MIKAILALSYS
Journal of Multidisciplinary Sciences

e-ISSN : 2987-2286
p-ISSN : 2987-3924

Index: Harvard, Boston, Sydney, Copernicus, Sinta 4, Scilit, Semantic, Google, etc

https://doi.org/10.58578/mikailalsys.v4i3.9373

Mathematical Analysis of Cholera Transmission Incorporating Media Coverage

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Article Info:

Submitted:	Revised:	Accepted:	Published:
Feb 11, 2026	May 9, 2026	Jul 23, 2026	Aug 7, 2026

Abstract

Cholera is an acute gastrointestinal disease caused by the bacterium *Vibrio cholerae* and is primarily transmitted through the consumption of food or water contaminated with fecal matter from infected individuals. Given its rapid transmission, widespread occurrence, and potentially fatal consequences, effective prevention and control require integrated interventions, including vaccination, water chlorination, and appropriate treatment. This study aims to develop and analyze a mathematical model of cholera transmission that incorporates the effect of media coverage on disease dynamics. The model was formulated using a system of differential equations, and its stability properties were examined by deriving the basic reproduction number, (R_0), through the next-generation matrix method. Numerical simulations were subsequently conducted to assess the influence of media awareness on cholera transmission. The findings indicate that increased media awareness substantially reduces the number of cholera cases, demonstrating the important role of public health communication in limiting disease spread. These results suggest that integrating media campaigns with established cholera prevention and control measures can

strengthen efforts to reduce outbreaks. This study contributes a mathematical framework for understanding the role of media coverage in cholera transmission dynamics and provides practical insights for incorporating public health communication into disease-control strategies.

Keywords: Cholera Transmission; Differential Equations; Mathematical Modeling; Media Awareness; Numerical Simulation

Introduction

Cholera is an infectious disease caused by the bacterium Vibrio cholerae, which attacks the gastrointestinal system and can lead to severe dehydration, with life-threatening complications possible if left untreated (Harris et al., 2012). The disease spreads mainly through contaminated food or water, making it prevalent in regions with poor sanitation and inadequate access to clean drinking water (Sack et al., 2004; World Health Organization, 2015). Biologically, Vibrio cholerae is a gram-negative, comma-shaped bacterium that thrives in aquatic environments, particularly in brackish waters and estuaries, where it can attach to plankton and other organisms, facilitating its persistence in the environment (Colwell, 1996). Due to the rapid spread and severe health consequences of cholera, effective public health interventions are crucial for disease control. Traditional cholera prevention strategies have focused on improving water quality, sanitation, and hygiene practices (Nelson et al., 2009). However, recent research has pointed out the role of media campaigns in disease prevention through influencing public behavior and increasing awareness of sanitation and hygiene practices (Levy et al., 2019). The media can also play a crucial role in reducing the transmission of diseases through behavioral changes, increased vaccine uptake, and increased use of safe drinking water sources, as evidenced by studies (Funk et al., 2019; Mahbub et al., 2022). Media-influenced mathematical models have also shown their effectiveness in the control of infectious diseases by changing public perception and exposure risk (Teng et al., 2021).

Due to the complexities of cholera transmission dynamics include biological, environmental, and social factors, mathematical modeling becomes necessary (Tuite et al., 2011). Epidemiological models allow quantification of key parameters such as basic reproduction number (R_0), assessment of effects of intervention strategies, including those involving media campaigns (Mukandavire et al., 2013). Other works include Hanani et al.,

media interventions in cholera control 2005; Chaturvedi et al., 2015.

