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/mm³ [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 \( \tau \) is incorporated in the variables of active humans who are infected by malaria and the coinfected humans while a discrete time delay \( \tau_m \) is incorporated in the variable of the infectious mosquitoes. After a time \( \tau \), susceptible people become infected by malaria while exposed individuals become infectious after the same time. On the other hand, mosquitoes become infectious after time \( \tau_m \). 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 \( \alpha \)). Let \( \alpha \) be a bounded and continuous function; then Riemann–Liouville fractional-order derivative of \( f(t) : [a, b] \rightarrow \mathbb{R} \) is defined as follows [27].

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

\[
R_L a^\alpha D_t^\alpha f(t) = \frac{1}{\Gamma(1-\alpha)} \frac{d}{dt} \int_a^t (t-\omega)^{-\alpha} f(\omega) d\omega,
\]

\(0 < \alpha \leq 1\)

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

\[
R_R b^\alpha D_t^\alpha f(t) = \frac{1}{\Gamma(1-\alpha)} \int_t^b (t-\omega)^{-\alpha} f(\omega) d\omega,
\]

\(0 < \alpha \leq 1\)

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

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

\[
C_L a^\alpha D_t^\alpha f(t) = \frac{1}{\Gamma(1-\alpha)} \int_a^t (t-\omega)^{-\alpha} f'(\omega) d\omega,
\]

\(0 < \alpha \leq 1\)

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

\[
C_R b^\alpha D_t^\alpha f(t) = -\frac{1}{\Gamma(1-\alpha)} \int_t^b (t-\omega)^{-\alpha} f'(\omega) d\omega,
\]

\(0 < \alpha \leq 1\)

**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 \( f(t) : [a, b] \rightarrow \mathbb{R} \) is defined as follows [27].

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

\[
R_L a^{\alpha(t)} D_t^{\alpha(t)} f(t) = \frac{1}{\Gamma(1-\alpha(t))} \frac{d}{dt} \int_a^t (t-\omega)^{-\alpha(t)} f(\omega) d\omega,
\]

\(0 < \alpha(t) \leq 1\)

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

\[
R_R b^{\alpha(t)} D_t^{\alpha(t)} f(t) = \frac{1}{\Gamma(1-\alpha(t))} \int_t^b (t-\omega)^{-\alpha(t)} f(\omega) d\omega,
\]

\(0 < \alpha(t) \leq 1\)

**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 \( f(t) : [a, b] \rightarrow \mathbb{R} \) is defined as follows [27].
Complexity

\[ (i) \text{ Left Caputo derivative of fractional-order } \alpha(t) \text{ is defined by } \]
\[ C_a^\alpha D^\alpha f(t) = \frac{1}{\Gamma(1-\alpha(t))} \int_0^t (t-\omega)^{-\alpha(t)} f'(\omega) d\omega, \quad 0 < \alpha(t) \leq 1 \]
\[ (ii) \text{ Right Caputo derivative of fractional-order } \alpha(t) \text{ is defined by } \]
\[ C_t^\alpha D^\alpha f(t) = -\frac{1}{\Gamma(1-\alpha(t))} \int_t^b (t-\omega)^{-\alpha(t)} f'(\omega) d\omega, \quad 0 < \alpha(t) \leq 1 \]

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
\[ C^\alpha D^\alpha y(t) = f\left(t, y(t), y(t-\varsigma)\right), \quad 0 \leq t \leq T; \]
\[ y(t) = g(t), \quad -\varsigma \leq t \leq 0 \]

where \( 0 < \alpha(t) \leq 1 \), \( T \in \mathbb{R}^+ \), 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 \( y_{p,n+1} \) is defined by
\[ y_{p,n+1} = y(0) + \frac{1}{\Gamma(\alpha(t_{n+1}))} \sum_{j=0}^{n} B_{j,n+1} f\left(t_j, y_j, y_{j-q}\right), \quad (10) \]
where
\[ B_{j,n+1} = \frac{I^\alpha(t_{n+1})}{\alpha(t_{n+1})} \left[ (n-j+1)^{\alpha(t_{n+1})} - (n-j)^{\alpha(t_{n+1})} \right], \quad (11) \]

