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Research Article

A Non-Integer Variable Order Mathematical Model of Human Immunodeficiency Virus and Malaria Coinfection with Time Delay

A. A. M. Arafa,[1] M. Khalil,[2] and A. Sayed[1]

[1]*Department of Mathematics and Computer Science, Faculty of Science, Port Said University, Port Said, Egypt*
[2]*Department of Mathematics, Faculty of Engineering, October University for Modern Sciences and Arts (MSA), Giza, Egypt*

Correspondence should be addressed to A. Sayed; amal_wa84@yahoo.com

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The purpose of this paper is to propose a variable fractional-order model with a constant time delay of the coinfection of HIV/AIDS and malaria. The proposed model describes the interaction between HIV/AIDS and malaria. This model is presented by using variable fractional-order derivative which is an extension of the constant fractional-order derivative to explain a certain pattern in the development of infection of several patients. The presented model has been solved numerically via the predictor-corrector scheme. The local and global stability conditions of the disease-free equilibrium are investigated. Also, numerical simulations are presented for different variable fractional-order derivatives in Caputo sense.

1. Introduction

The human immunodeficiency virus (HIV) and malaria are considered among the most challenging global public health issues in the last few decades. HIV and malaria are life-threatening diseases which have similar geographic distributions [1]. They cause millions of deaths every year in several areas especially in Africa, Asia, and Latin America. In 2017, HIV killed about one million people [2] while malaria killed roughly 435 000 people worldwide [3]. HIV can be transmitted through certain body fluids while malaria is transmitted through bites of infected mosquitoes.

HIV is considered as one of the most deadly infectious diseases which strikes the human immune system and destroy the CD4+ cells. AIDS is the last stage of HIV which occurs when the CD4+ cells of the human body count drops below 200 cells/mm [4]. In this stage, the immune system cannot defend the body against the attacks by several opportunistic diseases. On the other hand, if malaria parasite invades the bloodstream, then, it destroys red blood cells. So, malaria infection may be developed to anemia or cerebral malaria, which can cause disabilities and death [5]. The coinfection of HIV and malaria has become endemic in several developing countries. World health organization (WHO) reports indicating that more than two million people die every year because of the malaria/AIDS coinfection [6]. The interaction between HIV and malaria in Sub-Saharan Africa has become among the major public health problems [7] and has resulted in many economic disasters [1] by negatively affecting the contribution of the labor force to the national economy.

Recently, increasing research efforts have been made to obtain an effective vaccine to halt the progression and transmission of malaria. Vaccination target is to reduce the rate of human infection, the severity of the disease [8–10], and the parasite's transmission to mosquitoes. Clinical trials in Africa proved that a malaria vaccine is partially protective [11].

From mathematicians' perspective, mathematical models are significant tools that help us to understand the current state and the future progress of infectious diseases in human networks in order to control and prevent such diseases. Several mathematical models have been presented to study the prevalence and the coinfection of HIV and malaria, but most

of such models are integer or constant fractional-order models [12–22]. This paper is devoted to propose a delay variable fractional-order model for the coinfection of HIV/AIDS and malaria. In this model, a discrete time delay τ_h is incorporated in the variables of active humans who are infected by malaria and the coinfected humans while a discrete time delay τ_m is incorporated in the variable of the infectious mosquitoes. After a time τ_h, susceptible people become infected by malaria while exposed individuals become infectious after the same time. On the other hand, mosquitoes become infectious after time τ_m. Introducing such a time delay to the proposed model is essential to characterize the time needed to start in vaccination and treatments processes. The merits of the proposed model are clear from putting in the time delay with the variable fractional-order derivative which is an extension of the constant fractional-order in the same model. Hence, using the proposed variable fractional-order model with time delay gives a better understanding of the interaction between malaria and HIV. To the best of our knowledge, the presented model is the first variable fractional-order model with a time delay which describes the prevalence and interactions between HIV and malaria. In this model, the integer order derivative is used to distinguish the short memory of systems, while the variable fractional-order derivative is utilized to characterize the variable memory of systems.

This paper is organized as follows. In Section 2, some preliminaries of fractional calculus and the algorithm of the predictor-corrector method are presented while Section 3 describes the proposed model. In Section 4, the disease-free equilibrium and stability are presented. The basic reproduction number is computed in Section 5. Section 6 is devoted to the numerical results and discussions. Our conclusion is illustrated in Section 7.

2. Preliminaries

2.1. Fractional Calculus. The fractional calculus is considered as a mathematical tool for characterizing memory of biological and epidemiological systems. The classical integer order derivative can be used to describe the short memory of the dynamical systems, while fractional-order derivative has the merit of describing the long memory of dynamical systems. The variable fractional-order derivative is an extension of the constant fractional-order derivative and has been introduced in several scientific fields [23–25]. Also, it is a powerful tool to characterize memory that varies from point to point. Furthermore, the variable fractional-order derivative can be applied to describe the variable memory of dynamical systems [26].

In this section, we present some basic definitions of constant/variable fractional-order derivatives as follows.

Definition 1 (Riemann–Liouville derivatives of fractional-order α). Let α be a bounded and continuous function; then Riemann–Liouville fractional-order derivative of $f(t) : [a,b] \longrightarrow \mathbb{R}$ is defined as follows [27].

(i) Left Riemann–Liouville derivative of fractional-order α is defined by

$$ {}^{RL}_{a}D^{\alpha}_{t} f(t) = \frac{1}{\Gamma(1-\alpha)} \frac{d}{dt} \int_a^t (t-\omega)^{-\alpha} f(\omega)\, d\omega, \quad 0 < \alpha \leq 1 \tag{1} $$

(ii) Right Riemann–Liouville derivative of fractional-order α is defined by

$$ {}^{RL}_{t}D^{\alpha}_{b} f(t) = \frac{1}{\Gamma(1-\alpha)} \frac{d}{dt} \int_t^b (t-\omega)^{-\alpha} f(\omega)\, d\omega, \quad 0 < \alpha \leq 1 \tag{2} $$

Definition 2 (Caputo derivatives of fractional-order α). Let α be a bounded and continuous function; then the Caputo fractional-order derivative of $f(t) : [a,b] \longrightarrow \mathbb{R}$ is defined as follows [27].

(i) Left Caputo derivative of fractional-order α is defined by

$$ {}^{C}_{a}D^{\alpha}_{t} f(t) = \frac{1}{\Gamma(1-\alpha)} \int_a^t (t-\omega)^{-\alpha} f'(\omega)\, d\omega, \quad 0 < \alpha \leq 1 \tag{3} $$

(ii) Right Caputo derivative of fractional-order α is defined by

$$ {}^{C}_{t}D^{\alpha}_{b} f(t) = \frac{-1}{\Gamma(1-\alpha)} \int_t^b (t-\omega)^{-\alpha} f'(\omega)\, d\omega, \quad 0 < \alpha \leq 1 \tag{4} $$

Definition 3 (Riemann–Liouville derivatives of variable fractional-order $\alpha(t)$). Let $\alpha(t)$ be a bounded and continuous function; then Riemann–Liouville fractional-order derivative of $f(t) : [a,b] \longrightarrow \mathbb{R}$ is defined as follows [27].

