e-ISSN: 2564-7954 CUJSE 20(01): 035-052 (2023) Research Article

Çankaya University

Journal of Science and Engineering

https://dergipark.org.tr/cankujse

CUJSE

Modelling the Transmission Dynamics of Cholera Disease with the Impact of Control Strategies in Nigeria

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Keywords

Cholera, Modelling, Transmission, Control Measures, Epidemic, Reproduction Number

Abstract

Cholera remains a severe health concern in many developing nations, including Nigeria, and its control remains challenging. Therefore, a mathematical model for the mitigation of cholera disease in Nigeria is developed and analyzed. It includes vital dynamics that examine the impact of environmental sanitation, water body treatment, water hygiene, and therapeutic treatment as mitigation strategies for containing the disease. The impact of control techniques on the diseased population is investigated using numerical simulation. The model was simulated to determine the impacts of hygienic culture on the infected population at no, low, moderate, and high levels of vaccination and treatment, or both. The model under study demonstrates that the cholera pandemic might be eliminated from society with the right mix of preventative measures and determined effort. According to the model used, Nigeria will quickly rid itself of the disease if treatment, water hygiene, and environmental sanitation are highly monitored and improved.

1. Introduction

Cholera is a transmittable intestinal tract disease that is triggered by a bacterium called Vibrio cholerae. Cholera enters into an individual via the drinking of infected water as well as beverages or intake of food items infected with V. Cholerae. The disease quickly reduces life expectancy, especially when the epidemic is not early detected and when prompt medical assistance is not given. Children and mothers are the two groups most impacted by the disease breakout. Regardless of century studies on the disease, inning accordance with studies obtainable at WHO [1], it is reported that there are about 3-5 million cholera cases of which 11.4 per cent were children below 5 years of age consisting of aged individuals as well as 100,000120,000 fatalities as a result of Cholera annually. A lot more cases were unaccounted for, as a result of constraints in surveillance and anxiety of trade travel sanctions.

Cholera transmission exists in the location where there is ecological mismanagement, thus, environmental problems and war do play significant roles in spreading out the disease in communities. New outbreaks can easily take place regularly around poor locations where food items safety and security, hygiene, clean water and hygiene are not readily accessible. The greatest threat takes place in overcrowded areas and refugees through harmful consumption and poor hygiene. WHO reports revealed that the transference of this deadly disease is through infected food items or drinking water [1]. The two to five-day incubation period is also thought to have the potential to accelerate the eruptive tendency of outbreaks [2]. Several attempts were implemented to cope with the dispersal of cholera disease, but regardless of the continual attempts, it continues to trigger epidemic and pandemic infection, consequently, Cholera continues to be a worldwide public health threat.

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Received: March 16, 2023, Accepted: April 11, 2023

Therefore, comprehending the basic mechanism of the transmission of communicable disease is vital for reliable precautionary and mitigation tactics against the cholera upsurge. In light of this, mathematical modelling presents a special way to comprehend the dynamics of communicable diseases at a fundamental level. Mathematical models are frequently used for a better understanding of environmental and epidemic problems. They are particularly useful as a scientific tool to evaluate and compare mitigation and prevention strategies, as well as to examine the respective impacts of various biological, sociocultural, and ecological factors on the spread of disease [3]. Consequently, by exploring the possible impacts of disease management tactics including water chlorination, environmental hygiene etc., mathematical modelling can easily predict the mechanics of eruptive upsurges usually connected with cholera disease.

Many mathematical models have been developed as a way to better comprehend the complicated mechanics of cholera [4 –33]. For instance, a model that integrated the ecological constituent-, the amount of V. cholera in the water supply—into a standard SIR epidemiological model was proposed [34]. A logistic function was used to model the occurrence to depict the saturation effect. Based on the laboratory data, the model was enhanced to include a hyper-virulent stage of the bacteria, mimicking the eruptive infection rate of newly discharged V. cholerae [35]. This model was carefully examined using a limited pathogenic concentration for transmission as well as a mindful dialogue on individual-ecological interaction and in-reservoir disease aspects [36].

More recently, a model was proposed to examine the 2008-2009 cholera epidemics in Zimbabwe [37]. Both human-to-human, as well as environment-to-human transmission pathways, were examined by the model. The outcomes illustrated the importance of human-to-human transmission of diseases, particularly in places like Zimbabwe, an impoverished landlocked country in the centre of Africa. The twofold transmission channels were also incorporated in a water-borne infection model that was presented, and bilinear incidence rates were used for both the human-to-human and the ecosystem-to-human transmission paths [38]. No saturation effect was taken into consideration. An extensive worldwide stability study was carried out for most of the above-mentioned models. Furthermore, the model was modified to incorporate numerous control procedures [39]. As a result, they assessed the best mitigation tactics and ran numerical simulations using their model. No human-to-human transmission pathway was taken into account. In addition, considered three types of controls were considered: vaccination, therapeutic treatment, and water hygiene, the effect of environmental hygiene was not mentioned [40].

Consequently, our objective is to examine the transmission mechanics of Cholera considering human-to-human along with environment-to-human infection paths, the loss of immunity after cholera infection recovery and also the effect of applying mitigation strategies in curtailing its spread out in Nigeria which has not been extensively studied. Thus, we considered five control measures: water hygiene, immunization, therapeutic intervention, treatment of water bodies and environmental hygiene. For the exclusive scenario with consistent controls, we carefully assessed the stabilities of the corresponding autonomous dynamical system for the exclusive scenario with reliable controls and then utilized numerical simulation to investigate a variety of optimum control strategies entailing multiple controls.

2. Model Formulation

In this section, we discuss the application of a mathematical model to the cholera transmission mechanism. Considering the infection model for the **SIR-V** type of infection model.

2.1. Model Assumptions

The model formulation is guided by the following assumptions:
(i) The overall populace of people is not consistent.
(ii) Mitigation is applied consistently
(iii) Vaccination is offered to the vulnerable populace.
(iv) Therapeutic procedure is applied to the infected people
(v) Water hygiene causes the fatality of Vibrios.
(vi) Treatment of water

(vii) Environmental hygiene leads to the fatality of Vibrios.
(viii) On recovery, there's short-term immunity.

