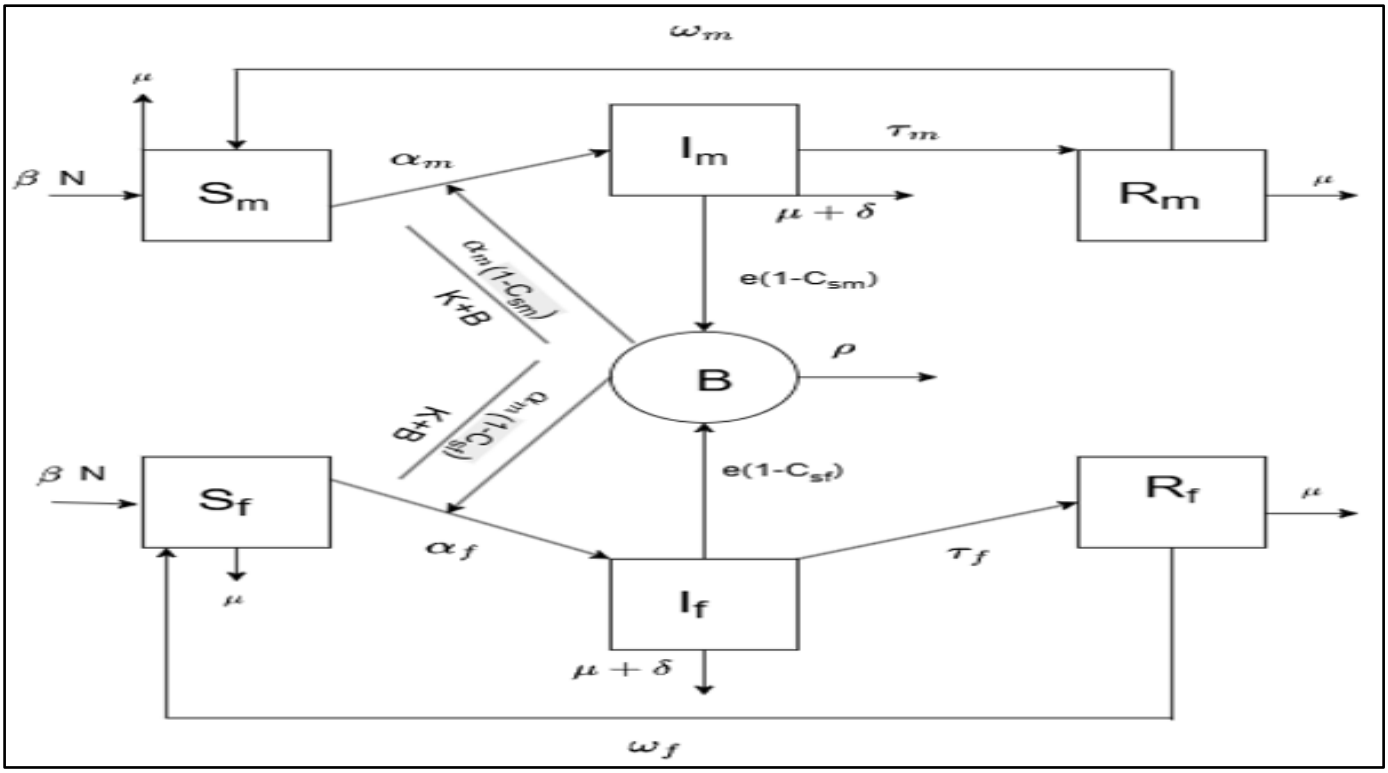

Fig 1 Flow Diagram of the Cholera Transmission Model.

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From the model description and the flow diagram of the chorela transmission model on Figure 1, we have the equation $N(t) = S_m(t) + S_f(t) + I_m(t) + I_f(t) + R_m(t) + R_f(t) + B(t)$ and the model equation in section 2.3. The population of the susceptible increase through immigration or birth rate of βN and recover rates (τ_m, τ_m). The mass action from $e(1 - C_{w_m})$ and $e\left(1 - C_{w_f}\right)$ is given as $\frac{\alpha_m(1-C_{wm})B}{N}$ and $\frac{\alpha_m\left(1-C_{wf}\right)B}{N}$ respectively.

➢ *Model Equations*

Based on the model assumptions and the flow diagram in Figure 1, we formulate the following *SIRB* model, which is a system of nonlinear ordinary differential equations.

$$\frac{dS_m}{dt} = \beta N - \frac{\alpha_m\left(1-C_{\omega m}\right)B}{K+B} S_m + \omega_m R_m - \mu S_m \quad (1)$$

$$\frac{dS_f}{dt} = \beta N - \frac{\alpha_f\left(1-C_{\omega f}\right)B}{K+B} S_f + \omega_f R_f - \mu S_f \quad (2)$$

$$\frac{dI_m}{dt} = \frac{\alpha_m\left(1-C_{\omega m}\right)B}{K+B} S_m - \left(\tau_m + e\left(1-C_{sm}\right) + \mu + \delta\right) I_m \quad (3)$$

$$\frac{dI_f}{dt} = \frac{\alpha_f\left(1-C_{\omega f}\right)B}{K+B} S_f - \left(\tau_f + e\left(1-C_{sf}\right) + \mu + \delta\right) I_f \quad (4)$$

$$\frac{dB}{dt} = e\left(1-C_{sm}\right) I_m + e\left(1-C_{sf}\right) I_f - \rho \mathrm{B} \quad (5)$$

$$\frac{dR_m}{dt} = \tau_m I_m - \left(\omega_m + \mu\right) R_m \quad (6)$$

$$\frac{dR_f}{dt} = \tau_f I_f - \left(\omega_f + \mu\right) R_f \quad (7)$$

With

$$N(t) = S_m + I_m + R_m + B + S_f + I_f + R_f \quad (8)$$

Considering the initial conditions of the model (1) - (7) gives

$$S_m(0) > 0, S_f(0) > 0, I_m(0) \geq 0, I_f(0) \geq 0, R_m(0) \geq 0, R_f(0) \geq 0, B(0) \geq 0 \quad (9)$$

➢ *Model Analysis*

- *Positivity and Boundedness of the Solution of the Cholera Model.*

Since this mathematical model monitors human population, we need to show that all the state variables remain non-negative for all times (t). In other word, mathematical solutions of the disease model system of equations with positive initial values will remain non-negative for all times $t \geq 0$.

✓ *Theorem 1: (Positivity Solution)*

Let

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$$\Omega = \left\{ \left(S_m, S_f, I_m, I_f, R_m, R_f B \right) \in \mathbb{R}_+^7 : S_m(0) > 0, S_f(0) > 0, I_m(0) > 0, I_f(0) > 0, R_m(0) > 0, R_f(0) > 0, B(0) > 0 \right\}$$

Then the solutions of the system of equations are non - negative for all $t \geq 0$.

✓ *Proof:*

From equation (1) we have

$$\Rightarrow \frac{dS_m}{dt} \geq \beta N - \mu S_m$$

$$\frac{dS_m}{dt} + \mu S_m = \beta N \tag{10}$$

Multiplying equation (10) through by the integrating factor (IF) and integrating we have

$$\frac{dS_m}{dt} \cdot e^{\mu t} + \mu S_m \cdot e^{\mu t} = \beta N \cdot e^{\mu t} \Rightarrow \int_0^t \frac{d}{dt}\left(S_m \cdot e^{\mu t} \right) = \int_0^t \beta N e^{\mu t}$$

$$\Rightarrow S_m(t) = \frac{\beta N}{\mu} + Ce^{-\mu t} \tag{11}$$

where C is a constant

With initial condition $S_m(0) = S_0^m, \Rightarrow C = S_0^m - \frac{\beta N}{\mu}$

$$\therefore S_m(t) = \frac{\beta N}{\mu} + \left(S_0^m - \frac{\beta N}{\mu} \right) e^{-\mu t} \tag{12}$$

Taking limit as $t \to \infty$, $S_m(t) = \frac{\beta N}{\mu} + \left(S_0^m - \frac{\beta N}{\mu} \right) e^{-\mu t} \geq 0$ (13)

Similarly,

From equation (2) we have

$$\Rightarrow \frac{dS_f}{dt} \geq \beta N - \mu S_f$$

$$\frac{dS_f}{dt} + \mu S_f = \beta N \tag{14}$$

Multiplying equation (14) through by the integrating factor (IF) and integrating we have

$$\frac{dS_f}{dt} \cdot e^{\mu t} + \mu S_f \cdot e^{\mu t} = \beta N \cdot e^{\mu t} \Rightarrow \frac{d}{dt}\left(S_f \cdot e^{\mu t} \right) = \beta N e^{\mu t}$$