Objectives of the Study

In this study, we aimed to:

i. Formulate a mathematical model for cholera transmission by incorporating the effect of media coverage.

ii. Determine the basic reproduction number R_0 of the proposed model.

iii. Perform stability analysis of the developed cholera transmission model.

iv. Obtain numerical solutions of the proposed model

Cholera Transmission Model with Media Coverage

In this work, we present the Susceptible (S), Exposed (E), Infected (I), and Recovered (R) model for the transmission of cholera outbreak within a community. Thus, we formulated the following equations:

$$\frac{dS}{dt} = \pi - \big(C_1 - C_2 f(I)\big)S \cdot I - \mu S \qquad (2.1.1)$$

$$\frac{dE}{dt} = \big(C_1 - C_2 f(I)\big)S \cdot I - (\lambda + \mu)E \qquad (2.1.2)$$

$$\frac{dI}{dt} = \lambda E - (\gamma + \mu)I \qquad (2.1.3)$$

$$\frac{dR}{dt} = \gamma I - \mu R \qquad (2.1.4)$$

Where:

$$f(I) = \frac{I}{1 + I}$$

Model Analysis

In this section we establish well posedness of the model by showing that its solution are positive and bounded.

Theorem 2.1

The solution of the differential equation in system (1) are bounded within the invariant region **Ω**, define by $\Omega = \{(S, E, I, R): N(t) \leq {}^{\pi}\}$, where $N(t) = S(t) + E(t) + I(t)$

$+ R(t)$ represent the μ total population π is the recruitment rate, and μ is the natural death rate.

Proof

Consider the total population $N(t)$, which is the sum of all compartments:

$$N(t) = S(t) + E(t) + I(t) + R(t)$$

Taking the time derivative of $N(t)$, we get:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR}{dt}$$

Substituting the system (1) into the above equation gives:

$$\frac{dN}{dt} = \pi - \mu S - (C_1 - C_2)f(I)SI - (\sigma + \mu)E - (\gamma + \mu)I - \mu R$$

Simplifying the above result, we obtain:

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

This simplifies to:

$$\frac{dN}{dt} = \pi - \mu N$$

Rearranging this differential equation gives:

$$\frac{dN}{dt} + \mu N = \pi$$

The integrating factor is:

$$e^{\mu t}$$

Multiplying the above equation by the integrating factor yields:

$$e^{\mu t}\frac{dN}{dt} + \mu e^{\mu t}N = \pi e^{\mu t}$$

This can be written as:

$$\frac{d}{dt}(e^{\mu t}N) = \pi e^{\mu t}$$

Integrating both sides with respect to t, we obtain:

$$e^{\mu t}N = \frac{\pi}{\mu}e^{\mu t} + C$$

where $C$is the constant of integration.

Solving for $N(t)$, we obtain:

$$N(t) = \frac{\pi}{\mu} + Ce^{-\mu t}$$

Given the initial condition $N(0)$, we can find Cas follows:

$$N(0) = \frac{\pi}{\mu} + C$$

$$C = N(0) - \frac{\pi}{\mu}$$

Thus, the solution becomes:

$$N(t) = \frac{\pi}{\mu} + \left(N(0) - \frac{\pi}{\mu}\right)e^{-\mu t}$$

Taking the limit as $t \to \infty$:

$$\lim_{t\to\infty} N(t) = \frac{\pi}{\mu}$$

Thus, we get:

$$0 \leq N(t) \leq \frac{\pi}{\mu}$$

Therefore, the solution $N(t)$is bounded within the invariant region Ω, demonstrating that the model is mathematically well-posed and biologically meaningful within the feasible region Ω.

Theorem 2

Let the initial conditions be:

$$\{S, (0), E, (0)I(0)R(0)\} \geq 0$$

within the feasible region Ω. Then the solution set:

$$\{S, (t), E, (t)I(t)R(t)\}$$

of the system (1) remains positive for all $t \geq 0$.

