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A MATHEMATICAL MODEL OF CHOLERA OUTBREAK WITH MULTIPLE TIME DELAYS

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Abstract

The past two decades witnessed Cholera outbreaks in several countries, such as Zimbabwe, Nigeria, DRC, South Sudan and Yemen. In this paper, we consider a system of four delay differential equations model of cholera outbreak with two time delays. These time delays represent incubation periods of vibrio cholerae transmitted indirectly from environment to human or directly from human to human. The model is analyzed and the stability properties have been concluded. Finally, numerical simulations are carried out to case of cholera outbreak in Zimbabwe. The results obtained show that neither of the delays leads to periodic oscillations.

1. Introduction

Mathematical modelling of cholera disease transmission dynamics is an important research topic for ecologists, biologists and applied mathematicians.

Differential equations have been effective tools to model epidemiological diseases. For example, the delayed and non-delayed HIV infection models [62, 6, 61], Hantavirus infection model [22], and COVID-19 models [63, 14, 5].

In the past few decades, attention has been drawn to cholera, and many mathematical models have been proposed to understand multiple transmission paths and control their propagation, for example, don't exclusive [11], [15], [47], [23], [39], [38], [55], [38], [4], [52], [13], [41], [3], [50], [57], [18], [56], [38], [29], [37], [58], [10], [44], [8], [44], [7], [21], [1], [45], [24], [30], [42], [19], [53], [40], [53], [27], [59].

The aforementioned epidemiological models above were formed using systems of ordinary differential equations. A challenge to consider including appropriate delay conditions on the model. Time delay plays an important role to reflect a realistic dynamic behaviors of models. There are few researchers in the literature that have proposed and analyzed cholera models involving time delays. Such examples include [9], [36], [51], [2], [60], [35], [32], [33], [46], [64], [28].

According to [58], Vibrio that is transmitted from human to human has a much higher rate of infection (up to 700 times higher) than the original Vibrio that is ingested from the environment.

Based on the model that was introduced in [38], this paper introduces a cholera model consisting of four delay differential equations with two time delays. These time delays represent two different kinds of incubation periods, referring to the incubation period of vibrio cholerae that is transmitted from environment to human and the incubation period of vibrio cholerae that is transmitted from human to human. The proposed model will be subject to qualitative analysis to investigate the impact of including time delays to the model.

This paper is organized as follows. In Section 2, we formulate the mathematical model. In Section 3, we study the qualitative behavior of the model via stability of the endemic equilibrium and Hopf bifurcation when time delays is considered as a bifurcation parameter. To verify our theoretical predictions, some numerical simulations are also included in Section 4. In Section 5 are the conclusions.

2. Model Formulation

The model has standard type SIR (susceptible-infected-recovered) compartments, with an additional compartment B that represents the concentration of the bacteria Vibro cholerae in the contaminated water.

We now extend model [38] by incorporating two time delays that represent incubation periods. Assuming that τ_1 denote the incubation period of the vibrio cholerae transmitted from environment to human, and τ_2 denote the incubation period of the vibrio cholerae transmitted from human to human. The new model takes the form:

$$\begin{aligned}
\frac{dS}{dt} &= \mu N - \eta \frac{SB}{k+B} - \alpha SI - \mu S, \\
\frac{dI}{dt} &= \eta \frac{S(t-\tau_1)B(t-\tau_1)}{k+B(t-\tau_1)} + \alpha S(t-\tau_2)I(t-\tau_2) - (\mu+\gamma)I, \\
\frac{dR}{dt} &= \gamma I - \mu R, \\
\frac{dB}{dt} &= \zeta I - \delta B.
\end{aligned} \tag{2.1}$$

In the equations above, the parameter k is the concentration of vibrios in contaminated water in the environment, η and α are rates of ingesting vibrios from the contaminated environment and through human-to-human interaction, respectively. The parameter μ represents the natural human birth/death rate, ζ, γ the recovery rate and δ the bacterial death rate. The total human population at time t, denoted by $N(t)$, is given by

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

For ecological reasons, we assume that the initial conditions for system (2.1) satisfies:

$$S_0(\theta) \geq 0,\ I_0(\theta) \geq 0,\ R_0(\theta) \geq 0,\ B_0(\theta) \geq 0, \quad \theta \in [-\tau, 0]. \tag{2.2}$$

The initial conditions for the model (2.1) is given by

$$S(\theta) = \phi_1(\theta),\ I(\theta) = \phi_2(\theta),\ R(\theta) = \phi_3(\theta),\ B(\theta) = \phi_4(\theta), \tag{2.3}$$

where $\phi = [\phi_1, \phi_2, \phi_3, \phi_4] \in C$, such that $\phi_i(\theta) = \phi_i(0) \geq 0$ for $\theta \in [-\tau, 0]$, $i = (1, 2, 3, 4)$ and C denotes the Banach space $C([-\tau, 0], R_+^4)$.

The solutions $(S(t), I(t), R(t), B(t))$ of system (2.1) with the initial conditions as stated above exist for all $t \geq 0$ and are unique [26].

From [38], we know that model (2.1) has a disease-free equilibrium (DFE) given by

$$E_0 = (N, 0, 0, 0), \tag{2.4}$$

and based on the next-generation matrix approach [54] the basic reproduction number $\mathcal{R}_0$ has the following expression:

$$\mathcal{R}_0 = \frac{N}{\delta k(\gamma + \mu)}[\zeta\eta + \delta k\alpha] = \mathcal{R}_e + \mathcal{R}_h.$$

Additionally, when $\mathcal{R}_0 > 1$, there is an endemic equilibrium given by

$$E_1 = (S^*, I^*, R^*, B^*), \tag{2.5}$$

where

$$S^* = N - \frac{(\gamma + \mu)I^*}{\mu},\ R^* = \frac{\gamma I^*}{\mu},\ B^* = \frac{\zeta I^*}{\delta},$$

and I^* is the positive root of the quadratic equation $aI^2 + bI + c = 0$ given by

$$I^* = \frac{-b + \sqrt{b^2 - 4ac}}{2a},$$

where

$$a = \alpha\zeta(\gamma + \mu),$$

$$b = -\alpha\mu\zeta N + (\gamma + \mu)(\zeta(\eta + \mu) + \alpha\delta k),$$

$$c = -\mu(\eta\zeta + \alpha\delta k)N + k\delta\mu(\gamma + \mu).$$

Such positive root exists if $b > 0$ and $c < 0$, that is $\alpha\mu\zeta N < (\gamma + \mu)(\zeta(\eta + \mu) + \alpha\delta k)$ and $(\eta\zeta + \alpha\delta k)N > k\delta(\gamma + \mu)$ or $b < 0$ and $c > 0$ (i.e., $\alpha\mu\zeta N > (\gamma + \mu)(\zeta(\eta + \mu) + \alpha\delta k)$ and $(\eta\zeta + \alpha\delta k)N < k\delta(\gamma + \mu)$).

3. Stability Analysis of Endemic Equilibrium E_1

In particular, when the time delays are zeros $\tau_1 = \tau_2 = 0$ the above system (2.1) is reduced to the original model that was developed in [38]. Based on their work, follow the results below directly:

Theorem 1. *The disease-free equilibrium* (*DFE*) *of the model* (2.1) $E_0 = (N, 0, 0, 0)$, *when* $R_0 < 1$ *with* $\tau_1 = \tau_2 = 0$ *is both locally and globally asymptotically stable.*

Theorem 2. *The endemic equilibrium of the model* (2.1) $E_1 = (S^*, I^*, R^*, B^*)$, *when* $\mathcal{R}_0 > 1$ *with* $\tau_1 = \tau_2 = 0$ *is locally and globally asymptotically stable.*

The details proof of Theorem 2 can be found on pages one and two of the Supporting information document of [38].

