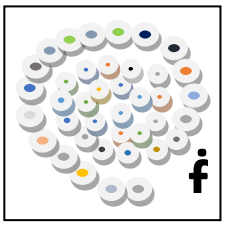

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Effects of quarantine and delay on transmission dynamics of cholera model with Holling Type - II incidence function

Ahmed Nakaka*and Adamu Shitu Hassan†

Abstract

A delay cholera model with Holling Type - II saturated incidence function and quarantine measure as intervention is formulated and dynamically analyzed with the aim of investigating the effects of time delay and quarantine measure. Basic properties of the model are investigated, and two equilibria are found to be locally and globally asymptotically stable whenever the control reproduction number is below and above unity, respectively. Quarantine intervention is found to reduce the infectivity of cholera when fully implemented. Although time delay does not alter the qualitative dynamics of the model, however it decreases the burden of cholera when the delay is beyond a critical value. Numerical simulations are conducted to illustrate all our results.

Keywords: Cholera; Holling Type -II; Delay; Quarantine; Global Stabilities.

1 Introduction

Cholera is an acute, water-borne, infectious diarrhoeal disease caused by bacterium known as *Vibrio Cholerae* serogroup $O1$ and $O139$. Only toxigenic strain of serogroup $O1$ and $O139$ have caused widespread epidemics and are reported to the World Health Organization (WHO). Humans are one of the reservoirs for pathogenic form of *Vibrio Cholerae* $O1$ and $O139$. The disease causes much havoc, especially to underdeveloped countries and massive threat to their economic development. Without prompt treatment, an infected individual may die of dehydration within hours especially in severe cases [5]. The incubation period of cholera range between few hours to 5 days [21, 26]. Some of the control and intervention measures for cholera include antibiotics, rehydration therapy, effective sanitation, proper waste management, personal hygiene and vaccination [4, 38]. After several years of steady increase, from 2007 the number of cholera reported cases to WHO indicate a considerable decrease [33]. The disease is still a threat to many countries. For instance in 2012 alone, a cumulative total of $245,393$ cases, including $3,034$ deaths with a case fatality of 1.2% were reported to WHO from all countries [1]. The disease still remains a major public health problem, affecting primarily developing world populations lacking access to adequate water and sanitation. The most recent is the September, 2018 cholera outbreak in Yobe state, North-east Nigeria which was declared by the state's ministry of Health. Two weeks after, the same outbreak was declared in the neighbouring Borno state. The cumulative number of recorded cases in the two states as of Thursday, 20 September, 2018 stands at $3,126$ including 97 deaths. This represents a cumulative case fatality rate of 7.8% [29].

*Department of Mathematical Sciences, Faculty of Physical Sciences, Bayero University, Kano, Nigeria
†Department of Mathematical Sciences, Faculty of Physical Sciences, Bayero University, Kano, Nigeria; hashitu.mth@buk.edu.ng

Mathematical models have been proposed to investigate the complex epidemic and endemic behavior of cholera using time delay as incubation period [16, 24, 32, 34] and some without delay [5, 23]. In 2001, Codeço [5] formulated a pioneer mathematical model of cholera that incorporated an environmental reservoir of *Vibrio cholerae* with the objective of exploring the role of aquatic reservoir on the persistence of endemic cholera and to define minimum conditions for the development of epidemic and endemic cholera. In [1], Berge, *et al* studied a two-patch model for the transmission of cholera using the environmental reservoir in [5] as pathogen compartment. In their model, they categorized the mode of transmission into two, namely; fast (human-human transmission) and slow (human-environment transmission) as reported in [5, 12]. The main aim of the study was to investigate the impact of human population movements between two cities (patches) in the transmission of cholera. They obtained the basic reproduction number R_0, which defends on the rate of human movement, in addition to other model parameters. Nirwani *et al* [23] analyzed $SIQR - B$ (Susceptible, Infectious, Quarantine, Removed and Pathogen) model for transmission of cholera with saturated incidence rate and quarantine as measure of intervention. The study focused on effects of quarantine and incidence on the spread of cholera. Wang and Wang [30] Studied a cholera model that incorporates human behaviour via modelling disease prevalence depending on contact rate for direct and indirect transmissions and infectious host shedding. Other related delay model is presented by Prasenjit, *et al* in 2007, [24] that incorporated delay as time between infection and infectiousness for the reservoir hosts in the model of American Cutaneous Leishmaniasis originally formulated in [9].

Quarantine is a disease control strategy that has been adopted as a mean of separating people, animals and goods that may have been exposed to communicable diseases. The separation can be in form of isolation, sanitary cordons, bills of health issued to ships, fumigation, disinfection and regulation of groups of persons, amongst other strategies [20]. In [7], quarantine is defined loosely as the temporary removal (from their immediate abode or the general population) of people suspected of being exposed to a communicable disease. The Holling type II otherwise called saturated incidence function is used widely in mathematical epidemiology to describe a situation where a population is saturated with susceptibles or infectives or both - see for example [17, 25].

In this paper, we study the transmission dynamics of cholera in human population which is divided into four compartments, and the population of *Vibrio cholerae* pathogens concentration with time delay as incubation period. Furthermore, we use a saturated incidence function of Holling type II and quarantine of infected individuals inform of isolation/treatment as control measure. The main objective is to investigate the effects of quarantine and time delay on both dynamics and infectivity on the model. This research is extension of the work in [23, 27, 31] in the following sense.

1. In Nirwani *et al* [23], No time delay and global stabilities of equilibria.
2. In Sun *et al* [27], No time delay.
3. In Wang and Wei [31], No any control strategy.

2 Model Formulation

The human population is divided into four compartments at any time t given as Susceptible $S(t)$, Infectious $I(t)$, Quarantine $Q(t)$ and Removed $R(t)$, while the concentration of cholera bacterium in the environment as $B(t)$. The total human population at time t is given by

$N(t) = S(t) + I(t) + Q(t) + R(t)$. The susceptible humans compartment is increasing with constant recruitment at rate (A), decrease due to infection described by Holling type II saturated incidence rate $(\frac{\beta_1 S(t)B(t)}{1+\alpha_1 B(t)} + \frac{\beta_2 S(t)I(t-\tau)}{1+\alpha_2 I(t-\tau)})$ and by natural death at rate (μ_1), which is also applicable to all human compartments. The parameters β_1 and β_2 are the human-environment and human-human contact rates respectively. Here, τ is time delay representing the incubation period of cholera, while $e^{-\mu_1 \tau}$ is the probability that infectives will survive natural death over the period $0 \leq t \leq \tau$. Similarly, α_1 and α_2 are constants of the incidence rate that determine the rate of new infection. Infectious compartment is increasing due to infection at the rate $\left(\frac{\beta_1 S(t)B(t)}{1+\alpha_1 B(t)} + \frac{\beta_2 S(t)I(t-\tau)e^{-\mu_1 \tau}}{1+\alpha_2 I(t-\tau)}\right)$, decrease by deaths due to infection and naturally at rates (d_1) and (μ_1), respectively, natural recovery and quarantine at rates (α) and (δ), respectively. Similarly, the quarantine compartment increases by the rate of infectious individuals been quarantined at rate (δ), decreases by the rate of recovery of quarantined individuals, natural death and disease induced at rates (ϵ), (μ) and (d_2), respectively. The removed class is increased by the recoveries of infectious and quarantined individuals at rates (α) and (ϵ), respectively, and decrease by the natural death of humans at rate (μ_1). Finally, the *Vibro cholarae* is increased by the infectious human contribution to the environment at rate (η) and decreased by the natural death of pathogens at rate (μ_2).