2.2. Model Description

A logistic (or Michaelis-Menten kind) function is used to describe environment-to-human transmission in the cholera model proposed in [37], and the conventional mass-action rule is used to represent human-to-human transmission. Vaccination, treatment, water hygiene, and proper hygiene are now added to this paradigm.
According to their disease status in the system, the model split the whole human populace at any given moment (t) into three subpopulations. The subpopulations of Susceptible humans (S), Infectious humans (I), and Recovered humans make up the total population, denoted as (R). The overall populace $N(t)$ is as follows:

$N(t) = S(t) + I(t) + R(t)$, where $N(0) = S(0)$.

$S(t)$ is the population that is at risk of contracting cholera disease. $I(t)$ are the people who are displaying the cholera disease symptoms, and $R(t)$ are the people who have recovered from the illness and received temporary resistance. The amount of vibrio bacteria present in the ecosystem is indicated by $V(t)$.

Table 1. Description of Parameter

Parameter	Description
Ω	Rate of recruitment of susceptible humans
α_I	Rate of contact of susceptible humans with infectious humans
α_V	Rate of the interaction of susceptible humans with contaminated water
γ	Rate of loss of immune by recovered individuals
μ	The natural death rate of human
τ	Rate of vaccination
ω	Disease-induced death rate
σ	The rate at which infectious humans contaminate the ecosystem
β	Rate of recovery
η	Rate of treatment of water bodies in the ecosystem
δ	The decay rate of Vibrios
a	A positive constant providing adjustment to the rate of infection
ρ	A positive constant providing adjustment to the rate of Vibrio ingestion in the ecosystem.
C_V	The density of vibrio in the ecosystem.
C_h	The density of Infection among humans

The following systems of linear equations are the given models of the disease.

$$\begin{cases} \dfrac{dS}{dt} = \Omega - \alpha_I SI - \alpha_V SV + \gamma R - \mu S - \tau S \\ \dfrac{dI}{dt} = \alpha_I SI + \alpha_V SV - (\omega + \sigma + \beta + \mu)I \\ \dfrac{dR}{dt} = \beta I - \tau S - (\gamma + \mu)R \\ \dfrac{dV}{dt} = \sigma I - \eta\delta V \end{cases} \quad (1)$$

Where $\alpha_I = \frac{C_h}{1+aI}$, $\alpha_V = \frac{c_V}{V+\rho}$

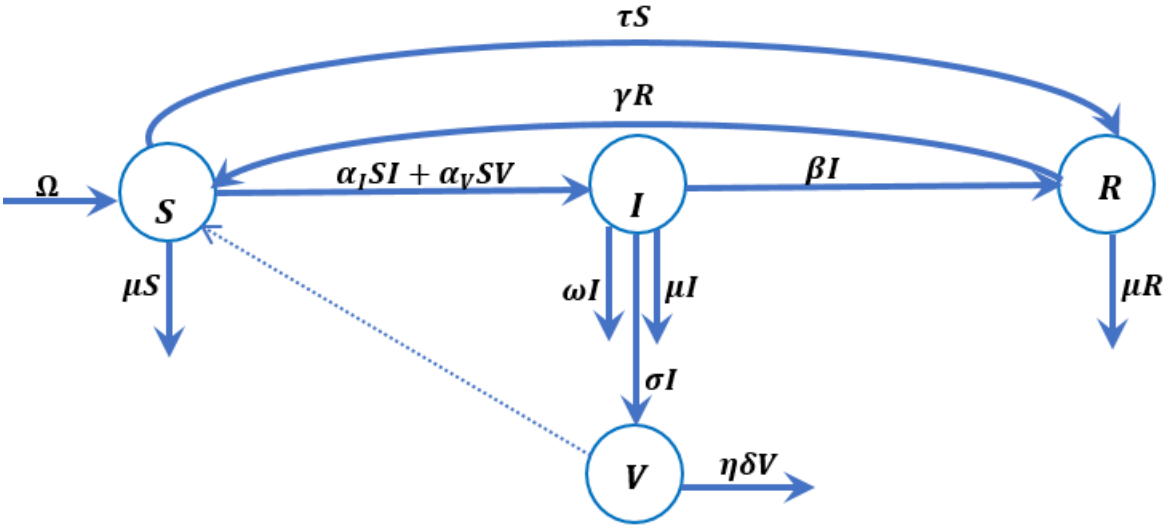

Figure 1. Schematic depiction of the Cholera model with control strategies

2.3. Positivity and Boundedness of Solution

Given that the entire human population at any given moment t, the dynamic system is uniformly confined in the appropriate subset $\theta \subset R_+^3$ is given by:

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

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

$$\frac{dN}{dt} = \Omega - \alpha_I SI - \alpha_V SV + \gamma R - \mu S - \tau S + \alpha_I SI + \alpha_V SV - (\omega + \sigma + \beta + \mu)I + \beta I - \tau S - (\gamma + \mu)R \quad (2)$$

$$\frac{dN}{dt} = \Omega - \mu S - \mu I - \omega I - \sigma I - \mu R$$

There is no cure when there are infections, that is, $I(t) = 0, R(t) = 0$. Originally $N(0) = S(0)$

$$\frac{dN}{dt} = \Omega - \mu S \quad (3)$$

$$\frac{dN}{dt} = \Omega - \mu N \quad (4)$$

Separating the variables to solve the differential equation

$$\frac{dN}{\Omega - \mu N} = dt \quad (5)$$

Then integrating yields

$$\int \frac{dN}{\Omega - \mu N} \leq \int dt$$

$$-\frac{1}{\mu}\ln|\Omega - \mu N| \leq t + A$$

$$\Omega - \mu N \geq \mathrm{e}^{-\mu(\mathrm{t}+\mathrm{A})} = e^{-\mu t}e^{-\mu A}$$

$$\Omega - \mu N \geq Ce^{-\mu t} \tag{6}$$

At $N(0) = N_0 \Longrightarrow t = 0, N = N_0$

$$(\Omega - \mu N_0) = C \tag{7}$$

$$(\Omega - \mu N) \geq (\Omega - \mu N_0)e^{-\mu t} \tag{8}$$

By rearranging and simplifying

$$\frac{\Omega}{\mu} - N \geq \frac{(\Omega - \mu N_0)}{\mu} e^{-\mu t}$$

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

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

Hence,

$$\theta_t = \left\{(S, I, R) \in R_+^3 : S + I + R \leq \frac{\Omega}{\mu}\right\} \tag{9}$$

The dynamic system of equations in the model has a solution that is constrained to the area where $\varphi = \theta_S + \theta_I + \theta_R$. This suggests that the model's dynamic system in the area is properly constructed.
If the starting values of all the variables are positive, the system's solution is always positive.
Theorem 1: *Assuming that the dynamic system area such that;*

$$\phi = \{(S(t), I(t), R(t)) \in R_+^3 : S(0) > 0, I(0) > 0, R(0) > 0\} \tag{10}$$