(i) Left Riemann–Liouville derivative of variable fractional-order $\alpha(t)$ is defined by

$$ {}^{RL}_{a}D^{\alpha(t)}_{t} f(t) = \frac{1}{\Gamma(1-\alpha(t))} \frac{d}{dt} \int_a^t (t-\omega)^{-\alpha(t)} f(\omega)\, d\omega, \quad 0 < \alpha(t) \leq 1 \tag{5} $$

(ii) Right Riemann–Liouville derivative of fractional-order α is defined by

$$ {}^{RL}_{t}D^{\alpha(t)}_{b} = \frac{1}{\Gamma(1-\alpha(t))} \frac{d}{dt} \int_t^b (t-\omega)^{-\alpha(t)} f(\omega)\, d\omega, \quad 0 < \alpha(t) \leq 1 \tag{6} $$

Definition 4 (Caputo derivatives of variable fractional-order $\alpha(t)$). Let $\alpha(t)$ be a bounded and continuous function; then the Caputo fractional-order derivative of $f(t) : [a,b] \longrightarrow \mathbb{R}$ is defined as follows [27].

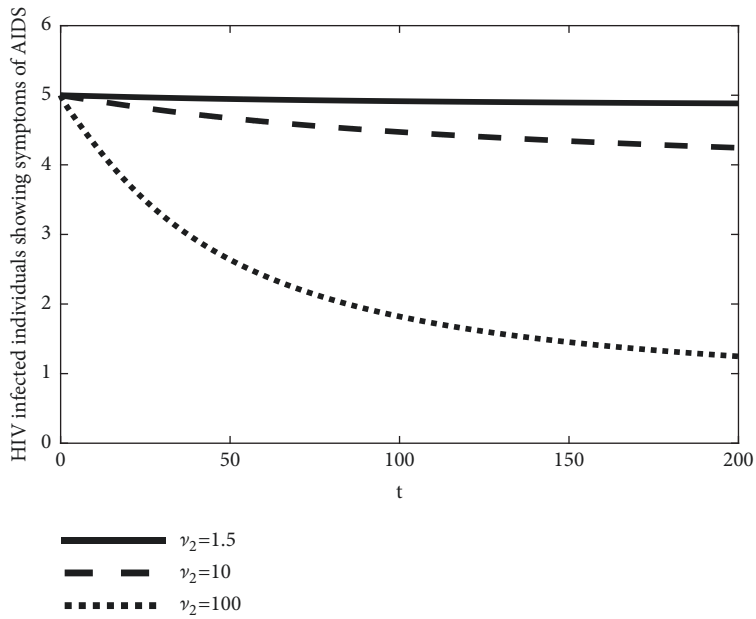

Figure 1: HIV infected individuals showing symptoms of AIDS at $\alpha(t) = 0.8$ with $\nu_2 = 1.5$ (solid line), $\nu_2 = 10$ (dashed line), and $\nu_2 = 100$ (dotted line). Parameters values are in Table 1 with $\beta_h = 0.01$.

(i) Left Caputo derivative of fractional-order $\alpha(t)$ is defined by

$$ {}_a^C D_t^{\alpha(t)} f(t) = \frac{1}{\Gamma(1-\alpha(t))} \int_a^t (t-\omega)^{-\alpha(t)} f'(\omega)\, d\omega, \quad 0 < \alpha(t) \le 1 \tag{7} $$

(ii) Right Caputo derivative of fractional-order $\alpha(t)$ is defined by

$$ {}_t^C D_b^{\alpha(t)} f(t) = \frac{-1}{\Gamma(1-\alpha(t))} \int_t^b (t-\omega)^{-\alpha(t)} f'(\omega)\, d\omega, \quad 0 < \alpha(t) \le 1 \tag{8} $$

2.2. Predictor-Corrector Method. There are many techniques for solving a delay variable fractional-order models such as finite difference [28], Hermite wavelet [29], and Adams-Bashforth-Morton [30] methods. In this section, we state a predictor-corrector method for solving a delay variable fractional-order model [31].

Let

$$ {}^C D^{\alpha(t)} y(t) = f\left(t, y(t), y(t-\varsigma)\right), \quad 0 \le t \le T, \qquad y(t) = g(t), \quad -\varsigma \le t \le 0 \tag{9} $$

where $0 < \alpha(t) \le 1$, $T \in \mathbb{R}^+$, and $g(t)$ is a smooth function. Suppose a uniform grid $\{t_j = jh : j = -q, q+1, \ldots, -1, 0, 1, \ldots, n\}$, where n and q are integers such that $n = T/h$ and $q = \varsigma/h$.

The predictor approximation y_{n+1}^p is defined by

$$ y_{n+1}^p = y(0) + \frac{1}{\Gamma(\alpha(t_{n+1}))} \sum_{j=0}^{n} B_{j,n+1} f\left(t_j, y_j, y_{j-q}\right), \tag{10} $$

where

$$ B_{j,n+1} = \frac{h^{\alpha(t_{n+1})}}{\alpha(t_{n+1})} \left[(n-j+1)^{\alpha(t_{n+1})} - (n-j)^{\alpha(t_{n+1})} \right], \quad 0 \le j \le n. \tag{11} $$

The corrector approximation y_{n+1} is defined by

$$ y_{n+1} = y(0) + \frac{h^{\alpha(t_{n+1})}}{\Gamma(\alpha(t_{n+1})+2)} f\left(t_{n+1}, y_{n+1}^p, y_{n+1-q}\right) + \frac{h^{\alpha(t_{n+1})}}{\Gamma(\alpha(t_{n+1})+2)} \sum_{j=0}^{n} A_{j,n+1} f\left(t_j, y_j, y_{j-q}\right) \tag{12} $$

where

$$ A_{j,n+1} = \begin{cases} n^{\alpha(t_{n+1})+1} - \left[n - \alpha(t_{n+1})\right](n+1)^{\alpha(t_{n+1})}, & j = 0, \\ (n-j+2)^{\alpha(t_{n+1})+1} - 2(n-j+1)^{\alpha(t_{n+1})+1} + (n-j)^{\alpha(t_{n+1})+1}, & 1 \le j \le n, \\ 1, & j = n+1. \end{cases} \tag{13} $$