In this section, we discuss the qualitative analysis of model (2.1) behaviour at the endemic equilibrium point (EEP) under the impact of the time-delays. We also derive the stability conditions for the endemic equilibrium point (EEP) and explore the possibility of Hopf-bifurcation.

We first linearize model (2.1) around the endemic equilibrium point (EEP) and determine the characteristic equation of the Jacobian matrix. Let $S = S^* + s$, $I = I^* + i$, $R = R^* + r$, $B = B^* + b$, where s, i, r and b are small perturbations around the equilibrium E_1. We then obtain the linear delay differential equation:

$$\dot{Z} = AZ(t) + CZ(t - \tau_1) + DZ(t - \tau_2), \text{ where } Z(t) = (S, I, R, B)^T. \quad (3.1)$$

The linearization of the system (2.1) is

$$\begin{bmatrix} -\frac{\eta B^*}{k+B^*} - \alpha I^* - \mu & -\alpha S^* & 0 & -\frac{\eta S^*}{k+B^*} + \frac{\eta S^* B^*}{(k+B^*)^2} \\ \frac{e^{-\tau_1\lambda}\eta B^*}{k+B^*} + e^{-\tau_2\lambda}\alpha I^* & -\mu - \gamma + e^{-\tau_2\lambda}\alpha S^* & 0 & e^{-\tau_1\lambda}\left(\frac{\eta S^*}{k+B^*} - \frac{\eta S^* B^*}{(k+B^*)^2}\right) \\ 0 & \gamma & -\mu & 0 \\ 0 & \zeta & 0 & -\delta \end{bmatrix}.$$

So, the characteristic equation of the linearized system of delayed differential (2.1) is given by

$$P_1(\lambda) + P_2(\lambda)e^{-(\lambda\tau_1)} + P_3(\lambda)e^{-(\lambda\tau_2)} = 0, \quad (3.2)$$

where $P_1(\lambda)$, $P_2(\lambda)$ and $P_3(\lambda)$ are polynomials, each of degree not exceeding 4.

In case of positive delays $\tau_1 \neq 0$ and $\tau_2 \neq 0$, the characteristic equation for the linearized model (2.1) given by (3.2) at the endemic equilibrium E_1 is given by:

$$\lambda^4 + U_1\lambda^3 + U_2\lambda^2 + U_3\lambda + U_4 + (Q_1\lambda^2 + Q_2\lambda + Q_3)e^{-\tau_1\lambda} + (V_1\lambda^3 + V_2\lambda^2 + V_3\lambda + V_4)e^{-\tau_2\lambda} = 0, \quad (3.3)$$

where

$$P_1(\lambda) = \lambda^4 + U_1\lambda^3 + U_2\lambda^2 + U_3\lambda + U_4,$$

$$P_2(\lambda) = Q_1\lambda^2 + Q_2\lambda + Q_3,$$

$$P_3(\lambda) = V_1\lambda^3 + V_2\lambda^2 + V_3\lambda + V_4.$$

In the above expressions of $P_1(\lambda)$, $P_2(\lambda)$ and $P_3(\lambda)$, $U_i's (i = 1, 2, 3, 4)$, $Q_i's (i = 1, 2, 3)$, $V_i's (i = 1, 2, 3, 4)$ are given as follows:

$$U_1 = \frac{I\alpha k + B^*\delta + B^*\eta + B^*\gamma + 3B^*\mu + B^*I^*\alpha + \delta k + \gamma k + 3k\mu}{k + B^*},$$

$$U_2 = \frac{B^{*2}I\delta\alpha + B^{*2}I\alpha\gamma + 2B^{*2}I^*\alpha\mu + 2B^*I^*\delta\alpha k + 2B^*I^*\alpha\gamma k + 4B^*I^*\alpha k\mu + I^*\delta\alpha k^2 + I^*\alpha\gamma k^2}{(k + B^*)^2}$$

$$+ \frac{2I^*\alpha k^2\mu B^{*2}\delta\eta + B^{*2}\delta\gamma + 3\,B^{*2}\delta\mu + B^{*2}\eta\gamma + 2\,B^{*2}\eta\mu + 2\,B^{*2}\gamma\mu + 3\,B^{*2}\mu^2 + B^*\delta\eta k + 2\,B^*\delta\gamma k}{(k + B^*)^2}$$

$$+ \frac{6B^*\delta k\mu + B^*\eta\gamma k + 2\,B^*\eta k\mu + 4\,B^*\gamma k\mu + 6\,B^*k\mu^2 + \delta\gamma k^2 + 3\delta k^2\mu + 2\gamma k^2\mu + 3k^2\mu^2}{(k + B^*)^2},$$

$$U_3 = \frac{\begin{array}{c} B^{*3}I^{*}\delta\alpha\gamma + 2\,B^{*3}I^{*}\delta\alpha\mu + B^{*3}I^{*}\alpha\gamma\mu + B^{*3}I^{*}\alpha\mu^2 \\ + 3\,B^{*2}I\delta\alpha\gamma k + 6B^{*2}I^{*}\delta\alpha k\mu + 3B^{*2}I^{*}\alpha\gamma k\mu \end{array}}{(k+B^{*})^3}$$

$$+ \frac{\begin{array}{c} 3\,B^{*2}I\alpha k\mu^2 + 3\,B^{*}I^{*}\delta\alpha\gamma k^{*2} + 6\,B^{*}I^{*}\delta\alpha k^2\mu + 3\,B^{*}I^{*}\alpha\gamma k^2\mu \\ + 3\,B^{*}I^{*}\alpha k^2\mu^2 + I^{*}\delta\alpha\gamma k^3 + 2I^{*}\delta\alpha k^3\mu \end{array}}{(k+B^{*})^3}$$

$$+ \frac{\begin{array}{c} I\alpha\gamma k^3\mu + I\alpha k^3\mu^2 + B^{*3}\delta\eta\gamma + 2\,B^{*3}\delta\eta\mu + 2\,B^{*3}\delta\gamma\mu \\ + 3\,B^{*3}\delta\mu^2 + B^{*3}\eta\gamma\mu + B^{*3}\eta\mu^2 + B^{*3}\gamma\mu^2 \end{array}}{(k+B^{*})^3}$$

$$+ \frac{\begin{array}{c} B^{*3}\mu^3 + 2\,B^{*2}\delta\eta\gamma k + 4\,B^{*2}\delta\eta k\mu + 6B^{*2}\delta\gamma k\mu + 9\,B^{*2}\delta k\mu^2 \\ + 2\,B^{*2}\eta\gamma k\mu + 2\,B^{*2}\eta k\mu^2 + 3\,B^{*2}\gamma k\mu^2 \end{array}}{(k+B^{*})^3}$$

$$+ \frac{\begin{array}{c} 3\,B^{*2}k\mu^3 + B^{*}\delta\eta\gamma k^2 + 2\,B^{*}\delta\eta k^2\mu + 6\,B^{*}\delta\gamma k^2\mu + 9B^{*}\delta k^2\mu^2 \\ + B^{*}\eta\gamma k^2\mu + B^{*}\eta k^2\mu^2 + 3\,B^{*}\gamma k^2\mu^2 \end{array}}{(k+B^{*})^3}$$

$$+ \frac{3B^{*}k^2\mu^3 + 2\delta\gamma k^3\mu + 3\delta k^3\mu^2 + \gamma k^3\mu^2 + k^3\mu^3}{(k+B^{*})^3},$$

