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Rahim Ud Din

King Mongkut's Institute of Technology Ladkrabang

Atta Ullah

King Mongkut's Institute of Technology Ladkrabang

Puntani Pongsumpun

`puntani.po@kmitl.ac.th`

King Mongkut's Institute of Technology Ladkrabang

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A Fractional Order Dengue and Malaria Co-infection Model with Hyers-Ulam Stability, Sensitivity Analysis, and Optimal Control Strategies in Thailand

Rahim Ud Din^a, Atta Ullah^a, Puntani Pongsumpun^{a*}

^a*Department of Mathematics, School of Science, King Mongkut's Institute of Technology Ladkrabang, Bangkok, Thailand,*

Abstract

Dengue fever and malaria remain major mosquito-borne diseases that continue to threaten public health, particularly in tropical countries such as Thailand, where co-infection may occur. This paper presents a novel Atangana-Baleanu fractional-order mathematical model to investigate the transmission dynamics of dengue–malaria co-infection in coupled human–mosquito populations. The proposed model extends the classical framework by incorporating exposed, infected, co-infected, hospitalized, and recovered human compartments together with mosquito dynamics. Fundamental mathematical properties, including positivity, boundedness, existence and uniqueness of solutions, disease-free equilibrium, and the basic reproduction number, are established. Hyers–Ulam stability and sensitivity analysis are performed to examine the robustness of the model and identify the most influential transmission parameters. Furthermore, four optimal control strategies are introduced to minimize disease transmission and intervention costs. Numerical simulations using the Modified Euler Method illustrate the influence of the fractional-order parameter and demonstrate that mosquito vector control is the most effective strategy for reducing co-infection. The proposed model provides valuable insights for understanding dengue–malaria co-infection dynamics and supports the development of effective disease control policies in Thailand.

Keywords: Co-infection; Fractional modeling; Sensitivity analysis; Optimal control; Numerical simulations.

1. Introduction

One serious vector-borne illness is dengue fever. According to numerous publications [1, 2, 3], the infectious dengue virus is transmitted by female Aedes mosquito bites. The dengue virus enters the mosquito's bloodstream after it feeds on an infectious. Following infection, the mosquito acts as a vector, transmitting the dengue virus to a susceptible individual during subsequent blood feeding [4, 5]. According to a 2012 survey, people in over 100 nations are at risk of getting dengue fever [6, 7]. In recent decade, multiple dengue outbreaks in different nations, which have seriously damaged public health [8, 9, 10, 11]. According to [12], dengue fever might result in the rapid reduction in platelet count and a breakdown of white

Email addresses: 67056116@kmitl.ac.th (Rahim Ud Din^a), 68056118@kmitl.ac.th (Atta Ullah^a), puntani.po@kmitl.ac.th (Puntani Pongsumpun^{a*})

blood cells. High temperature, vomiting, stomach problem or discomfort, frequent vomiting, diagnostic fluid accumulation, Mucosal blood loss, lethargy, and enlarged liver are further signs of dengue fever [13]. Dengue fever has been detected over the entire country of Thailand. Every year, it emerges on a regular basis, peaking in June and August during the wet season. The most crucial element in the spread of the dengue virus is the quantity of rainfall [14].

According to the World Health Organization (WHO) [15], female anopheles mosquitoes carry the plasmodium parasite, which causes malaria. Compared to other mosquito carrying diseases like dengue, malaria has a longer and more hazardous history. A substantial rise in malaria incidence was observed, increasing from 2.16×10^9 cases in 2016 to 3.20×10^7 in 2017 [16, 17], with an estimated 2.41×10^7 cases worldwide and an estimated 627,000 malaria fatalities in 2020 [18]. In 2020, the African region bore the highest burden of malaria, representing 95% of cases and 96% of associated deaths globally [18, 19]. Children below five years old represented 80% of all malaria-related deaths in the region. The illness has reappeared in areas where control measures were previously successful, despite decades of worldwide eradication and control efforts [19]. Fever, perspiration, chills or shivering, nausea, headaches, diarrhea, and muscle aches are typical signs of malaria. Malaria has a latent stage that lasts seven to fourteen days [15, 16, 17, 18, 19, 20]. [21] The purpose of the study was to use statistical modeling to describe the prevalence of malaria in Thai nationals in seven different provinces in the upper region of southern Thailand. Planning, prevention, and management of malaria in the higher region of southern Thailand can benefit from these findings.