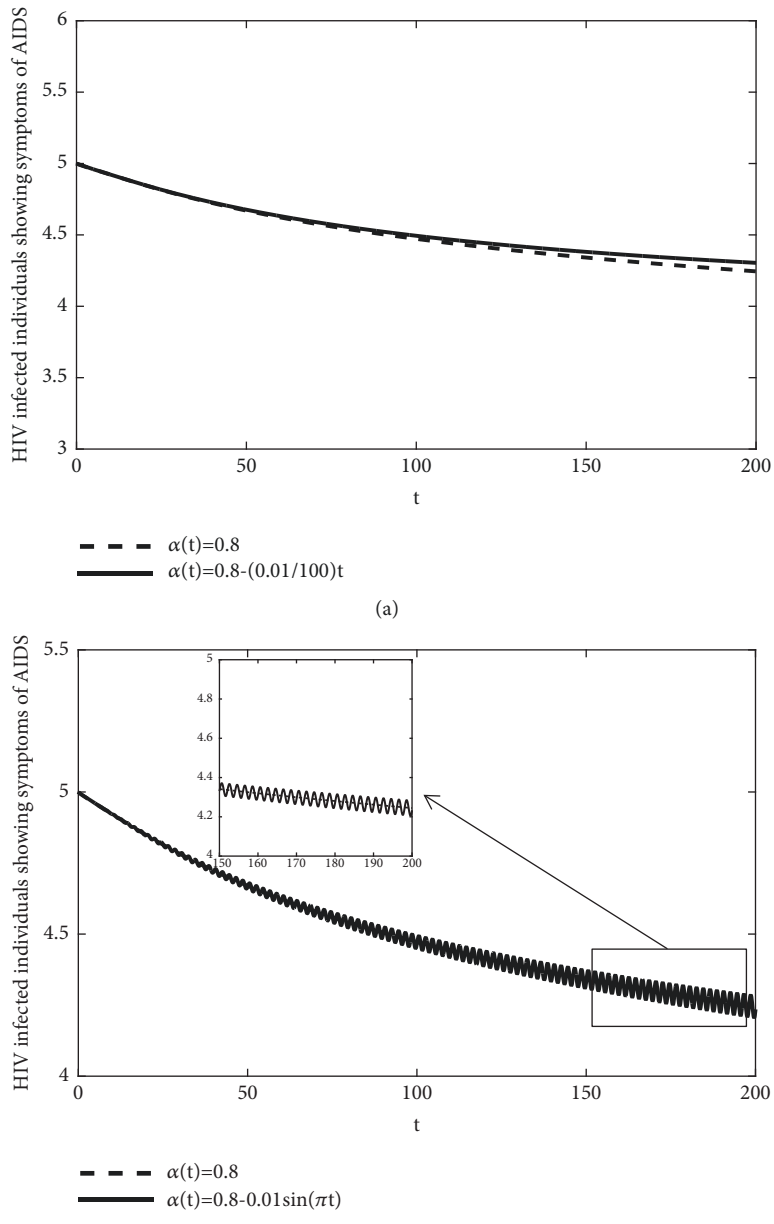

Figure 2: HIV infected individuals showing symptoms of AIDS with $\nu_2 = 10$. Parameters values are in Table 1 with $\beta_h = 0.01$. (a) $\alpha(t) = 0.8$ comparing with $\alpha(t) = 0.8 - (0.01/100)t$; (b) $\alpha(t) = 0.8$ comparing with $\alpha(t) = 0.8 - 0.01\sin(\pi t)$.

3. The Model

The proposed variable fractional-order model with a constant delay in this paper is based on the constant fractional delay model proposed in [32]. This model consists of 12 compartments, as follows:

$$
\begin{aligned}
&{}^{C}D^{\alpha(t)}N_h(t) \\
&\quad = A_h - a_{h1}\left[I_h(t) + (1-\theta_2)Y_h(t)\right] - \tau a_{h1} I_{mhiv}(t) \\
&\qquad - \left[\tau a_{h1} + \psi\delta_H\right] A_{mhiv}(t) - \delta_H A_{hiv}(t) \\
&\qquad - \mu_h N_h(t), \\
&{}^{C}D^{\alpha(t)}S_h(t) \\
&\quad = (1-p)A_h - f_h(t)S_h(t) - \beta_{hiv}(t)S_h(t) \\
&\qquad + r_h\left[I_h(t) + \theta_1 Y_h(t)\right] + \sigma V_h(t) - \mu_h S_h(t), \\
&{}^{C}D^{\alpha(t)}V_h(t) \\
&\quad = pA_h - f_h(t)(1-\gamma)V_h(t) - \left[\sigma + \mu_h\right]V_h(t), \\
&{}^{C}D^{\alpha(t)}I_h(t)
\end{aligned}
$$

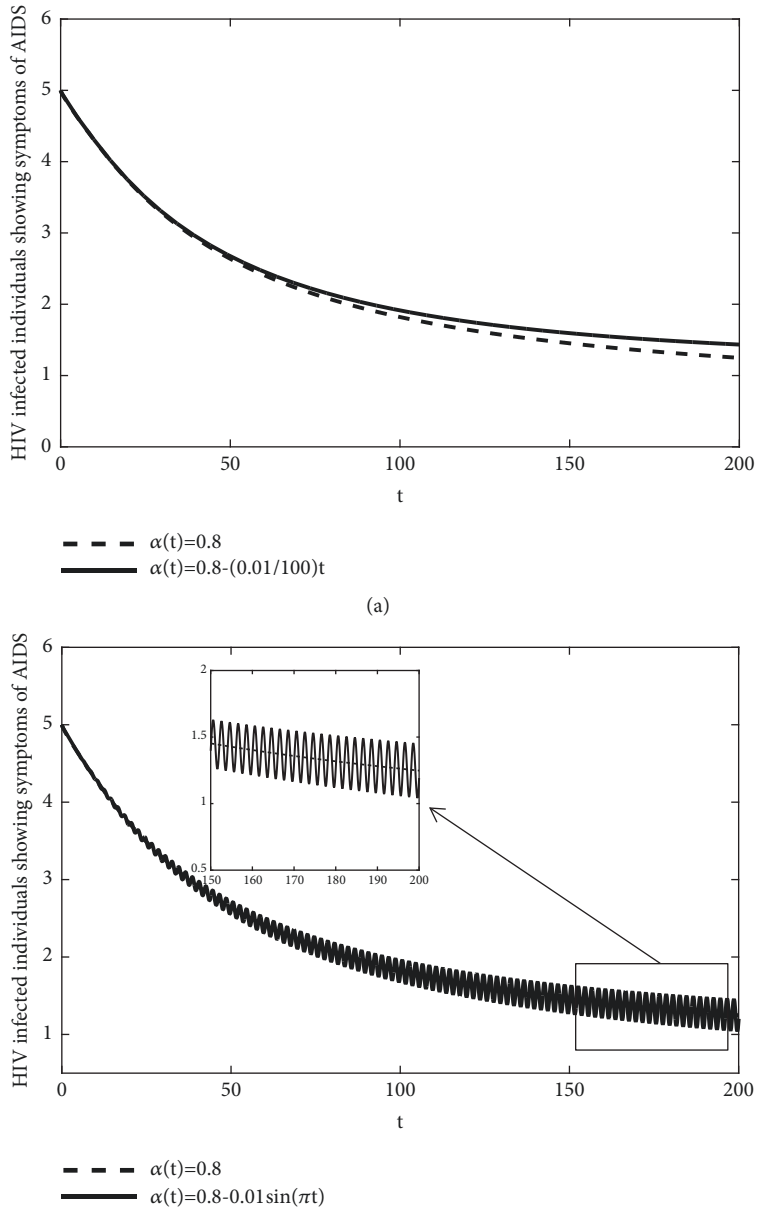

Figure 3: HIV infected individuals showing symptoms of AIDS with $\nu_2 = 100$. Parameters values are in Table 1 with $\beta_h = 0.01$. (a) $\alpha(t) = 0.8$ comparing with $\alpha(t) = 0.8 - (0.01/100)t$; (b) $\alpha(t) = 0.8$ comparing with $\alpha(t) = 0.8 - 0.01\sin(\pi t)$.