$$U_4 = \frac{\begin{array}{c} \mu(B^{*3}I^{*}\delta\alpha\gamma + B^{3}I\delta\alpha\mu + 3\,B^{*2}I\delta\alpha\gamma k + 3B^{*2}I\delta\alpha k\mu \\ + 3\,B^{*}I^{*}\delta\alpha\gamma k^2 + 3\,B^{*}I^{*}\delta\alpha k^2\mu + I^{*}\delta\alpha\gamma k^3) \end{array}}{(k+B^{*})^3}$$

$$+ \frac{\begin{array}{c} \mu(I^{*}\delta\alpha k^3\mu + B^{*3}\delta\eta\gamma + B^{*3}\delta\eta\mu + B^{*3}\delta\gamma\mu + B^{*3}\delta\mu^2 \\ + 2\,B^{*2}\delta\eta\gamma k + 2B^{*2}\delta\eta k\mu + 3B^{*2}\delta\gamma k\mu) \end{array}}{(k+B^{*})^3}$$

$$+ \frac{\begin{array}{c} \mu(3B^{*2}\delta k\mu^2 + B^{*}\delta\eta\gamma k^2 + B^{*}\delta\eta k^2\mu + 3\,B^{*}\delta\gamma k^2\mu \\ + 3\,B^{*}\delta k^2\mu^2 + \delta\gamma k^3\mu + \delta k^3\mu^2) \end{array}}{(k+B^{*})^3},$$

$$Q_1 = \frac{B^{*2}S^*\alpha\eta + B^*S^*\alpha\eta k + S^*\zeta\eta k}{(k + B^*)^2},$$

$$Q_2 = \frac{\begin{array}{c} B^{*3}S^*\delta\alpha\eta + B^{*3}S^*\alpha\eta\mu + 2\,B^{*2}S^*\delta\alpha\eta k + 2\,B^{*2}S^*\alpha\eta k\mu \\ + B^*I^*,\ S^*\zeta\alpha\eta k + B^*S^*\delta\alpha\eta k^2 \end{array}}{(k + B^*)^3}$$

$$+ \frac{\begin{array}{c} B^*S^*\alpha\eta k^2\mu + I^*S^*\zeta\alpha\eta k^2 + 2\,B^*S^*\zeta\eta^2 k + 2\,B^*S^*\zeta\eta k\mu \\ + 2S^*\zeta\eta k^2\mu \end{array}}{(k + B^*)^3},$$

$$Q_3 = \frac{\begin{array}{c} \mu(B^3S^*\delta\alpha\eta + 2\,B^{*2}S^*\delta\alpha\eta k + B^*I^*S^*\zeta\alpha\eta k + B^*S^*\delta\alpha\eta k^2 \\ + I^*S^*\zeta\alpha\eta k^2 + 2\,B^*S^*\zeta\eta^2 k) \end{array}}{(k + B^*)^3}$$

$$+ \frac{\mu(B^*S^*\zeta\eta k\mu + S^*\zeta\eta k^2\mu)}{(k + B^*)^3},$$

and

$$V_1 = \frac{-B^*S^*\alpha - S^*\alpha k}{k + B^*},$$

$$V_2 = \frac{\begin{array}{c} -B^{*2}S^*\delta\alpha - B^{*2}S^*\alpha\eta - 2\,B^{*2}S^*\alpha\mu - 2\,B^*S^*\delta\alpha k \\ - B^*S^*\alpha\eta k - 4\,B^*S^*\alpha k\mu - S^*\delta\alpha k^2 - 2\,S^*\alpha k^2\mu \end{array}}{(k + B^*)^2},$$

$$V_3 = \frac{\begin{array}{l} - B^{*3}S^*\delta\alpha\eta - 2\,B^{*3}S\delta\alpha\mu - B^{*3}S^*\alpha\eta\mu - B^{*3}S^*\alpha\mu^2 \\ - 2\,B^{*2}S\delta\alpha\eta k - 6\,B^{*2}S\delta\alpha k\mu \end{array}}{(k + B^*)^3},$$

$$+ \frac{\begin{array}{l} - 2\,B^{*2}S^*\alpha\eta k\mu - 3\,B^{*2}S^*\alpha k\mu^2 + B^*I^*S^*\zeta\alpha\eta k \\ - B^*S^*\delta\alpha\eta k^2 - 6\,B^*S^*\delta\alpha k^2\mu - B^*S^*\alpha\eta k^2\mu \end{array}}{(k + B^*)^3},$$

$$+ \frac{- 3B^*S^*\alpha k^2\mu^2 + I^*S^*\zeta\alpha\eta k^2 - 2\,S^*\delta\alpha k^3\mu - S^*\alpha k^3\mu^2}{(k + B^*)^3},$$

$$V_4 = \frac{\begin{array}{l} \mu(- B^{*3}S^*\delta\alpha\eta - B^{*3}S^*\delta\alpha\mu - 2\,B^{*2}S^*\delta\alpha\eta k \\ - 3\,B^{*2}S^*\delta\alpha k\mu + B^*I^*S^*\zeta\alpha\eta k - B^*S^*\delta\alpha\eta k^2) \end{array}}{(k + B^*)^3},$$

$$+ \frac{\mu(- 3B^*S^*\delta\alpha k^2\mu + I^*S^*\zeta\alpha\eta k^2 - S^*\delta\alpha k^3\mu)}{(k + B^*)^3}.$$

Theorem 3. *Necessary and sufficient conditions for the endemic equilibrium* E_1 *to be asymptotically stable for all delays* $\tau_1 \geq 0$ *and* $\tau_2 \geq 0$ *are as follows*:

- *The real parts of all the roots of characteristic equation* $\phi(\lambda, \tau_j) = 0$ *are negative.*

- *For all* ω_j *and* $\tau_j \geq 0$ $\phi(i\omega_j, \tau_j) \neq 0$, *where* $j = 1, 2$.

Now we analyze the corresponding characteristic equation that correspond the endemic equilibrium E_1. Analysis of the general case is very complicated when choosing two delays as parameters of systems (2.1). Hence, we study two cases:

Case 1 $(\tau_1 > 0, \tau_2 = 0)$**.** In this case, we regard $\tau_1 > 0$ as the bifurcation parameter so the characteristic polynomial (3.3) is reduced to

$$\lambda^4 + (U_1 + V_1)\lambda^3 + (U_2 + V_2)\lambda^2 + (U_3 + V_3)\lambda + (U_4 + V_4) + (Q_1\lambda^2 + Q_2\lambda + Q_3)e^{-\tau_1\lambda} = 0. \quad (3.4)$$

We investigate existence of Hopf-bifurcation, following the methods in [25, 20, 49, 34, 36, 33]. We want to determined if the solution curve of characteristic equation (3.4) crosses the imaginary axis so we suppose $\lambda = i\omega$ when $\omega > 0$ is root of Equation (3.4) if and only if

$$((i\omega)^4 + D_1(i\omega)^3 + D_2(i\omega)^2 + D_3(i\omega) + D_4) + (Q_1(i\omega)^2 + Q_2(i\omega) + Q_3)e^{-\tau_1(i\omega)} = 0, \quad (3.5)$$

and separating the real and imaginary parts we obtain the following transcendental equations:

$$D_3\omega - D_1\omega^3 = -Q_2\omega\cos(\omega\tau_1) + (Q_3 - Q_1\omega^2)(\sin(\omega\tau_1)), \quad (3.6)$$

$$\omega^4 - D_2\omega^2 + D_4 = -(Q_3 - Q_1\omega^2)\cos(\omega\tau_1) - Q_2\omega(\sin(\omega\tau_1)). \quad (3.7)$$

Squaring and adding Equations (3.6) and (3.7) and letting $\omega^2 = \chi$, we get the following equation:

$$h(\chi) = \chi^4 + Z_1\chi^3 + Z_2\chi^2 + Z_3\chi + Z_4 = 0, \quad (3.8)$$

where

$$Z_1 = D_1{}^2 - 2D_2,$$

$$Z_2 = -2D_1D_3 + D_2{}^2 + 2D_4 - Q_1^2,$$

$$Z_3 = D_3{}^2 - 2D_2D_4 + 2Q_1Q_3 - Q_2^2,$$

$$Z_4 = D_4{}^2 - Q^2{}_3.$$

Lemma 1. (1) *If the coefficients in* $h(\chi)$ *satisfy the conditions of the Routh-Hurwitz criterion* ([43], [17]), *then Equation* (3.3) *will not have any positive real root of* ω_2, *and the positive equilibrium* E_1 *is locally asymptotically stable for all delay* $\tau_1 > 0$.