Proof

Starting with the first equation of the system (1), we write:

$$\frac{dS}{dt} = \pi - \big(C_1 - C_2 f(I)\big)SI - \mu S$$

Since $\pi > 0$and all other terms in the equation are non-negative, we have:

$$\frac{dS}{dt} \geq -\big(C_1 - C_2 f(I)\big)SI - \mu S$$

$$\frac{dS}{dt} \geq -\Big(\big(C_1 - C_2 f(I)\big)I + \mu\Big)S$$

By separating variables and integrating both sides, we obtain:

$$\int \frac{dS}{S} \geq -\int \Big(\big(C_1 - C_2 f(I)\big)I + \mu\Big)\,dt$$

This can be written as:

$$\ln S\,(t) \geq -\Big(\big(C_1 - C_2 f(I)\big)I + \mu\Big)t + C_1$$

Exponentiating both sides, we get:

$$S(t) \geq Ke^{-\left((C_1 - C_2 f(I))I + \mu\right)t}$$

where:

$$K = e^{C_1}$$

Thus:

$$S(t) \geq 0$$

for all $t \geq 0$.

Next, we consider the second equation from system (1):

$$\frac{dE}{dt} = \big(C_1 - C_2 f(I)\big)SI - (\sigma + \mu)E$$

This can also be written as:

$$\frac{dE}{dt} \geq -(\sigma + \mu)E$$

By integrating the above equation, we have:

$$\int \frac{dE}{E} \geq -\int (\sigma + \mu)dt$$

Taking the logarithm:

$$\ln E\,(t) \geq -(\sigma + \mu)t + C_2$$

Exponentiating both sides, we obtain:

$$E(t) \geq K_1 e^{-(\sigma+\mu)t}$$

Given that:

$$E(0) = K_1$$

it follows that:

$$E(t) \geq E(0)e^{-(\sigma+\mu)t}$$

Thus:

$$E(t) \geq 0$$

for all $t \geq 0$.

For the third equation of system (1), we write:

$$\frac{dI}{dt} = \sigma E - (\gamma + \mu)I$$

This can also be written as:

$$\frac{dI}{dt} \geq -(\gamma + \mu)I$$

By integrating the above equation after separating variables, we get:

$$\int \frac{dI}{I} \geq -\int (\gamma + \mu)dt$$

Taking the logarithm:

$$\ln I\,(t) \geq -(\gamma + \mu)t + C_3$$

Exponentiating both sides, we get:

$$I(t) \geq K_2 e^{-(\gamma+\mu)t}$$

Given that:

$$I(0) = K_2$$

it follows that:

$$I(t) \geq I(0)e^{-(\gamma+\mu)t}$$

Thus:

$$I(t) \geq 0$$

for all $t \geq 0$.

The last equation of system (1) can be written as:

$$\frac{dR}{dt} = \gamma I - \mu R$$

This is similar to:

$$\frac{dR}{dt} \geq -\mu R$$

Integrating the above equation, we obtain:

$$\int \frac{dR}{R} \geq -\int \mu \, dt$$

Taking the logarithm:

$$\ln R\,(t) \geq -\mu t + C_4$$

Exponentiating both sides, we get:

$$R(t) \geq K_3 e^{-\mu t}$$

where:

$$K_3 = e^{C_4}$$

Given:

$$R(0) = K_3$$

it follows that:

$$R(t) \geq R(0)e^{-\mu t}$$

Thus:

$$R(t) \geq 0$$

for all $t \geq 0$.

Therefore, all state variables $S(t), E(t), I(t), R(t)$are positive for all $t \geq 0$. The solutions are non-negative for $t \geq 0$and are bounded within the invariant region Ω, making the system mathematically well-posed and biologically meaningful within the feasible region Ω.

Basic Reproductive Number.

The Basic Reproductive Number R_0 is a crucial parameter in epidemiological modeling. It represents the average number of secondary infections generated by a single infected individual in a fully susceptible population. In the context of mathematical model for cholera with media influence, we use the next-generation matrix approach to derive R_0.

Thus we identify matrices F and V respectively.