$$= f_h(t-\tau_h) S_h(t-\tau_h) e^{-\mu_h \tau_h} - \varepsilon_2 \beta_{hiv}(t) I_h(t) - [r_h + a_{h1} + \mu_h] I_h(t),$$

$${}^C D^{\alpha(t)} I_{mhiv}(t) = \upsilon_1 f_h(t-\tau_h) I_{hiv}(t-\tau_h) e^{-\mu_h \tau_h} + \varepsilon_2 \beta_{hiv}(t) I_h(t) - [\zeta a_{h2} + \phi_2 + \tau a_{h1} + \mu_h] I_{mhiv}(t),$$

$${}^C D^{\alpha(t)} I_{hiv}(t) = \beta_{hiv}(t) S_h(t) + \phi_2 I_{mhiv}(t) - \upsilon_1 f_h(t) I_{hiv}(t) - [a_{h2} + \mu_h] I_{hiv}(t),$$

$${}^C D^{\alpha(t)} Y_h(t) = f_h(t-\tau_h)(1-\gamma) V_h(t-\tau_h) e^{-\mu_h \tau_h} - [\theta_1 r_h + (1-\theta_2) a_{h1} + \mu_h] Y_h(t),$$

$${}^C D^{\alpha(t)} A_{mhiv}(t) = \zeta a_{h2} I_{mhiv}(t) + \upsilon_2 f_h(t-\tau_h) A_{hiv}(t-\tau_h) e^{-\mu_h \tau_h} - [\mu_h + \phi_3 + \tau a_{h1} + \psi \delta_H] A_{mhiv}(t),$$

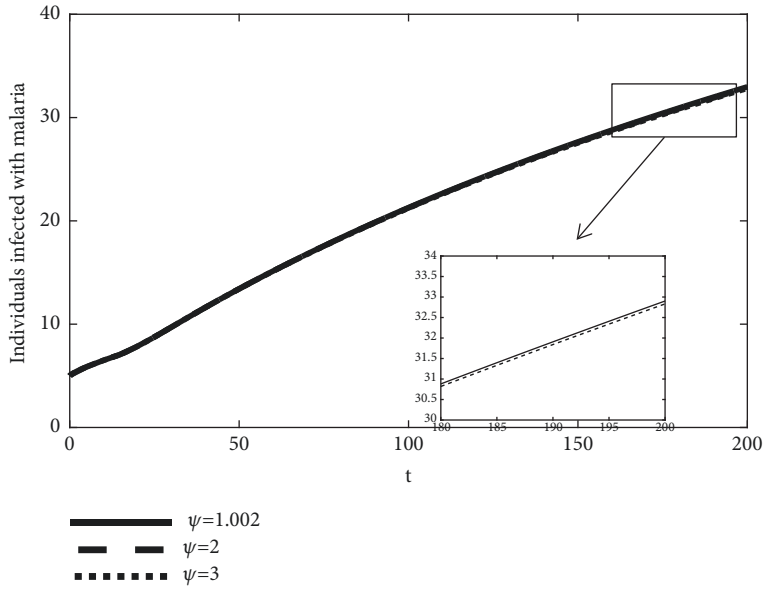

FIGURE 4: Individuals infected with malaria at $\alpha(t) = 0.8$ with $\psi = 1.002$ (solid line), $\psi = 2$ (dashed line), and $\psi = 3$ (dotted line). Parameters values are in Table 1 with $\beta_h = 0.05$.

$$
\begin{aligned}
&{}^{C}D^{\alpha(t)}A_{hiv}(t)\\
&\quad = a_{h2}I_{hiv}(t) + \phi_3 A_{mhiv}(t) - \upsilon_2 f_h(t) A_{hiv}(t)\\
&\qquad - \left[\mu_h + \delta_H\right] A_{hiv}(t),\\
&{}^{C}D^{\alpha(t)}N_m(t) = A_m - a_m I_m(t) - \mu_m N_m(t),\\
&{}^{C}D^{\alpha(t)}S_m(t) = A_m - f_m(t) S_m(t) - \mu_m S_m(t),\\
&{}^{C}D^{\alpha(t)}I_m(t)\\
&\quad = f_m(t-\tau_m) S_m(t-\tau_m) e^{-\mu_m \tau_m}\\
&\qquad - \left[\mu_m + a_m\right] I_m(t).
\end{aligned}
\tag{14}
$$

where the population of mosquitoes as follows:

$$N_m(t) = I_m(t) + S_m(t), \tag{15}$$

where $I_m(t)$ are the infectious mosquitoes and $S_m(t)$ are the susceptible mosquitoes.

And the population of human $N_h(t)$ is divided into the following classes:

S_h are the susceptible individuals

V_h are the individuals vaccinated against malaria

I_h are the individuals infected with malaria

Y_h are individuals infected and vaccinated against malaria

I_{mhiv} are the coinfected individuals showing no symptoms of AIDS

I_{hiv} are the individuals asymptomatically infected with HIV/AIDS

A_{hiv} are the HIV infected individuals showing symptoms of AIDS

A_{mhiv} are the coinfected individuals showing symptoms of AIDS

Besides, all human are subject to natural death, occurring at a rate μ_h. Susceptible individuals get in the human population at a rate A_h. The parameter p is the proportion of individuals successfully vaccinated, where $(1-p)A_h$ is the proportion getting in the class $S_h(t)$ and pA_h is the proportion getting in the class $V_h(t)$. Susceptible individuals enter the class $I_h(t)$ after some time τ_h. The rate of infection by malaria parasite of susceptible individuals $f_h(t)$ is given by

$$f_h(t) = \beta_h c (1 - bz) \frac{I_m(t)}{N_h(t)} \tag{16}$$

where $0 < b \leq 1$ is the proportion of individuals in the community and $0 < z \leq 1$ models the efficacy of adopted strategies for individuals protection. c is the rate of female mosquitoes' bites. The value of the probability that a bite of an infectious mosquito leads to the infection of a susceptible human is β_h. The efficacy of the preerythrocytic vaccine is given by $0 < \gamma \leq 1$. Vaccinated individuals may become susceptible at a rate σ. The rate of infection with HIV/AIDS of susceptible individuals is $\beta_{hiv}(t)$:

$$
\begin{aligned}
&\beta_{hiv}(t)\\
&= \frac{\beta_H \left[I_{hiv}(t) + \eta_{HM} I_{mhiv}(t) + \eta_A \left[A_{hiv}(t) + \eta_{HM} A_{mhiv}(t)\right]\right]}{N_h(t)}
\end{aligned}
\tag{17}
$$

where $\eta_A > 1$ is the infectiousness of individuals in the AIDS stage of HIV infection. β_H is the effective contact rate for HIV infection. Infectiousness to malaria of coinfected individuals showing symptoms of AIDS is $\eta_{HM} > 1$.