(2) *If the coefficients in* $h(\chi)$ *do not satisfy the Routh-Hurwitz criterion in this case a simple assumption for the existence of a positive root of Equation* (3.8) *is* $Z_4 < 0$, *we have* $h(0) = 0$ *and* $\lim_{\chi\to\infty h(\chi)} = \infty$ *so Equation* (3.4) *has a pair of pure imaginary roots* $\pm\omega i$.

Remark 1. We can easily show that for $\tau_1 > 0$, the local stability condition, of the Routh-Hurwitz criterion ([43], [17]) $Z_4 > 0$, $\frac{Z_1Z_2 - Z_3}{Z_1} > 0$, $\frac{Z_3(Z_1Z_2 - Z_3)}{Z_1Z_2 - Z_3} - \frac{Z_1^2Z_4}{Z_1Z_2 - Z_3} > 0$ is automatically satisfied.

Proposition 3.1. *If the Routh-Hurwitz criterion are satisfied, then endemic equilibrium* E_1 *is asymptotically stable for all delay* $\tau_1 > 0$.

Case 2 ($\tau_2 > 0$, $\tau_1 = 0$)**.** In this case, we regard $\tau_1 = 0$ and $\tau_2 > 0$ as the bifurcation parameter so the characteristic polynomial (3.3) is reduced to

$$\lambda^4 + U_1\lambda^3 + (U_2 + Q_1)\lambda^2 + (U_3 + Q_2)\lambda + (U_4 + Q_3) + (V_1\lambda^3 + V_2\lambda^2 + V_3\lambda + V_4)e^{-\tau_2\lambda} = 0. \quad (3.9)$$

Again to show Hopf-bifurcation, we must have a pair of purely imaginary roots of characteristic equation (3.9) [25, 20, 49, 34, 36, 33].

We suppose $\lambda = i\omega_2$ when $\omega_2 > 0$ is root of Equation (3.9) if and only if

$$(i\omega_2)^4 + U_1(i\omega_2)^3 + L_2(i\omega_2)^2 + L_3(i\omega_2) + L_4 + (V_1(i\omega_2)^3 + V_2(i\omega_2)^2 + V_3(i\omega_2) + V_4)e^{-\tau_2\lambda} = 0, \quad (3.10)$$

and separating real and imaginary parts, we get the following transcendental equations:

$$-U_1\omega_2^3 + L_3\omega_2 = -(V_3\omega_2 - V_1\omega_2^3)(\cos(\omega_2\tau_2)) - (V_2\omega_2^2 - V_4)(\sin(\omega_2\tau_2)), \tag{3.11}$$

$$\omega_3^4 - L_2\omega_2^2 + L_4 = (V_2\omega_2^2 - V_4)(\cos(\omega_2\tau_2)) - (V_3\omega_2 - V_1\omega^3)(\sin(\omega_2\tau_2)). \tag{3.12}$$

Squaring and adding Equations (3.11) and (3.12) and letting $\omega_3^2 = \chi_1$, we obtained the following equation:

$$h_1(\chi_1) = \chi_1^4 + Z_6\chi_1^3 + Z_7\chi_1^2 + Z_8\chi_1 + Z_9 = 0, \tag{3.13}$$

where

$$Z_6 = U_1{}^2 - V_1{}^2 - 2L_2,$$

$$Z_7 = -2U_1L_3 + L_2{}^2 + 2V_1V_3 - V_2{}^2 + 2L_4,$$

$$Z_8 = -2L_2L_4 + L_3{}^2 + 2V_2V_4 - V_3{}^2,$$

$$Z_9 = L_4{}^2 - V_4{}^2.$$

Lemma 2.

• *If the coefficients in* $h(\chi)$ *satisfy the conditions of the Routh-Hurwitz criterion* ([43], [17]), *then Equation* (3.13) *will not have any positive real root of* ω_2, *and the positive equilibrium* E_1 *is locally asymptotically stable for all delay* $\tau_2 > 0$.

• *If the coefficients in* $h(\chi)$ *do not satisfy the Routh-Hurwitz criterion in this case a simple assumption for the existence of a positive root of Equation* (3.8) *is* $Z_9 < 0$, *we have* $h(0) = 0$ *and* $\lim_{\chi\to\infty} h(\chi) = \infty$ *so Equation* (3.4) *has a pair of pure imaginary roots* $\pm\omega_2 i$.

Remark 2. We can also easily show that for $\tau_2 > 0$, the local stability condition, of the Routh-Hurwitz criterion ([43], [17]) $Z_9 > 0, \frac{Z_6 Z_2 - Z_3}{Z_6} > 0, \frac{Z_3(Z_6 Z_7 - Z_8)}{Z_6 Z_7 - Z_8} - \frac{Z_6^2 Z_4}{Z_6 Z_7 - Z_8} > 0$ is automatically satisfied.

Proposition 3.2. *If the Routh-Hurwitz criterion satisfied, then endemic equilibrium* E_1 *is asymptotically stable for all delay* $\tau_2 > 0$.

4. Numerical Simulations

The 2008-2009 cholera epidemic in Zimbabwe resulted in 98,585 reported cases and caused more than 4,000 deaths [16].

The cholera epidemic began in August 2008, not only ravaging all 10 districts of Zimbabwe, but also rapidly spreading to Botswana, Mozambique, South Africa and Zambia. The primary cause of the outbreak was the collapse of Zimbabwe's public health system. By the end of November 2008, three of Zimbabwe's four main hospitals were closed, and many places did not have essential medicines, medicine and water supplies long enough during the outbreak. On December 4, 2008, the Zimbabwean government declared a national emergency [32].

In this section, we present a numerical simulation of the model system (2.1) to confirm our analytical results, using MATLAB. The parameters values are according to Table 1. These parameter values have been taken from [38].