The corrector approximation \( y_{n+1} \) is defined by
\[ y_{n+1} = y(0) + \frac{I^\alpha(t_{n+1})}{\Gamma(\alpha(t_{n+1})+2)} \sum_{j=0}^{n} A_{j,n+1} f\left(t_j, y_j, y_{j-q}\right), \quad (12) \]
where
\[ A_{j,n+1} = \begin{cases} 
(n \alpha(t_{n+1})+1) - [n - \alpha(t_{n+1})] (n+1)^{\alpha(t_{n+1})}, & j = 0, \\
(n-j+2)^{\alpha(t_{n+1})+1} - 2(n-j+1)^{\alpha(t_{n+1})+1} + (n-j)^{\alpha(t_{n+1})+1}, & 1 \leq j \leq n, \\
1, & j = n+1.
\end{cases} \quad (13) \]

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 \( \beta_h = 0.01 \).
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:

\[ C \frac{D^\alpha(t)}{\alpha(t)} N_h(t) \]

\[ = A_h - a_{h1} [I_h(t) + (1 - \theta_2) Y_h(t)] - \tau a_{h1} I_{mhi}(t) \]

\[ - [\tau a_{h1} + \sigma h] A_{mhi}(t) - \delta_h A_{hiv}(t) \]

\[ - \mu_h N_h(t) , \]

\[ C \frac{D^\alpha(t)}{\alpha(t)} S_h(t) \]

\[ = (1 - p) A_h - f_h(t) S_h(t) - \beta_{hiv} (t) S_h(t) \]

\[ + r_h [I_h(t) + \theta_1 Y_h(t)] + \sigma V_h(t) - \mu_h S_h(t) , \]

\[ C \frac{D^\alpha(t)}{\alpha(t)} V_h(t) \]

\[ = p A_h - f_h(t) (1 - \gamma) V_h(t) - [\sigma + \mu_h] V_h(t) , \]

\[ C \frac{D^\alpha(t)}{\alpha(t)} I_h(t) \]
Figure 3: HIV infected individuals showing symptoms of AIDS with $\gamma_2 = 100$. 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 $\alpha(t) = 0.8 - 0.01 \sin(\pi t)$. 

$$
\begin{align*}
\frac{d}{dt} I_h(t) &= f_h(t) S_h(t) (1 - \tau_h) e^{-\mu_h t} - \beta_h(t) S_h(t) I_h(t) - \left[ \rho_h + a_{h1} + \mu_h \right] I_h(t), \\
\frac{d}{dt} I_{mhiv}(t) &= v_1 f_h(t - \tau_h) I_{mhiv}(t - \tau_h) e^{-\mu_{mhiv} t} + \beta_{mhiv}(t) S_h(t) I_{mhiv}(t) - \left[ \rho_{mhiv} + a_{mh1} + \mu_{mhiv} \right] I_{mhiv}(t), \\
\frac{d}{dt} Y_h(t) &= f_h(t - \tau_h) (1 - \gamma) V_h(t - \tau_h) e^{-\mu_h t} - \left[ \theta_1 r_h + (1 - \theta_2) a_{h1} + \mu_h \right] Y_h(t), \\
\frac{d}{dt} A_{mhiv}(t) &= \xi a_{hm2} I_{mhiv}(t) + v_2 f_h(t - \tau_h) A_{mhiv}(t - \tau_h) e^{-\mu_{mhiv} t} - \left[ \mu_h + \phi_3 + \tau a_{h1} + \psi \delta H \right] A_{mhiv}(t),
\end{align*}
$$
\[ C \frac{d(t)}{A_{hiv}(t)} \]