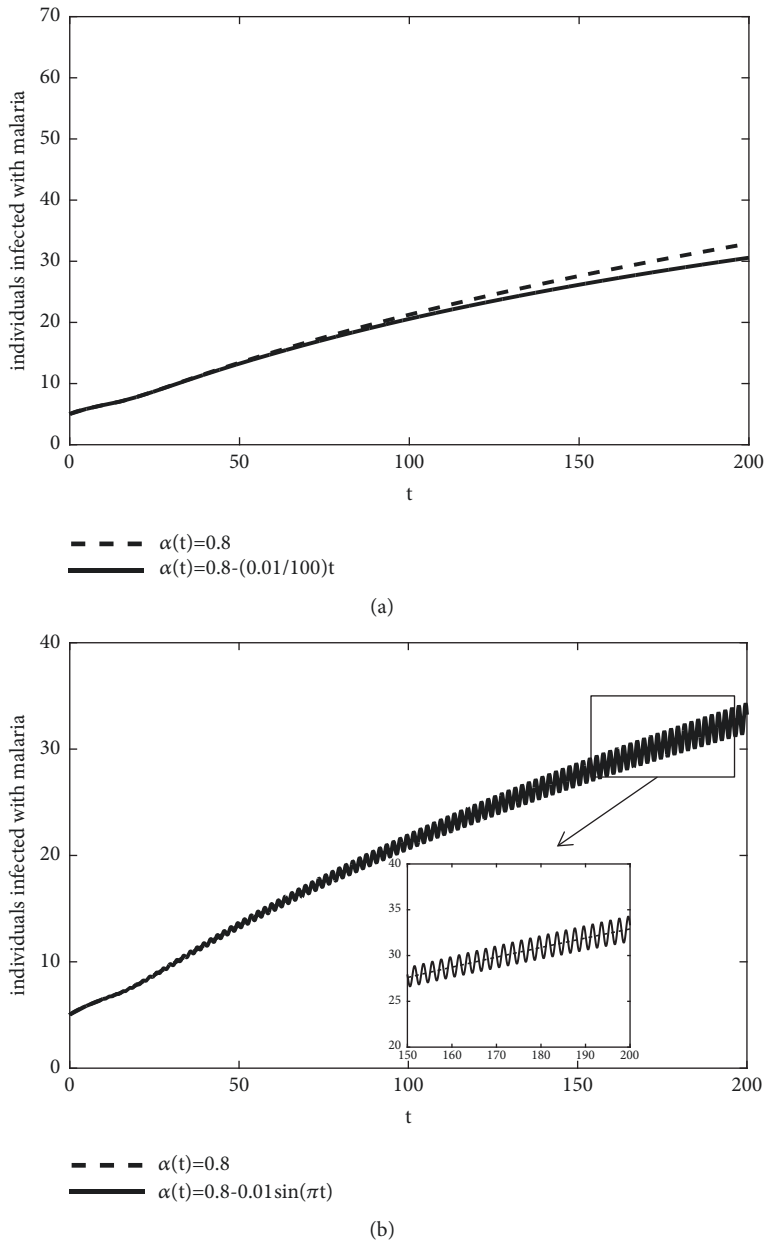

FIGURE 5: Individuals infected with malaria with $\psi = 2$. Parameters values are in Table 1 with $\beta_h = 0.05$. (a) $\alpha(t) = 0.8$ comparing with $\alpha(t) = 0.8 - (0.01/100)t$; (b) $\alpha(t) = 0.8$ comparing with $\alpha(t) = 0.8 - 0.01\sin(\pi t)$.

Parameter $0 < \theta_2 \leq 1$ models the effect of the preerythrocytic vaccine in the raising of the recovery. Parameter $\theta_1 \geq 1$ models the effect of the preerythrocytic vaccine in the decreasing of mortality due to disease. The rate of recovery of individuals infected with malaria and going to the susceptible class is r_h. The rate of death of individuals infected with malaria is a_{h1}. $\varepsilon_2 < 1$ models the decrease in sexual activity due to malaria disease. ϕ_2 is the rate of recovery of the coinfected individuals showing no symptoms of AIDS from malaria. τ refers to the increased malaria mortality of individuals coinfected with HIV. ψ indicates the rise in HIV mortality due to the coinfection with malaria. a_{h2} is the rate of development of $I_{hiv}(t)$ to AIDS. The rate of death from AIDS is δ_H. The rate of natural death of $I_{hiv}(t)$ is μ_h. υ_1 is the assumed rise in susceptibility to malaria as a result of HIV infection. The rate of recovery of $A_{mhiv}(t)$ from malaria is ϕ_3. υ_2 is the rise in susceptibility to malaria of individuals of $A_{hiv}(t)$. $\zeta > 1$ defines those coinfected individuals develop to AIDS faster than those infected only with HIV.

The rate of natural death of mosquitoes is μ_m. The rate of infection by the Anopheles parasite of susceptible mosquitoes $f_m(t)$ is given by

$$f_m(t) = \beta_m c(1 - bz) \cdot \frac{I_h(t) + I_{mhiv}(t) + (1 - \varepsilon) Y_h(t) + A_{mhiv}(t)}{N_h(t)} \quad (18)$$

where $\varepsilon \in [0,1]$ defines the decreasing of transmission from vaccinated humans to susceptible mosquitoes. The probability that a mosquito's bite in a malaria infective human tends to infection of the mosquito is β_m. The exposed mosquitoes turn infectious after time τ_m. The rate of increasing mortality due to the presence of the parasite in the body is a_m. In other words, all mosquitoes are subjected to a natural death, at a rate of μ_m. It is assumed that the infectious mosquitoes are subjected to death rate because of the presence of the parasite in their bodies at a rate a_m and that they do not recover before they die [32].

4. The Disease-Free Equilibrium and Stability

The equilibrium point of a dynamical system is a solution that does not change with time.

To obtain the disease-free equilibrium of model (14), let

$$\begin{aligned} {}^{C}D^{\alpha(t)}N_h(t) &= {}^{C}D^{\alpha(t)}S_h(t) = {}^{C}D^{\alpha(t)}V_h(t) \\ &= {}^{C}D^{\alpha(t)}I_h(t) = {}^{C}D^{\alpha(t)}I_{mhiv}(t) \\ &= {}^{C}D^{\alpha(t)}I_{hiv}(t) = {}^{C}D^{\alpha(t)}Y_h(t) \\ &= {}^{C}D^{\alpha(t)}A_{mhiv}(t)\,{}^{C}D^{\alpha(t)}A_{hiv}(t) \\ &= {}^{C}D^{\alpha(t)}N_m(t) = {}^{C}D^{\alpha(t)}S_m(t) \\ &= {}^{C}D^{\alpha(t)}I_m(t) = 0 \end{aligned} \tag{19}$$

Then the disease-free equilibrium E_0 is

$$E_0 = \left(\frac{A_h}{\mu_h}, \frac{(1-p)A_h(\sigma+\mu_h) + \sigma p A_h}{\mu_h(\sigma+\mu_h)}, \frac{pA_h}{\sigma+\mu_h}, 0, 0, 0, 0, 0, \frac{A_m}{\mu_m}, \frac{A_m}{\mu_m}, 0 \right) \tag{20}$$

The stability of disease-free equilibrium is defined by a sign of real part of eigenvalues of the Jacobian matrix evaluated at disease-free equilibrium. The Jacobian matrix is the matrix of partial derivatives of the right-hand side with respect to state variables.