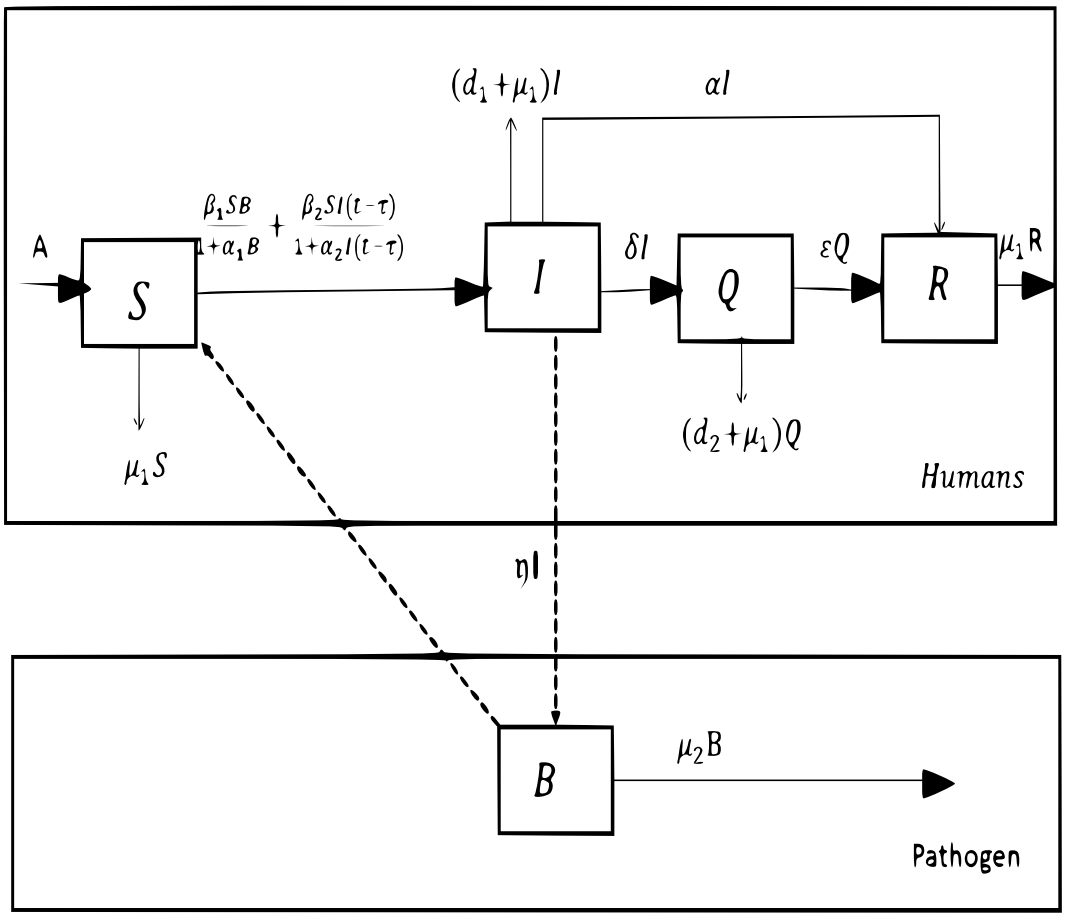


Figure 1: Schematic diagram for the model (2.1)–(2.5) illustrating movements between compartments for transmission of cholera.

It should be noted that in Figure 1, the solid arrows indicate movements among the human population due to change of status (compartment) in the course of infection or recovery, while the dotted arrows indicate interaction between human population and the cholera bacterium in

Variable/Parameter	Description	Units
S	Susceptible individuals	individual
I	Infectious individuals	individual
Q	Quarantine individuals	individual
R	Removed individuals	individual
B	Pathogen concentration in water	cell ml^{-1}
A	Recruitment of susceptible individuals	day^{-1}
μ_1	Natural death human of population	day^{-1}
μ_2	Death rate of pathogen in the environment	day^{-1}
β_1	Human-environment contact rate	$cell^{-1}\ ml^{-1}\ day^{-1}$
β_2	Human-human contact rate	$individual^{-1}\ day^{-1}$
d_1	Death rate of infected individuals due to disease	day^{-1}
d_2	Death rate of quarantine individuals due to disease	day^{-1}
δ	Quarantine rate of infected individuals	day^{-1}
ϵ	Rate of recovery of after quarantine	day^{-1}
α	Rate of recovery of infected individuals	day^{-1}
η	Infected human contributions to the pathogen	cell $ml^{-1}\ day^{-1}\ indl.^{-1}$
α_1	Determinant of new infections through Pathogen	day^{-1}
α_2	Determinant of new infections through infectious humans	day^{-1}

Table 1: Descriptions of Variables and Parameters for System (2.1)–(2.5).

the environment. The model is therefore described by the flow diagram Figure 1 and represented with the following system of delay differential equations:

$$\frac{dS(t)}{dt} = A - \frac{\beta_1 S(t)B(t)}{1+\alpha_1 B(t)} - \frac{\beta_2 S(t)I(t-\tau)e^{-\mu_1\tau}}{1+\alpha_2 I(t-\tau)} - \mu_1 S(t), \tag{2.1}$$

$$\frac{dI(t)}{dt} = \frac{\beta_1 S(t)B(t)}{1+\alpha_1 B(t)} + \frac{\beta_2 S(t)I(t-\tau)e^{-\mu_1\tau}}{1+\alpha_2 I(t-\tau)} - (d_1+\mu_1+\alpha+\delta)I(t), \tag{2.2}$$

$$\frac{dQ(t)}{dt} = \delta I(t) - (\epsilon+\mu_1+d_2)Q(t), \tag{2.3}$$

$$\frac{dB(t)}{dt} = \eta I(t) - \mu_2 B(t). \tag{2.4}$$

$$\frac{dR(t)}{dt} = \alpha I(t) + \epsilon Q(t) - \mu_1 R(t). \tag{2.5}$$

The model (2.1)–(2.5) satisfy the initial conditions

$$S(\theta) = \psi_1(\theta),\ I(\theta) = \psi_2(\theta),\ Q(\theta) = \psi_3(\theta),\ B(\theta) = \psi_4(\theta),\ R(\theta) = \psi_5(\theta), \tag{2.6}$$

for all $\theta \in [-\tau, 0]$. where $\psi_i(\theta)$, $i = 1,2,3,4,5 \in \mathcal{C}$, the Banach space $\mathcal{C}([-\tau,0],\mathbb{R}^5)$ of continuous functions mapping$[-\tau,0] \to \mathbb{R}^5$, with $\|\psi_i(\theta)\| = \sup_{-\tau\le\theta\le 0} |\psi_i|$, $i = 1,2,3,4,5$. We further assume that $\psi_i(\theta) \ge 0$ for $i = 1,...,5$.

3 Basic properties of the model

In this section, we present results for basic properties of the model such as existence, uniqueness, positivity and boundedness of solution. We also present existence and stability of equilibria.

Theorem 1 *The solution* $(S(t), I(t), Q(t), B(t), R(t))$ *of system* (2.1)–(2.5)*, exists and is bounded for all time* $t \geq 0$ *under the initial conditions* (2.6)*. Moreover, the solution is positive for all* $t > 0$ *and the region*

$$\Omega = \left\{ (S, I, Q, B, R) \in \mathbb{R}^5_+ : S + I + Q + R \leq \frac{A}{\mu_1}, B \leq \frac{K}{\mu_2} \right\}$$

is positive invariant, where $K = \eta \frac{A}{\mu_1}$.

Proof. We first prove that the solution $(S(t), I(t), Q(t), B(t), R(t))$ exists and is unique as shown below.
Let $X(t) = (S(t),\ I(t),\ Q(t),\ R(t),\ B(t))$. The system (2.1)–(2.5), can be written as

$$\frac{dX}{dt} = f(t, X_t),$$

where $X_t(\theta) = X(t + \theta)$, for $\theta \in [-\tau, 0]$, f is Lipschitz in X. It then follows from Theorems 2.1 and 2.3 in [11] that the system (2.1)–(2.5) has a unique solution $(S(t),\ I(t),\ Q(t),\ R(t),\ B(t))$ satisfying the initial data (2.6).