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Integrating both side, we have

$$\Rightarrow S_f(t) = \frac{\beta N}{\mu} + Ce^{-\mu t} \tag{15}$$

where C is a constant

With initial condition $S_f(0) = S_0^f, \Rightarrow C = S_0^f - \frac{\beta N}{\mu}$

$$\therefore S_f(t) = \frac{\beta N}{\mu} + \left(S_0^f - \frac{\beta N}{\mu}\right)e^{-\mu t} \tag{16}$$

Taking limit as $t \to \infty$

$$S_f(t) = \frac{\beta N}{\mu} + \left(S_0^f - \frac{\beta N}{\mu}\right)e^{-\mu t} \geq 0 \tag{17}$$

Similarly, considering equation (3) we have

$$\Rightarrow \frac{dI_m}{dt} \geq -(\tau_m + \mu + \delta) I_m$$

$$\frac{dI_m}{dt} + (\tau_m + \mu + \delta) I_m = 0 \tag{18}$$

Multiply equation (18) through by the integrating factor (IF) and integrating, we have

$$\frac{dI_m}{dt}.e^{(\tau_m+\mu+\delta)t} + (\tau_m + \mu + \delta) I_m.e^{(\tau_m+\mu+\delta)t} = 0 \Rightarrow \int_0^t \frac{d}{dt}\left(I_m e^{(\tau_m+\mu+\delta)t}\right) = \int_0^t 0 dt$$

$$I_m(t)e^{(\tau_m+\mu+\delta)t} = C \tag{19}$$

Where C is a constant

With initial conditions,

$$t = 0 \text{ and } I_m(0) = I_0^m, \Rightarrow C = I_0^m$$

$$\therefore I_m(t)e^{(\tau_m+\mu+\delta)t} = I_0^m \tag{20}$$

Taking limit as $t \to \infty, I_m(t) = I_0^m e^{-(\tau_m+\mu+\delta)t} \geq 0$ (21)

Similarly, from equation (4) we have

$$\Rightarrow \frac{dI_f}{dt} \geq -(\tau_f + \mu + \delta) I_f$$

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$$\frac{dI_f}{dt}+\left(\tau_f+\mu+\delta\right)I_f=0 \tag{22}$$

Multiply equation (22) through by the integrating factor (IF) and integrating, we have

$$\frac{dI_f}{dt}.e^{\left(\tau_f+\mu+\delta\right)t}+\left(\tau_f+\mu+\delta\right)I_f.e^{\left(\tau_f+\mu+\delta\right)t}=0\Rightarrow\int_0^t\frac{d}{dt}\left(I_f e^{\left(\tau_f+\mu+\delta\right)t}\right)=\int_0^t 0dt$$

$$I_f\left(t\right)e^{\left(\tau_f+\mu+\delta\right)t}=C \tag{23}$$

where C is a constant

With initial conditions, $t=0$ and $I_f\left(0\right)=I_0^f,\Rightarrow C=I_0^f$

$$\therefore I_f\left(t\right)e^{\left(\tau_f+\mu+\delta\right)t}=I_0^f \tag{24}$$

Taking limit as $t\to\infty, I_f\left(t\right)=I_0^f e^{-\left(\tau_f+\mu+\delta\right)t}\geq 0$ (25)

Similarly, form equation (5) we have

$$\Rightarrow\frac{dB}{dt}\geq-\rho B$$

$$\frac{dB}{dt}+\rho B=0 \tag{26}$$

Multiply equation (26) through by the integrating factor (IF) and integrating, we have

$$\frac{dB}{dt}.e^{\rho t}+\rho B.e^{\rho t}=0$$

$$\int_0^t\frac{d}{dt}\left(Be^{\rho t}\right)=\int_0^t 0dt$$

$$B\left(t\right)e^{\rho t}=C \tag{27}$$

where C is a constant

With initial conditions, $t=0$ and $\mathrm{B}\left(0\right)=B_0,\Rightarrow C=B_0$

$$\therefore B\left(t\right)e^{\rho t}=B_0 \tag{28}$$

Taking limit as $t\to\infty,\quad B\left(t\right)=B_0e^{-\rho t}\geq 0$ (29)

Similarly, from equation (6), we have

$$\Rightarrow\frac{dR_m}{dt}\geq-\left(\omega_m+\mu\right)R_m$$

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$$\frac{dR_m}{dt}+\left(\omega_m+\mu\right)R_m=0 \tag{30}$$

Multiply equation (30) through by the integrating factor (IF) and integrating, we have

$$\frac{dR_m}{dt}.e^{(\omega_m+\mu)t}+\left(\omega_m+\mu\right)R_m.e^{(\omega_m+\mu)t}=0\Rightarrow\int_0^t\frac{d}{dt}R_me^{(\omega_m+\mu)t}=\int_0^t 0\,dt$$

$$R_m\left(t\right)e^{(\omega_m+\mu)t}=C \tag{31}$$

where C is a constant

With initial conditions,

$$t=0 \text{ and } \mathrm{R}_m\left(0\right)=R_0^m,\Rightarrow C=R_0^m$$

$$\therefore R_m\left(t\right)e^{(\omega_m+\mu)t}=R_0^m \tag{32}$$

Taking limit as $t\to\infty$,

$$R_m\left(t\right)=R_0^me^{-(\omega_m+\mu)t}\geq 0 \tag{33}$$

Finally from equation (7) we have

$$\Rightarrow\frac{dR_f}{dt}\geq-\left(\omega_f+\mu\right)R_f$$

$$\frac{dR_f}{dt}+\left(\omega_f+\mu\right)R_f=0 \tag{34}$$

Multiply equation (34) through by the integrating factor (IF) and integrating, we have

$$\frac{dR_f}{dt}.e^{\left(\omega_f+\mu\right)t}+\left(\omega_f+\mu\right)R_f.e^{\left(\omega_f+\mu\right)t}=0\Rightarrow\int_0^t\frac{d}{dt}R_fe^{(\omega f+\mu)t}=\int_0^t 0$$

$$R_f\left(t\right)e^{\left(\omega_f+\mu\right)t}=C \tag{35}$$

where C is a constant

With initial conditions,

$$t=0 \text{ and } \mathrm{R}_f\left(0\right)=R_0^f,\Rightarrow C=R_0^f$$

$$\therefore R_f\left(t\right)e^{\left(\omega_f+\mu\right)t}=R_0^f \tag{36}$$

Taking limit as $t\to\infty$

$$R_f\left(t\right)=R_0^fe^{-\left(\omega_f+\mu\right)t}\geq 0 \tag{37}$$

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Since the function is always positive and the initial population of susceptible individual $S_m(0)$ and $S_f(0)$ is assumed to be non-negative. Therefore $S_m(t_n)$ and $S_f(t_n)$ are always non-negative for all t. Hence, we have that $S_m(0) > 0,\ S_f(0) > 0,\ I_m(0) > 0,\ I_f(0) > 0,\ R_m(0) > 0,\ R_f(0) > 0,\ B(0) > 0,$

➢ *Invariant Region of the Model.*

The system of Equation (1) - (7) have solutions which are contained in the feasible region. We obtained invariant region from equation (8), the total population $N(t)$;

Now,

$$\frac{dN(t)}{dt} = \frac{dS_m(t)}{dt} + \frac{dS_f(t)}{dt} + \frac{dI_m(t)}{dt} + \frac{dI_f(t)}{dt} + \frac{dR_m(t)}{dt} + \frac{dR_f(t)}{dt} + \frac{dB(t)}{dt} \tag{38}$$

Substituting equation (1) to equation (7) into (8) we have

$$\frac{dN(t)}{dt} = \beta N - \mu S_m + \beta N - \mu S_f - \left(\tau_m + e\left(1-C_{s_m}\right) + \mu + \delta\right) I_m - \left(\tau_f + e\left(1-C_{s_f}\right) + \mu + \delta\right) I_f - \left(\omega_m + \mu\right) R_m - \left(\omega_f + \mu\right) R_f - \rho B$$

When $\tau_m = \tau_f = e\left(1-C_{s_m}\right) = e\left(1-C_{s_f}\right) = \delta = \omega_m = \omega_f = \rho = o$

$$\frac{dN(t)}{dt} = \beta N - \mu S_m + \beta N - \mu S_f - \mu I_m - \mu I_f - \mu R_m - \mu R_f - B$$

$$\frac{dN(t)}{dt} = \beta N - \left(S_m + S_f + I_m + I_f + R_m + R_f + B\right)\mu$$

$$\frac{dN(t)}{dt} = \beta N - N\mu \tag{39}$$

Solving (39) when $\frac{dN(t)}{dt} = 0$, we have

$$N = \frac{\beta N}{\mu} \quad \Omega = \left\{\left(S_m, S_f, I_m, +I_f, +R_m, +R_f, +B\right) \in \mathbb{R}_+^7 \ : 0 \le N \le \frac{\beta}{\mu}\right\}$$

Lemma (1)

The region $$\Omega = \left\{\begin{array}{l}\left(S_m, S_f, I_m, I_f, R_m, R_f B\right) \in \mathbb{R}_+^7 : 0 \le S_m(0), 0 \le S_f(0), 0 \le I_m(0), 0 \le I_f(0), 0 \le R_m(0), 0 \le R_f(0), 0 \le B(0), \\ S_m + S_f + I_m + I_f + R_m + R_f \ge \frac{\beta N}{\mu}\end{array}\right\}$$

where the model makes biological sense, it solution exist and shown to be positively invariant and globally attracting in $\mathbb{R}_+^7$ with respect to the system of ODE described in our model.