The solution set $\{(S(t), l(t), R(t))\}$ *is positive for* $t \geq 0$

3. Equilibria and Reproduction Number

3.1. Existence of Disease Free Equilibrium (DFE)

At DFE, $I = R = V = 0$, $\frac{dS}{dt} = 0$

$$\frac{dS}{dt} = \Omega - \alpha_S SI - \alpha_V SV + \gamma R - \mu S - \tau S \tag{11}$$

$$\Omega - \mu S + \tau S = 0 \tag{12}$$

$$S^* = \frac{\Omega}{\mu + \tau} \tag{13}$$

$$(S^*, 0,0,0) = \left(\frac{\Omega}{\mu + \tau}, 0,0,0\right) \tag{14}$$

3.2. Stability of the Disease Free Equilibrium

Proposition 2: *Disease-free equilibrium point is locally asymptomatically stable if the basic reproduction number* $R_0 < 1$ *and unstable otherwise.*

Proof: DFE is given by $\left(\frac{\Omega}{\mu+\tau}, 0,0,0\right)$ has

$$R_0 = \frac{\Omega}{\mu+\tau} - (\mu + \omega + \Omega + \beta) \tag{15}$$

such that; $R_0 < 0$

$$\frac{\Omega}{\mu+\tau} - (\mu + \omega + \sigma + \beta) < 0 \tag{16}$$

$$\frac{\alpha\Omega}{(\mu+\tau)(\mu+\omega+\sigma+\beta)} < 1 \tag{17}$$

and $R_0 < 1$. Given that $R_0 < 1$, hence, the DFE point is locally asymptomatically stable.

3.3. Basic Reproduction Number

This threshold controls how quickly a disease spreads among people. Whether the illness would linger or disappear over time in the body. The basic reproduction rate predicts or indicates the progression of illness over time. By computing the Jacobian of the system at the disease-free equilibrium and imposing the requirement that all eigenvalues of the related characteristic equation have negative real components, the basic reproductive number may be determined.

Taking into account the differential equations' Jacobian matrix:

$$\mathcal{J}(S^*, I^*, V^*, R^*) = \begin{pmatrix} \frac{\partial S}{\partial S} & \frac{\partial S}{\partial I} & \frac{\partial S}{\partial V} & \frac{\partial S}{\partial R} \\ \frac{\partial I}{\partial S} & \frac{\partial I}{\partial I} & \frac{\partial I}{\partial N} & \frac{\partial I}{\partial R} \\ \frac{\partial V}{\partial S} & \frac{\partial V}{\partial I} & \frac{\partial V}{\partial V} & \frac{\partial V}{\partial R} \\ \frac{\partial R}{\partial S} & \frac{\partial R}{\partial I} & \frac{\partial R}{\partial V} & \frac{\partial R}{\partial R} \end{pmatrix} \tag{18}$$

$$\mathcal{J}(S^*, I^*, V^*, R^*) = \begin{pmatrix} -\alpha_I I - \alpha_V V - \mu - \tau & -\alpha_S S & -\alpha_V S & \gamma \\ \alpha_I I & \alpha_I S - (\mu + \omega + \sigma + \beta) & -\alpha_V S & 0 \\ 0 & \sigma & -\eta\delta & 0 \\ \tau & \beta & 0 & -(\gamma + \mu) \end{pmatrix} \tag{19}$$

Where $S = \frac{\Omega}{\mu+\tau}, \alpha_I = \frac{C_h}{1+aI}, \alpha_V = \frac{C_V}{V+\rho}$

The Jacobian matrix at DFE

$$\mathcal{J}(S^*, I^*, V^*, R^*) = \begin{pmatrix} -\mu - \tau & -\alpha_S \frac{\Omega}{\mu+\tau} & -\alpha_V \frac{\Omega}{\mu+\tau} & \gamma \\ 0 & \alpha_I \frac{\Omega}{\mu+\tau} - (\mu + \omega + \sigma + \beta) & -\alpha_V \frac{\Omega}{\mu+\tau} & 0 \\ 0 & \sigma & -\eta\delta & 0 \\ \tau & \beta & 0 & -(\gamma + \mu) \end{pmatrix} \tag{20}$$

Then the determinant of equation (20) is given by

$$\begin{vmatrix} -\mu-\tau-A & -\alpha_S\dfrac{\Omega}{\mu+\tau} & -\alpha_V\dfrac{\Omega}{\mu+\tau} & \gamma \\ 0 & \alpha_I\dfrac{\Omega}{\mu+\tau}-(\mu+\omega+\sigma+\beta)-\mathrm{A} & -\alpha_V\dfrac{\Omega}{\mu+\tau} & 0 \\ 0 & \sigma & -\eta\delta-A & 0 \\ \tau & \beta & 0 & -(\gamma+\mu)-\mathrm{A} \end{vmatrix} = 0 \quad (21)$$

A stands for the eigenvalues.

By finding the values and choosing the dominant eigenvalues. The determinant is as follows

$$\frac{\alpha_I\Omega}{\mu+\tau}-(\mu+\omega+\Omega+\beta) \quad (22)$$

Hence, the basic reproduction is obtained as:

$$\boldsymbol{R_0 = \frac{\alpha_I\Omega}{\mu+\tau}-(\mu+\omega+\Omega+\beta)} \quad (\mathbf{23})$$

3.4. Endemic Equilibrium

The endemic equilibrium points would take the form $(EE) = (S^*, I^*, V^*, R^*)$ by evaluating the state variables and bringing the system of equations to zero

$$\frac{dI}{dt} = \alpha_I SI + \alpha_V SV - (\omega+\sigma+\beta+\mu)I = 0$$

$$S^* = \frac{(\omega+\sigma+\beta+\mu)I}{\alpha_I+\alpha_V V} \quad (24)$$

Also;

$$\frac{dR}{dt} = \beta I - \tau S - (\gamma+\mu)R = 0$$

$$I = \frac{\tau S + (\gamma+\mu)R^*}{\beta}$$

$$I^* = \frac{\tau S + (\gamma+\mu)R^*}{\beta}$$

$$\frac{dS}{dt} = \Omega - \alpha_I SI - \alpha_V SV + \gamma R - \mu S - \tau S = 0$$

$$\Omega - (\alpha_I I + \mu + \tau + \alpha_V V)S + \gamma R = 0 \quad (25)$$

$$\gamma R = (\alpha_l I + \mu + \tau + \alpha_V V)S - \Omega$$

$$R^* = \frac{(\alpha_I I + \mu + \tau + \alpha_V V)S^* - \Omega}{\gamma} \quad (26)$$

$$\frac{dV}{dt} = \sigma I - \eta\delta V = 0$$

$$V^* = \frac{\sigma}{\eta\delta}\frac{\tau S + (\gamma+\mu)R^*}{\beta} \quad (27)$$