The Jacobian matrix of model (14) around the disease-free equilibrium E_0 is

$$J(E_0) = \begin{bmatrix}
-\mu_h & 0 & 0 & -a_{h1} & -\tau a_{h1} & 0 & -a_{h1}(1-\theta_2) & -\tau a_{h1} - \psi\delta_H & -\delta_H & 0 & 0 & 0 \\
0 & -\mu_h & \sigma & r_h & -\eta_{HM}G_3 & G_3 & r_h\theta_1 & \eta_A\eta_{HM}G_3 & \eta_A G_3 & 0 & 0 & G_4 \\
0 & 0 & -(\sigma+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & G_5 \\
0 & 0 & 0 & -(r_h + a_{h1} + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -e^{-\mu_h\tau_h}G_4 \\
0 & 0 & 0 & 0 & -(\zeta a_{h2} + \phi_2 + \tau a_{h1} + \mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \phi_2 - \eta_{HM}G_3 & -G_3 - (a_{h2} + \mu_h) & 0 & -\eta_A\eta_{HM}G_3 & -\eta_A G_3 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & -(\theta_1 r_h + (1-\theta_2)a_{h1} + \mu_h) & 0 & 0 & 0 & 0 & -e^{-\mu_h\tau_h}G_5 \\
0 & 0 & 0 & 0 & & 0 & 0 & -(\mu_h + \phi_3 + \tau a_{h1} + \psi\delta_H) & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & a_{h2} & 0 & \phi_3 & -(\mu_h + \delta_H) & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_m & 0 & -a_m \\
0 & 0 & 0 & G_1 & G_1 & 0 & (1-\varepsilon)G_1 & G_1 & 0 & 0 & -\mu_m & 0 \\
0 & 0 & 0 & G_2 & G_2 & 0 & (1-\varepsilon)G_2 & G_2 & 0 & 0 & 0 & -(\mu_m + a_m)
\end{bmatrix} \tag{21}$$

where

$$\begin{aligned}
G_1 &= -\beta_m c(1-bz)\frac{\mu_h A_m}{\mu_m A_h}, \\
G_2 &= \beta_m c(1-bz)\frac{\mu_h A_m}{\mu_m A_h}e^{-\tau_m(\lambda+\mu_m)}, \\
G_3 &= -\beta_H\left(\frac{\sigma+\mu_h(1-p)}{\sigma+\mu_h}\right), \\
G_4 &= -\beta_h c(1-bz)\frac{\sigma+\mu_h(1-p)}{\sigma+\mu_h}, \\
G_5 &= -\beta_h c(1-bz)(1-\gamma)\frac{p\mu_h}{\sigma+\mu_h}.
\end{aligned} \tag{22}$$

The eigenvalues of the Jacobian matrix are

$$\begin{aligned}
\lambda_{1,2} &= -\mu_h, \\
\lambda_{3,4} &= -\mu_m, \\
\lambda_5 &= -(\sigma+\mu_h), \\
\lambda_6 &= -(\zeta a_{h2} + \phi_2 + \tau a_{h1} + \mu_h), \\
\lambda_7 &= -(\mu_h + \phi_3 + \tau a_{h1} + \psi\delta_H)
\end{aligned} \tag{23}$$

The remaining five eigenvalues are obtained from the following matrix:

$$M = \begin{bmatrix}
F_1 & 0 & 0 & 0 & F_2 \\
0 & F_3 & 0 & F_4 & 0 \\
0 & 0 & F_5 & 0 & F_6 \\
0 & F_7 & 0 & F_8 & 0 \\
F_9 & 0 & F_{10} & 0 & F_{11}
\end{bmatrix} \tag{24}$$

where

$$
\begin{aligned}
F_1 &= -\left(r_h + a_{h1} + \mu_h\right),\\
F_2 &= \beta_h c\left(1 - bz\right)\frac{\sigma + \mu_h\left(1 - p\right)}{\sigma + \mu_h}e^{-\tau_h(\lambda+\mu_h)}\\
F_3 &= \beta_H\frac{\sigma + \mu_h\left(1 - p\right)}{\sigma + \mu_h} - \left(a_{h2} + \mu_h\right),\\
F_4 &= \beta_H\eta_h\frac{\sigma + \mu_h\left(1 - p\right)}{\sigma + \mu_h}\\
F_5 &= -\left(\theta_1 r_h + \left(1 - \theta_2\right)a_{h1} + \mu_h\right),\\
F_6 &= \beta_h c\left(1 - bz\right)\left(1 - \gamma\right)\frac{p\mu_h}{\sigma + \mu_h}e^{-\tau_h(\lambda+\mu_h)}\\
F_7 &= a_{h2},\\
F_8 &= -\left(\mu_h + \delta_H\right),\\
F_9 &= \beta_m c\left(1 - bz\right)\frac{\mu_h A_m}{\mu_m A_h}e^{-\tau_m(\lambda+\mu_m)}\\
F_{10} &= \beta_m c\left(1 - bz\right)\left(1 - \varepsilon\right)\frac{\mu_h A_m}{\mu_m A_h}e^{-\tau_m(\lambda+\mu_m)},\\
F_{11} &= -\left(\mu_m + a_m\right)
\end{aligned}
\tag{25}
$$

That matrix M has the characteristic equation

$$\lambda^5 + M_1\lambda^4 + M_2\lambda^3 + M_3\lambda^2 + M_4\lambda + M_5 = 0 \tag{26}$$

where

$$M_1 = -\left(F_1 + F_3 + F_5 + F_8 + F_{11}\right) \tag{27}$$

$$
\begin{aligned}
M_2 = {} & F_{11}\left(F_1 + F_3 + F_5 + F_8\right) + F_5F_8 - F_{10}F_6\\
& + \left(F_1 + F_3\right)\left(F_5 + F_8\right) + F_1F_3 - F_7F_4 - F_2F_9
\end{aligned}
\tag{28}
$$

$$
\begin{aligned}
M_3 = {} & -F_5F_8\left(F_1 + F_3 + F_{11}\right) + F_{10}F_6\left(F_1 + F_3 + F_8\right)\\
& - \left(F_1 + F_3\right)\left(F_5 + F_8\right)F_{11}\\
& - F_1F_3\left(F_5 + F_8 + F_{11}\right)\\
& + F_7F_4\left(F_1 + F_5 + F_{11}\right)\\
& + F_2F_9\left(F_3 + F_5 + F_8\right)
\end{aligned}
\tag{29}
$$

$$
\begin{aligned}
M_4 = {} & F_3F_5F_{11}\left(F_1 + F_3\right) + F_1F_3F_{11}\left(F_5 + F_8\right)\\
& + F_1F_3F_5F_8 - F_7F_4\left(F_1F_{11} + F_1F_5 + F_5F_{11}\right)\\
& - F_6F_{10}\left(F_1F_8 + F_3F_8 + F_1F_3 - F_4F_7\right)\\
& - F_2F_9(F_5F_8 + F_3F_5 + F_3F_8 - F_4F_7
\end{aligned}
\tag{30}
$$

$$
\begin{aligned}
M_5 = {} & -F_1F_5F_{11}\left(F_3F_8 - F_4F_7\right)\\
& + F_6F_{10}\left(F_1F_3F_8 - F_1F_4F_7\right)\\
& + F_2F_9\left(F_3F_5F_8 - F_4F_5F_7\right)
\end{aligned}
\tag{31}
$$

Using the results in [33], the disease-free equilibrium E_0 is locally asymptotically stable if the Routh-Hurwitz determinants $\Delta_1, \Delta_2, \Delta_3, \Delta_4, \Delta_5$ are

$$
\begin{aligned}
\Delta_1 &= M_1,\\
\Delta_2 &= \begin{vmatrix} M_1 & 1\\ M_3 & M_2\end{vmatrix},\\
\Delta_3 &= \begin{vmatrix} M_1 & 1 & 0\\ M_3 & M_2 & M_1\\ M_5 & M_4 & M_3\end{vmatrix}\\
\Delta_4 &= \begin{vmatrix} M_1 & 1 & 0 & 0\\ M_3 & M_2 & M_1 & 1\\ M_5 & M_4 & M_3 & M_2\\ 0 & 0 & M_5 & M_4\end{vmatrix},\\
\Delta_5 &= \begin{vmatrix} M_1 & 1 & 0 & 0 & 0\\ M_3 & M_2 & M_1 & 1 & 0\\ M_5 & M_4 & M_3 & M_2 & M_1\\ 0 & 0 & M_5 & M_4 & M_3\\ 0 & 0 & 0 & 0 & M_5\end{vmatrix}
\end{aligned}
\tag{32}
$$

satisfying $\Delta_i > 0$, $i = 1, 2, 3$, $\Delta_4 = 0$, and $M_5 > 0$. These conditions are the needed sufficient conditions to verify $|\arg(\lambda)| > \alpha(t)\pi/2$ for $\alpha(t) \in [0, 1)$.