- *Existence of Cholera Disease Free Equilibrium (DFE) Point of The Model*

At the cholera disease free equilibrium (DFE), the human population is free of infection, therefore we let the disease compartment to equal zero, that is $I_m(t) = 0,\ I_f(t) = 0,\ R_m(t) = 0,\ R_f(t) = 0,\ B(t) = 0.$

The model Equations (1)-(7) are therefore solved by setting the derivatives (RHS) to zero. Hence, we have

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$$\beta N - \frac{\alpha_m \left(1 - C_{\omega_m}\right) B}{K + B} S_m + \omega_m R_m - \mu S_m = 0 \tag{40}$$

$$\beta N - \frac{\alpha_f \left(1 - C_{\omega_f}\right) B}{K + B} S_f + \omega_f R_f - \mu S_f = 0 \tag{41}$$

$$\frac{\alpha_m \left(1 - C_{\omega m}\right) B}{K + B} S_m - \left(\tau_m + e\left(1 - C_{sm}\right) + \mu + \delta\right) I_m = 0 \tag{42}$$

$$\frac{\alpha_f \left(1 - C_{\omega f}\right) B}{K + B} S_f - \left(\tau_f + e\left(1 - C_{sf}\right) + \mu + \delta\right) I_f = 0 \tag{43}$$

$$\tau_m I_m - \left(\omega_m + \mu\right) R_m = 0 \tag{44}$$

$$\tau_f I_f - \left(\omega_f + \mu\right) R_f = 0 \tag{45}$$

$$e\left(1 - C_{sm}\right) I_m + e\left(1 - C_{sf}\right) I_f - \rho \mathrm{B} = 0 \tag{46}$$

Substituting $I_m = I_f = R_m = R_f = B = 0$ into (35), we have

$$\beta N - \mu S_m(t) = 0$$

$$S_m(t) = \frac{\beta N}{\mu},\ S_f(t) = \frac{\beta N}{\mu}$$

Therefore,

$$E_0 = \left\{S_0^m(t), S_0^f(t), I_0^m(t), I_0^f(t), R_0^m(t), R_0^f(t), B_0(t)\right\}$$

$$E_0 = \left\{\frac{\beta N_m}{\mu}, \frac{\beta N_f}{\mu}, 0, 0, 0, 0, 0\right\} \tag{47}$$

- *Existence of Cholera Disease Endemic Equilibrium (DEE) Point for the Model*

Cholera-endemic refers to the persistent present of Vibrio cholera and its attendant symptoms such as diarrhea, dehydration, vomiting, leg crimp, and watery stool in the population. Therefore, the solution of the system of the model equation (1) – (7) where all the state variables are positive and denoted by $\boldsymbol{E}^*$ is given by

$$E^* = \left\{S_m^*(t), S_f^*(t), I_m^*(t), I_f^*(t), R_m^*(t), R_f^*(t), B^*(t)\right\} \tag{48}$$

We set the RHS of the system of equation (1)–(7) to zeros and let

$$\lambda_1 = \frac{\alpha_m \left(1 - C_{\omega_m}\right) B}{K + B},\ \lambda_2 = \frac{\alpha_f \left(1 - C_{\omega_f}\right) B}{K + B},$$

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we then have the following system of equations.

$$\beta N + \omega_m R_m - (\lambda_1 + \mu) S_m = 0 \quad (49)$$

$$\beta N + \omega_f R_f - (\lambda_2 + \mu) S_f = 0 \quad (50)$$

$$\lambda_1 S_m - (\tau_m + e(1 - C_{sm}) + \mu + \delta) I_m = 0 \quad (51)$$

$$\lambda_2 S_f - (\tau_f + e(1 - C_{sf}) + \mu + \delta) I_f = 0 \quad (52)$$

$$\tau_m I_m - (\omega_m + \mu) R_m = 0 \quad (53)$$

$$\tau_f I_f - (\omega_f + \mu) R_f = 0 \quad (54)$$

$$e(1 - C_{sm}) I_m + e(1 - C_{sf}) I_f - \rho B = 0 \quad (55)$$

From system of equations (49) - (55) we let

$$K_1 = \lambda_1 + \mu, \quad K_2 = \lambda_2 + \mu, \quad K_3 = \tau_m + e(1 - C_{sm}) + \mu + \delta$$

$$K_4 = \tau_f + e(1 - C_{s_f}) + \mu + \delta, \quad K_5 = \omega_m + \mu, \quad K_6 = \omega_f + \mu$$

$$K_7 = e(1 - C_{s_m}), \quad K_8 = e(1 - C_{s_f})$$

Substituting $K_1, K_2, K_3, \cdots K_8$ into the system of equations (49) – (55) we have

$$\beta N + \omega_m R_m - K_1 S_m = 0 \quad (56)$$

$$\beta N + \omega_f R_f - K_2 S_f = 0 \quad (57)$$

$$\lambda_1 S_m - K_3 I_m = 0 \quad (58)$$

$$\lambda_2 S_f - K_4 I_f = 0 \quad (59)$$

$$\tau_m I_m - K_5 R_m = 0 \quad (60)$$

$$\tau_f I_f - K_6 R_f = 0 \quad (61)$$

$$K_7 I_m + K_8 I_f - \rho B = 0 \quad (62)$$

Solving equations (57) - (62) simultaneously we have;

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$$S_m^* = \frac{\beta N K_3 K_5}{K_1 K_3 K_5 - \omega_m \lambda_1 \tau_m}, \quad I_m^* = \frac{\beta N \lambda_1 K_5}{K_1 K_3 K_5 - \omega_m \lambda_1 \tau_m}, \quad S_f^* = \frac{K_4 K_6 \beta N}{K_2 K_4 K_6 - \lambda_2 \tau_f \omega_f}$$

$$I_f^* = \frac{\beta N \lambda_1 K_5}{K_1 K_3 K_5 - \omega_m \lambda_1 \tau_m}, \quad R_m^* = \frac{\tau_m \beta N \lambda_1}{K_1 K_3 K_5 - \omega_m \lambda_1 \tau_m}, \quad R_f^* = \frac{\tau_f \beta N \lambda_2}{K_2 K_4 K_6 - \omega_f \lambda_1 \tau_f}$$

$$B^* = \frac{K_5 K_7 \beta N \lambda_1 \left(K_2 K_4 K_6 - \omega_f \lambda_2 \tau_f\right) + K_6 K_8 \beta N \lambda_2 \left(K_1 K_3 K_5 - \omega_m \lambda_1 \tau_m\right)}{\tau \left(K_1 K_3 K_5 - \omega_m \lambda_1 \tau_m\right)\left(K_2 K_4 K_6 - \omega_f \lambda_2 \tau_f\right)}$$

III. RESULTS

➢ *Computation of the Basic Reproduction Number* (R_0)*for the Cholera Transmission Model*

We follow the approach of [24] and [25] also refers to as the next generation matrix method. This procedure converts a system of ordinary differential equations of a model of infectious disease dynamics to an operator (or matrix) that translate from one generation of infectious individuals to the next. The basic reproduction number is then defined as the spectral radius (dominant eigenvalue) of this operator. We deduce that whether a disease becomes persistent or dies out in a community it totally depends on the value of the basic reproduction ratio R_0. Furthermore, stability of equillibria can be analyzed using $R_0 < 1$, which means that every infectious individual will cause less than one secondary infection and hence the disease will die out and when$R_0 > 1$, every infectious individual will cause more than one secondary infection and hence the disease will persist in the population.

A large value of R_0 may indicate the possibility of a major epidemic. For the case of a model with single infected class, R_0 is simply the product of the infection rate and the duration of the infection.