Therefore, the endemic equilibrium state (S^*, I^*, V^*, R^*)

$$\left(\begin{array}{c} \frac{(\omega+\sigma+\beta+\mu)I}{\alpha_I+\alpha_V V}, \frac{\tau S+(\gamma+\mu)R^*}{\beta}, \frac{\sigma}{\eta\delta}\frac{\tau S+(\gamma+\mu)R^*}{\beta}, \\ \frac{(\alpha_I I+\mu+\tau+\alpha_V V)S^*-\Omega}{\gamma} \end{array}\right) \tag{28}$$

3.5. Stability of the Endemic Equilibrium

Consider the Lyapunov function

$$\mathcal{L}(S^*, I^*, V^*, R^*) = \begin{cases} \left(S - S^* - S^* \ln\left(\frac{S^*}{S}\right)\right) + \left(I - I^* - I^* \ln\left(\frac{r^*}{I}\right)\right) \\ + \left(V - V^* - V^* \ln\left(\frac{V^*}{V}\right)\right) + \left(R - R^* - R^* \ln\left(\frac{R^*}{R}\right)\right) \end{cases} \tag{29}$$

The derivative of $\mathcal{L}$ along the solution of the system is direct;

$$\frac{d\mathcal{L}}{dt} = \left(\frac{S-S^*}{S}\right)\frac{dS}{dt} + \left(\frac{I-I^*}{I}\right)\frac{dI}{dt} + \left(\frac{V-V^*}{V}\right)\frac{dV}{dt} + \left(\frac{R-R^*}{dR}\right)\frac{dR}{dt}$$

$$\frac{d\mathcal{L}}{dt} = \begin{cases} \left(\frac{S-S^*}{S}\right)[\Omega - \alpha_I SI - \alpha_V SV + \gamma R - \mu S - \tau S] \\ + \left(\frac{I-F^*}{I}\right)[\alpha_I SI + \alpha_V SV - (\omega+\sigma+\beta+\mu)I] \\ + \left(\frac{V-V^*}{V}\right)[\sigma I - \eta\delta V] + \left(\frac{R-R^*}{R}\right)[\beta I - \tau S - (\gamma+\mu)R] \end{cases} \tag{30}$$

By expansion and simplification;

$$\frac{d\mathcal{L}}{dt} = \begin{cases} \Omega - \alpha SI - \mu S + \gamma R - \frac{\Omega S^*}{S} + \alpha S^* I + \mu S^* - \frac{\gamma R S^*}{S} + \alpha SI \\ -\alpha S^* I + (\mu+\omega+\sigma+\beta)\frac{IS^*}{S} + \sigma I - | \eta V - \frac{\sigma S^* I}{S} \\ + \frac{\eta V S^*}{S} + \beta I - \frac{(\gamma+\mu)S^* R}{S} \end{cases} \tag{31}$$

Suppose,

$$\frac{d\mathcal{L}}{dt} = P - Q$$

where P denotes the positive terms and Q denotes the negative terms, so that

$$P = \Omega + \mu S^* + (\mu+\omega)I + \eta V + \frac{\beta I S^*}{S} + (\gamma+\mu)R \tag{32}$$

The largest invariant set in is $\left\{(S^*, I^*, V^* < R^*) \in \theta: \frac{d\mathcal{L}}{dt} = 0\right\}$ a unit set of E^* where E^* is the endemic equilibrium. Signifying that the endemic is globally asymptomatically stable.

4. Numerical Simulation

4.1. Result

The behaviour of the model using some parameter values that are compatible with Cholera [41] as given in Table 1 and by considering the initial conditions

$S(0) = 49900$, $I(0) = 100$, $R(0) = 0$, $V(0) = 1000000$, $N = 50000$

The numerical simulation is done and plotted against time (years) using MATLAB and the results are shown in Figure 2 – 9 to illustrate the effect of assessment control i.e water hygiene, environmental sanitation,

inculcating vaccination and therapeutic treatment. The rate of contact of susceptible humans with infectious humans α_I and the rate of susceptibility with contaminated water α_V are respectively expressed as

$$\alpha_I = \frac{C_h}{1+aI}$$

$$\alpha_V = \frac{C_V}{V+\rho}$$

where C_h and C_v is respectively the human-to-human contact rate and rate of vibrio ingestion in the ecosystem. ρ is the density of vibrio cholerae in water.

Furthermore, C_h and C_v can further be expressed as follows:

$$C_h = k_h(1 - C_w), \qquad C_v = k_e(1 - C_s)$$

where k_h, k_e , C_w and C_s is the rate of exposure by humans, rate of exposure to contaminated water, rate of compliance with water hygiene and rate of compliance with environmental sanitation respectively.

Table 2. Model parameters and values used for simulation

Parameters	Values
Ω	0.03/day
k_e	0.091371323/day
ρ	0.878455198/day
k_h	0.56341910/day
τ	0.46791193/day
μ	0.000048/day
ω	0.00132473/day
β	0.00116620/day
σ	0.001/day
δ	0.27707643/day
C_w	0 – 1
C_s	0 – 1
η	0 – 1