We can put system (14) in the following form:

$$
\begin{aligned}
{}^{C}D^{\alpha(t)}y_i\left(t\right) &= f\left(t, y_i\left(t\right), y_i\left(t - \tau_h\right), y_i\left(t - \tau_m\right)\right), \quad 0 \le t \le T,\\
y_i\left(t\right) &= g\left(t\right), \quad -\tau \le t \le 0,\ i = 1, 2, \ldots, 12
\end{aligned}
\tag{33}
$$

Let $y_i(t) = u_i$, $y_i(t - \tau_h) = w_i$, $y_i(t - \tau_m) = z_i$; then $f(t, u_i, w_i, z_i) \in C([0, T] \times \mathbb{R}^{12})$ is continuous with respect to t and globally Lipschitz continuous with respect to u_i, w_i, and z_i in the following norm: that is,

$$
\begin{aligned}
&\left\|f\left(t, u_1, w_1, z_1\right) - f\left(t, u_2, w_2, z_2\right)\right\|\\
&\quad \le L_1\left\|u_1 - u_2\right\| + L_2\left\|w_1 - w_2\right\| + L_3\left\|z_1 - z_2\right\|
\end{aligned}
\tag{34}
$$

for some Lipschitz constants $L_1 > 0$, $L_2 > 0$, and $L_3 > 0$, and $t \in [0, T]$, $u_1, u_2, w_1, w_2, z_1, z_2 \in \mathbb{R}^{12}$. So $f(t, u_i, w_i, z_i)$ satisfies the standard conditions for the existence and uniqueness of solutions [34].

Also, let y^* be an equilibrium point of system (33). To determine the local stability of the system (33) we can use the indirect method of Lyapunov which uses the linearization of a system [35].

The linearization of the system (33) is

$${}^{C}D^{\alpha(t)}y_i\left(t\right) = B_0u_i + B_1w_i + B_2z_i \tag{35}$$

where $B_0 = \partial f(t, u_i, w_i, z_i)/\partial u_i$, $B_1 = \partial f(t, u_i, w_i, z_i)/\partial w_i$, and $B_2 = \partial f(t, u_i, w_i, z_i)/\partial z_i$ are 12×12 matrices evaluated at

the disease-free equilibrium (essentially a Jacobian matrix for each time delay) [36].

It follows that, for each fixed t, the remainder is

$$f_1(t, u_i, w_i, z_i) = f(t, u_i, w_i, z_i) - B_0 u_i - B_1 w_i - B_2 z_i \tag{36}$$

And the remainder tends to zero as u_i, w_i, z_i tend to zero. But, the remainder may not tend to zero uniformly. So we need a stronger condition which is

$$\begin{aligned} \lim_{\|u_i\| \longrightarrow 0} \sup_{t \geq 0} \frac{\|f_1(t, u_i, w_i, z_i)\|}{\|u_i\|} &= 0, \\ \lim_{\|w_i\| \longrightarrow 0} \sup_{t \geq 0} \frac{\|f_1(t, u_i, w_i, z_i)\|}{\|w_i\|} &= 0, \\ \lim_{\|z_i\| \longrightarrow 0} \sup_{t \geq 0} \frac{\|f_1(t, u_i, w_i, z_i)\|}{\|z_i\|} &= 0 \end{aligned} \tag{37}$$

If (37) holds, then system (35) is the linearization of the system (33). Once the linearization exits, its stability defines the local stability of the original nonlinear system.

Let B_0, B_1, B_2 be bounded. If y^* is a uniformly asymptotically stable equilibrium point of system (35) then y^* is a locally uniformly asymptotically stable equilibrium point of system (33).

5. The Basic Reproduction Number R_0

In epidemiology, the basic reproduction number is defined as the number of secondary infections due to a single infection in a totally susceptible population. It is useful since it decides if or not an infectious disease can spread through a population. When $R_0 > 1$, the infection will be able to spread in a population. But if $R_0 < 1$, the infection will disappear. For $R_0 > 1$, there was, at least, one stable endemic equilibrium [32]. In some cases, the basic reproduction number is not enough to predict the spread of epidemics because bifurcation may occur.

The basic reproduction number of the model (14) is shown in [32]

$$R_0 = \max(R_m, R_{hiv}) \tag{38}$$

where R_m is the basic reproduction number of malaria model and R_{hiv} is the basic reproduction of HIV model as follows:

$$R_m = \left(\frac{\mu_h \beta_h \beta_m A_m e^{-\mu_h \tau_h} e^{-\mu_m \tau_m} c^2 (1 - bz)^2}{(a_m + \mu_m) \mu_m A_h (\sigma + \mu_h)} \right) \cdot \left(\frac{\sigma + \mu_h (1 - p)}{r_h + \mu_h + a_{h1}} + \frac{(1 - \gamma)(1 - \varepsilon) \mu_h p}{\theta_1 r_h + (1 - \theta_2) a_{h1} + \mu_h} \right) \tag{39}$$

$$R_{hiv} = \frac{\beta_H (\mu_h + \delta_H + \eta_A a_{h2})(1 - p)}{(a_{h2} + \mu_h)(\mu_h + \delta_H)} \tag{40}$$

Theorem 5 (see [43]). *If $R_0 < 1$, then the disease-free equilibrium E_0 is globally asymptotically stable in Ω.*

$\Omega = (A_h, p, \sigma, \eta_{HM}, c, A_m, \phi_2, \phi_3, b, z, r_h, a_{h1}, a_{h2}, \beta_m, \beta_h, \varepsilon_2, \delta_H, \mu_h, \gamma, \varepsilon, \mu_m, \tau, \psi, \theta_1, \theta_2, \upsilon_1, \upsilon_2, \eta_A, a_m, \tau_h, \tau_m, \zeta, \beta_H$ such that the solution of the system (14) is positive.

Proof. From the previous section according to Routh-Hurwitz conditions M_5 defined by (31) must be greater than zero so we will rewrite M_5 in terms of R_{hiv} and R_m after some manipulation as follows:

$$\begin{aligned} M_5 &= (r_h + a_{h1} + \mu_h)(\theta_1 r_h + (1 - \theta_2) a_{h1} + \mu_h) \\ &\cdot (\mu_m + a_m)(a_{h2} + \mu_h)(\mu_h + \delta_H)(1 - R_{hiv})(1 - R_m) \\ &> 0. \end{aligned} \tag{41}$$

Thus $M_5 > 0$ if $R_{hiv} < 1$ and $R_m < 1$ so the disease-free equilibrium E_0 is globally asymptotically stable in Ω. □

6. Numerical Results and Discussions

Applying the predictor-corrector method to solve model (14) with initial conditions,

$$\begin{aligned} N_h(0) &= 430, \\ S_h(0) &= 300, \\ V_h(0) &= 100, \\ I_h(0) &= 5, \\ I_{mhiv}(0) &= 5, \\ I_{hiv}(0) &= 5, \\ Y_h(0) &= 5, \\ A_{mhiv}(0) &= 5, \\ A_{hiv}(0) &= 5, \\ N_m(0) &= 450, \\ S_m(0) &= 430, \\ I_m(0) &= 20 \end{aligned} \tag{42}$$

And the values of parameters are shown in Table 1

We investigate the model behavior in two cases. Firstly, the variable fractional-order is $\alpha(t) = 0.8 - (0.01/100)t$. Secondly, the variable fractional-order is a periodic function $\alpha(t) = 0.8 - 0.01 \sin(\pi t)$.