The R_0.threshold is derived from the rates at which individuals move from being susceptible to exposed, and from exposed to infected.The infection matrix F represents the rate at which new Infections occur. Specifically, it captures the rate at which susceptible individuals become exposed due to contact with infected individuals, and this can be written as:

$$F = \begin{bmatrix} C_1\pi & 0 \\ 0 & \mu \end{bmatrix}$$

$$V = \begin{bmatrix} \sigma + \mu & 0 \\ -\sigma & \gamma + \mu \end{bmatrix}$$

The inverse of Vcan be calculated as:

$$V^{-1} = \frac{1}{(\sigma + \mu)(\gamma + \mu)} \begin{bmatrix} \gamma + \mu & 0 \\ \sigma & \sigma + \mu \end{bmatrix}$$

Let $K = F \cdot V^{-1}$. Since Fand Vare non-negative matrices, $F \cdot V^{-1}$is non-negative and hence:

$$R_0 = \varrho\left(F \cdot V^{-1}\right)$$

where $V^{-1}$is the inverse of the matrix V.

Multiplying Fby V^{-1}gives the next generation matrix as follows:

$$K = F \cdot V^{-1}$$

By substitution, we have:

$$K = \begin{bmatrix} C_1\pi & 0 \\ 0 & \mu \end{bmatrix} \frac{1}{(\sigma + \mu)(\gamma + \mu)} \begin{bmatrix} \gamma + \mu & 0 \\ \sigma & \sigma + \mu \end{bmatrix}$$

Simplifying, we get:

$$K = \frac{C_1 \pi \sigma}{\mu(\sigma + \mu)(\gamma + \mu)} \begin{bmatrix} 1 & 0 \\ -\sigma & \gamma + \mu \end{bmatrix}$$

Thus, the expression for the basic reproductive number is:

$$R_0 = \frac{C_1 \pi \sigma}{\mu(\sigma + \mu)(\gamma + \mu)}$$

This $R_0$is the basic reproductive number, which determines whether the disease will spread in the population. If $R_0 > 1$, the disease will spread; if $R_0 < 1$, the disease will die out.

The existence and stability of these equilibrium points are analyzed using the basic reproduction number R_0, which was derived using the next-generation matrix approach. Stability analysis helps us understand the conditions under which the disease either spreads or dies out within the population. Since:

$$R(t) = N(t) - E(t) - I(t)$$

the recovered compartment $R(t)$is often excluded from the stability analysis.

Disease-Free Equilibrium Point (DFE)

The disease-free equilibrium, denoted by E_0, is the point where the disease is entirely absent from the population. At this equilibrium, there are no infected individuals, so the exposed (E), infectious (I), and recovered (R) compartments are all zero. The remaining population is fully susceptible.

From the first equation (1), the DFE can be derived as follows:

$$\frac{dS}{dt} = \pi - \big(C_1 - C_2 f(I)\big) S \cdot I - \mu S = 0$$
$$\frac{dE}{dt} = \big(C_1 - C_2 f(I)\big) S \cdot I - (\sigma + \mu) E = 0$$
$$\frac{dI}{dt} = \sigma E - (\gamma + \mu) I = 0$$
$$\frac{dR}{dt} = \gamma I - \mu R = 0$$

Solving the above equations gives:

$$\frac{dS}{dt} = \pi - \mu S = 0$$
$$\pi - \mu S = 0$$

$$S = \frac{\pi}{\mu}, \ \frac{dE}{dt} = 0, \ \frac{dI}{dt} = 0, \ \frac{dR}{dt} = 0$$

This implies that:

$$E_0 = (S^*, E^*, I^*, R^*) = \left(\frac{\pi}{\mu}, 0, 0, 0\right)$$

Thus, the DFE $E_0$of the system is given by:

$$E_0 = \left(\frac{\pi}{\mu}, 0, 0, 0\right)$$

The basic reproduction number $R_0$is a critical threshold parameter that determines the behavior of the disease within the population.

Theorem 3

The Disease-Free Equilibrium (DFE) is locally asymptotically stable if $R_0 < 1$and unstable if $R_0 > 1$.