For the positivity of solution $(S(t),\ I(t),\ Q(t),\ R(t),\ B(t))$, we assume that the initial condition in (2.6) hold true, then we prove that $S(t) > 0$, $I(t) > 0$, $R(t) > 0$, $B(t) > 0$ for all time $t \in (0, \infty)$. We proceed by contradiction by assuming that the solution is non-positive. This will implies that the corresponding derivatives are also non-positive (≤ 0). Then there exists a time $t_1 > 0$ such that

$$\min\{S(t_1), I(t_1), R(t_1), B(t_1)\} = 0,$$

and

$$\min\{S(t), I(t), Q(t), R(t), B(t)\} > 0 \text{ for all } \ t \in [0, t_1).$$

If $\min\{S(t_1), I(t_1), Q(t_1), R(t_1), B(t_1)\} = I(t_1) = 0$, then from (2.4),

$$\frac{dB(t)}{dt} \ \geq \ -\mu_2 B(t), \text{ for all } \ t \in [0, \hat{t}],$$

hence

$$B(t_1) \ > \ B(0) \exp(-\mu_2 t_1) > 0,$$

which contradicts our earlier assumption. Hence there is no such time $t_1 > 0$, which implies that $B(t) > 0$ for all $t > 0$.

Similarly, from (2.3), we get

$$\frac{dQ(t)}{dt} \ \geq \ -(\epsilon + \mu_1 + d_2) Q(t), \text{ for all } \ t \in [0, t_1],$$

so that

$$Q(t_1) \ > \ Q(0) \exp(\epsilon + \mu_1 + d_2) t_1 > 0,$$

which also contradicts our earlier assumption. Hence there is no such time $t_1 > 0$, which implies that $Q(t) > 0$ for all $t > 0$.

Suppose also the $\min\{S(t_1), I(t_1), Q(t_1), R(t_1), B(t_1)\} = S(t_1) = 0$, then from (2.2) we have

$$\frac{dI(t)}{dt} \ \geq \ -(d_1 + \mu_1 + \alpha + \delta) I(t), \text{ for all } \ t \in [0, t_1],$$

hence

$$I(t_1) \quad > \quad I(0)\exp((d_1+\mu_1+\alpha+\delta)t_1) > 0.$$

This also contradicts our assumption. Hence there is no such time $t_1 > 0$, hence, $I(t) > 0$ for all $t > 0$. Similar approaches can be followed to show that $S(t)$, $R(t)$ are positive at all time $t > 0$.

Thus, starting with positive initial values, all solutions of the model (2.1)–(2.5) will be positive at all time $t > 0$.

For boundedness of solution, we prove that the total population of humans ($N(t)$) and the pathogen concentration ($B(t)$) at time t are bounded as shown below: Adding equations (2.1)–(2.3) and (2.5) we have

$$\frac{dN(t)}{dt} \leq A - \mu_1 N(t). \tag{3.1}$$

Solving (3.1) we get

$N(t) \leq \frac{A}{\mu_1} + \left[N(0) - \frac{A}{\mu_1}\right] e^{-\mu_1 t}$.
If $N(0) \leq \frac{A}{\mu_1}$, then
$0 \leq N(t) \leq \frac{A}{\mu_1}$.

Furthermore, it follows from equation (2.4) that since $I(t) \leq N(t) \leq \frac{A}{\mu_1}$, it implies that $I(t) \leq \frac{A}{\mu_1}$. Then

$$\frac{dB(t)}{dt} \leq K - \mu_2 B(t) \tag{3.2}$$

Using Gronwall's Inequality [10],

$0 \leq B(t) \leq \frac{K}{\mu_2}$.

This completes the proof of Theorem 1. ■

4 Equilibria points and Stability Analysis

At equilibrium points, $I(t) = I(t-\tau) = I^*$. It is observed that in system (2.1)–(2.5), $R(t)$ appear only in equation (2.4). Therefore, without loss of generality, the dynamics of the system can be considered by dropping (2.5).

To determine equilibria of the system, we set the right hand sides of equations (2.1)–(2.4) to zero. Thus

$$A - \frac{\beta_1 S(t)B(t)}{1+\alpha_1 B(t)} - \frac{\beta_2 S(t)I(t-\tau)e^{-\mu_1\tau}}{1+\alpha_2 I(t-\tau)} - \mu_1 S = 0, \tag{4.1}$$

$$\frac{\beta_1 S(t)B(t)}{1+\alpha_1 B(t)} + \frac{\beta_2 S(t)I(t-\tau)e^{-\mu_1\tau}}{1+\alpha_2 I(t-\tau)} - (d_1+\mu_1+\alpha+\delta)I = 0, \tag{4.2}$$

$$\delta I(t) - (\epsilon+\mu_1+d_1)Q(t) = 0, \tag{4.3}$$

$$\eta I(t) - \mu_2 B(t) = 0. \tag{4.4}$$

From equation (4.4),

$$B^* = \frac{\eta I^*}{\mu_2}. \tag{4.5}$$

Similarly, from (4.3),

$$Q^* = \frac{\delta I^*}{(\epsilon + \mu_1 + d_1)}. \tag{4.6}$$

Substituting (4.5) in (4.2) we have

$$\left[\frac{\beta_1\eta}{\mu_2 + \alpha_1\eta I^*} + \frac{\beta_2 e^{-\mu_1\tau}}{1 + \alpha_2 I^*}\right] S^* I^* - (d_1 + \mu_1 + \alpha + \delta)I^* = 0. \tag{4.7}$$

Adding (4.1) and (4.2) gives

$$S^* = \frac{A - (d_1 + \mu_1 + \alpha + \delta)I^*}{\mu_1}. \tag{4.8}$$

Substituting equation (4.8) into (4.7) we have

$$\left[\left(\frac{\beta_1\eta}{\mu_2 + \alpha_1\eta I^*} + \frac{\beta_2 e^{-\mu_1\tau}}{1 + \alpha_2 I^*}\right)\left(\frac{A - (d_1 + \mu_1 + \alpha + \delta)}{\mu_1}\right)\right] I^* - (d_1 + \mu_1 + \alpha + \delta)I^* = 0. \tag{4.9}$$

The trivial case, when $I^* = 0$, give rise to the disease free equilibrium (DFE) denoted by $E_1 = (S^0, I^0, Q^0, B^0)$ given by,

$$E_1 = \left(\frac{A}{\mu_1}, 0, 0, 0\right). \tag{4.10}$$

.

However, for non trivial solution, ($I^* \neq 0$), we get

$$\left[\frac{\beta_1\eta}{\mu_2 + \alpha_1\eta I^*} + \frac{\beta_2 e^{-\mu_1\tau}}{1 + \alpha_2 I^*}\right]\left[\frac{A - (d_1 + \mu_1 + \alpha + \delta)I^*}{\mu_1}\right] - (d_1 + \mu_1 + \alpha + \delta) = 0 \tag{4.11}$$

.

After simplifying equation (4.11), we obtain the following quadratic equation in I^*.

$$C_1(I^*)^2 + C_2 I^* + C_3 \quad = 0, \tag{4.12}$$

where $C_1 = [(\beta_1\alpha_2\eta + \beta_2\alpha_1\eta)(d_1 + \mu_1 + \alpha + \delta) + \alpha_1\alpha_2\eta\mu_1(d_1 + \mu_1 + \alpha + \delta)]$,
$C_2 = [(\beta_1\eta + \beta_2\mu_2)(d_1 + \mu_1 + \alpha + \delta) + (\mu_2\alpha_2 + \alpha_1\eta)(d_1 + \mu_1 + \alpha + \delta)\mu_1 - (\beta_1\alpha_2\eta + \beta_2\alpha_1\eta)A]$, and
$C_3 = -\mu_1\mu_2(d_1 + \mu_1 + \alpha + \delta)[\mathcal{R}_q - 1]$.