The basic reproduction ratio is obtained by taking the dominant eigenvalue of FV^{-1} given by equation (63) below

$$FV^{-1} = \left[\frac{\delta F_i(E_0)}{\delta x_j}\right]\left[\frac{\delta V_i(E_0)}{\delta x_j}\right]^{-1} \tag{63}$$

where F_i is the rate of appearance of new infection in compartment I_m and I_f, V_i is the transfer of infections from one compartment I_m and I_f to another, and E_0 is the disease free equilibrium. From the system of differential equations (1) – (7), we first identify the system of equations with infectious classes as rearranged the equation beginning with the cholera disease infected compartments.

$$\frac{dI_m}{dt} = \frac{\alpha_m\left(1-C_{\omega m}\right)B}{K+B} S_m - \left(\tau_m + e\left(1-C_{sm}\right) + \mu + \delta\right) I_m \tag{64}$$

$$\frac{dI_f}{dt} = \frac{\alpha_f\left(1-C_{\omega f}\right)B}{K+B} S_f - \left(\tau_f + e\left(1-C_{sf}\right) + \mu + \delta\right) I_f \tag{65}$$

$$\frac{dB}{dt} = e\left(1-C_{sm}\right) I_m + e\left(1-C_{sf}\right) I_f - \rho \mathrm{B} \tag{66}$$

$$\frac{dS_m}{dt} = \beta N - \frac{\alpha_m\left(1-C_{\omega m}\right)B}{K+B} S_m + \omega_m R_m - \mu S_m \tag{67}$$

$$\frac{dS_f}{dt} = \beta N - \frac{\alpha_f\left(1-C_{\omega f}\right)B}{K+B} S_f + \omega_f R_f - \mu S_f \tag{68}$$

$$\frac{dR_m}{dt} = \tau_m I_m - \left(\omega_m + \mu\right) R_m \tag{69}$$

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$$\frac{dR_f}{dt} = \tau_f I_f - \left(\omega_f + \mu\right) R_f \tag{70}$$

Using the method in [24], [25] and Lemma (1) We split equation (1) – (7) into new cases of infection and other cases of infection denoted by F and V.

$$F_i = \begin{bmatrix} \frac{\alpha_m \left(1 - C_{\omega m}\right) B}{K + B} S_m \\ \frac{\alpha_f \left(1 - C_{\omega_f}\right) B}{K + B} S_f \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \tag{71}$$

Similarly,

$$V_i = \begin{bmatrix} \left(\tau_m + \mu + \delta\right) I_m \\ \left(\tau_f + \mu + \delta\right) I_f \\ -e\left(1 - C_{s_m}\right) I_m - e\left(1 - C_{s_f}\right) I_f + \rho B \\ 0 \\ 0 \\ 0 \end{bmatrix} \tag{72}$$

We then find the partial derivative of equation (1) - (7) with respect to all compartment, starting with the infected classes,

$$F = \begin{bmatrix} 0 & 0 & -\frac{\alpha_m \left(1 - C_{\omega_m}\right) S_m^0}{K} \\ 0 & 0 & -\frac{\alpha_f \left(1 - C_{\omega_f}\right) S_f^0}{K} \\ 0 & 0 & 0 \end{bmatrix} \tag{73}$$

$$V = \begin{bmatrix} \tau_m + \mu + \delta & 0 & 0 \\ 0 & \tau_f + \mu + \delta & 0 \\ -e\left(1 - C_{s_m}\right) & -e\left(1 - C_{s_f}\right) & \rho \end{bmatrix} \tag{74}$$

We find the inverse of equation (4.12) which gives us

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$$V^{-1}=\begin{bmatrix} \frac{1}{\tau_m+\mu+\delta} & 0 & 0 \\ 0 & \frac{1}{\tau_f+\mu+\delta} & 0 \\ \frac{-e\left(1-C_{s_m}\right)}{(\tau_m+\mu+\delta)\rho} & \frac{-e\left(1-C_{s_f}\right)}{(\tau_f+\mu+\delta)\rho} & \frac{1}{\rho} \end{bmatrix} \tag{75}$$

From equation (73) and equation (75), we let $M_1=\tau_m+\mu+\delta$, $M_2=\tau_f+\mu+\delta$, $M_3=-e\left(1-C_{s_m}\right)$,

$M_4=-e\left(1-C_{s_f}\right)$, $W_1=\frac{\alpha_m\left(1-C_{w_m}\right)}{K}$, $W_2=\frac{\alpha_f\left(1-C_{w_f}\right)}{K}$

$S_f=S_f^0$, $S_m=S_m^0$

$$\therefore FV^{-1}=\begin{bmatrix} \frac{W_1S_m^0M_3}{M_1\rho} & \frac{W_1S_m^0M_4}{M_2\rho} & -\frac{W_1S_m^0}{\rho} \\ \frac{W_2S_f^0M_3}{M_1\rho} & \frac{W_2S_f^0M_4}{M_2\rho} & -\frac{W_2S_f^0}{\rho} \\ 0 & 0 & 0 \end{bmatrix} \tag{76}$$

From the eigenvalue of equation (76), we obtained the basic reproduction number R_0 which is the spectral radius (dominant eigenvalue) as

$$R_0=\frac{S_f^0\omega_fM_1M_4+S_m^0\omega_mM_2M_3}{M_1M_2\rho} \tag{77}$$

➢ *Global Stability of Disease-Free Equilibrium (E_0) of the Cholera Model.*

Theorem 2 The DFE of the Model equation (1) – (7) is globally asymptomatically stable (GAS) in Ω whenever $R_0=\max(FV^{-1})\le 1$.

- Proof: Let consider the model equation (1) – (7) and the linear Lyapunov function.

$$V=\omega_fM_1I_f+\omega_mM_2I_m+M_1M_2B \tag{78}$$

$$=\omega_fM_1\left[\alpha_f\left(\frac{1-C_{\omega_f}}{K+B}\right)S_f-\left(I_f+K_2+\mu+\delta\right)I_f\right]$$

$$+\omega_mM_2\left[\left(\frac{\alpha_m\left(1-C_{\omega_m}\right)}{K+B}\right)S_m-\left(I_m+K_1+\mu+\delta\right)I_m\right]+M_1M_2\left[K_1I_m+K_2I_f-\rho B\right] \quad . \tag{79}$$

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$$= \omega_f M_1 \left[\alpha_f \left(\frac{1-C_{\omega_f}}{K+B} \right) S_f - \left(M_2 + K_2 \right) I_f \right]$$

$$+\omega_m M_2 \left[\left(\frac{\alpha_m \left(1-C_{\omega_m} \right)}{K+B} \right) S_m - \left(M_1 + K_1 \right) I_m \right] + M_1 M_2 \left[K_1 I_m + K_2 I_f - \rho B \right] \quad . \quad . \quad (80)$$

$$= \omega_f M_1 \alpha_f \frac{\left(1-C_{\omega_f} \right)}{K+B} S_f - \omega_f M_1 \left(M_2 + K_2 \right) I_f$$

$$+ \frac{\omega_m M_2 \alpha_m \left(1-C_{\omega f} \right)}{K+B} S_m - \omega_m M_2 \left(M_1 + K_1 \right) I_m + M_1 M_2 K_1 I_m + K_2 I_f M_1 M_2 - M_1 M_2 \rho B \quad (81)$$

Where $K_1 = \alpha_m \left(1-C_{\omega_m} \right)$ and $K_2 = \alpha_f \left(1-C_{\omega_f} \right)$

$$= \frac{1}{K+B} \left[\omega_f M_1 \alpha_f \left(1-C_{\omega_f} \right) S_f - \omega_m M_2 \alpha_m \left(1-C_{\omega m} \right) S_m \right] +$$

$$- \omega_f M_1 I_f \left(M_2 - K_2 \right) - K_2 I_f M_1 M_2 \omega_m M_2 I_m \left(-M_1 + K_1 \right) + K_1 M_1 M_2 I_m - M_1 M_2 \rho B \quad (82)$$

$$= \frac{1}{K+B} \left[\omega_f M_1 \alpha_f \left(1-C_{\omega_f} \right) S_f + \omega_m M_2 \alpha_m \left(1-C_{\omega m} \right) S_m \right] - \omega_f M_1 I_f \left(M_2 - K_2 \right) + \omega_m M_2 I_m \left(-M_1 + K_1 \right)$$

$$= \frac{M_1 M_2 \rho}{K+B} \left[\frac{\omega_f M_1 \alpha_f \left(1-C_{\omega_f} \right) S_f + \omega_m M_2 \alpha_m \left(1-C_{\omega_m} \right) S_f}{M_1 M_2 \rho} - \frac{\omega_f I_f \left(M_2 + K_2 \right)}{M_2 \rho} + \frac{\omega_m I_m \left(K_1 - M_1 \right)}{M_1 \rho} + \frac{K_2 I_f + K_1 I_m - \rho B}{\rho} \right] \quad (83)$$

Since $S_m \le N_m$ and $S_f \le N_f$ in the domain that forms the invariant set Ω, it follows that

$$\dot{V} \le \frac{M_1 M_2 \rho}{K+B} \left(R_0 - K_3 \right) \quad . \quad . \quad . \quad (84)$$

Where $K_3 = \frac{\omega_f I_f \left(M_2 + K_2 \right)}{M_2 \rho} - \frac{\omega_m I_m \left(K_1 - M_1 \right)}{M_1 \rho} - \frac{\left(K_2 I_f + K_2 I_m - \rho B \right)}{\rho}$

Hence, $\dot{V} \le 0$ whenever $R_0 = \max(FV^{-1})$ and all parameters and variables of the model equations (3.1) – (3.7) are non-negative, it follows that $\dot{V} \le 0$ when $R_0 < 1$ and $\dot{V} = 0$ if and only if $I_f = I_m = B = 0$.