It is feasible for a person to contract simultaneously Malaria and Dengue at the same time, since both diseases are vector-borne by similar mosquito species and share overlapping initial symptoms [22, 23, 24, 25, 26, 28]. With respect to transmission and effects of human health, dengue fever and malaria are similar. Both diseases are vector-borne, meaning that mosquito bites can transmit them to humans. Certain species of mosquitoes carry the parasites that cause malaria and the viruses that cause dengue illness. Additionally, these diseases have affected societies worldwide throughout human history. Both diseases can result in high temperature, fatigue, and physical discomfort, which can be extremely painful for those who contract them. Furthermore, these disorders can have major repercussions and, in severe cases, can result in death if they are misdiagnosed or improperly treated. Given the many parallels between these two illnesses, it is critical to prioritize the execution of thorough preventative and preventative strategies in order to effectively stop the transmission of both infections. In 2016, dengue-malaria co-infection was reported across several countries, with the highest number of cases in India (26), followed by Pakistan (4), and isolated cases in Cambodia, Malaysia, Bangladesh, Japan, and East Timor [22], also in Thailand in 2019.

A very reliable and useful method for comprehending biological and physical issues and patterns is mathematical modeling [29, 30, 31, 32]. In study of mathematical and biological models have been essential for understand the dynamics of infection flow in order to analyze and control diseases [33, 34, 35]. Researchers can model multiple situations, investigate different measures, and direct public policy strategies making by fusing mathematical tools with epidemiological knowledge [36, 37, 38]. Kermack and McKendrick [39], laid

the foundation of modern epidemiological modeling by introducing the deterministic SIR model in 1927, which was later extended to the SEIR framework. For a variety of illnesses, including coronavirus, tuberculosis, whooping cough, and cancer, different mathematical systems have been put forth in the past over time [40, 41, 42].

The established Atangana Balean derivatives in the Caputo definition was incorporated into modified **SEIR** host community and **SI** rodent population non integral order equation for dengue transmission by the authors in [6]. The authors present an optimal control strategies with treatment as the control parameter and added two more compartments to the host population: the isolated visitors's compartment and the treatment compartment. In a similar vein, the authors of [7] demonstrated the importance of the suggested model and control techniques by proposing an *SEIHR* dengue infection system with optimal prevention for real data of world. On the bases of the Caputo non-integral order calculus was presented by the authors in [8]. This concept divides the entire human population into four groups: those who are susceptible, those who are infected (both carriers and symptomatic), those who are partially immune, and the recovered class. Similarly, susceptible and infected populations comprise the whole vector population. The authors used Caputo fractional derivatives to illustrate and validate the essential features of the suggested epidemic model. They also emphasized the advantages of fractional derivatives, namely the inclusion of memory effects.

There are also a number of mathematical models for malaria, such as those described in [16, 17, 18], each of which is predicated on a particular set of assumptions and control techniques. Mathematical formulation and two preventative methods human safety measure and mosquito safty were the main topics of [16]. Both tactics have a significant impact on the prevention of disease, but self-precaution is the more effective method. When at least 40% of vulnerable people adopt the necessary safeguards, the model reaches a disease-free state. In a similar vein, the authors of [18] described an efficient control strategy known as awareness-based intervention using media for managing malaria. Their mathematical model incorporates a dynamic awareness variable and assesses treatments (such as mosquito nets). This approach links recovery to awareness-dependent therapies by classifying individuals based on awareness, taking into account transitions from awareness to unawareness. In an effort to reduce the cost of controlling malaria, the model includes dependent-time preventative functions for electronic media ads, pesticides, and therapy. The authors of [20] used partial differential equations to study a new mathematical system for malaria by separating the host and vector populations into discrete compartments. Using both traditional and impulsive techniques, they examined the efficacy of spraying in the prevention of malaria. According to their research, uniform spraying produced outcomes that were similar to those of a non-spatial model.

There aren't many articles in the current studies that address dengue and malaria co-infection. One noteworthy example can be found in [22], which presents a comprehensive investigation of dengue-malaria infection. The clinical signs and diagnostic difficulties related to the co-infection were thoroughly examined in this study. In a similar vein, the authors of another study clarified the specifics of the first dengue-malaria

infection case reported in Nepal [25]. By providing in-depth case investigations, both of these articles advance our knowledge of dengue-malaria disease. In [28], a deterministic model of non-linear equations for the infection of the dengue malaria disease in form of the model. A classical system of equation for dengue and malaria disease was put out by the authors. It study with two sub models, dengue sub model and malaria sub model, and then integrates them into a co-infection model of Malaria and dengue. The goal of the suggested mathematical model is to give a succinct overview of the illness and to analyze the model's stability using analytical and numerical techniques. Although the paper covers a relatively small portion of the topic and more work, such as sensitivity and control analysis, can be done, it is fundamental and thorough for the motivation of epidemiologists. However, as of right now, no study offers thorough understanding, in-depth research, and useful control methods specifically for dengue and malaria co-infection.