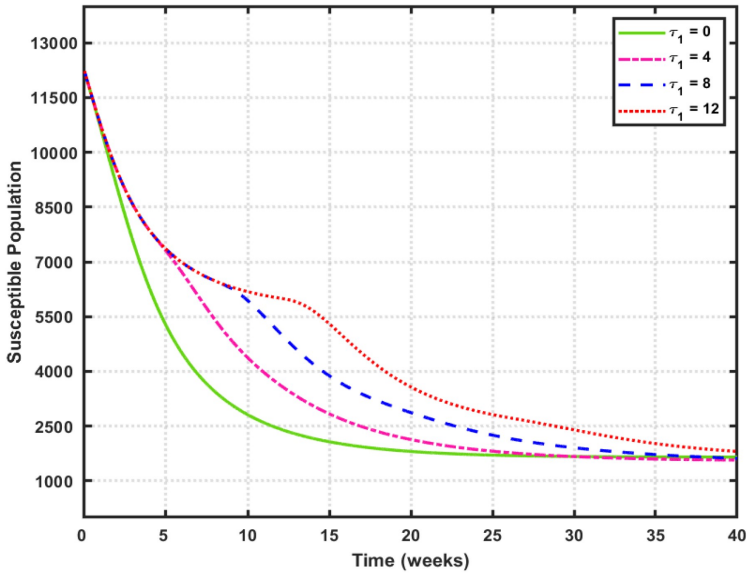
Table 1. Parameter values for model (2.1)

Parameter	Definition	Value	Unit	Source
N	Total population size	12347	person	[38]
μ	Natural death rate	0.000442	per week	[38]
k	Half saturation rate	10^6	cells per mL	[23]
η	Indirect transmission rate	0.525	per person per week	[56]
α	Direct transmission rate	1.1×10^{-4}	per person per week	[56]
γ	Recovery rate	1.40	person per week	[48], [31], [23], [55]
ζ	Shedding rate	70	cells ml week per person	[39, 15]
δ	Bacterial net death rate	0.23		[23], [12]

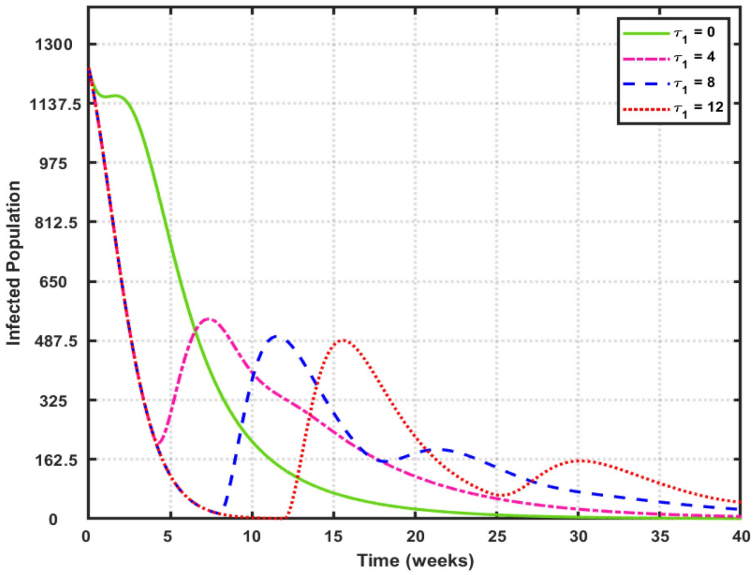
The basic reproduction number is $\mathcal{R}_0 = 2.2702 > 1$. The positive equilibrium for this data is $E_1 = (5440.7852, 2.1801, 6904.0347, 654.0389)$.

We considered four values {0, 4, 8, 12} for each of the two time delays τ_1 and τ_2. The values $\tau_1 = 0$ and $\tau_2 = 0$ correspond the solutions of ODEs.

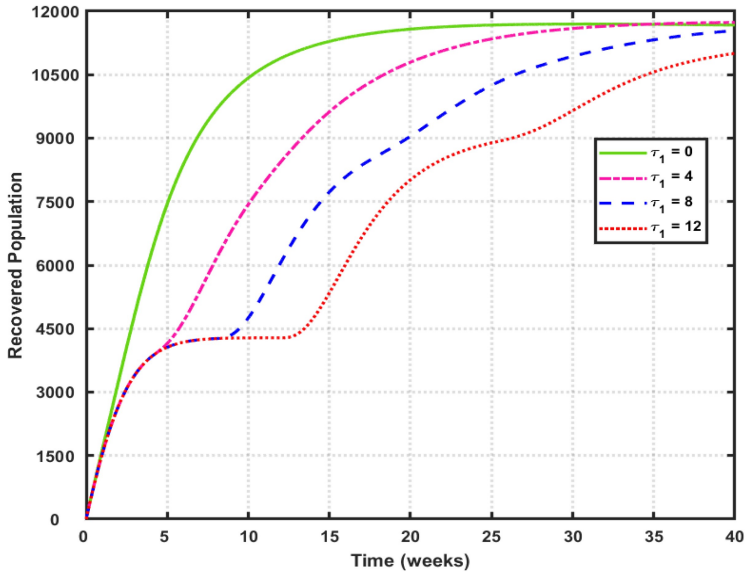
For $\tau_1 > 0$ and $\tau_2 = 0$, Equation (3.8) does not have positive roots. It may be noted that two roots are zeros and two roots are complex. Hence, the endemic equilibrium E_1 is locally asymptotically stable in the $\tau_1 > 0$ and $\tau_2 = 0$. From Figure 1, we see that for $\tau_1 \in \{0, 4, 8, 12\}$, the model variables approach the equilibrium points E_1, which confirms the stability of endemic equilibrium E_1 for $\tau_1 > 0$.



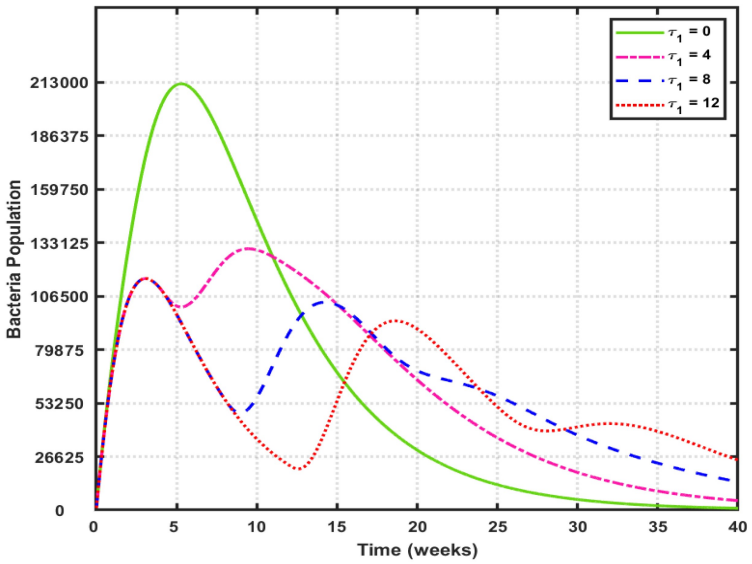
(a) Susceptible population.



(b) Infected population.



(c) Recovered population.



(d) Bacteria concentration.

Figure 1. Solution of cholera model for $\tau_1 \in \{1, 4, 8, 12\}$.

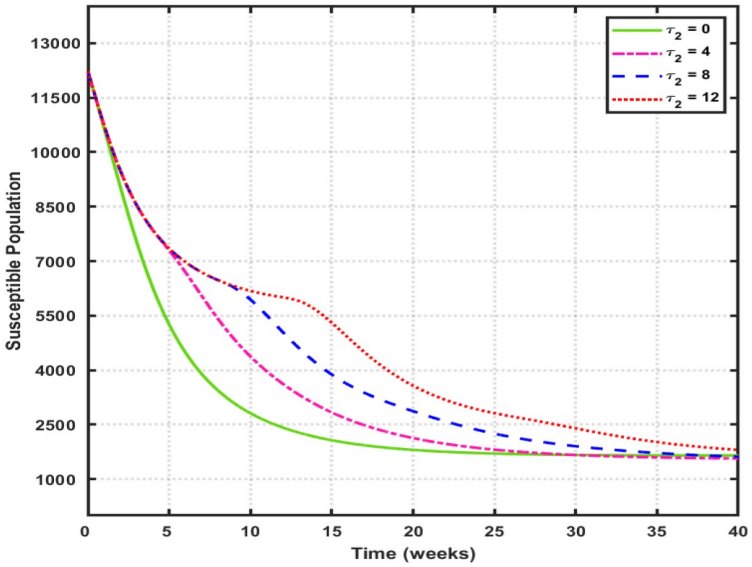
In the case $\tau_2 > 0$ and $\tau_1 = 0$, Equation (3.13) has two complex roots and two roots are zeros. Hence, the endemic equilibrium E_1 is locally asymptotically stable in this case.