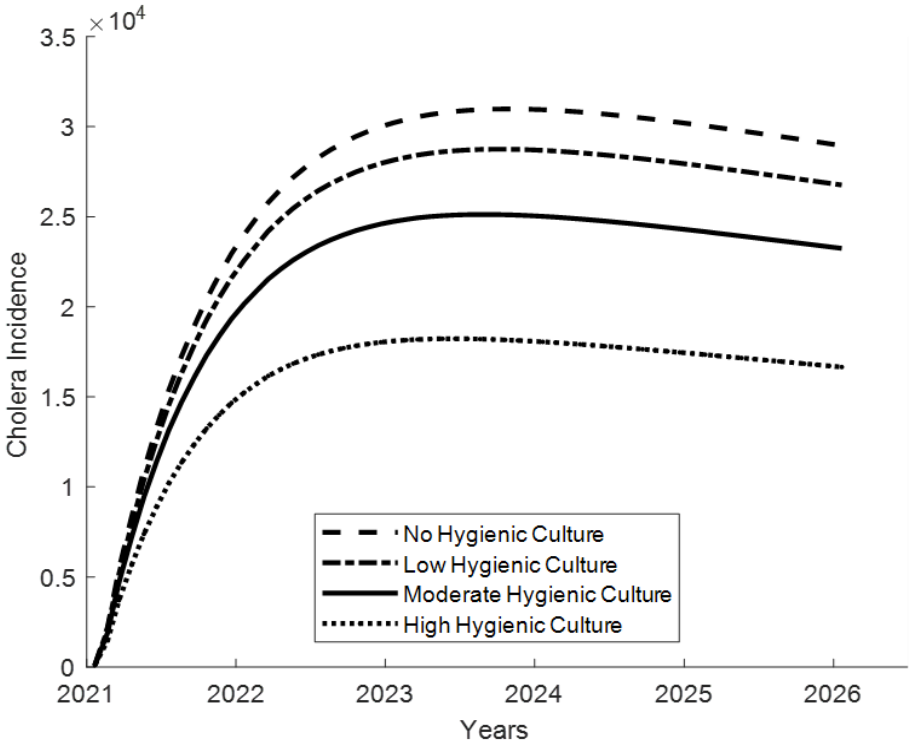

Figure 2. Infection Population profile measuring hygienic culture in the absence of vaccination

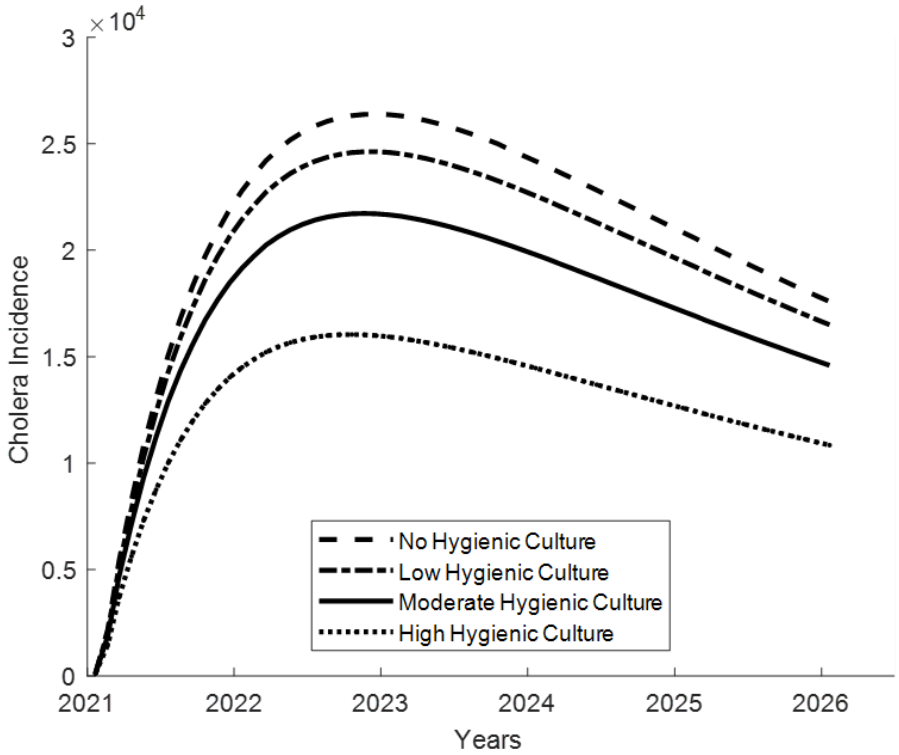

Figure 3. Infection Population profile measuring hygienic culture with low level of vaccination and low level of treatment

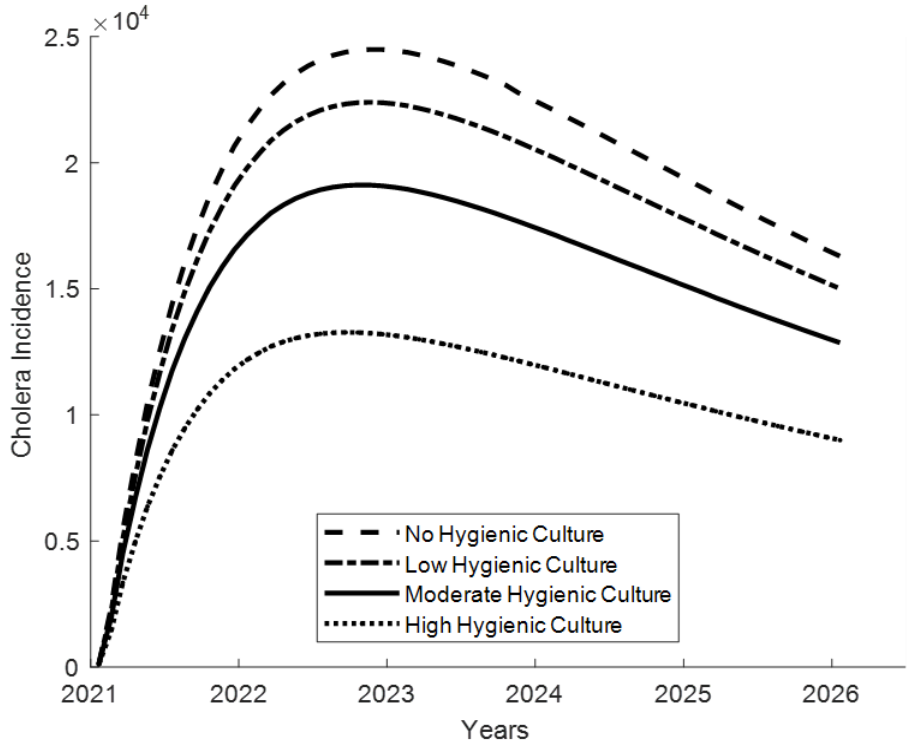

Figure 4. Infection Population profile measuring hygienic culture with a moderate level of vaccination and low level of treatment