The novelty of this study lies in the development of a comprehensive dengue-malaria co-infection model that explicitly incorporates mosquito population dynamics, co-infected individuals, hospitalization, and recovery compartments using Thailand epidemiological data. The proposed model combines rigorous mathematical analysis, including positivity, boundedness, equilibrium analysis, basic reproduction number computation, and the relatively unexplored Hyers-Ulam stability analysis, with sensitivity analysis and multiple optimal control strategies. Furthermore, the model evaluates the comparative effectiveness of different intervention policies and demonstrates that vector reduction through mosquito spraying is the most cost-effective strategy. This integrated theoretical and computational framework provides a more realistic and policy-oriented approach for understanding and controlling dengue-malaria co-infection than existing models. The foundation of this research is, section 2 have the brief presentation of model formulation, section 3 has the basic characteristic of the co-infection model, section 4 present the fundamental properties, while section 5 have Hyers-Ulam stability, and sensitivity of the model is present in section 6. Optimal is study in section 7, numerical analysis in section 8, while the last section is conclude with conclusion.

2. Model formulation

The mathematical model (1) describes the dynamics of Dengue and malaria co-transmission in a coupled human-vector densities. The total density is divided into human and mosquito populations to capture the biological interaction between hosts and vectors responsible for disease transmission. The human density is structured into eight epidemiological compartments representing different stages of disease and recovery. The susceptible density of human, denoted by $\mathcal{S}_h(t)$, has the individuals which are healthy but vulnerable to contracting either dengue or malaria through contact with infected individuals or infected vectors. Once susceptible population are exposed to malaria infection, they go into the malaria exposed compartment $\mathcal{E}_m(t)$, which represents individuals who have been infected but are still in the incubation period and are not yet able of carrying the infection. After the incubation stage, these individuals progress to the infectious malaria compartment $\mathcal{I}_m(t)$ at the progression rate σ_m . Similarly, susceptible individuals who acquire dengue infection move into the exposed dengue compartment $\mathcal{E}_d(t)$, which represents individuals who carry the dengue virus

but remain in the incubation period. These individuals then progress to the infectious dengue compartment $\mathcal{I}_d(t)$ at the rate σ_d . The model further incorporates the possibility of simultaneous infection through the co-infected compartment $\mathcal{I}_c(t)$, which represents individuals infected with both dengue and malaria. The transition into this class is governed by the interaction parameter η , which reflects the increased likelihood that individuals infected with one disease may acquire the second infection. Individuals in the co-infection class may experience more severe health conditions and therefore may be transferred to the hospitalized class $\mathcal{H}_h(t)$ at the rate δ . This compartment represents individuals receiving medical treatment or isolation in order to reduce further disease transmission. Recovered individuals are moved into the recovered class $\mathcal{R}_h(t)$, which accumulates individuals who recover from malaria, dengue, co-infection, or hospitalize treatment. Recovery occurs at rates ν_m , ν_d , ν_{md} , and ν_q , respectively. Natural mortality occurs in all human compartments at rate μ_h , while the new birth of new susceptible density into the rate β_h . These demographic parameters maintain the population balance and reflect natural birth and death processes in the human population. In addition to the human community, the model also incorporates the mosquito density, which serves as the primary vector carrying for disease flow. The mosquito density is divided into susceptible $\mathcal{S}_a(t)$ density compartment and infected $\mathcal{I}_a(t)$ density compartment. Susceptible individuals become infected after effective interaction with infected individuals at transmission rate k_a . The new birth of mosquitoes is represent by β_a , while natural mosquito mortality occurs at rate μ_a . The parameters k_{ma} , k_{mh} , k_{dh} , and k_{rh} represent the active flow rates between susceptible individuals and infected human or vectors, determining the speed at which malaria and dengue infections spread within the population. The parameters σ_m and σ_d represent the progression rates from the exposed classes to the infected classes for malaria and dengue, respectively, reflecting the biological incubation periods of the diseases. The recovery parameters ν_m , ν_d , ν_{md} , and ν_q describe the rates at which infected individuals recover from malaria, dengue, co-infection, and hospitalize treatment. The parameter δ represents the rate at which co-infected are transferred into hospitalized, capturing the effect of healthcare intervention strategies. Furthermore, the interaction parameter η plays a important role in the dynamics of the model, The dynamics of all above dissuasion is transfer into mathematical formulation as below.