Here, we define

$$\mathcal{R}_q = \frac{A(\beta_1\eta + \beta_2\mu_2 e^{-\mu_1\tau})}{\mu_1\mu_2(d_1 + \mu_1 + \alpha + \delta)}, \tag{4.13}$$

to be the control reproduction number. The basic control reproduction number is defined as the expected number of secondary cases produced by introducing one infected person and cholera pathogens in a completely susceptible population in the presence of intervention. $\mathcal{R}_q$ can also be calculated using the next infection operator matrix as shown in [36, 37],

From (4.12), it can be observed that $C_1 > 0$, and $C_3 < 0$ if $\mathcal{R}_q > 1$, while C_2 can be > 0 or < 0. Therefore, irrespective of the sign of C_2, equation (4.12) will admit a unique positive endemic equilibrium in I^*.

Therefore, the endemic equilibrium (EE) is given by

$$E_2 = (S^*, I^*, Q^*, B^*),$$

where

$$S^* = \frac{(d_1+\mu_1+\alpha+\delta)}{\left[\frac{\beta_1\eta}{\mu_2+\alpha_1\eta I^*}+\frac{\beta_2 e^{-\mu_1\tau}}{1+\alpha_2 I^*}\right]},\ Q^* = \frac{\delta I^*}{(\epsilon+\mu_1+d_2)},\ B^* = \frac{\eta I^*}{\mu_2},$$

with I^* the unique positive root of equation (4.12). Thus the endemic equilibrium exists whenever $\mathcal{R}_q > 1$.

In order to study the stability of equilibria, we first linearize the system (2.1)–(2.4) about an arbitrary equilibrium to have,

$$\frac{dZ}{dt} = J_1 Z(t) + J_2 Z(t-\tau), \tag{4.14}$$

where $Z(t) = (S(t), I(t), Q(t), B(t))^T$ is a vector while J_1 and J_2 are the Jacobian matrices of the system given as

$$J_1 = \begin{pmatrix} -\frac{\beta_1 B(t)}{1+\alpha_1 B(t)} - \frac{\beta_2 I(t-\tau)e^{-\mu_1\tau}}{1+\alpha_2 I(t-\tau)} - \mu_1 & 0 & 0 & \frac{\alpha_1\beta_1 S(t)B(t) - (1+\alpha_1 B(t))\beta_1 S(t)}{(1+\alpha_1 B(t))^2} \\ \frac{\beta_1 B(t)}{1+\alpha_1 B(t)} + \frac{\beta_2 I(t-\tau)e^{-\mu_1\tau}}{1+\alpha_2 I(t-\tau)} & -(d_1+\mu_1+\alpha+\delta) & 0 & 0 \\ 0 & \delta & -(d_2+\mu_1+\epsilon) & 0 \\ 0 & \eta & 0 & -\mu_2 \end{pmatrix},$$

$$J_2 = \begin{pmatrix} 0 & \frac{\alpha_2\beta_2 S(t)I(t-\tau)e^{-\mu_1\tau} - \beta_2 S(1+\alpha_2 I(t-\tau)e^{-\mu_1\tau})}{(1+\alpha_2 I(t-\tau))^2} & 0 & 0 \\ 0 & \frac{\beta_2 S(1+\alpha_2 I(t-\tau)e^{-\mu_1\tau}) - \alpha_2\beta_2 SI(t-\tau)e^{-\mu_1\tau}}{(1+\alpha_2 I(t-\tau))^2} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

Assuming a solution of the form

$$Y(t) = \mathbf{c}e^{\lambda t},$$

where $\mathbf{c}$ is a constant vector and λ is a complex eigenvalue.

For a non trivial solution ($ce^{\lambda t} \neq 0$) therefore, we have

$$J_1 + J_2 e^{-\lambda\tau} = 0, \tag{4.15}$$

as the transcendental equation.

4.1 Local stability of disease free equilibrium

To investigate the local stability of DFE E_1, we state and prove the following result.

Theorem 2 *The disease free equilibrium E_1 is absolutely stable if $\mathcal{R}_q < 1$ for all delay $\tau \geq 0$ and unstable otherwise.*

Proof. We substitute the disease free equilibrium E_1 into equation (4.15) to get the corresponding transcendental equation as

$$\begin{aligned} H_1(\lambda) = \quad & (\lambda+\mu_1)(\lambda+d_2+\mu_1+\epsilon)[(\lambda-\beta_2\tfrac{A}{\mu_1}e^{-\lambda\tau}+d_1+\mu_1+\alpha+\delta)(\lambda+\mu_2) \\ & -\eta\beta_1\tfrac{A}{\mu_1})] = 0. \end{aligned} \tag{4.16}$$

Clearly, H_1 has two negative real roots. Thus,

$\lambda_1 = -\mu_1$ and $\lambda_2 = -(d_2+\mu_1+\epsilon)$. Hence we concentrate on the remaining part of (4.16) defined by,

$$\begin{aligned} H_2(\lambda) = \lambda^2 &+ (d_1+\mu_1+\alpha+\delta+\mu_2-\beta_2\frac{A}{\mu_1}e^{-\lambda\tau})\lambda + (d_1+\mu_1+\alpha+\delta \\ &- \beta_2\frac{A}{\mu_1}e^{-\lambda\tau})\mu_2 - \eta\beta_1\frac{A}{\mu_1}. \end{aligned} \tag{4.17}$$

Assume $\tau = 0$. From (4.17) we have

$$\begin{aligned} H_2(\lambda) =& \lambda^2 + (d_1+\mu_1+\alpha+\delta+\mu_2-\beta_2\frac{A}{\mu_1})\lambda + (d_1+\mu_1+\alpha+\delta-\beta_2\frac{A}{\mu_1})\mu_2 - \eta\beta_1\frac{A}{\mu_1} \\ =& \lambda^2 + \lambda\left[d_1+\mu_1+\alpha+\delta+\mu_2-\frac{\beta_2\mu_2(d_1+\mu_1+\alpha+\delta)\mathcal{R}_q}{\beta_1\eta+\beta_2\mu_2}\right] \\ &+ \mu_2(d_1+\mu_1+\alpha+\delta)[1-\mathcal{R}_q] \\ =& \lambda^2 + \lambda\left[\mu_2+(d_1+\mu_1+\alpha+\delta)(1-\frac{\beta_2\mu_2\mathcal{R}_q}{\beta_1\eta+\beta_2\mu_2}\right] + \mu_2(d_1+\mu_1+\alpha+\delta)[1-\mathcal{R}_q]. \end{aligned}$$

This implies that, when $\mathcal{R}_q < 1$, the coefficient of λ and the constant term are positive. Therefore, by Descarte's rule of signs, $H_2(\lambda)$ has no positive real roots. Hence, $H_2(\lambda)$ is stable when $\tau = 0$.

If $\tau \neq 0$, then from (4.17), and substituting $\lambda = iy$ to be a root, where $y \in \mathbb{R}_+$, we have

$$\begin{aligned} F_1(y) = y^4 &+ \left[(d_1+\mu_1+\mu_2+\alpha+\delta)+\beta_2\frac{A}{\mu_1}\right]\left[\mu_2+(d_1+\mu_1+\alpha+\delta)(1-\frac{\beta_2\mu_2R_q}{\beta_1\eta+\beta_2\mu_2}\right]y^2 \\ &+ \left[(d_1+\mu_1+\alpha+\delta)\mu_1\mu_2+A(\eta\beta_1+\mu_2\beta_2)\right](d_1+\mu_1+\alpha+\delta)\mu_1\mu_2\left[1-R_q\right]. \end{aligned}$$

Thus, when $\mathcal{R}_q < 1$, the coefficient of y^2 and the constant term are positive. Therefore, by the application of Descarte's rule of sign, $F_1(y) = 0$ has no positive real root and there is no $y \in \mathbb{R}_+$ such that $\lambda = iy$ can be a root of $F_1(y)$. Hence, the disease free equilibrium E_1 is absolutely stable when $\mathcal{R}_q < 1$ and unstable otherwise. ■

4.2 Global stability of disease free equilibrium

In order to be certain about the eradication of cholera irrespective of the initial population started with, we prove the global attractiveness of the DFE as shown below.