\[ = \theta(I_{m}(t)) + \phi A_{n_{hiv}}(t) - \nu_{2} f_{h}(t) A_{hiv}(t) \]

\[ - [\mu_{h} + \delta_{H}] A_{hiv}(t), \]

\[ C \frac{d(t)}{N_{m}(t)} = A_{m} - a_{m} I_{m}(t) - \mu_{m} N_{m}(t), \]

\[ C \frac{d(t)}{S_{m}(t)} = A_{m} - f_{m}(t) S_{m}(t) - \mu_{m} S_{m}(t), \]

\[ C \frac{d(t)}{I_{m}(t)} = f_{m}(t - \tau_{m}) S_{m}(t - \tau_{m}) e^{-\mu_{m} \tau_{m}} \]

\[ - [\mu_{m} + a_{m}] I_{m}(t). \]

(14)

where the population of mosquitoes as follows:

\[ N_{m}(t) = I_{m}(t) + S_{m}(t), \]

(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
- \( I_{m_{hiv}} \) are the coinfected individuals showing no symptoms of AIDS
- \( I_{hiv} \) are the individuals asymptotically infected with HIV/AIDS

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

\[ A_{m_{hiv}} \] are the coinfected individuals showing symptoms of AIDS

Besides, all human are subject to natural death, occurring at a rate \( \mu_{h} \). 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 \( p A_{h} \) is the proportion getting in the class \( V_{h}(t) \). Susceptible individuals enter the class \( I_{h}(t) \) after some time \( \tau_{h} \). The rate of infection by malaria parasite of susceptible individuals \( f_{h}(t) \) is given by

\[ f_{h}(t) = \beta_{h} c (1 - b z) \frac{I_{m}(t)}{N_{h}(t)} \]

(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 \( \beta_{h} \). The efficacy of the preerythrocytic vaccine is given by \( 0 < \gamma \leq 1 \). Vaccinated individuals may become susceptible at a rate \( \sigma \). The rate of infection with HIV/AIDS of susceptible individuals is \( \beta_{hiv}(t) \):

\[ \beta_{hiv}(t) = \frac{\beta_{h} I_{m}(t) + \eta_{HIV} I_{m_{hiv}}(t) + \eta_{A} [A_{hiv}(t) + \eta_{HIV} A_{m_{hiv}}(t)]}{N_{h}(t)} \]

(17)

where \( \eta_{A} > 1 \) is the infectiousness of individuals in the AIDS stage of HIV infection. \( \beta_{HIV} \) is the effective contact rate for HIV infection. Infectiousness to malaria of coinfected individuals showing symptoms of AIDS is \( \eta_{HIV} > 1 \).
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 $a_{h1}$. $\epsilon_2 < 1$ models the decrease in sexual activity due to malaria disease. $\phi_2$ is the rate of recovery of the coinfected individuals showing no symptoms of AIDS from malaria. $\tau$ refers to the increased malaria mortality of individuals coinfected with HIV. $\psi$ 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 $\delta_{hiV}$. The rate of natural death of $I_{hiV}(t)$ is $\mu_h$. $\nu_1$ 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 $\phi_3$. $\phi_2$ 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 $\mu_m$. The rate of infection by the Anopheles parasite of susceptible mosquitoes $f_m(t)$ is given by

$$f_m(t) = \beta_m \epsilon (1 - b_z) \cdot I_h(t) + I_{mhiv}(t) + (1 - \epsilon) Y_h(t) + A_{mhiv}(t) \over N_h(t)$$  \hspace{1cm} (18)
They die due to the presence of the parasite in the body is a natural death, at a rate of $\mu_m$. The exposed mosquitoes are subjected to a natural death, at a rate of $\mu_e$. 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

$$C D^{\alpha(t)} N_h(t) = C D^{\alpha(t)} S_h(t) = C D^{\alpha(t)} I_h(t)$$

$$= C D^{\alpha(t)} I_{mhiv}(t)$$

\[ (E_0) \]