$$\begin{aligned}
\text{Human population:} \quad & \begin{cases} {}^{ABC}D_t^\zeta \mathcal{S}_h = \beta_h - k_{ma}\mathcal{S}_h\mathcal{I}_a - k_{mh}\mathcal{S}_h\mathcal{I}_m - k_{dh}\mathcal{S}_h\mathcal{I}_d - \mu_h\mathcal{S}_h, \\ {}^{ABC}D_t^\zeta \mathcal{E}_m = k_{ma}\mathcal{S}_h\mathcal{I}_a + k_{mh}\mathcal{S}_h\mathcal{I}_m - (\sigma_m + \mu_h)\mathcal{E}_m, \\ {}^{ABC}D_t^\zeta \mathcal{I}_m = \sigma_m\mathcal{E}_m - (\eta\mathcal{I}_d + \nu_m + \mu_h)\mathcal{I}_m, \\ {}^{ABC}D_t^\zeta \mathcal{E}_d = k_{rh}\mathcal{S}_h\mathcal{I}_d - (\sigma_d + \mu_h)\mathcal{E}_d, \\ {}^{ABC}D_t^\zeta \mathcal{I}_d = \sigma_d\mathcal{E}_d - (\eta\mathcal{I}_m + \nu_d + \mu_h)\mathcal{I}_d, \\ {}^{ABC}D_t^\zeta \mathcal{I}_c = \eta\mathcal{I}_m\mathcal{I}_d - (\nu_{md} + \delta + \mu_h)\mathcal{I}_c, \\ {}^{ABC}D_t^\zeta \mathcal{H} = \delta\mathcal{I}_c - (\nu_q + \mu_h)\mathcal{H}, \\ {}^{ABC}D_t^\zeta \mathcal{R}_h = \nu_m\mathcal{I}_m + \nu_d\mathcal{I}_d + \nu_{mv}\mathcal{I}_c + \nu_q\mathcal{H} - \mu_h\mathcal{R}_h. \end{cases} \\
\text{Mosquito population:} \quad & \begin{cases} {}^{ABC}D_t^\zeta \mathcal{S}_a = \beta_a - k_a\mathcal{S}_a\mathcal{I}_d - \mu_a\mathcal{S}_a, \\ {}^{ABC}D_t^\zeta \mathcal{I}_a = k_a\mathcal{S}_a\mathcal{I}_d - \mu_a\mathcal{I}_a. \end{cases}
\end{aligned} \tag{1}$$

With initial condition $(\mathcal{S}_h, \mathcal{E}_m, \mathcal{E}_d, \mathcal{I}_m, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a) \geq 0$.

The flow chart of the above equations is present in the follow figure (Fig.1)

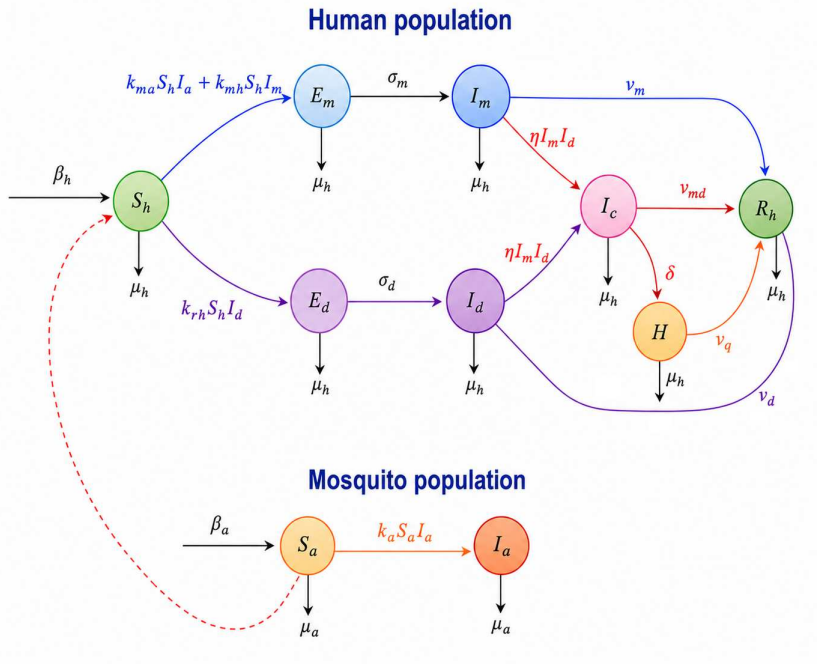


Figure 1: Compartmental flow of the co-infection model (1)