Figure 2 shows the dynamic of the delayed cholera model for $\tau_1 = 0$ and $\tau_2 \in \{0, 4, 8, 12\}$. In all cases, the dynamics approaches the endemic equilibrium point E_1.

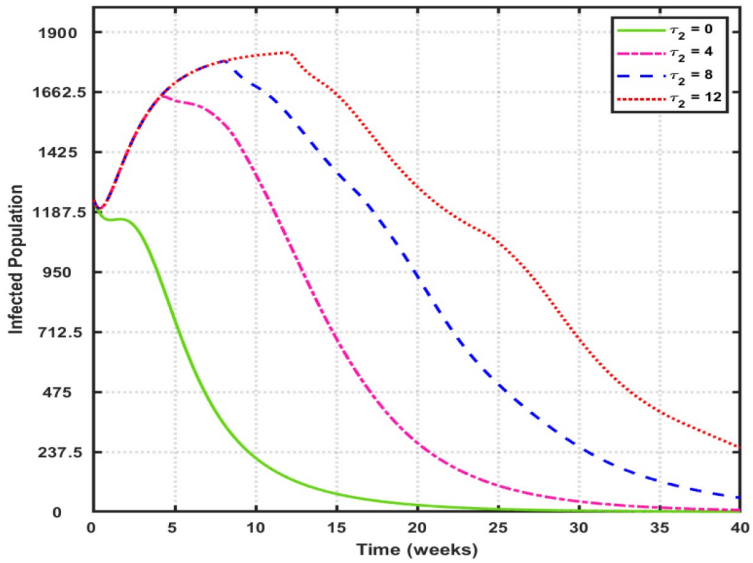
5. Conclusions and Discussions

This paper is devoted to the formulation and analysis of a time delayed mathematical model for cholera outbreak inspired by the work in Mukandavire et al. [38]. It represents a coupling between multiple transmission pathways of cholera and multiple time delays.

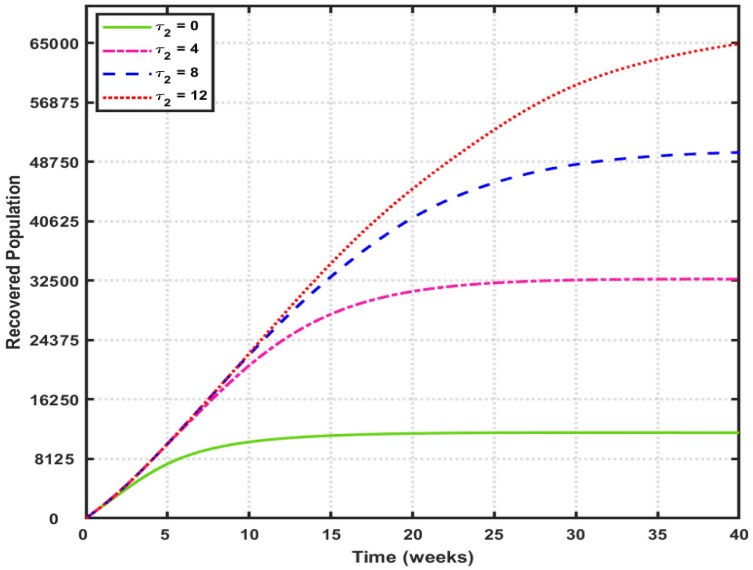
We stated the conditions under which endemic equilibrium point E_1 can exist. Then, we analyzed the corresponding characteristic equations of the linearized model to investigate the local stability of E_1 when $\tau_i > 0$, $i = 1, 2$.



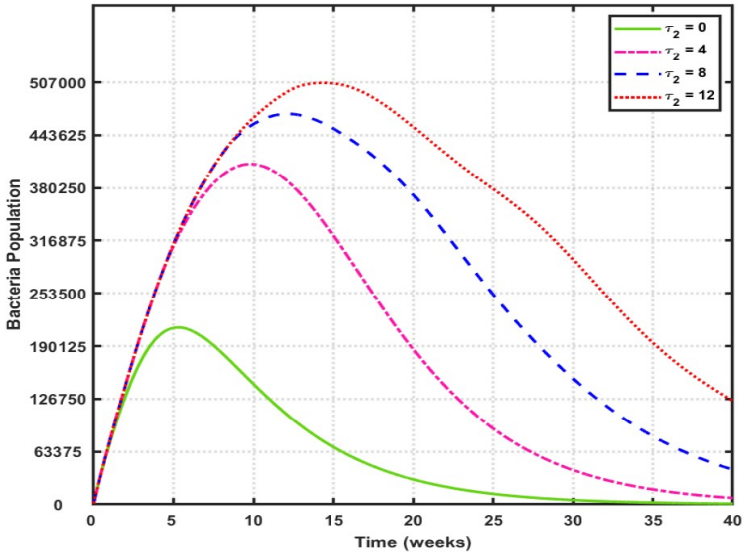
(a) Susceptible population.



(b) Infected population.



(c) Recovered population.



(d) Bacteria concentration.

Figure 2. Solution of cholera model for $\tau_2 \in \{1, 4, 8, 12\}$.

We investigated the local stability of the endemic equilibrium point under two cases for time delays. In each of the two cases, we stated the conditions under which the endemic equilibrium is locally stable.

It is found that in the case of cholera outbreak in Zimbabwe, changing either of the two delays does not cause a Hopf bifurcation, and hence the model under investigation does not have periodic solutions.

Finally, we considered the case of cholera outbreak in Zimbabwe for the numerical illustration. It is found that changing either of the two delays does not cause a Hopf bifurcation, and hence the model under investigation does not have periodic solutions.

References

[1] Falaye Adeyinka Adesuyi, E. E. E. Akarawak, A. T. Cole, Evans O. Patience, Oluyori David Adeyemi, Falaye Roseline Adunola and Adama Ndako Victor, A mathematical model for capturing cholera spread and containment options, International Journal of Mathematical Sciences and Computing 4(1) (2018), 15-40.

DOI: https://doi.org/10.5815/ijmsc.2018.01.02

[2] Manju Agarwal and Vinay Verma, Modeling and analysis of the spread of an infectious disease cholera with environmental fluctuations, Applications and Applied Mathematics: An International Journal 7(1) (2012); Article 27.

[3] Mo'tassem Al-Arydah, Abubakar Mwasa, Jean M. Tchuenche and Robert J. Smith, Modeling cholera disease with education and chlorination, Journal of Biological Systems 21(4) (2013); Article 1340007.

DOI: https://doi.org/10.1142/S021833901340007X

[4] Alen Alexanderian, Matthias K. Gobbert, K. Renee Fister, Holly Gaff, Suzanne Lenhart and Elsa Schaefer, An age-structured model for the spread of epidemic cholera: Analysis and simulation, Nonlinear Analysis: Real World Applications 12(6) (2011), 3483-3498.

DOI: https://doi.org/10.1016/j.nonrwa.2011.06.009

[5] Mohammad Aslefallah, Şuayip Yüzbaşi and Saeid Abbasbandy, A numerical investigation based on exponential collocation method for nonlinear SITR model of COVID-19, Computer Modeling in Engineering & Sciences 136(2) (2023), 1687-1706.

DOI: https://doi.org/10.32604/cmes.2023.025647

[6] Attaullah, Rashid Jan and Şuayip Yüzbaşi, Dynamical behaviour of HIV infection with the influence of variable source term through Galerkin method, Chaos, Solitons & Fractals 152 (2021); Article 111429.