Therefore, V is a Lyapunov function in Ω. Thus, it follows from Lasalle's invariance principle [26] that $\{I_f(t), I_m(t), B(t)\} \to (0,0,0)$ as $t \to \infty$. This result shows that, for the case of infected male, infected female and concentration of Vibrio cholera population; the DFE of the model (1) – (7) is GAS whenever $R_0 \le 1$. Hence, Cholera will be eliminated from the population.

➢ *Sensitivity Analysis of the Model Parameters*

The sensitivity analysis aims to identify which parameters most strongly influence the spread of cholera within the proposed mathematical model. This step is crucial in understanding how variations in model parameters, arising from measurement errors or uncertainties in data affect the accuracy and stability of the model's predictions. Essentially, sensitivity analysis helps highlight which parameters should be prioritized in intervention or control strategies.

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Following the forward sensitivity index method outlined by [27], denoted by $S_\omega = \frac{\omega}{R_0} \times \frac{\partial R_0}{\partial \omega}$, where ω is the given parameter in R_0, each model parameter's sensitivity was quantified in relation to the basic reproduction number (R_0). The results reveal that parameters with positive sensitivity values ($S_\omega > 0$), such as male compliance with environmental sanitation and the bacterial contribution rate from infected individuals enhance disease transmission when increased. Conversely, parameters with negative sensitivity indices ($S_\omega < 0$), such as male and female recovery rates, bacterial growth rate in the aquatic environment, and cholera-induced death rate indicate that improvements or increases in these parameters reduce the value of R_0, thereby curbing transmission.

Table 2 Sensitivity Indices for the Model Parameter

Parameter	Sensitivity Indices Value	Sign
$\boldsymbol{\tau_m}$	-0.534	Negative
$\boldsymbol{\tau_f}$	-0.6356	Negative
$\boldsymbol{\omega_1}$	0.0000	-
$\boldsymbol{\omega_2}$	0.0000	-
$\boldsymbol{C_{s_m}}$	0.00004	Positive
$\boldsymbol{C_{s_f}}$	0.000159	Positive
$\boldsymbol{\rho}$	-0.00025	Negative
$\boldsymbol{\mu}$	-0.016716	Negative
$\boldsymbol{e}$	0.970370	Positive
$\boldsymbol{\delta}$	-0.000025	Negative

Graphical analysis of these sensitivity indices (Figure 2) reinforces these trends, showing how small variations in parameter values can either amplify or suppress cholera spread. The findings underscore the importance of targeted interventions especially promoting sanitation compliance and treatment recovery to effectively control cholera outbreaks.

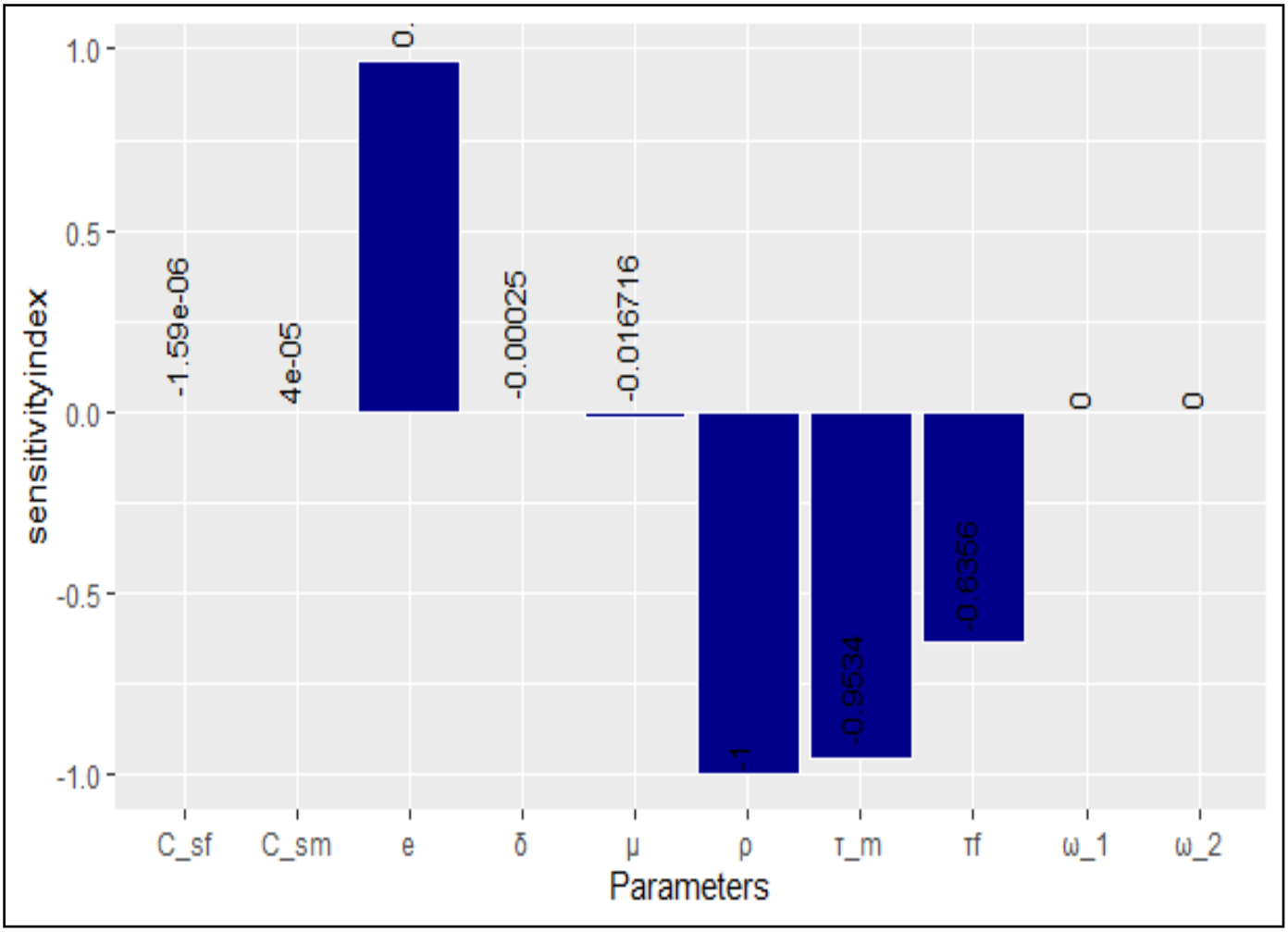

Fig2 Sensitivity Index Plot for the Model Parameter

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➢ *Numerical Simulation of the Model*

The Cholera Disease Transmission Model in the previous section, demonstrated in the model equation (1) – (7) was solved numerically using Rung –Kutta Fehiiberg 5th order method and implemented using Maple 17 software. The parameters used in the implementation of the model are shown in Table 3 below. Parameters were chosen in consonance with the threshold values obtained in the stability analysis of both the disease-free equilibrium and the endemic equilibrium state of the model.