Proof

To analyze the local stability of the DFE of the system (1), we use the Routh-Hurwitz criterion. This method provides a systematic way to determine the stability of the equilibrium by examining the characteristic polynomial of the Jacobian matrix:

$$J = \begin{bmatrix} -(C_1 - C_2)f(I)I - \mu & 0 & -(C_1 - C_2)f(I)SI + f'(I)S & 0 \\ (C_1 - C_2)f(I)I & -(\sigma + \mu) & (C_1 - C_2)f(I)SI + f'(I)S & 0 \end{bmatrix}$$

At the DFE:

$$(S, E, I, R) = \left(\frac{\pi}{\mu}, 0, 0, 0\right)$$

the Jacobian matrix becomes:

$$J = \begin{bmatrix} -\mu & 0 & -(C_1 - C_2)\pi & 0 \\ 0 & -(\sigma + \mu) & (C_1 - C_2)\pi & 0 \\ 0 & \sigma & -(\gamma + \mu) & 0 \\ 0 & 0 & \gamma & -\mu \end{bmatrix}$$

To calculate the characteristic equation, we write:

$$\det(\lambda I - J) = 0$$

Expanding this determinant, we get:

$$(\lambda+\mu)^2\left[\lambda^2+(\sigma+\gamma+2\mu)\lambda+(\sigma+\mu)(\gamma+\mu)-\sigma(C_1-C_2)\pi\right]=0$$

This expands into a fourth-degree polynomial:

$$\lambda^4+a_1\lambda^3+a_2\lambda^2+a_3\lambda+a_4=0$$

where the coefficients $a_1, a_2, a_3, a_4$are functions of the system parameters $\mu, \sigma, \gamma, C_1, C_2, \pi$, respectively.

$$a_1=\sigma+\gamma+4\mu$$
$$a_2=\mu^2+2\mu(\sigma+\gamma+2\mu)+(\sigma+\mu)(\gamma+\mu)-\sigma(C_1-C_2)\pi$$
$$a_3=2\mu[(\sigma+\mu)(\gamma+\mu)-\sigma(C_1-C_2)\pi]$$
$$a_4=\mu^2[(\sigma+\mu)(\gamma+\mu)-\sigma(C_1-C_2)\pi]$$

The Routh array for a fourth-order polynomial is given as follows:

λ^4	1	a_2	a_4
λ^3	a_1	a_3	0
λ^2	b_1	b_2	0
λ^1	c_1	0	0
λ^0	d_1	0	0

The entries of the above matrix can be defined as follows:

$$b_1=\frac{a_1a_2-a_3}{a_1}$$
$$b_2=a_4$$
$$c_1=\frac{b_1a_3-a_1b_2}{b_1}$$

For the system to be stable, all elements in the first column of the Routh array must be positive. That is:

$$a_1>0,\ b_1>0,\ c_1>0,\ d_1>0$$

We further check each condition of stability as follows:

$$a_1=\sigma+\gamma+4\mu>0$$
$$a_2=\mu^2+2\mu(\sigma+\gamma+2\mu)+(\sigma+\mu)(\gamma+\mu)-\sigma(C_1-C_2)\pi>0$$
$$a_3=2\mu[(\sigma+\mu)(\gamma+\mu)-\sigma(C_1-C_2)\pi]>0$$
$$a_4=\mu^2[(\sigma+\mu)(\gamma+\mu)-\sigma(C_1-C_2)\pi]>0$$

Since all coefficients in the first column of the Routh array are positive, the system is stable, indicating that $\mathbf{R_0 < 1}$at the DFE.

For the local asymptotic stability of the DFE:

- All coefficients in the first column of the Routh array should be positive.
- If any coefficient is non-positive, the system is unstable.