Theorem 3 *The DFE E_1 is globally asymptotically stable for all delay $\tau \geq 0$ when $\mathcal{R}_q < 1$ and unstable otherwise.*

Proof. We assume a continuously differentiable Lyapunov function

$$V = \mu_2\eta I + \mu_2(d_1 + \mu_1 + \alpha + \delta)B. \tag{4.18}$$

Differentiating equation (4.18) along the solution of model (2.1)–(2.5), we have

$$V' = \mu_2\eta I' + \mu_2(d_1 + \mu_1 + \alpha + \delta)B'. \tag{4.19}$$

Substituting I', B' in (4.19) we get

$$\begin{aligned} V' =& \mu_2\eta\left[\frac{\beta_1 SB}{1+\alpha_1 B} + \frac{\beta_2 SIe^{-\mu_1\tau}}{1+\alpha_2 I} - (d_1+\mu_1+\alpha+\delta)I\right] + \mu_2(d_1+\mu_1+\alpha+\delta)\left[\eta I - \mu_2 B\right], \\ =& \frac{\mu_2\eta\beta_1 SB}{1+\alpha_1 B} + \frac{\mu_2\eta\beta_2 SIe^{-\mu_1\tau}}{1+\alpha_2 I} - \mu_2^2(d_1+\mu_1+\alpha+\delta)B. \end{aligned}$$

From Theorem 1, $I(t) \leq \frac{A}{\mu_1}$, $S(t) \leq \frac{A}{\mu_1}$ and $B \leq \frac{\eta A}{\mu_1\mu_2}$, thus

$$\begin{aligned} V' \leq& \frac{\eta^2\beta_1 AS}{\mu_1} + \frac{\mu_2\eta\beta_2 ASe^{-\mu_1\tau}}{\mu_1} - \frac{\mu_2\eta(d_1+\mu_1+\alpha+\delta)A}{\mu_1}, \\ \leq& \frac{\eta S}{\mu_1}\left(\eta\beta_1 A + \mu_2\beta_2 Ae^{-\mu_1\tau} - \mu_1\mu_2(d_1+\mu_1+\alpha+\delta)I\right) \\ \leq& \eta\mu_2(d_1+\mu_1+\alpha+\delta)S\left[\left(\frac{\eta\beta_1 A + \mu_2\beta_2 Ae^{-\mu_1\tau}}{\mu_1\mu_2(d_1+\mu_1+\alpha+\delta)}\right) - 1\right], \\ \leq& \eta\mu_2(d_1+\mu_1+\alpha+\delta)S\left(\mathcal{R}_q - 1\right). \end{aligned}$$

Thus,

$$V' \leq \eta\mu_2(d_1+\mu_1+\alpha+\delta)S\left(\mathcal{R}_q - 1\right). \tag{4.20}$$

All parameters and variables of the model are non-negative, therefore, $V' \leq 0$ when $\mathcal{R}_q < 1$ with equality if and only if $S = \frac{A}{\mu_1}$, $I = B = 0$. Hence, V is a Lyapunov function in the feasible region Ω, and it can easily be shown that E_1 is the largest invariant set in Ω. Hence by Lasalle's Invariance Principle [19], the disease free equilibrium is globally asymptotically stable when $\mathcal{R}_q < 1$ for all $\tau \geq 0$ and unstable if $\mathcal{R}_q > 1$. ■

4.3 Local stability of endemic equilibrium (EE)

Here we establish the local stability of endemic equilibrium point E_2 as follows.

Theorem 4 *The endemic equilibrium E_2, is locally asymptotically stable if $\mathcal{R}_q > 1$ for all $\tau \geq 0$ and unstable otherwise.*

Proof. To investigate the local stability of EE first we substitute E_2 into the Jacobian matrix (4.15) to get the transcendental equation as

$$\begin{aligned} H_3(\lambda) =& (\lambda + K_1 + \mu_1)(\lambda + d_2 + \mu_1 + \epsilon)[(\lambda + K_2e^{-\lambda\tau} + (d_1+\mu_1+\alpha+\delta))(\lambda+\mu_2) \\ &+ \eta K_3] = 0, \end{aligned} \tag{4.21}$$

where

$$\begin{aligned} K_1 &= \frac{\beta_1 B^*}{1+\alpha_1 B^*} + \frac{\beta_2 I^*}{1+\alpha_2 I^*},\ K_2 = \frac{\alpha_2\beta_2 S^* I^* - \beta_2 S^*(1+\alpha_2 I^*)}{(1+\alpha_2 I^*)^2} = -\frac{\beta_2 S^*}{(1+\alpha_2 I^*)^2}, \\ K_3 &= \frac{(1+\alpha_1 B^*)\beta_1 S^* - \alpha_1\beta_1 S^* B^*}{(1+\alpha_1 B^*)^2} = \frac{\beta_1 S^*}{(1+\alpha_1 B^*)^2}. \end{aligned}$$

Clearly, H_3 has two negative real roots: $\lambda_1 = -K_1 - \mu_1 < 0$ and $\lambda_2 = -(d_2 + \mu_1 + \epsilon) < 0$. Therefore, stability of E_2 depends on the remaining part of H_3 defined as

$$\begin{aligned} H_4(\lambda) =& \lambda^2 + (d_1 + \mu_1 + \mu_2 + \alpha + \delta + K_2 e^{-\lambda\tau})\lambda + \mu_2(d_1 + \mu_1 + \alpha + \delta) \\ &+ \mu_2 K_2 e^{\lambda\tau} + \eta K_3. \end{aligned} \tag{4.22}$$

When $\tau = 0$,

$$H_4(\lambda) = \lambda^2 + (d_1 + \mu_1 + \mu_2 + \alpha + \delta + K_2)\lambda + \mu_2(d_1 + \mu_1 + \alpha + \delta) + \mu_2 K_2 + \eta K_3.$$

It can be observed that all the coefficients of H_4 are positive. By Discarte's rule of sign, $H_4(\lambda)$ has no positive real roots, hence, it is stable.

However, when $\tau \neq 0$, using Theorem (1) in [6], and letting $\lambda = iy$ for $y \in \mathbb{R}_+$ as a root of H_4, we have

$$\begin{aligned} F_2(y) = y^4 &+ \left[(d_1 + \mu_1 + \mu_2 + \alpha + \delta) + \frac{\beta_2 S^*}{(1+\alpha_2 I^*)^2}\right]\left[(d_1 + \mu_1 + \mu_2 + \alpha + \delta) + K_2\right] y^2 \\ &+ \left[(d_1 + \mu_1 + \alpha + \delta)\mu_2 + \left[\frac{\eta\beta_1}{(1+\alpha_1 B^*)^2} + \frac{\mu_2\beta_2}{(1+\alpha_2 I^*)^2}\right] S^*\right] \\ &\left[(d_1 + \mu_1 + \alpha + \delta)\mu_2 + \left[\frac{\eta\beta_1}{(1+\alpha_1 B^*)^2} - \frac{\mu_2\beta_2}{(1+\alpha_2 I^*)^2}\right] S^*\right]. \end{aligned}$$

Whenever, $\mathcal{R}_q > 1$, the coefficient of y^2 and the constant term are positive values. Therefore, by the application of Descarte's rule of sign, $F_2(y) = 0$ has no positive real root. Therefore there is no $y \in \mathbb{R}_+$ such that $\lambda = iy$ can be a root of $F_2(y) = 0$. Hence, the endemic equilibrium E_2 is locally asymptotically stable when $\mathcal{R}_q > 1$ for all $\tau \geq 0$ and unstable otherwise. ■

4.4 Global stability of endemic equilibrium

In this section the global asymptotic stability for the endemic equilibrium E_2 is shown. This result will present the conditions for existence of endemic cholera in the population irrespective of initial populations started with.