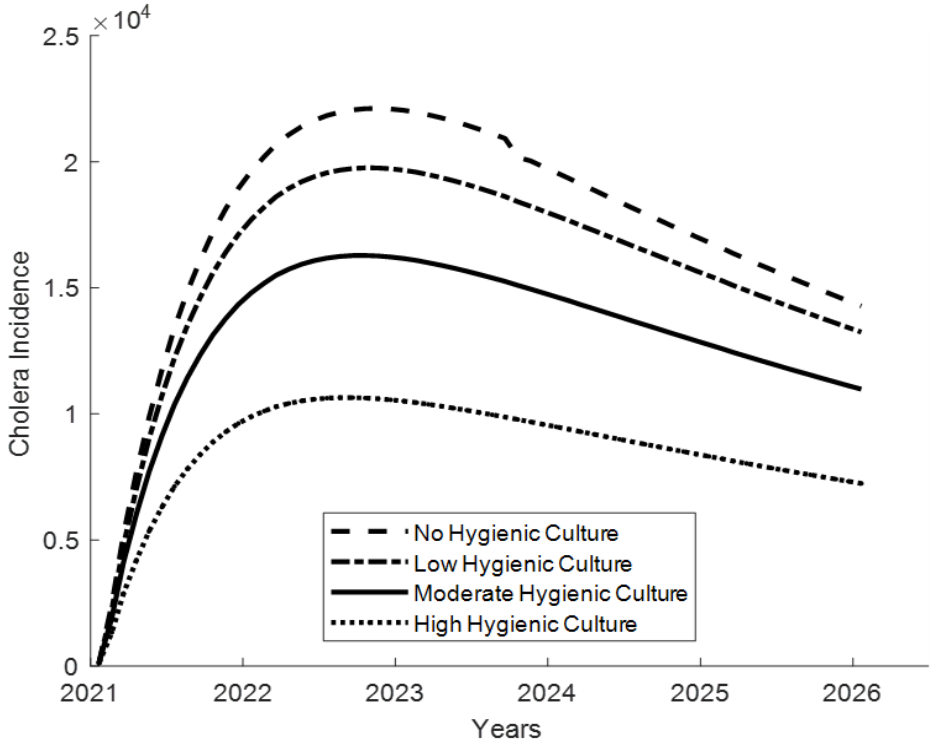

Figure 5. Infection Population profile measuring hygienic culture with a high level of vaccination and low level of treatment

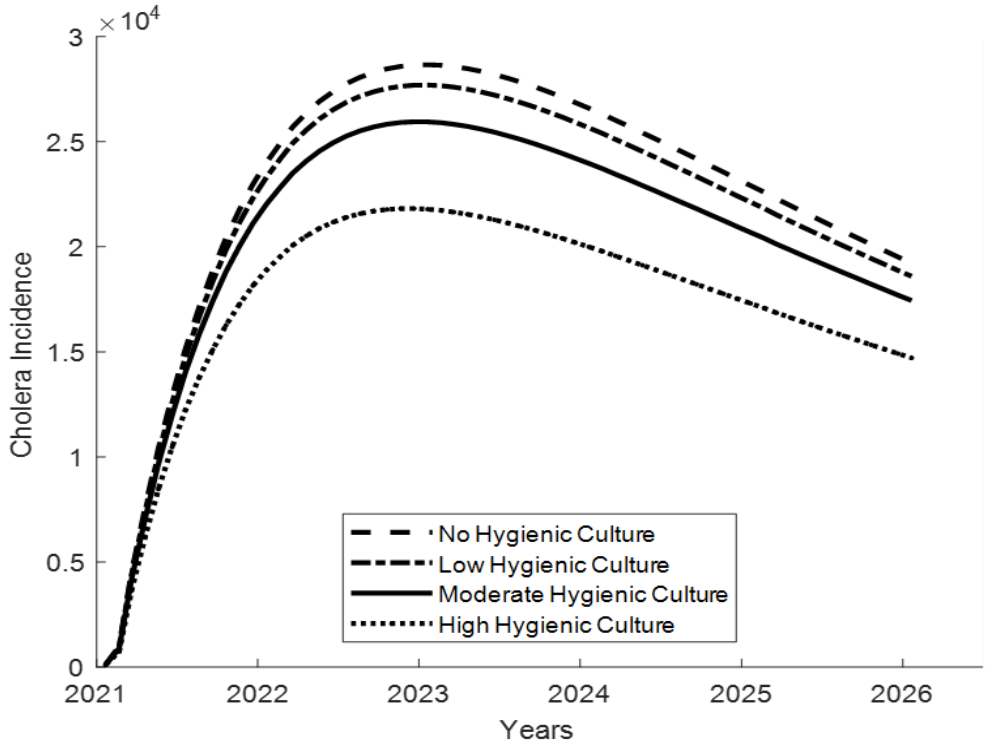

Figure 6. Infection Population profile measuring hygienic culture with a very low level of vaccination and treatment

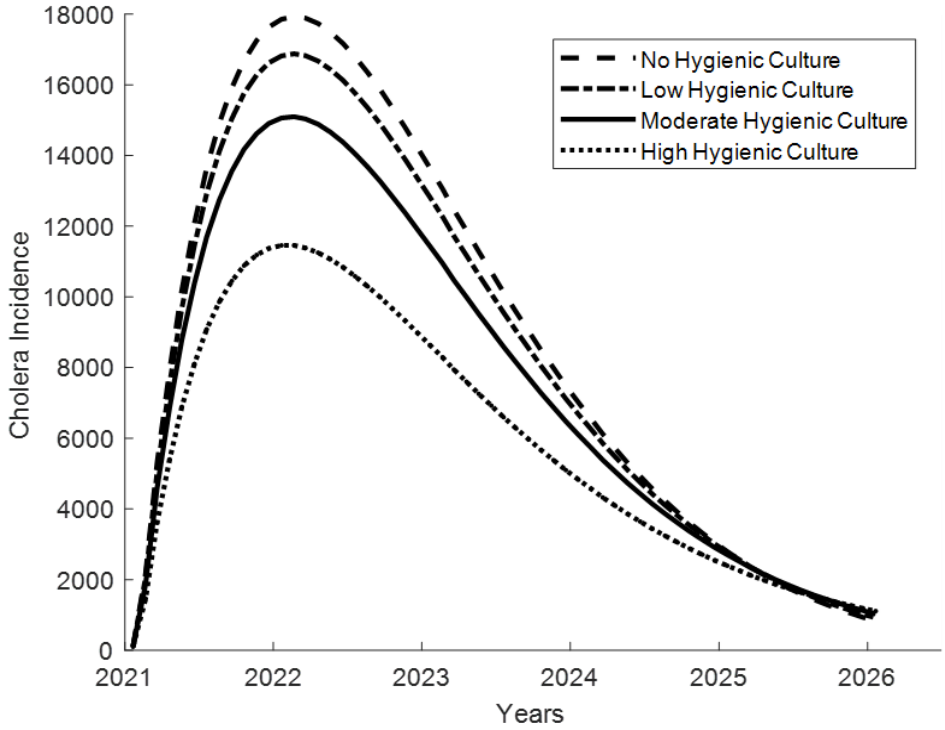

Figure 7. Infection Population profile measuring hygienic culture with a low level of vaccination and treatment