Thus, we show that:

$$R_0 < 1 \Rightarrow \text{the system is locally asymptotically stable}$$
$$R_0 > 1 \Rightarrow \text{the system is unstable}$$

Numerical Results

Numerical simulation is a key tool for evaluating complex models where analytical solutions may not be feasible. In this study, numerical simulation of SEIR model that incorporates media as a control measure to assess its effectiveness in reducing disease transmission. By influencing the infection rate through behavioral changes, media campaigns are modeled as interventions that reduce contact rates or promote preventive actions. To exermine an analyse the transmission of cholera epidemic, Our formulated model incoorperating media coverage is now numerically solved. In order to acertain the validity of our model, different parameters values from previous studies are used in our simulation, (See Table 1).

Table 1. Table of parameters Parameters

PARAMETERS	VALUES	SOURCE
π	1000	Assumed
C_1	0.02	Chengjun sun et,al,.(2011)
C_2	0.9	Chengjun sun et,al,.(2011)
μ	$1/(78 \times 360)$	Chengjun sun et,al,.(2011)
σ	0.23	Assumed
γ	1/10	Chengjun sun et,al,.(2011)
$f(I)$	$\frac{I}{1+I}$	Chengjun sun et,al,.(2011)

In the absence of any intervention, the cholera out break increase exponentially (see Figuire 1). We notice in the first few days of the outbreak, The cholera transmission was

about 500 cases, However after around 20 days of the epidemic, the transmission infected more than 8000 individuals.

However, when the media compaign was used as a control strategy, The outbreak decays exponentially (Figure 2). One can observed that in the first few days, it cholera cases was reported to be about 400. In contrast with the media intervention the disease almost dies out after 40 days of the outbreak .