Theorem 5 *If $\mathcal{R}_q > 1$, the endemic equilibrium E_2 is globally asymptotically stable for all delay $\tau \geq 0$ and unstable otherwise.*

Proof. Considering equations (2.1)–(2.4) of the model, it is observed that, equation (2.4) is independent of the other equations, hence can be drop. At equilibrium, from equations (2.1)–

(2.3), we have

$$A = \frac{\beta_1 S^* B^*}{1+\alpha_1 B^*} + \frac{\beta_2 S^* I^* e^{-\mu_1 \tau}}{1+\alpha_2 I^*} + \mu_1 S^*, \tag{4.23}$$

$$(d_1+\mu_1+\alpha+\delta) = \frac{1}{I^*}\left[\frac{\beta_1 S^* B^*}{1+\alpha_1 B^*} + \frac{\beta_2 S^* I^* e^{-\mu_1 \tau}}{1+\alpha_2 I^*}\right], \tag{4.24}$$

$$\eta = \frac{\mu_2 B^*}{I^*}. \tag{4.25}$$

We follow the approach in [22] and define a Lyapunov function as

$$\mathcal{F} = (S - S^* \ln S) + G(I - I^* \ln I) + H(B - B^* \ln B) \tag{4.26}$$

By definition $\mathcal{F} > 0$, where G and H are positive constants to be determined.

Differentiating $\mathcal{F}$ along the solution curves of the model, we have

$$\begin{aligned}
\mathcal{F}' &= \left(1-\frac{S^*}{S}\right)S' + G\left(1-\frac{I^*}{I}\right)I' + H\left(1-\frac{B^*}{B}\right)B' \\
&= \left(1-\frac{S^*}{S}\right)\left[A - \frac{\beta_1 SB}{1+\alpha_1 B} - \frac{\beta_2 SIe^{-\mu_1\tau}}{1+\alpha_2 I} - \mu_1 S\right] \\
&\quad + G\left(1-\frac{I^*}{I}\right)\left[\frac{\beta_1 SB}{1+\alpha_1 B} + \frac{\beta_2 SIe^{-\mu_1\tau}}{1+\alpha_2 I} - (d_1+\mu_1+\alpha+\delta)I\right] \\
&\quad + H\left(1-\frac{B^*}{B}\right)\left[\eta I - \mu_2 B\right].
\end{aligned}$$

Substituting equations (4.23)–(4.25) in $\mathcal{F}'$, we have

$$\begin{aligned}
\mathcal{F}' &= \left(1-\frac{S^*}{S}\right)\left[\frac{\beta_1 S^* B^*}{1+\alpha_1 B^*} + \frac{\beta_2 S^* I^* e^{-\mu_1\tau}}{1+\alpha_2 I^*)} + \mu_1 S^* - \frac{\beta_1 SB}{1+\alpha_1 B} - \frac{\beta_2 SIe^{-\mu_1\tau}}{1+\alpha_2 I} - \mu_1 S\right] \\
&\quad + G\left(1-\frac{I^*}{I}\right)\left[\frac{\beta_1 SB}{1+\alpha_1 B} + \frac{\beta_2 SIe^{-\mu_1\tau}}{1+\alpha_2 I} - \frac{I}{I^*}\left[\frac{\beta_1 S^* B^*}{1+\alpha_1 B^*} + \frac{\beta_2 S^* I^* e^{-\mu_1\tau}}{1+\alpha_2 I^*}\right]\right] \\
&\quad + H\left(1-\frac{B^*}{B}\right)\left[\frac{\mu_2 B^* I}{I^*} - \mu_2 B\right], \\
&= \left(1-\frac{S^*}{S}\right)\mu_1(S^*-S) + \left(1-\frac{S^*}{S}\right)\left[\frac{\beta_1 S^* B^*}{1+\alpha_1 B^*}\right] + \left(1-\frac{S^*}{S}\right)\left[\frac{\beta_2 S^* I^* e^{-\mu_1\tau}}{1+\alpha_2 I^*)}\right] \\
&\quad - \left(1-\frac{S^*}{S}\right)\left[\frac{\beta_1 SB}{1+\alpha_1 B}\right] - \left(1-\frac{S^*}{S}\right)\left[\frac{\beta_2 SIe^{-\mu_1\tau}}{1+\alpha_2 I} - \mu_1 S\right] + G\left(1-\frac{I^*}{I}\right)\left[\frac{\beta_1 SB}{1+\alpha_1 B}\right] \\
&\quad + G\left(1-\frac{I^*}{I}\right)\left[\frac{\beta_2 SIe^{-\mu_1\tau}}{1+\alpha_2 I}\right] - G(\frac{I}{I^*}-1)\left[\frac{\beta_1 S^* B^*}{1+\alpha_1 B^*}\right] - G(\frac{I}{I^*}-1)\left[\frac{\beta_2 S^* I^* e^{-\mu_1\tau}}{1+\alpha_2 I^*}\right] \\
&\quad + H\mu_2 B^*\left(1-\frac{B^*}{B}\right)\left[\frac{I}{I^*} - \frac{B}{B^*}\right], \\
&= -\mu_1\frac{(S-S^*)^2}{S} + \frac{\beta_1 S^* B^*}{1+\alpha_1 B^*}\left[\left(1-\frac{S^*}{S}\right) - G\left(\frac{I}{I^*}-1\right)\right] \\
&\quad + \frac{\beta_1 S^* B^*}{1+\alpha_1 B}\left[G\left(1-\frac{I^*}{I}\right)\frac{SB}{S^*B^*} - \left(1-\frac{S^*}{S}\right)\frac{SB}{S^*B^*}\right] + \frac{\beta_2 S^* I^* e^{-\mu_1\tau}}{1+\alpha_2 I^*}\left[\left(1-\frac{S^*}{S}\right) - G\left(\frac{I}{I^*}-1\right)\right] \\
&\quad + \frac{\beta_2 S^* I^* e^{-\mu_1\tau}}{1+\alpha_2 I}\left[G\left(1-\frac{I^*}{I}\right)\frac{SI}{S^*I^*} - \left(1-\frac{S^*}{S}\right)\frac{SI}{S^*I^*}\right] + H\mu_2 B^*\left(1-\frac{B^*}{B}\right)\left[\frac{I}{I^*} - \frac{B}{B^*}\right],
\end{aligned}$$

Let $x = \frac{S}{S^*}$, $y = \frac{B}{B^*}$, $z = \frac{I}{I^*}$ *and* $\beta_1 = \beta_2 = \beta$, such that

$$\begin{aligned}\mathcal{F}' =& -\mu_1\frac{(S-S^*)^2}{S} + \frac{\beta S^*B^*}{1+\alpha_1 B^*}\left[(1-\frac{1}{x}) - G(z-1)\right]\\ &+ \frac{\beta S^*B^*}{1+\alpha_1 B}\left[G(1-\frac{1}{z})xy - (1-\frac{1}{x})xy\right] + \frac{\beta S^*I^*e^{-\mu_1\tau}}{1+\alpha_2 I^*}\left[(1-\frac{1}{x}) - G(z-1)\right]\\ &+ \frac{\beta S^*I^*e^{-\mu_1\tau}}{1+\alpha_2 I}\left[G(1-\frac{1}{z})xz - (1-\frac{1}{x})xz\right] + H\mu_2 B^*(1-\frac{1}{y})(z-y),\end{aligned}$$