DOI: https://doi.org/10.1016/j.chaos.2021.111429

[7] A. A. Ayoade, M. O. Ibrahim, O. J. Peter and F. A. Oguntolu, A mathematical model on cholera dynamics with prevention and control, Covenant Journal of Physical and Life Sciences 6(1) (2018), 46-54.

[8] S Balamuralitharan and M Radha, Stability analysis of cholera-carrier dependent infectious disease, International Journal of Pure and Applied Mathematics 113(11) (2017), 234-242.

[9] Yibeltal Negussie Bayleyegn, Mathematical Analysis of a Model of Cholera Transmission Dynamics, African Institute for Mathematical Sciences (AIMS), South Africa, 2009.

[10] Musundi O. Beryl, Lawi O. George and Nyamwala O. Fredrick, Mathematical analysis of a cholera transmission model incorporating media coverage, International Journal of Pure and Applied Mathematics 111(2) (2016), 219-231.

DOI: https://doi.org/10.12732/ijpam.v111i2.8

[11] V. Capasso and S. L. Paveri-Fontana, A mathematical model for the 1973 cholera epidemic in the European Mediterranean region, Revue d'Épidémiologie et de Santé Publique 27(2) (1979), 121-132.

[12] Richard Carr and Martin Strauss, Excreta-related infections and the role of sanitation in the control of transmission, Water Quality: Guidelines, Standards and Health (2001), 89-113.

[13] Dennis L. Chao, Ira M. Longini and J. Glenn Morris, Modeling Cholera Outbreaks, In Cholera Outbreaks, Pages 195-209, Springer, 2013.

DOI: https://doi.org/10.1007/82_2013_307

[14] Amar Nath Chatterjee and Bashir Ahmad, A fractional-order differential equation model of COVID-19 infection of epithelial cells, Chaos, Solitons & Fractals 147 (2021); Article 110952.

DOI: https://doi.org/10.1016/j.chaos.2021.110952

[15] Cláudia Torres Codeço, Endemic and epidemic dynamics of cholera: The role of the aquatic reservoir, BMC Infectious Diseases 1(1) (2001); Article 1.

DOI: https://doi.org/10.1186/1471-2334-1-1

[16] C. Nicholas Cuneo, Richard Sollom and Chris Beyrer, The cholera epidemic in Zimbabwe, 2008-2009: A review and critique of the evidence, Health and Human Rights 19(2) (2017), 249-264.

[17] Edmund X. DeJesus and Charles Kaufman, Routh-Hurwitz criterion in the examination of eigenvalues of a system of nonlinear ordinary differential equations, Physical Review A 35(12) (1987); Article 5288.

DOI: https://doi.org/10.1103/PhysRevA.35.5288

[18] Stephen Edward and Nkuba Nyerere, A mathematical model for the dynamics of cholera with control measures, Applied and Computational Mathematics 4(2) (2015), 53-63.

DOI: https://doi.org/10.11648/j.acm.20150402.14

[19] Subchan, Irma Fitria and Ahmad M. Syafi'i, An Epidemic Cholera Model with Control Treatment and Intervention, In Journal of Physics: Conference Series, Volume 1218, Page 012046. IOP Publishing, 2019.

DOI: https://doi.org/10.1088/1742-6596/1218/1/012046

[20] Jonathan Erwin Forde, Delay Differential Equation Models in Mathematical Biology, University of Michigan, 2005.

[21] Isaac Chun-Hai Fung, Cholera transmission dynamic models for public health practitioners, Emerging Themes in Epidemiology 11(1) (2014); Article 1.

DOI: https://doi.org/10.1186/1742-7622-11-1

[22] Ahmet Gökdoğan, Mehmet Merdan and Ahmet Yildirim, A multistage differential transformation method for approximate solution of Hantavirus infection model, Communications in Nonlinear Science and Numerical Simulation 17(1) (2012), 1-8.

DOI: https://doi.org/10.1016/j.cnsns.2011.05.023

[23] David M. Hartley, J. Glenn Morris Jr. and David L. Smith, Hyperinfectivity: A critical element in the ability of V. Cholerae to cause epidemics?, PLoS Medicine 3(1) (2006), e7.

DOI: https://doi.org/10.1371/journal.pmed.0030007

[24] Daihai He, Xueying Wang, Daozhou Gao and Jin Wang, Modeling the 2016–2017 Yemen cholera outbreak with the impact of limited medical resources, Journal of Theoretical Biology 451 (2018), 80-85.

DOI: https://doi.org/10.1016/j.jtbi.2018.04.041

[25] Xiaowu Jiang, Xueyong Zhou, Xiangyun Shi and Xinyu Song, Analysis of stability and Hopf bifurcation for a delay-differential equation model of HIV infection of $CD4^+$ T-cells, Chaos, Solitons & Fractals 38(2) (2008), 447-460.

DOI: https://doi.org/10.1016/j.chaos.2006.11.026

[26] Yang Kuang, Delay Differential Equations: with Applications in Population Dynamics, Academic Press, 1993.

[27] Pushpendra Kumar and Vedat Suat Erturk, Dynamics of cholera disease by using two recent fractional numerical methods, Mathematical Modelling and Numerical Simulation with Applications 1(2) (2021), 102-111.

DOI: https://doi.org/10.53391/mmnsa.2021.01.010

[28] Ana P. Lemos-Paião, Helmut Maurer, Cristiana J. Silva and Delfim F. M. Torres, A SIQRB delayed model for cholera and optimal control treatment, Mathematical Modelling of Natural Phenomena 17 (2022); Article 25.

DOI: https://doi.org/10.1051/mmnp/2022027

[29] Ana P. Lemos-Paião, Cristiana J. Silva and Delfim F. M. Torres, An epidemic model for cholera with optimal control treatment, Journal of Computational and Applied Mathematics 318 (2017), 168-180.

DOI: https://doi.org/10.1016/j.cam.2016.11.002

[30] Ana P. Lemos-Paião, Cristiana J. Silva and Delfim F. M. Torres, A cholera mathematical model with vaccination and the biggest outbreak of world's history, AIMS Mathematics 3(4) (2018), 448-463.

DOI: https://doi.org/10.3934/Math.2018.4.448

[31] Myron M. Levine, James B. Kaper, Deirdre Herrington, Genevieve Losonsky, J. Glenn Morris, M. L. Clements, Robert E. Black, B. Tall and Robert Hall, Volunteer studies of deletion mutants of Vibrio Cholerae o1 prepared by recombinant techniques, Infection and Immunity 56(1) (1988), 161-167.

DOI: https://doi.org/10.1128/iai.56.1.161-167.1988

[32] Shu Liao and Fang Fang, Stability analysis and optimal control of a cholera model with time delay, Journal of Computational Analysis & Applications 22(6) (2017), 1055-1073.

[33] Shu Liao and Weiming Yang, Cholera model incorporating media coverage with multiple delays, Mathematical Methods in the Applied Sciences 42(2) (2019), 419-439.

DOI: https://doi.org/10.1002/mma.5175

[34] A. K. Misra, S. N. Mishra, A. L. Pathak, Peeyush Misra and Ram Naresh, Modeling the effect of time delay in controlling the carrier dependent infectious disease – cholera. Applied Mathematics and Computation 218(23) (2012), 11547-11557.

DOI: https://doi.org/10.1016/j.amc.2012.04.085

[35] A. K. Misra, S. N. Mishra, A. L. Pathak, P. K. Srivastava and Peeyush Chandra, A mathematical model for the control of carrier-dependent infectious diseases with direct transmission and time delay, Chaos, Solitons & Fractals 57 (2013), 41-53.