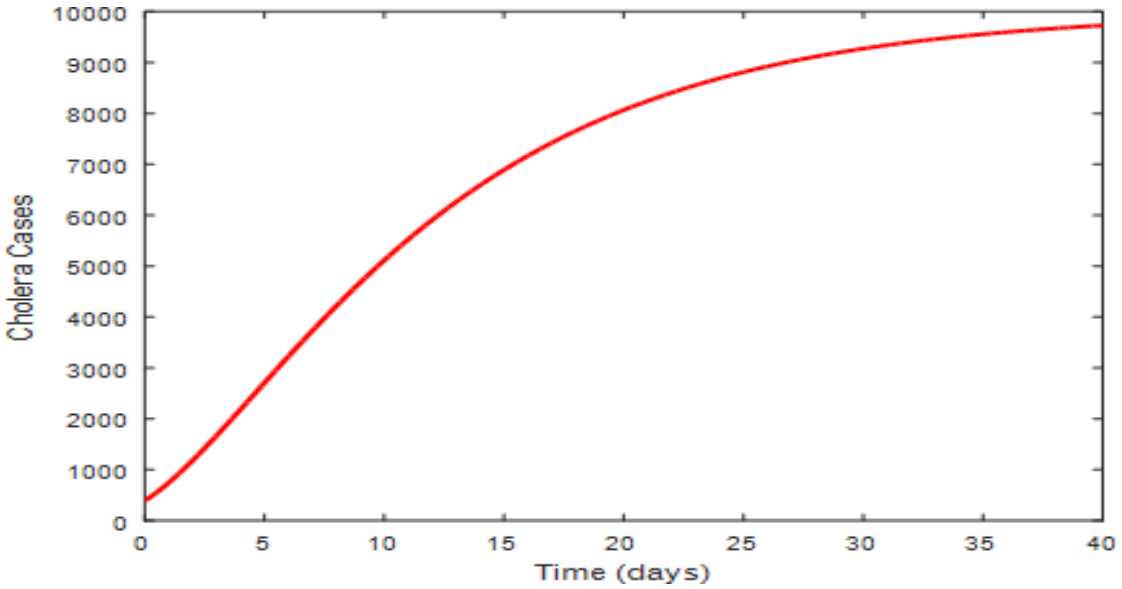

Figure 1; Cholera simulation in the absence of media

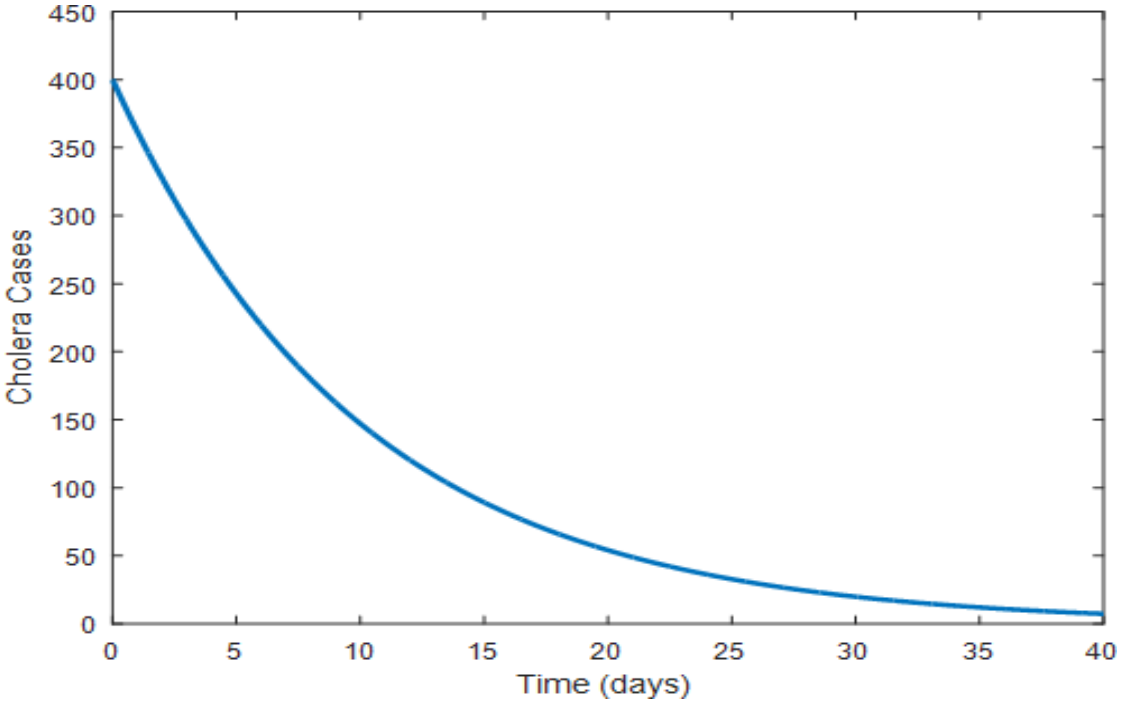

Figure 2; Cholera Simulation in the Presence of Media.

In order to clearly see the differentce between the two transmission with or (without) media coverage we depict the two trajectories in figure 3. While without media intervention the disease increases exponentially the outbreak decays exponentially with media intervention.