Table 3 Estimated values of the parameters used in the Numerical simulations

Variables/ Parameters	Values	Source	Parameters	Values	Source
$S_m(0)$	5750	[36]	K	1,000,000,000	[38]
$S_f(0)$	6000	[36]	τ_m	0.3	Assumed
$I_m(0)$	25	Assumed	τ_f	0.2	[39]
$I_f(0)$	100	Assumed	C_{ω_m}	0.4 year^{-1}	Assumed
$B(0)$	2750	[36]	C_{ω_f}	0.5 year^{-1}	[37]
$R_m(0)$	0	[36]	C_{s_m}	(0 – 2) year^{-1}	Assumed
$R_f(0)$	0	[41]	C_{s_f}	(0 – 1) year^{-1}	[40]
$N(t)$	15625	Assumed	δ	0.00008 day^{-1}	[41]
β	0.017	[23]	ω_m	0.7 year^{-1}	Assumed
μ	0.0000526 day^{-1}	[37]	ω_f	0.5 year^{-1}	[37]
$\boldsymbol{\alpha_m}$	0.05	Assumed	e	10 cell/ml/day	[38]
$\boldsymbol{\alpha_f}$	0.1	[37]	ρ	0.54year^{-1}	[40]

➢ *List of Numerical Experiment of the Model*

The following numerical experiments are carried out;

- To determine the effect of Cholera transmission when the hygiene incidence rate of male is greater than female $(\alpha_m < \alpha_f)$ and the rate of compliance with water hygiene of male is greater than female $(C_{\omega m} > C_{\omega f})$.
- To determine the effect of cholera transmission when the hygiene incidence rate of female is greater than male $(\alpha_f < \alpha_m)$ and the rate of compliance with water hygiene of female is greater than male $(C_{\omega f} > C_{\omega m})$.
- To determine the effect of cholera transmission when the hygiene incidence rate of male is equal with female $(\alpha_m = \alpha_f)$ and the rate of compliance with water hygiene of male is equal to female $(C_{\omega m} = C_{\omega f})$.
- To determine the effect of high treatment rate on Cholera transmission when the rate of compliance of both male and female with environmental sanitation $(C_{\omega m}, C_{\omega f})$ is high and the contribution rate of infected persons to the population of Vibrio cholera in the aquatic environment e is also high
- To determine the effect of high rate on Cholera transmission when the rate of compliance of both male and female with environmental sanitation $(C_{\omega m}, C_{\omega f})$ is high and the contribution rate of infected persons to the population of Vibrio cholera in the aquatic environment e is low.

➢ *Graphical Representation of Results*

- Experiment 1: The effect of treatment on Cholera transmission when the hygiene incidence rate of male is greater than female $(\alpha_m < \alpha_f)$ and the compliance with water hygiene of male is greater than female $(C_{\omega m} > C_{\omega f})$

$(\alpha_m = 0.01,\ \alpha_f = 0.003,\ C_{\omega m} = 0.4,\ C_{\omega f} = 0.2)$

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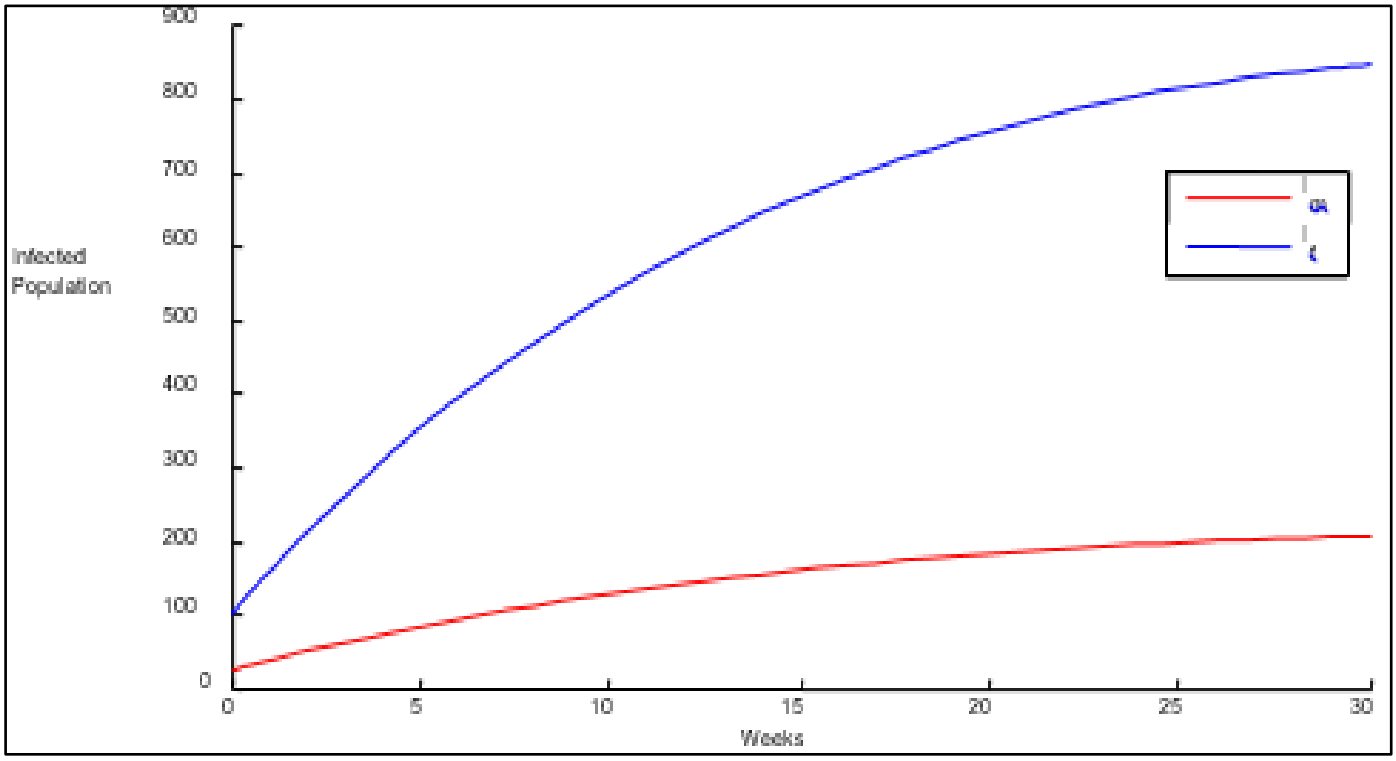

Fig 3 Prevalence of Cholera Infection when the Hygiene Incidence Rate of Male is Greater than Female.

$\left(\alpha_m = 0.01,\ \alpha_f = 0.003\ ,\ C_{\omega m} = 0.4,\ C_{\omega f} = 0.2\right)$

- Experiment 2: The effect of treatment on cholera transmission when the hygiene incidence rate of female is greater than male $\left(\alpha_f < \alpha_m\right)$ and the rate of compliance with water hygiene of female is greater than male $\left(C_{\omega f} > C_{\omega m}\right)$

$\left(\alpha_m = 0.04,\ \alpha_f = 0.01\ ,\ C_{\omega m} = 0.02,\ C_{\omega f} = 0.04\right)$

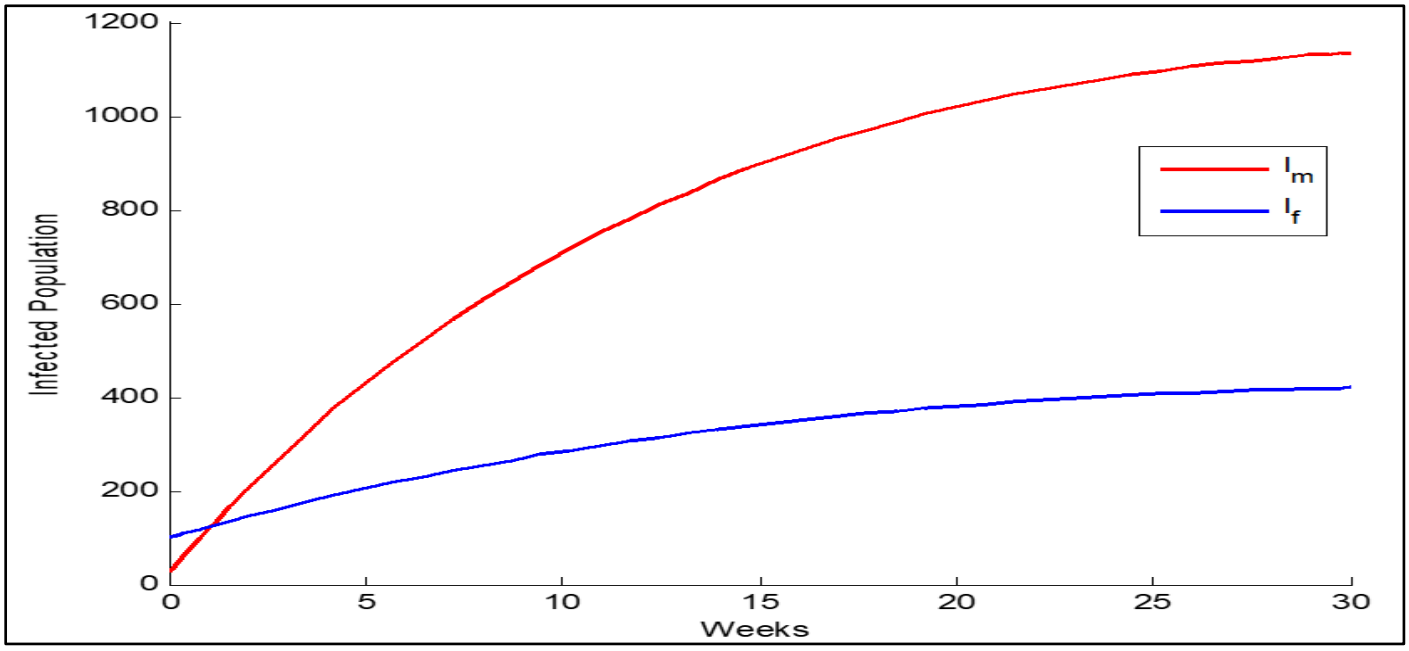

Fig 4 Prevalence of Cholera Infection When the Hygiene Incidence Rate of Female is Greater than Male