DOI: https://doi.org/10.1016/j.chaos.2013.08.002

[36] A. K. Misra and Vishal Singh, A delay mathematical model for the spread and control of water borne diseases, Journal of Theoretical Biology 301 (2012), 49-56.

DOI: https://doi.org/10.1016/j.jtbi.2012.02.006

[37] Ahmed A. Muhseen and Xueyong Zhou, On the dynamical behaviors of a cholera model with Holling type II functional response, Al-Nahrain Journal of Science 19(1) (2016), 156-167.

DOI: https://doi.org/10.22401/JNUS.19.1.18

[38] Zindoga Mukandavire, Shu Liao, Jin Wang, Holly Gaff, David L. Smith and J. Glenn Morris, Estimating the reproductive numbers for the 2008–2009 cholera outbreaks in Zimbabwe, Proceedings of the National Academy of Sciences 108(21) (2011), 8767-8772.

DOI: https://doi.org/10.1073/pnas.1019712108

[39] Rachael L. Miller Neilan, Elsa Schaefer, Holly Gaff, K. Renee Fister and Suzanne Lenhart, Modeling optimal intervention strategies for cholera, Bulletin of Mathematical Biology 72(8) (2010), 2004-2018.

DOI: https://doi.org/10.1007/s11538-010-9521-8

[40] O. M. Ogunmiloro and O. Ojo, Transmission dynamic response of cholera epidemic model to indirect and direct infectious contact: Stability and homotopy analysis method, Journal of Computer Science and Computational Mathematics 10(1) (2020), 13-18.

DOI: https://doi.org/10.20967/jcscm.2020.01.003

[41] Jeffrey M. Ochoche, A mathematical model for the transmission dynamics of cholera with control strategy, International Journal of Science and Technology 2(11) (2013), 797-803.

[42] O. M. Ogunmiloro and T. O. Ogunlade, Transmission dynamics of cholera epidemic model with latent and hygiene compliant class, Electronic Journal of Mathematical Analysis and Applications 7(2) (2019), 138-150.

[43] Geert Jan Olsder and J. W. Van der Woude, Mathematical Systems Theory, Volume 4, VSSD Delft, 2005.

[44] Musundi Beryl Onyuma, Modelling Cholera Transmission Incorporating Media Coverage, PhD Thesis, Moi University, 2017.

[45] Kennedy Jackob Owade, Modelling the Role of Rehydration and Antibiotic Treatment on Reduction of Cholera Mortality, PhD. Thesis, Mmust, 2018.

[46] Prabir Panja, Plankton population and cholera disease transmission: A mathematical modeling study, International Journal of Bifurcation and Chaos 30(4) (2020); Article 2050054.

DOI: https://doi.org/10.1142/S0218127420500546

[47] Mercedes Pascual, Menno J. Bouma and Andrew P. Dobson, Cholera and climate: Revisiting the quantitative evidence, Microbes and Infection 4(2) (2002), 237-245.

DOI: https://doi.org/10.1016/S1286-4579(01)01533-7

[48] M. Mujibur Rahaman, M. A. Majid, A. K. M. Jamiul Alam and M. Rafiqul Islam, Effects of doxycycline in actively purging cholera patients: A double-blind clinical trial, Antimicrobial Agents and Chemotherapy 10(4) (1976), 610-612.

DOI: https://doi.org/10.1128/aac.10.4.610

[49] S. Saber, E. Bashier, S. Alzahrani and I. Noaman, A mathematical model of glucose-insulin interaction with time delay, Journal of Applied & Computational Mathematics 7(3) (2018), 416.

DOI: https://doi.org/10.4172/2168-9679.1000416

[50] Anulekha Samadder, Koushik Ghosh and Kripasindhu Chaudhuri, A dynamical model for the spread of cholera near an aquatic reservoir, World Journal of Modelling and Simulation 10(4) (2014), 273-279.

[51] Zhisheng Shuai, Joseph H. Tien and P. Van den Driessche, Cholera models with hyperinfectivity and temporary immunity, Bulletin of Mathematical Biology 74(10) (2012), 2423-2445.

DOI: https://doi.org/10.1007/s11538-012-9759-4

[52] Zhisheng Shuai and P. Van den Driessche, Global dynamics of cholera models with differential infectivity, Mathematical Biosciences 234(2) (2011), 118-126.

DOI: https://doi.org/10.1016/j.mbs.2011.09.003

[53] Chenwei Song, Rui Xu, Ning Bai, Xiao Hong Tian and Jia Zhe Lin, Global dynamics and optimal control of a cholera transmission model with vaccination strategy and multiple pathways, Mathematical Biosciences and Engineering 17(4) (2020), 4210-4224.

DOI: https://doi.org/10.3934/mbe.2020233

[54] Pauline Van den Driessche and James Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Mathematical Biosciences 180(1-2) (2002), 29-48.

DOI: https://doi.org/10.1016/s0025-5564(02)00108-6

[55] Jin Wang and Chairat Modnak, Modeling cholera dynamics with controls, Canadian Applied Mathematics Quarterly 19(3) (2011), 255-273.

[56] Xueying Wang, Daozhou Gao and Jin Wang, Influence of human behavior on cholera dynamics, Mathematical biosciences 267 (2015), 41-52.

DOI: https://doi.org/10.1016/j.mbs.2015.06.009

[57] Xueying Wang and Jin Wang, Analysis of cholera epidemics with bacterial growth and spatial movement, Journal of Biological Dynamics 9(sup1) (2015), 233-261.

DOI: https://doi.org/10.1080/17513758.2014.974696

[58] Xueying Wang and Jin Wang, Modeling the within-host dynamics of cholera: Bacterial-viral interaction, Journal of Biological Dynamics 11(sup2) (2017), 484-501.

DOI: https://doi.org/10.1080/17513758.2016.1269957

[59] Xueying Wang, Ruiwen Wu and Xiao-Qiang Zhao, A reaction-advection-diffusion model of cholera epidemics with seasonality and human behavior change, Journal of Mathematical Biology 84(5) (2022), 1-30.

DOI: https://doi.org/10.1007/s00285-022-01733-3

[60] Yujie Wang and Junjie Wei, Global dynamics of a cholera model with time delay, International Journal of Biomathematics 6(1) (2013); Article 1250070.

DOI: https://doi.org/10.1142/S1793524512500702

[61] Şuayip Yüzbaşi and Mohammad Izadi, Bessel-quasilinearization technique to solve the fractional-order HIV-1 infection of $CD4^+$ T-cells considering the impact of antiviral drug treatment, Applied Mathematics and Computation 431 (2022); Article 127319.

DPO: https://doi.org/10.1016/j.amc.2022.127319

[62] Şuayip Yüzbaşi and Murat Karaçayır, A Galerkin-type method for solving a delayed model on HIV infection of $CD4^+$ T-cells, Iranian Journal of Science and Technology, Transactions A: Science 42(3) (2018), 1087-1095.

DOI: https://doi.org/10.1007/s40995-018-0529-5

[63] Şuayip Yüzbaşi and Gamze Yıldırım, A Pell-Lucas collocation approach for an SIR model on the spread of the novel coronavirus (SARS CoV-2) pandemic: The case of turkey, Mathematics 11(3) (2023); Article 697.

DOI: https://doi.org/10.3390/math11030697

[64] Xue-Yong Zhou, Xiang-Yun Shi and Jing-An Cui, Dynamic behavior of a delay cholera model with constant infectious period, Journal of Applied Analysis & Computation 10(2) (2020), 598-623.

DOI: https://doi.org/10.11948/20190006

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