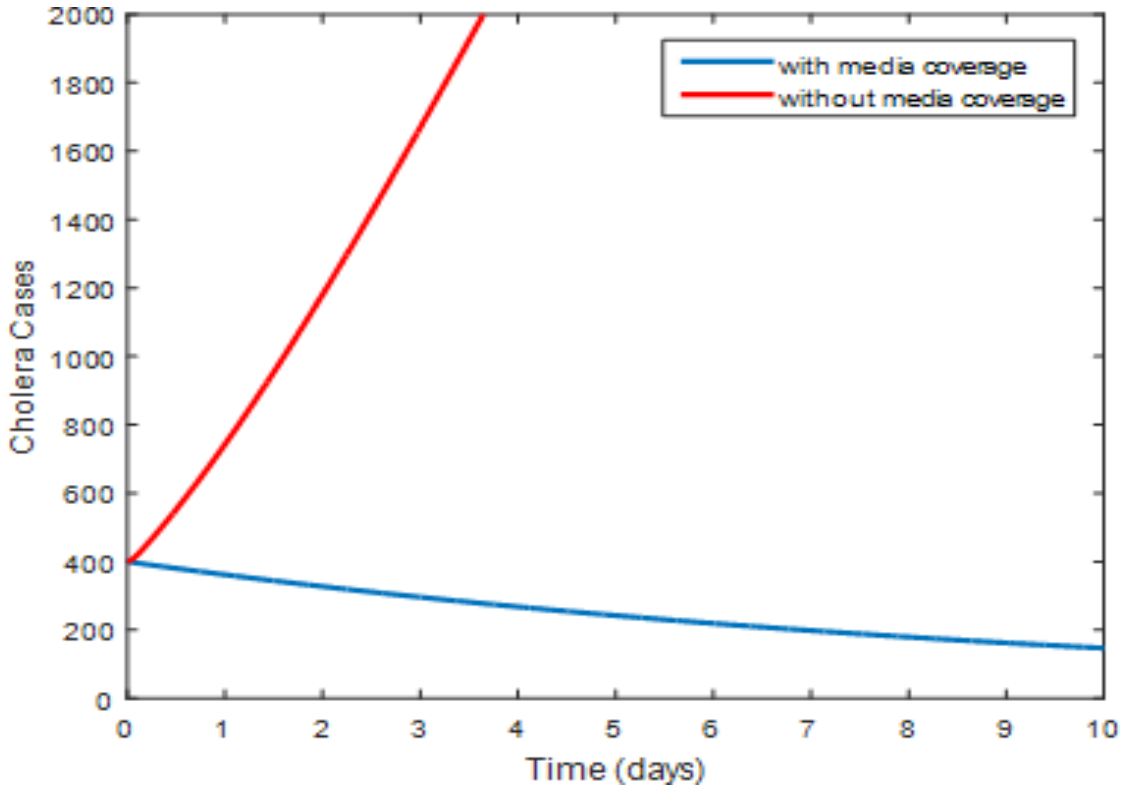

Figure3. Cholera Simulation in the Presence and absence of Media.

Discussion

Cholera remains one of the significant public health problems, especially in areas where environmental conditions are marked by poor sanitation and a shortage of clean water. Several measures have been utilized to reduce its spread, which include vaccination, improved water treatment, and public health education. A mathematical model which incorporates media coverage as a control measure is presented in this study to examine the impact of the media on the transmission of cholera. The results reveal that media campaigns greatly reduce the basic reproduction number, R_0, hence the number of infections. This result corroborates earlier studies on the efficiency of public awareness in infectious disease control (Funk et al., 2019; Mahbub et al., 2022). Numerical simulations show that media

interventions play a significant role in changing individual behavior, which increases the adoption of preventive measures such as proper hygiene and safe water usage. Stability analysis confirms that when R_0 is less than unity, the disease dies out asymptotically, hence the need for sustained public health communication.

The differential equation Model implemented in this research provides an appropriate estimation of cholera transmission dynamics and underlines the impact of media-driven interventions. The developed approaches have been applied to epidemiological modeling, demonstrating stability and providing reliable results in predicting the dissemination of a disease (Nava & Guevara-Jordan, 2023; Frei & Singh, 2024).

Conclusion

This work is on the formulation and analysis of a mathematical model that incorporates media coverage in the dynamics of cholera transmission as a control strategy. It has been realized that media campaigns are relatively effective in reducing infection rates through influencing the public to adopt preventive measures. Numerical results have confirmed that increased media coverage leads to a significant decrease in disease prevalence, hence highlighting its importance as a complementary strategy to traditional cholera control methods.

Furthermore, it is also supported by the model's stability analysis that cholera can be eliminated if adequate public awareness is sustained. The model framework in this work turns out to be useful for a simulation of cholera dynamics, testing different strategies of intervention. Future efforts must be made by incorporating other parameters, like environmental conditions and socio-economic influences, to enhance predictive capability and find more holistic methods of disease control.

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