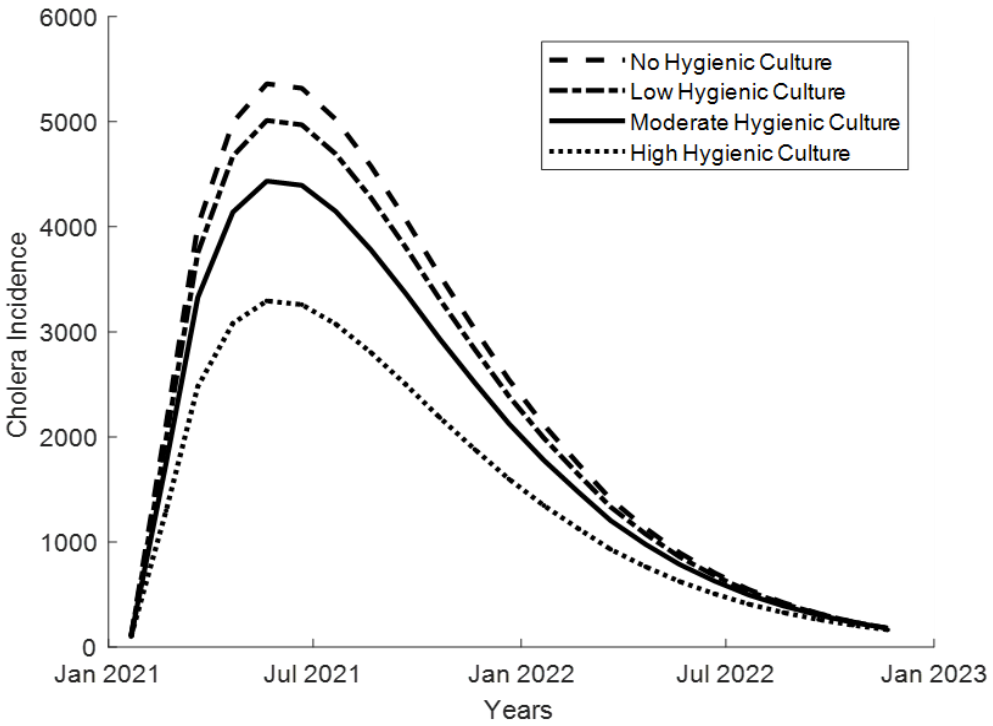

Figure 8. Infection Population profile measuring hygienic culture with a medium level of vaccination and treatment

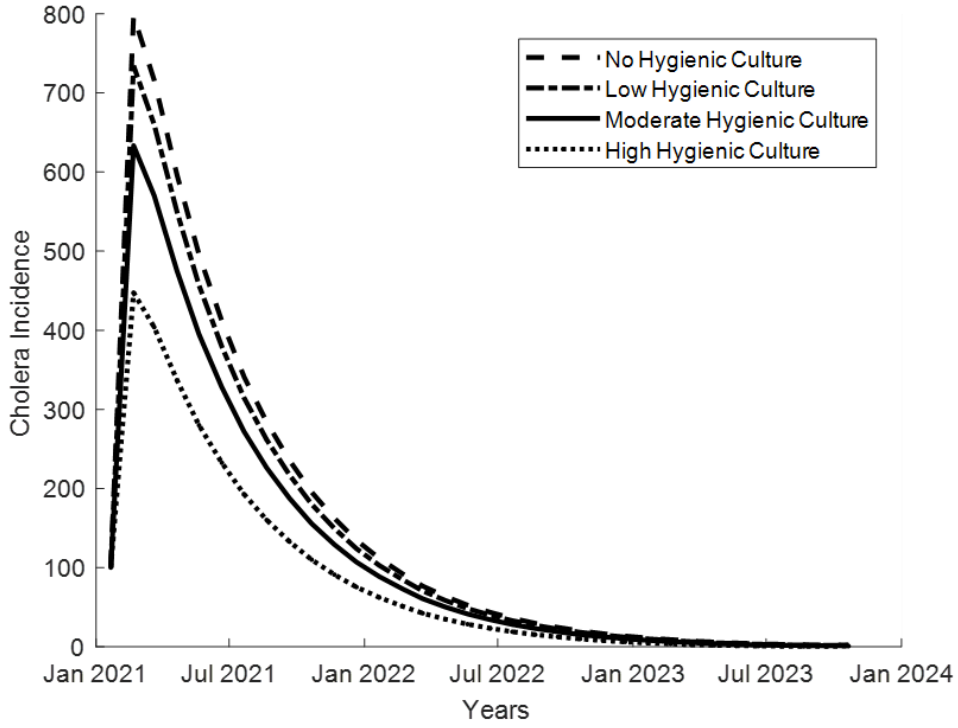

Figure 9. Infection Population profile measuring hygienic culture with a high level of vaccination and treatment

4.2. Discussion

Figure 2 – 9 illustrates the effect of complying with assessment control i.e water hygiene, environmental sanitation, inculcating vaccination and treatment. Using the baseline values in Table 2 for variables depicted in Table 1, the following measures are taken to study the effect of imbibing hygienic culture:

(i) No hygienic culture: This is a situation where there's no compliance with water hygiene, environmental sanitation and treatment of water bodies ($C_w = C_s = \eta = 0$))

(ii) Low hygienic culture: This is a situation where there is a low (25%) rate of compliance with water hygiene, environmental sanitation and treatment of water bodies ($C_w = C_s = \eta = 0.25$)

(iii) Moderate hygienic culture: This is a situation where there is a moderate (50%) rate of compliance with water hygiene, environmental sanitation and treatment of water bodies ($C_w = C_s = \eta = 0.50$)

(iv) High hygienic culture: This is a situation where there is a high (75%) rate of compliance with water hygiene, environmental sanitation and treatment of water bodies ($C_w = C_s = \eta = 0.75$)

Figure 2 presents the infection population profile measuring hygienic culture in the absence of vaccination and treatment. The effect of maintaining a hygienic culture in the absence of vaccination and treatment. It is observed that the rate of incidence of the infected individuals increases and continues at a constant rate. Additionally, with no hygienic culture, the incidence of infected individuals is about 30,000, when low hygienic culture is in place, there is a reduction to 28,000 owing to about a 6.67% reduction in the number of incidences. Maintaining a moderate level of hygienic culture brings about a 14.29% reduction and by imbibing a high level of hygienic culture, the number of infected individuals reduces to 16,000 (about 33%). This implies that we would continue to live with cholera but practising a high level of hygienic culture would reduce the incidence of infected individuals, which is not reasonable enough as there is a need to ensure cholera is wiped out.