$\left(\alpha_m = 0.04,\ \alpha_f = 0.01\ ,\ C_{\omega m} = 0.02,\ C_{\omega f} = 0.04\right)$

- Experiment 3: The effect of cholera treatment rate on cholera transmission when the hygiene incidence rate of

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male is equal with female $\left(\alpha_m = \alpha_f\right)$ and the rate of compliance with water hygiene of male is equal to female $\left(C_{\omega m} = C_{\omega f}\right)$

$\left(\alpha_m = \alpha_f = 0.04 \text{ , } C_{\omega m} = C_{\omega f} = 0.02\right)$

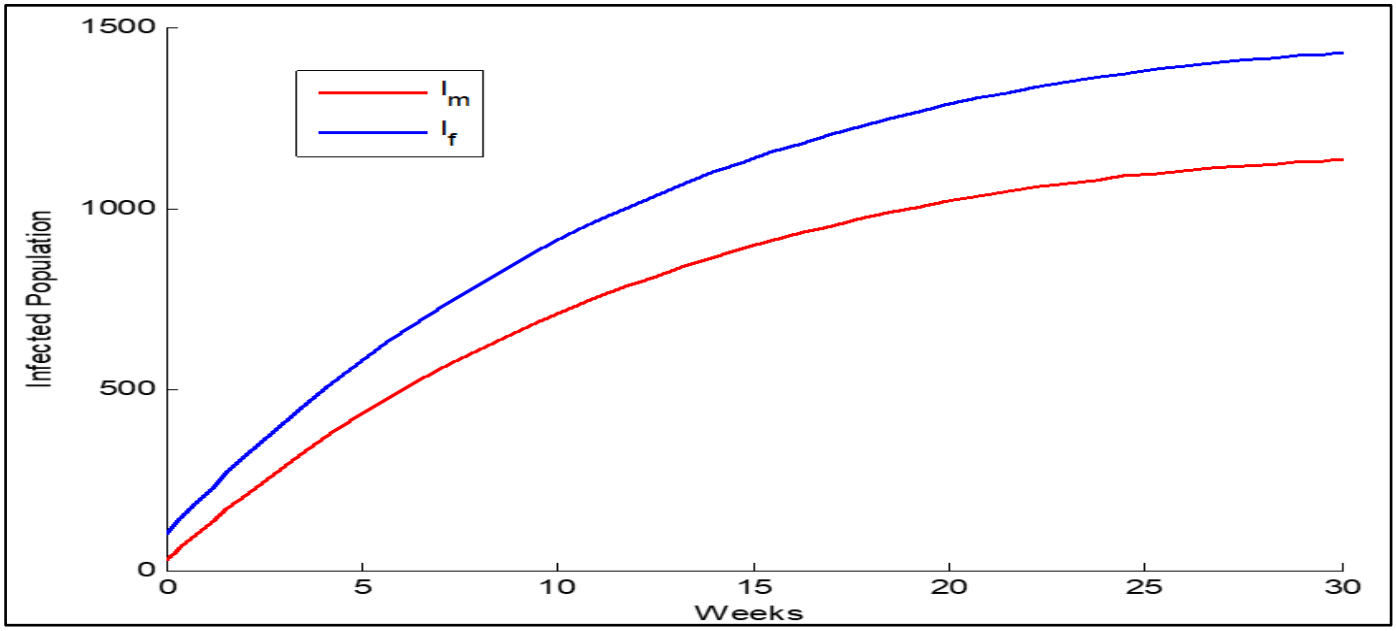

Fig 5 Prevalence of Cholera Infection when the Hygiene Incidence Rate of Male is Equal with Female

$\left(\alpha_m = \alpha_f = 0.04,\ C_{\omega m} = C_{\omega f} = 0.02\right)$

- Experiment 4: The effect of high treatment rate on Cholera transmission when the rate of compliance of both male and female with environmental sanitation $\left(C_{sm},\ C_{sf}\right)$ is high and the contribution rate of infected persons to the population of Vibrio cholera in the aquatic environment e is also high $\left(\tau_m = \tau_f = 0.09,\ C_{\omega m} = C_{\omega f} = 0.09 \text{ and } e = 0.10\right)$

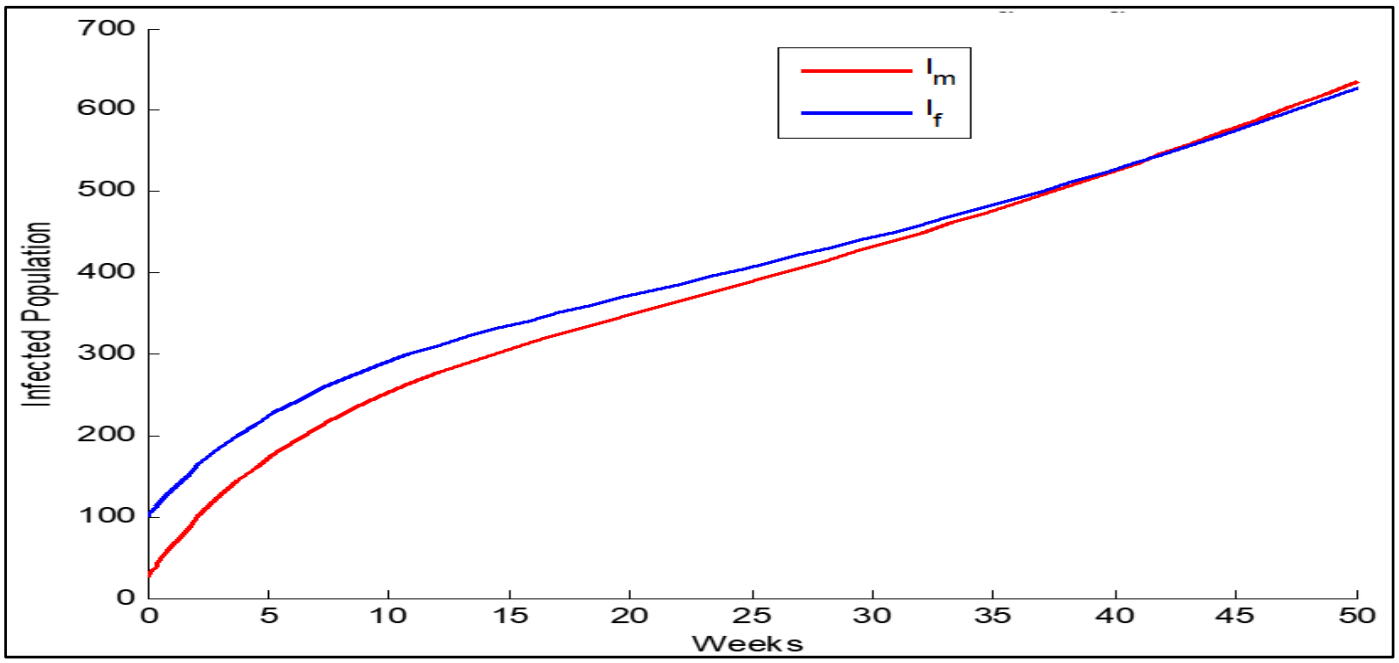

Fig 6 Prevalence of Cholera Infection Treatment Rate when the Rate of Compliance of Both Male and Female with Environmental Sanitation $\left(C_{sm},\ C_{sf}\right)$ is High and the Contribution Rate of Infected Persons to the Population of Vibrio Cholera in the Aquatic Environment e is Also High $\left(\tau_m = \tau_f = 0.09,\ C_{\omega m} = C_{\omega f} = 0.09 \text{ and } e = 0.10\right)$

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- Experiment 5: The effect of high treatment rate on Cholera transmission when the rate of compliance of both male and female with environmental sanitation $\left(C_{sm},\ C_{sf}\ \right)$ is high and the contribution rate of infected persons to the population of Vibrio cholera in the aquatic environment e is low $\left(\tau_m = \tau_f = 0.09,\ \mathrm{C}_{\omega m} = \mathrm{C}_{\omega f} = 0.09 \text{ and } e = 0.00001\right)$