3. Preliminaries and Basic Characteristic

In this section, we introduce the fundamental concepts, definitions, and analytical tools required for the formulation and analysis of the proposed Atangana-Baleanu fractional-order co-infection model. Furthermore, we investigate some essential qualitative properties of the model, including the existence of unique

solutions, the non-negativity, and the boundedness of the solution trajectories within the biologically feasible region.

Definition 3.1. [27] Suppose that $f \in C^1([0, T])$ and let $0 < \zeta < 1$. The Atangana-Baleanu fractional derivative in the Caputo sense (ABC derivative) is defined as

$${}_0^{ABC}D_t^\zeta f(t) = \frac{B(\zeta)}{1-\zeta} \int_0^t f'(\tau) E_\zeta\left(-\frac{\zeta}{1-\zeta}(t-\tau)^\zeta\right) d\tau,$$

where $E_\zeta(\cdot)$ denotes the Mittag-Leffler function of order ζ , and $B(\zeta)$ represents the normalization function holds the conditions $B(0) = B(1) = 1$.

Definition 3.2. [27] The Atangana-Baleanu fractional integral corresponding to the ABC derivative is expressed as

$${}_0^{AB}I_t^\zeta f(t) = \frac{1-\zeta}{B(\zeta)} f(t) + \frac{\zeta}{B(\zeta)\Gamma(\zeta)} \int_0^t (t-\tau)^{\zeta-1} f(\tau) d\tau,$$

where $\Gamma(\cdot)$ denotes the Gamma function.

Lemma 3.3. [27] Consider the fractional initial value problem

$${}_0^{ABC}D_t^\zeta \phi(t) = \kappa(t),$$

subject to the initial condition

$$\phi(0) = \phi_0.$$

If the forcing function satisfies $\kappa(0) = 0$, then the corresponding solution is given by

$$\phi(t) = \phi_0 + \frac{1-\zeta}{\mathcal{N}(\zeta)} \kappa(t) + \frac{\zeta}{\mathcal{N}(\zeta)\Gamma(\zeta)} \int_0^t (t-\eta)^{\zeta-1} \kappa(\eta) d\eta.$$

Furthermore, let $\mathbb{X} = AC(\mathcal{J})$ denote the Banach space of absolutely continuous functions defined on $\mathcal{J}$, equipped with the supremum norm

$$\|\phi\|_{\mathbb{X}} = \max_{t \in \mathcal{J}} |\phi(t)|.$$

3.1. Positivity, boundedness and invariant region

Let

$$\Omega = \left\{ (\mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a) \in \mathbb{R}_+^{10} \right\}$$

be the feasible region of the model. Assume that all initial conditions are non-negative. Define the total human density as

$$\mathcal{N}_h(t) = \mathcal{S}_h + \mathcal{E}_m + \mathcal{I}_m + \mathcal{E}_d + \mathcal{I}_d + \mathcal{I}_c + \mathcal{H}_h + \mathcal{R}_h.$$

Applying the ABC fractional derivative and adding all human equations of model (1), which follow

$${}_0^{ABC}D_t^\zeta \mathcal{N}_h = \beta_h - \mu_h \mathcal{N}_h - \delta \mathcal{I}_c.$$

Since $\delta \mathcal{I}_c \geq 0$, we obtain

$${}^{ABC}D_t^\zeta \mathcal{N}_h \leq \beta_h - \mu_h \mathcal{N}_h.$$

Using the comparison principle for ABC fractional derivatives, it follows that

$$\mathcal{N}_h(t) \leq \frac{\beta_h}{\mu_h}, \quad \forall t \geq 0.$$

Hence, the human population remains bounded by

$$0 \leq \mathcal{N}_h(t) \leq \frac{\beta_h}{\mu_h}.$$

Similarly, define the total mosquito population

$$\mathcal{N}_a(t) = \mathcal{S}_a + \mathcal{I}_a.$$

Summing the mosquito equations gives

$${}^{ABC}D_t^\zeta \mathcal{N}_a = \beta_a - \mu_a \mathcal{N}_a.$$

Thus,

$$\mathcal{N}_a(t) \leq \frac{\beta_a}{\mu_a}.$$

Therefore, the biologically feasible region is

$$\Omega = \left\{ \mathcal{N}_h \leq \frac{\beta_h}{\mu_h}, \mathcal{N}_a \leq \frac{\beta_a}{\mu_a} \right\}.$$

Hence, Ω is positively invariant.