Now,

$$\mathcal{F}' = -\mu_1\frac{(S-S^*)^2}{S} + f(x,y,z,\beta), \tag{4.27}$$

where,

$$\begin{aligned}f(x,y,z,\beta) =& \frac{\beta S^*B^*}{1+\alpha_1 B^*}\left[(1-\frac{1}{x}) - G(z-1)\right] + \frac{\beta S^*B^*}{1+\alpha_1 B}\left[G(1-\frac{1}{z})xy - (1-\frac{1}{x})xy\right]\\ &+ \frac{\beta S^*I^*e^{-\mu_1\tau}}{1+\alpha_2 I^*}\left[(1-\frac{1}{x}) - G(z-1)\right] + \frac{\beta S^*I^*e^{-\mu_1\tau}}{1+\alpha_2 I}\left[G(1-\frac{1}{z})xz - (1-\frac{1}{x})xz\right]\\ &+ H\mu_2 B^*(1-\frac{1}{y})(z-y),\\ =& \frac{\beta S^*B^*}{1+\alpha_1 B^*}\left[(1-\frac{1}{x}) - G(z-1)\right] + \frac{\beta S^*B^*}{1+\alpha_1 B}\left[G(1-\frac{1}{z})xy - xy + y\right]\\ &+ \frac{\beta S^*I^*e^{-\mu_1\tau}}{1+\alpha_2 I^*}\left[(1-\frac{1}{x}) - G(z-1)\right] + \frac{\beta S^*I^*e^{-\mu_1\tau}}{1+\alpha_2 I}\left[G(1-\frac{1}{z})xz - xz + z\right]\\ &+ H\mu_2 B^*(1+z-y-\frac{z}{y}).\end{aligned}$$

Now, we can choose the values of G and H such that f is non-positive for all $x, y, z \in \mathbb{R}_+$. Assume $G = 1 \quad$ and $\quad H = \frac{1}{\mu_2 B^*}$, then

$$\begin{aligned}f(x,y,z,\beta) =& (2-\frac{1}{x}-z)\beta S^*\left[\frac{B^*}{1+\alpha_1 B^*} + \frac{I^*e^{-\mu_1\tau}}{1+\alpha_2 I^*}\right] + \beta S^*\left[\frac{B^*}{1+\alpha_1 B}(y-\frac{xy}{z}) + \frac{I^*e^{-\mu_1\tau}}{1+\alpha_2 I}(z-x)\right]\\ &+ (1+z-y-\frac{z}{y})\end{aligned} \tag{4.28}$$

Substituting $x = z = 1$ in equation (4.28) implies that

$$f(x,y,z,\beta) = (1+z-y-\frac{z}{y}),$$

which show that $f(x,y,z,\beta) \leq 0$ by the arithmetic mean - geometric mean inequality, and with equality to zero if $y = z = 1$.
Thus, $\mathcal{F} \leq 0$, with equality if and only if $S = S^*$. Hence, $\mathcal{F}$ is a Lyapunov function and it can also be shown that the largest invariant set in Ω is E_2. Thus by LaSalle's Invariance Principle, the endemic equilibrium is globally asymptotically stable for all $\tau \geq 0$ when $\mathcal{R}_q > 1$ and unstable otherwise. ■

5 Analysis for the effect of time delay

In this section, we will investigate the effect of delay (increase or decrease) on the infectivity of cholera in a population. To start with, we use the unique threshold quantity of the model

(control reproduction number, $\mathcal{R}_q$). The idea is to determine a critical delay value above (below) which the number of infected individuals will decrease (increase).

Since $\mathcal{R}_q = 1$ is a bifurcation parameter point where stability changes, we consider it a threshold value for infection and determine the critical delay as follows:

$$\begin{aligned}
&\frac{A(\eta\beta_1 + \mu_2\beta_2 e^{-\mu_1\tau})}{\mu_1\mu_2(d_1+\mu_1+\alpha+\delta)} = 1\\
&A(\eta\beta_1 + \mu_2\beta_2 e^{-\mu_1\tau}) = \mu_1\mu_2(d_1+\mu_1+\alpha+\delta),\\
&\mu_2\beta_2 e^{-\mu_1\tau} = \frac{\mu_1\mu_2(d_1+\mu_1+\alpha+\delta)}{A} - \eta\beta_1,\\
&e^{-\mu_1\tau} = \frac{\mu_1\mu_2(d_1+\mu_1+\alpha+\delta)}{A\mu_2\beta_2} - \frac{\eta\beta_1}{\mu_2\beta_2}.
\end{aligned}$$

Thus, solving for τ, we get the critical delay as

$$\tau_{crit} = \frac{ln\left[\frac{1}{\mu_2\beta_2}\left(\frac{\mu_1\mu_2(d_1+\mu_1+\alpha+\delta)}{A} - \eta\beta_1\right)\right]}{-\mu_1}. \tag{5.1}$$

To determine the nature of (4.13) as either increasing or decreasing, we differentiate it with respect to time delay parameter (τ).

$$\begin{aligned}
\frac{\partial R_q}{\partial\tau} &= \frac{\partial}{\partial\tau}\left[\frac{A(\eta\beta_1 + \mu_2\beta_2 e^{-\mu_1\tau})}{\mu_1\mu_2(d_1+\mu_1+\alpha+\delta)}\right]\\
&= \frac{\partial}{\partial\tau}\left[\frac{A\eta\beta_1}{\mu_1\mu_2(d_1+\mu_1+\alpha+\delta)}\right] + \frac{\partial}{\partial\tau}\left[\frac{A\mu_2\beta_2 e^{-\mu_1\tau}}{\mu_1\mu_2(d_1+\mu_1+\alpha+\delta)}\right]\\
&= -\left[\frac{A\beta_2 e^{-\mu_1\tau}}{(d_1+\mu_1+\alpha+\delta)}\right].
\end{aligned}$$

The negative sign of the derivative above, implies that, increasing time delay beyond the critical delay τ_{crit} with fixed values of all parameters involved will lead to the decrease in number of infectious individuals. Hence, this indicates that time delay has effect on the infectivity of cholera in the population. This result can be summarized as follows.

Proposition 6 *The increase (decrease) of time delay in the model* (2.1)–(2.5) *will reduce (increase) the infectivity of cholera whenever the delay is greater than the threshold value* τ_{crit}.

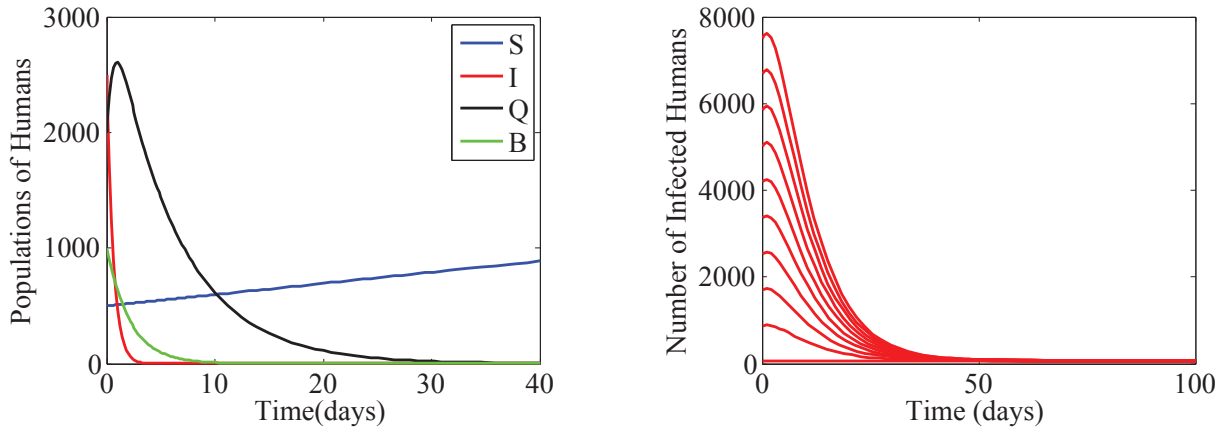
6 Numerical simulations

In this section, numerical simulations are conducted using MATLAB (R2014a) to illustrate the theoretical results proved in the previous sections, the effects of time delay and quarantine in the model. Parameter values from Table 2 are used as obtained from literature and some reasonable estimates.

Figure 2(a) illustrates that, starting with initial population close to disease free equilibrium E_1, the population will remain healthy as time progress (irrespective of the length of delay), this implies that E_1 is locally asymptotically stable as proved in Theorem 2. In Figure 2(b), it is shown that, irrespective of the initial population started with, the population will remain healthy as time progresses, the equilibrium E_1 is globally asymptotically stable as indicated in Theorem 5. In both Figures 2(a) and (b), the values of basic control reproductive number $\mathcal{R}_q$, are 0.8725 and 0.5412, (< 1) respectively for any value of τ.