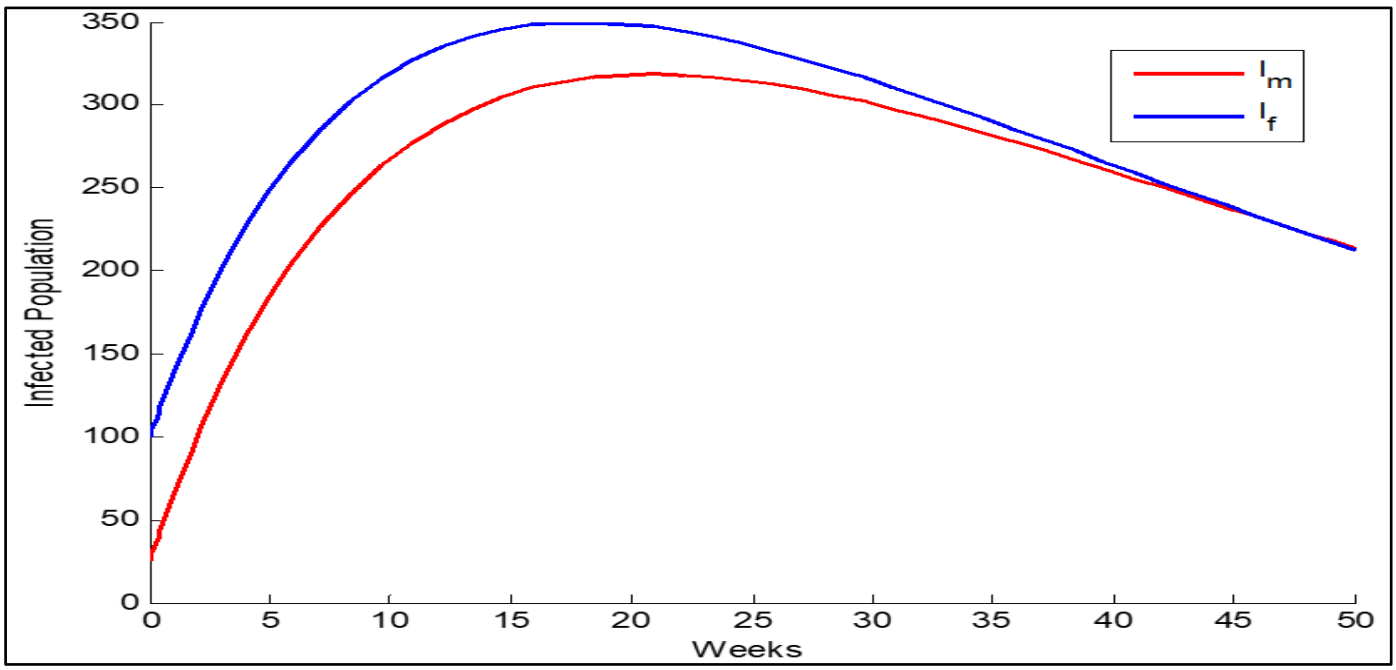

Fig 7 The Prevalence of Cholera Infection when the Rate of Compliance of Both Male and Female Population with Environmental Sanitation $\left(C_{sm},\ C_{sf}\ \right)$ is High and the Contribution Rate of Infected Persons to the Population of Vibrio Cholera in the Aquatic Environment e is Low $\left(\mathrm{C}_{\omega m} = \mathrm{C}_{\omega f} = 0.09\ ,\ e = 0.00001\right)$

IV. DISCUSSION

We provide a thorough examination of the simulation and numerical experiment results from the gender-hygiene-driven cholera model. The simulations investigate how disease dynamics are impacted by environmental pollution, gender-specific exposure rates, and behavioral hygiene compliance. The findings illustrate the intricate relationship between social structure and the spread of disease, and their validity and originality are demonstrated by a critical comparison with previous research.

➢ *Gender and Hygiene Compliance as a Behavioral Driver of Infection*

Looking at Figure 3 and Figure 4, under differential hygiene standards (that is the model incorporated unequal exposure due to gender-specific societal roles), the simulation results show a considerable variation in the transmission of cholera between genders. According to the model, female's infection burden is higher than that of males when their hygiene compliance is weaker (for example, as a result of caring responsibilities or exposure during water collection) as shown in Figure 3. This is consistent with the public health findings of [19], who found that poor sanitation access and caring exposure increase the incidence of diarrheal illness among women in Cape Town's informal settlements. Also, [28] demonstrated that preventive hygiene behaviors, especially access to safe water and hand washing, led to the extinction of cholera in high compliance scenarios. [22] similarly argued that hygiene-based interventions are most effective when behavior change is included, which aligning with our use of gender-modulated behavior functions in our model

Also from Figure 5, simulations reveals that when hygiene compliance is equal in both gender, females still suffer a higher peak infection rate due to differential contact parameters. This corroborate [21], who observed that the role of women's exposure in household sanitation disproportionately exposed women to health risks, especially waterborne disease like cholera due to their social and domestic roles like fetching water, caring for sick children, cooking and cleaning. Other recent studies that validate our model include [29] emphasized on the policy value of gender-differentiated hygiene strategies in India and [30] reported that gender-focused WASH programmes in refugee camp reduced cholera cases more rapidly than when general intervention is employed. This align with our model findings that cholera control is most effective when informed by gender-specific behavioral pattern.

➢ *Bacterial Concentration in Water Equals Environmental Persistence*

The environmental class $B(t)$ representing the concentration of Vibrio cholerae in water, introduces a feedback loop in the model. Simulation graphs (Figures 4 and

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Figure 6) show that if bacterial decay is slow (low), even small hygiene lapses can cause significant outbreaks. This phenomenon corresponds with real-life outbreaks in Haiti and Zimbabwe, where contaminated water persisted even after human cases declined as reported in [31]. [32] similarly modeled bacterial regrowth in water and stressed the importance of environmental disinfection alongside human interventions, noting that if bacteria persist in water, outbreaks can continue or will reappear in future, even if people recover. Hence, improving hygiene alone is not enough, water treatment is essential to break the transmission cycle of cholera.

- *Impact of Contaminated Water Reservoir*

The reservoir of Vibrio cholerae $B(t)$ drives secondary infection cycles. When bacterial decay is low, simulations show a prolonged epidemic tail even after infected human compartments decline [32]. Variable population SIRB model emphasized the long-term effect of aquatic reservoirs and suggested chlorination and bacterial decay as critical parameters. [33] and [34] showed similar feedback loops between water contamination and infection, but did not disaggregate much gender impacts. Looking at Figure 6, even when the treatment rate for cholera is extremely high and the compliance of both male and female with environmental sanitation is high, if the contribution rate of infected persons to the population of Vibrio cholera in the aquatic environment is also high, cholera become an endemic and persist over time. But when the contribution rate of infected person to the population of Vibrio cholera in the aquatic environment is low, then the prevalence of infection grows shortly and then decline.

- *Result from Basic Reproduction Number and Sensitivity Analysis*

The basic reproduction number R_0 was derived and used to access the outbreak potential of cholera. The simulation revealed as shown in figure 2 that R_0 is highly sensitive to three key parameters namely rate of compliance of male with environmental sanitation, rate of compliance of female with environmental sanitation, and the rate of contribution of infected persons to the aquatic environment (e). Sensitivity analysis using Latin Hypercube Sampling confirm that hygiene-related parameter are most influential. This finding support the conclusion of [35] whose model similarly showed hygiene and water-related variables as the most impactful in cholera related dynamics.

V. CONCLUSION

This study developed and analyzed a deterministic cholera transmission model that integrates gender-specific hygiene behaviors within a compartmental SIRB framework. By stratifying the population into male and female groups with distinct hygiene compliance rates, the model captures the social dynamics influencing exposure risk. It further incorporates an environmental reservoir to reflect the persistence of Vibrio cholerae in contaminated water sources, a key driver of sustained outbreaks.

Findings reveal that gender differences in hygiene practices significantly affect cholera prevalence. Women, due to their domestic and caregiving roles, experience higher infection risks, highlighting the importance of gender-sensitive interventions. The results also show that when community hygiene compliance exceeds approximately sixty percent, infection rates decline sharply, underscoring behavioral change as a critical control mechanism. Sensitivity analysis identified hygiene compliance, bacterial decay, and recovery rates as the most influential parameters determining the basic reproduction number (Ro).

The study contributes to mathematical epidemiology by introducing a socially informed modeling framework that links behavioral patterns to disease dynamics. It demonstrates that cholera control extends beyond biomedical interventions to include education, gender equity, and sanitation behavior.

For policy and practice, the findings advocate scaling up hygiene education programs, targeting women and children, ensuring continuous water treatment, and incorporating gender-disaggregated data into public-health planning.

Future extensions may include stochastic or spatial versions of the model to capture random or regional variations in transmission. In essence, this research confirms that cholera is not only an environmental and biological disease but also a behavioral one—requiring integrated strategies that combine gender-sensitive hygiene promotion with sustained environmental sanitation for effective long-term control.

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