In Figure 1, we show the effect of the parameter ν_2 which is the susceptibility to malaria of individuals showing symptoms of AIDS. It is shown that when ν_2 increases; the number of HIV infected individuals showing symptoms of AIDS decreases. Besides, when we use the variable fractional-order $\alpha(t) = 0.8 - 0.01 \sin(\pi t)$ means the memory of the model is described as a periodic function; hence the behavior of the model is also periodic. Also, when we use the variable fractional-order $\alpha(t) = 0.8 - (0.01/100)t$ means the memory

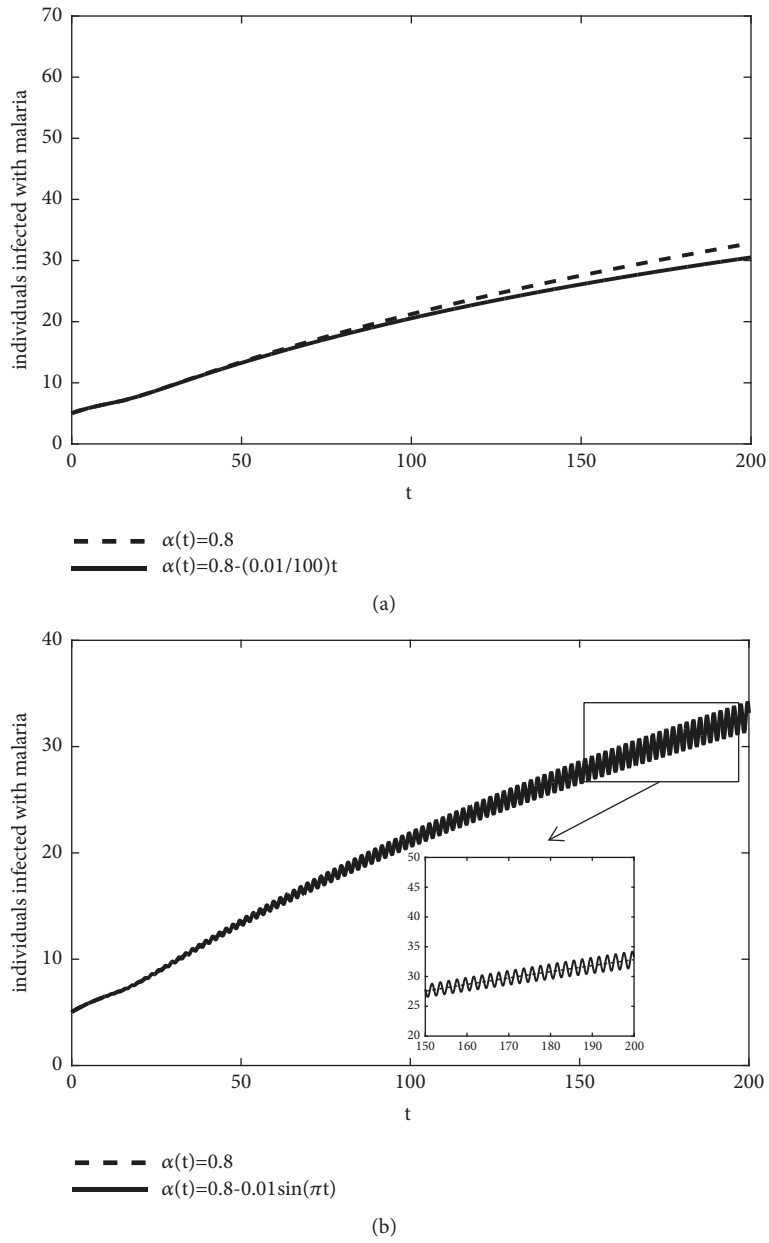

FIGURE 6: Individuals infected with malaria with $\psi = 3$. Parameters values are in Table 1 with $\beta_h = 0.05$. (a) $\alpha(t) = 0.8$ comparing with $\alpha(t) = 0.8 - (0.01/100)t$; (b) $\alpha(t) = 0.8$ comparing with $\alpha(t) = 0.8 - 0.01\sin(\pi t)$.

in the model is described by a decreasing function so the model behavior is slower with time as in Figures 2 and 3.

In Figure 4, we show the effect of the parameter ψ which is HIV mortality due to the coinfection with malaria. It is shown that when ψ increases; it leads to decreasing of new cases of malaria. Besides, when we use the variable fractional-order $\alpha(t) = 0.8 - 0.01\sin(\pi t)$ means the memory of the model is described as a periodic function; hence the behavior of the model is also periodic. Also, when we use the variable fractional-order $\alpha(t) = 0.8 - (0.01/100)t$ means the memory in the model is described by a decreasing function so the model behavior is slower with time as in Figures 5 and 6.

The presented numerical results indicate that the proposed delay variable fractional-order model is a generalization of the constant fractional-order model with a time delay which has been presented in [32].

7. Conclusion

A delay variable fractional-order model for the coinfection of HIV/AIDS and malaria which includes malaria vaccination and personal protection strategies is proposed in this paper. Also, the basic reproduction number and stability of the disease-free equilibrium have been studied. The numerical results showed the impact of changing the parameters values

Table 1: The values of parameters used in the numerical results.

Parameter	Value	Reference
A_h	0.05	[32]
p	0.4	[37]
σ	0.009	[37]
η_{HM}	1.5030	[32]
c	0.5	[38]
A_m	6	[37]
ϕ_2	0.002	[39]
ϕ_3	0.0005	[39]
b	0.3	[32]
z	0.9	[32]
r_h	0.005	[37]
a_{h1}	0.0004	[32]
a_{h2}	0.004	[32]
β_m	0.83	[40]
ε_2	0.8	[32]
δ_H	0.000913	[39]
μ_h	0.000039	[41]
γ	0.64	[37]
ε	0.86	[37]
τ_h	14	[37]
τ_m	12	[42]
μ_m	0.04	[37]
τ	1.001	[39]
ψ	1.002	[39]
θ_1	4.1	[37]
θ_2	0.06	[37]
υ_1	1.002	[39]
υ_2	1.5	[39]
η_A	1.4	[39]
a_m	0.01	[37]
ζ	1.002	[39]
β_H	0.001	[39]

such as ν_2 and ψ on the number of the infected individuals with malaria/HIV, coinfected individuals, and infectious mosquitoes as well. The variable fractional-order derivative in the proposed model is used to distinguish the effect of the memory that changes over time on the disease progression of distinct patients. In Our future work, comparisons between the numerical results and real data will be held in order to examine the numerical simulation results at different variable fractional-order $\alpha(t)$.

Data Availability

No data were used to support this study.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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