$$\begin{bmatrix}
-\mu_h & 0 & 0 & -\alpha_{h1} & -\beta_{m1} & 0 & -\alpha_h(1-\theta_1) & -\tau_{a_h} & -\psi S_h & -\delta_{1h} & 0 & 0 \\
0 & -\mu_n & \sigma & r_h & -\eta_{m1} & G_h & \tau_{a1} & \eta_{m2} & G_{m2} & 0 & 0 \\
0 & 0 & -\sigma & r_m & 0 & G_m & \eta_{m3} & G_{m3} & \eta_{m4} & 0 & 0 \\
0 & 0 & 0 & -\tau_{m1} & a_m & G_m & \eta_{m5} & G_{m5} & \eta_{m6} & 0 & 0 \\
0 & 0 & 0 & 0 & -\tau_{m2} & a_m & G_m & \eta_{m7} & G_{m7} & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & -\tau_{m3} & a_m & G_m & \eta_{m8} & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & -\tau_{m4} & a_m & G_m & \eta_{m9} & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & -\tau_{m5} & a_m & G_m & \eta_{m10} \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\tau_{m6} & a_m & G_m \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\tau_{m7} & a_m \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\tau_{m8} & a_m \\
\end{bmatrix}$$

where

$$G_1 = -\beta_{m1} (1-bz) \frac{\mu_h A_m}{\mu_m A_h}$$

$$G_2 = \beta_{m1} (1-bz) \frac{\mu_h A_m}{\mu_m A_h} e^{-\tau_m (\lambda+\mu_m)}$$

$$G_3 = -\beta_{m1} \left( \frac{\sigma + \mu_h}{\sigma + \mu_m} \right)$$

$$G_4 = -\beta_{m1} (1-bz) \frac{\sigma + \mu_h (1-p)}{\sigma + \mu_m}$$

$$G_5 = -\beta_{m1} (1-bz) (1-\gamma) \frac{\mu_h}{\sigma + \mu_h}$$

The eigenvalues of the Jacobian matrix are

\[ \lambda_{1,2} = -\mu_h, \]

complexity

\[ \lambda_3,4 = -\mu_m, \]

\[ \lambda_5 = -\left( \sigma + \mu_h \right), \]

\[ \lambda_6 = -\left( \xi_{a_{h2}} + \phi_2 + 2 a_{h1} + \mu_h \right), \]

\[ \lambda_7 = -\left( \mu_h + \phi_3 + 2 a_{h1} + \psi S_h \right) \]

The remaining five eigenvalues are obtained from the following matrix:

\[ M = \begin{bmatrix}
F_1 & 0 & 0 & 0 & F_2 \\
0 & F_3 & 0 & 0 & F_4 \\
0 & 0 & F_5 & 0 & F_6 \\
F_7 & 0 & F_8 & 0 & F_9 \\
0 & F_{10} & 0 & F_{11} & 0 \\
\end{bmatrix} \]
where
\[
F_1 = -(r_h + a_{i1} + \mu_h),
\]
\[
F_2 = \beta_m c (1 - b z) \frac{\sigma + \mu_h (1 - p)}{\sigma + \mu_h} e^{-\tau_m (\lambda + \mu_h)}
\]
\[
F_3 = \beta_H \frac{\sigma + \mu_h (1 - p)}{\sigma + \mu_h} - (a_{i2} + \mu_h),
\]
\[
F_4 = \beta_H \eta_h \frac{\sigma + \mu_h (1 - p)}{\sigma + \mu_h},
\]
\[
F_5 = -(\theta_1 r_h + (1 - \theta_2) a_{i1} + \mu_h),
\]
\[
F_6 = \beta_m c (1 - b z) (1 - \gamma) \frac{p(t) \mu_h}{\sigma + \mu_h} e^{-\tau_m (\lambda + \mu_h)}
\]
\[
F_7 = a_{i2},
\]
\[
F_8 = -(\mu_h + \delta_H),
\]
\[
F_9 = \beta_m c (1 - b z) \frac{\mu_h A_m}{\mu A_h} e^{-\tau_m (\lambda + \mu_m)}
\]
\[
F_{10} = \beta_m c (1 - b z) (1 - \gamma) \frac{\mu_h A_m}{\mu A_h} e^{-\tau_m (\lambda + \mu_m)},
\]
\[
F_{11} = -(\mu_m + a_m)
\]
That matrix \(M\) has the characteristic equation
\[
\lambda^5 + M_1 \lambda^4 + M_2 \lambda^3 + M_3 \lambda^2 + M_4 \lambda + M_5 = 0
\]
where
\[
M_1 = -(F_1 + F_3 + F_5 + F_8 + F_{11})
\]
\[
M_2 = F_{11} (F_1 + F_3 + F_5 + F_8) + F_3 F_8 - F_{10} F_6 + (F_1 + F_3) (F_5 + F_8) + F_1 F_3 - F_4 F_7 - F_2 F_9
\]
\[
M_3 = -F_2 F_8 (F_1 + F_3 + F_{11}) + F_{10} F_6 (F_1 + F_3 + F_8) - (F_1 + F_3) (F_5 + F_8) F_{11}
- F_1 F_6 (F_5 + F_8 + F_{11}) + F_2 F_4 (F_1 + F_5 + F_{11}) + F_4 F_2 (F_3 + F_5 + F_8)
\]
\[
M_4 = F_3 F_8 F_{11} (F_1 + F_3) + F_1 F_3 F_{11} (F_5 + F_8)
+ F_1 F_2 F_6 - F_7 F_4 (F_1 F_3 + F_5 F_8)
\]
\[
M_5 = -F_1 F_5 F_{11} (F_5 F_8 - F_4 F_7)
+ F_6 F_{10} (F_1 F_3 F_8 - F_4 F_7)
+ F_2 F_5 (F_3 F_5 F_8 - F_4 F_7)
\]

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
\[
\Delta_1 = M_1,
\]
\[
\Delta_2 = \begin{vmatrix} M_1 & 1 \\ M_3 & M_2 \end{vmatrix},
\]
\[
\Delta_3 = \begin{vmatrix} M_1 & 1 & 0 \\ M_3 & M_2 & M_1 \\ M_5 & M_4 & M_3 \end{vmatrix},
\]
\[
\Delta_4 = \begin{vmatrix} M_1 & 1 & 0 & 0 \\ M_3 & M_2 & M_1 & 1 \\ 0 & 0 & M_5 & M_4 \end{vmatrix},
\]
\[
\Delta_5 = \begin{vmatrix} M_1 & 1 & 0 & 0 & 0 \\ M_3 & M_2 & M_1 & 1 & 0 \\ 0 & 0 & M_5 & M_4 & M_3 \\ 0 & 0 & 0 & M_5 & M_4 \end{vmatrix}
\]

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

We can put system (14) in the following form:
\[
C \frac{D^\alpha(t)}{C^\alpha(t)} y_1(t) = f(t, y_1(t), y_1(t - \tau), y_1(t - \tau_m)),
\]
\[
0 \leq t \leq T.
\]

Let \(y_1(t) = u_i, y_i(t - \tau) = w_i, y_i(t - \tau_m) = z_i;\) 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,
\[
\|f(t, u_1, w_1, z_1) - f(t, u_2, w_2, z_2)\| \leq L_1 \|u_1 - u_2\| + L_2 \|w_1 - w_2\| + L_3 \|z_1 - z_2\|
\]

for some Lipschitz constants \(L_1 > 0, L_2 > 0, \) and \(L_3 > 0,\) and \(t \in [0, T], u_i, u_1, w_1, w_2, z_1, z_i \in \mathbb{R}^{12}.\) 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
\[
C \frac{D^\alpha(t)}{C^\alpha(t)} y_1(t) = B_0 u_i + B_1 w_i + B_2 z_i
\]
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 \(12 \times 12\) 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

\[
 f_1 (t, u_i, w_i, z_i) = f (t, u_i, w_i, z_i) - B_i u_i - B_i w_i - B_i z_i
\]