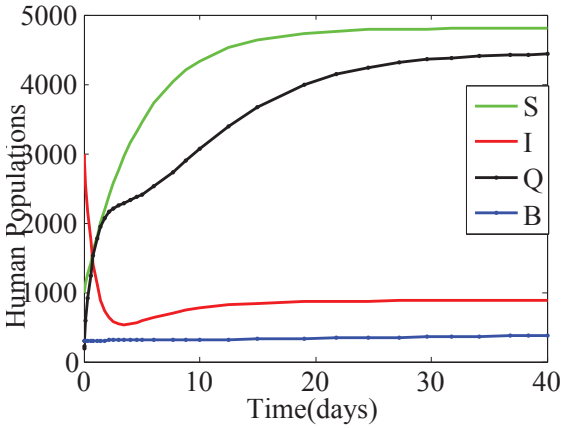
Parameter	Value	Unit	Reference
A	100	day^{-1}	Assumed
μ_1	0.000046	day^{-1}	$(WHO)(2016)$
μ_2	0.00046	day^{-1}	$(WHO)(2016)$
β_1	0.0000012	$individual^{-1}day^{-1}$	[28]
β_2	0.0000012	$individual^{-1}\ day^{-1}$	[28]
d_1	0.4	day^{-1}	[28]
d_2	0.2	day^{-1}	[28]
δ	0.0085	day^{-1}	[28]
α	0.0085	day^{-1}	[1]
ϵ	0.05	day^{-1}	[1]
η	0.036	$cellml^{-1}day^{-1}individual^{-1}$	Assumed
α_1	0.056	day^{-1}	Assumed
α_2	0.06	day^{-1}	Assumed

Table 2: Numerical values for Parameters of the model



(a) (b)

Figure 2: Numerical simulations showing (a) local and (b) global asymptotic stability of disease free equilibrium E_1 using parameter values in Table 2, except for $\tau = 3$, 10 respectively.



(a)

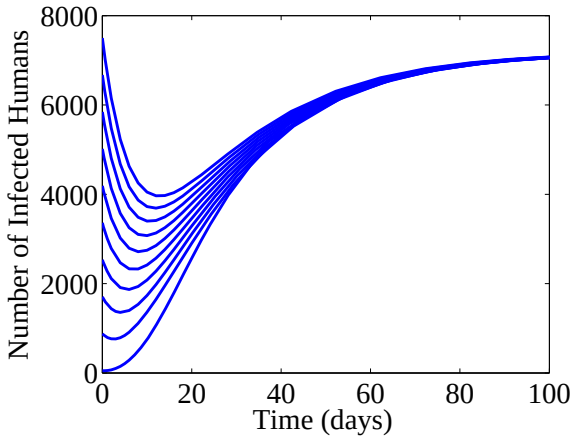
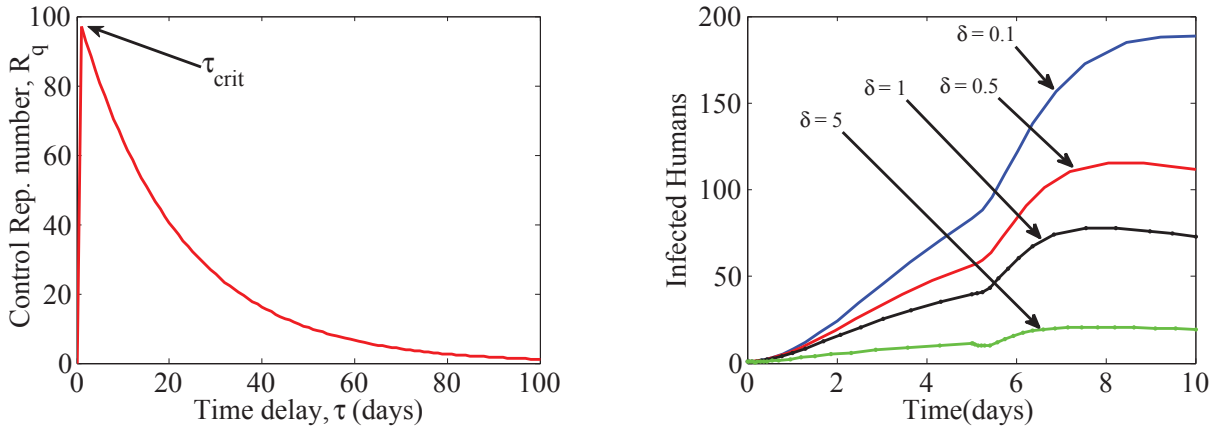


Figure 3: Numerical simulations showing (a) local and (b) global asymptotic stability of endemic equilibrium E_2 using parameter values from Table 2, except for $\beta_1 = 0.00012 = \beta_2$, $\tau = 3, 5$.



(a) (b)

Figure 4: Numerical simulations showing impacts of (a) time delay as a function of $\mathcal{R}_q$ and (b) quarantine on the model using parameter values from Table 2 with δ values as indicated.

The asymptotic stabilities of endemic equilibrium E_2 are shown in Figures 3 (a) and (b). It can be seen in Figure 3(a) that with initial data close to the endemic equilibrium and parameter values in Table 2, the subpopulations of human converge to E_2 asymptotically. Using $\beta_1 = 0.00012 = \beta_2$, $\tau = 3$, the value of $\mathcal{R}_q = 3.4175 > 1$. This illustrates the result in Theorem 4. Similarly, in Figure 3 (b), the global attractiveness of E_2 is shown as proved in Theorem 5. With parameter values from Table 2 except for $\beta_1 = 0.00012 = \beta_2$, $\tau = 10$, using different initial values, so that $\mathcal{R}_q = 2.8317 > 1$.

In Figure 4 (a), the control reproduction number $\mathcal{R}_q$ is plotted as a function of time delay to illustrates its impact on the infectivity of cholera in the population. It can be seen from the figure that initially $\mathcal{R}_q$ increases with increase in the time delay before τ_{crit} is reached. However, after passing τ_{crit}, as time delay τ increases, the value of $\mathcal{R}_q$ decreases drastically as stated in Preposition 6. This result tallies with the ones in [25, 31]. Similarly, the impact of quarantine is illustrated in Figure 4 (b). As shown in the figure, number of infected people decreases with increase in the quarantine rate δ.

7 Conclusion

A deterministic time delay Cholera model with quarantine as control strategy is formulated and analyzed. The main objectives are to investigate the effects of quarantine and time delay on the model with Holling type II incidence functional. The main results are summarized as follows:

(i) Basic properties of the model are proved and a Threshold quantity (Control Reproduction Number) $\mathcal{R}_q$ is obtained.

(ii) The model has a unique disease free equilibrium E_1, which is absolutely and globally asymptotically stable when $\mathcal{R}_q < 1$ for all delay $\tau \geq 0$. This result indicates that cholera

can be eradicated in the population whenever basic control reproduction number is brought and kept below unity.

(iii) When $\mathcal{R}_q > 1$, there exist an endemic equilibrium E_2, which is shown to be locally and globally asymptotically stable for all delay $\tau \geq 0$. This indicates that cholera will persists in the population.

(iv) Although time delay has no effect on the qualitative dynamics of the model, it however have effect on the disease burden whenever the time delay is above a threshold value (the critical delay value τ_{crit}).

(v) Quarantine intervention is also found to reduce the infectivity of cholera by increasing the quarantine rate. Indeed, cholera can be eradicated when at least 80% of infected individuals are quarantined in the given population.

(vi) Numerical simulations are used to illustrate our theoretical and numerical results.

In future work, we would like to investigate additional control strategies such as pulse vaccination for speedy eradication of cholera. Double patch model for the transmission of cholera with time delay would also be considered to investigate the effect of time delay in the presence of movement of individuals from one patch to another during epidemic.

Disclosure statement

The authors hereby declare that there is no conflicts of interest whatsoever.

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