Figure 3 represents the infection population profile measuring hygienic culture with a low level of vaccination and low level of treatment. With the introduction of the vaccine (low level of vaccination τ = 0.46791193 and low level of treatment β = 0.00011620), there is a decline in the curve with a peak of about 25500 infected individuals for no hygienic culture practice. With a low level of hygienic culture, the number of incidents reduces to 24,000 (about 5.9%). At a moderate level of hygienic culture, the number of incidents reduces to about 20,000 (16.67%). Lastly, at a high level of hygienic culture, the number of incidents reduces to about 14,000 (30%).

The infection population profile measuring hygienic culture with a moderate level of vaccination and low level of treatment is displayed in Figure 4. For a moderate level of vaccination τ = 0.66791193 and a low level of treatment β = .00011620), there is a decline in the curve with a peak of about 24000 infected individuals with no hygienic culture practice. With a low level of hygienic culture, the number of incidents reduces to 22,000 (about 8.3%). At a moderate level of hygienic culture, the number of incidents reduces to about 18,000 (18.18%). Lastly, at a high level of hygienic culture, the number of incidents reduces to about 12,000 (33%).

Figure 5 displays the infection population profile measuring hygienic culture with a high level of vaccination and a low level of treatment. For a high level of vaccination τ = 0.96791193 and a low level of treatment β = 0.00011620), there is a decline in the curve with a peak of about 22000 infected individuals for no hygienic culture practice. With a low level of hygienic culture, the number of incidents reduces to 18,000 (about 18.18%). At a moderate level of hygienic culture, the number of incidences reduces to about 15,000 (16%). Lastly, at a high level of hygienic culture, the number of incidents reduces to about 10,000 (33%).

Figure 6 demonstrates the infection Population profile measuring hygienic culture with a very low level of vaccination and treatment. Considering a very low level of vaccination τ = 0.16791193 and a very low level of treatment β = 2e−4), there's a decline in the curve with a peak of about 28,000 infected individuals with no hygienic culture practice. With a low level of hygienic culture, the number of incidents reduces to 27,000 (about 3.6%). At a moderate level of hygienic culture, the number of incidents reduces to about 25,000 (7.41%). Lastly, at a high level of hygienic culture, the number of incidents reduces to about 20,000 (20%).

The infection population profile measuring hygienic culture with a low level of vaccination and treatment is shown in Figure 7. Considering the low level of vaccination τ = 0.46791193 and the low level of treatment β = 0.0011620), there's a decline in the curve with a peak of about 18,000 infected individuals with no hygienic

culture practice. With a low level of hygienic culture, the number of incidents reduces to 16,500 (about 8.3%). At a moderate level of hygienic culture, the number of incidents reduces to about 15,000 (9.09%). In addition, at a high level of hygienic culture, the number of incidents reduces to about 11,000 (26.67%). Also, It is noticed that cholera won't eradicate until the year 2026.

Figure 8 shows the infection population profile measuring hygienic culture with a moderate level of vaccination and treatment. Considering the moderate level of vaccination β = 0.66791193 and a moderate level of treatment β = 0.011620), there's a decline in the curve with a peak of about 5400 infected individuals with no hygienic culture practice. With a low level of hygienic culture, the number of incidents reduces to 4900 (about 9.25%). At a moderate level of hygienic culture, the number of incidents reduces to about 4300 (12.24%). In addition, at a high level of hygienic culture, the number of incidents reduces to about 3100 (27.91%). Additionally, it is noted that by January 2023, the incidence would have reduced to almost zero.

Figure 9 shows the infection population profile measuring hygienic culture with a high level of vaccination and treatment. Considering a very low level of vaccination τ = 0.96791193 and a very low level of treatment β = 0.11620), there's a decline in the curve with a peak of about 800 infected individuals with no hygienic culture practice. With a low level of hygienic culture, the number of incidents reduces to 750 (about 6.25%). At a moderate level of hygienic culture, the number of incidents reduces to about 640 (14.67%). In addition, at a high level of hygienic culture, the number of incidents reduces to about 450 (29.69%). Furthermore, it is observed that by July 2022, the incidence would have reduced to almost zero.

5. Conclusions

In this work, we examined the dynamics of Cholera disease transmission and its mitigation in Nigeria. Thus, we considered five control measures: water hygiene, immunization, therapeutic intervention, treatment of water bodies and environmental hygiene. We calculate the basic reproduction number and do thorough stability and equilibrium assessments. It is discovered that both the endemic equilibrium condition and the disease-free equilibrium condition are locally asymptotically stable. According to the study's findings, the most reliable technique to curb cholera outbreak is to ensure the effectiveness and wide coverage of cholera vaccination as well as cholera treatment via the use of medicines in cholera endemic areas. From the numerical results, the study concludes that an increase in infection rate would trigger a high increase in the number of Vibrio that would be exposed and thus contaminated resulting in the individual populace entering extinction. To have a stable individual populace, the recovery rate should be increased and the infection rate between the individual populace and Virbio populace should be reduced.

Based on our findings, we recommend that the general population be effectively educated and made aware of the dangers of public defecation and bathing in sources of drinking water through pertinent authorities and NGOs. This will minimize how much each polluted person contributes to the water bodies. Likewise, we contend that the Federal authorities need to supply the populace with accessible water sources to prevent the intake of unclean water. As a result, there will be reduced exposure to infected water. To reduce the incidence of interaction between the contaminated and the vulnerable, it is also advised that cholera patients be immediately isolated. As cholera kills in a relatively short period, it is critically essential that all cholera patients receive early treatment. Those entering locations where there is a cholera outbreak should be restricted; doing so will assist to contain the spread of the illness. A vaccine strategy should be implemented in areas where cholera is chronic.

Acknowledgements

Declaration of Competing Interest

No conflict of interest was declared by the authors.

Authorship Contribution Statement

Sefiu Onitilo: Writing, Methodology, and Reviewing
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