(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

\[
 \lim_{t \to \infty} \sup_{t \in [0, T]} \left\| f_1 (t, u_i, w_i, z_i) \right\| = 0,
\]

(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 \( R_0, B_1, B_2 \) 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]

\[
 R_0 = \max (R_m, R_{mIV})
\]

(38)

where \( R_m \) is the basic reproduction number of malaria model and \( R_{mIV} \) is the basic reproduction of HIV model as follows:

\[
 R_m = \frac{\mu_0 \beta_0 A_m e^{\mu_0 t} e^{-\mu_m t} \sigma^2 (1 - e^{-\mu_m t})^2}{\mu_m + \mu_h} A_h (\sigma + \mu_h)
\]

(39)

\[
 R_{mIV} = \frac{\beta_{mIV} (\mu_h + \delta_{mIV} + \eta_4 h) (1 - p)}{\mu_h + \delta_{mIV}}
\]

(40)

\[
 \Omega = (A_m, \rho, \sigma, \eta_{HIV}, c, A_m, \phi_3, \phi_3, b, z, r_h, a_{h1}, a_{h2}, \beta_{m}, \beta_{h}, \epsilon_2, \delta_{m}, \mu_{m}, \gamma, e, \mu_m, \tau, \psi, \theta_1, \theta_2, \psi_1, \psi_2, \eta_A, \alpha_m, \gamma_h, \tau_m, \zeta, \beta_{HIV})
\]

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 \( R_{mIV} \) and \( R_m \) after some manipulation as follows:

\[
 M_5 = (r_h + a_{h1} + \mu_h) (\theta_1 + (1 - \theta_2) a_{h1} + \mu_h)
\]

\[
 \cdot (\mu_m + a_m) (\alpha_{h2} + \mu_h) (\mu_h + \delta_{mIV}) (1 - R_{mIV}) (1 - R_m)
\]

(41)

\[
 > 0
\]

Thus \( M_5 > 0 \) if \( R_{mIV} < 1 \) and \( R_m < 1 \) so the disease-free equilibrium \( E_0 \) is globally asymptotically stable in \( \Omega \).

6. Numerical Results and Discussions

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

\[
 N_h (0) = 430,
\]

\[
 S_h (0) = 300,
\]

\[
 V_h (0) = 100,
\]

\[
 I_h (0) = 5,
\]

\[
 I_{mIV} (0) = 5,
\]

\[
 I_{hIV} (0) = 5,
\]

\[
 Y_h (0) = 5,
\]

\[
 A_{mIV} (0) = 5,
\]

\[
 A_{hIV} (0) = 5,
\]

\[
 N_m (0) = 450,
\]

\[
 S_m (0) = 430,
\]

\[
 I_m (0) = 20
\]

(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 \( \nu_2 \) which is the susceptibility to malaria of individuals showing symptoms of AIDS. It is shown that when \( \nu_2 \) 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
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 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 \( \psi \) which is HIV mortality due to the coinfection with malaria. It is shown that when \( \psi \) 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.

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 \( \alpha(t) = 0.8 - 0.01 \sin(\pi t) \).
Table 1: The values of parameters used in the numerical results.

<table>
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<tr>
<th>Parameter</th>
<th>Value</th>
<th>Reference</th>
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<td>[32]</td>
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<td>$p$</td>
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<td>$\sigma$</td>
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<tr>
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<td>[32]</td>
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<tr>
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<tr>
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<td>[39]</td>
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</table>

such as $\nu_2$ and $\psi$ 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 $a(t)$.

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.

References