To prove positivity, we evaluate the first equation of system (1):

$${}^{ABC}D_t^\zeta \mathcal{S}_h \geq -\mu_h \mathcal{S}_h.$$

Using the generalized comparison lemma for ABC fractional derivatives, give us

$$\mathcal{S}_h(t) \geq 0, \quad \forall t \geq 0.$$

Similarly, the remaining equations of the co-infection model are positive. Therefore,

$$\Omega = \left\{ (\mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a) \in \mathbb{R}_+^{10} \mid \mathcal{N}_h \leq \frac{\beta_h}{\mu_h}, \mathcal{N}_a \leq \frac{\beta_a}{\mu_a} \right\}$$

Hence, all state compartment parameters remain positive for $t \geq 0$.

Therefore, for positive initial conditions, all solutions of co-infection model (1) remain bounded and positive for all $t \geq 0$. Hence, the feasible region Ω is non-negatively invariant and the system is mathematically and epidemiologically well posed.

3.2. Existence and uniqueness of solution

Establishing the uniqueness and existenceness of solutions is crucial for validating the well-posedness of fractional-order models driven by the Atangana-Baleanu derivative. Owing to its inherent nonlocal and nonsingular characteristics, this derivative presents analytical challenges that differ significantly from those encountered with classical operators, thereby requiring more refined mathematical techniques. In addressing these complexities, the analysis leverages fixed-point theory and Lipschitz-type conditions specifically adapted to the ABC framework, ensuring that a unique solution can be identified within an appropriate function space. This section presents the necessary theoretical foundations and assumptions that underpin the uniqueness and existenceness of the system under consideration.

$${}^{ABC}D_t^\zeta \mathcal{X}(t) = \mathcal{F}(t, \mathcal{X}(t)), \quad 0 < \zeta \leq 1. \quad (2)$$

Within this framework, the state variables are given by

$$\mathcal{X}(t) = (\mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a)^T,$$

where $\mathcal{X}(t)$ is a continuous-valued vector function. For clarity, the vector function $\mathcal{F} : \mathbb{R}^{10} \rightarrow \mathbb{R}^{10}$ can be explicitly described as:

$$\mathcal{F}(t, \mathcal{X}) = (F_1, F_2, \dots, F_{10})^T,$$

where $F_i, i = 1, 2, \dots, 10$, correspond to the right-hand sides of system (1).

The initial conditions are appropriately defined as

$$\mathcal{X}(0) = \mathcal{X}_0 \in \mathbb{R}_+^{10}.$$

Moreover, the function $\mathcal{F}(t, \mathcal{X})$ satisfies the Lipschitz condition described as follows:

$$\|\mathcal{F}(t, \mathcal{X}_1) - \mathcal{F}(t, \mathcal{X}_2)\| \leq L \|\mathcal{X}_1 - \mathcal{X}_2\|, \quad \forall \mathcal{X}_1, \mathcal{X}_2 \in \mathbb{R}^{10}, \quad (3)$$

where $L > 0$ is a Lipschitz constant.

Next, the uniqueness and existence of system solutions (1) are established as follows.

Theorem 3.4. *A unique solution to the co-infection fractional system (1) is guaranteed provided that the following condition is satisfied*

$$\frac{(1-\zeta)L}{\mathcal{B}(\zeta)} + \frac{\zeta L T^\zeta}{\mathcal{B}(\zeta)\Gamma(\zeta+1)} < 1, \quad (4)$$

where L is the Lipschitz constant associated with the nonlinear function $\mathcal{F}$, $T > 0$, and $\mathcal{B}(\zeta)$ denotes the normalization constant of the Atangana–Baleanu fractional operator.

Proof. To derive the desired result, we utilize Definition 3 of the Atangana–Baleanu fractional integral on system (2), yielding the following outcome:

$$\mathcal{X}(t) = \mathcal{X}(0) + \frac{1-\zeta}{\mathcal{B}(\zeta)} \mathcal{F}(t, \mathcal{X}(t)) + \frac{\zeta}{\mathcal{B}(\zeta)\Gamma(\zeta)} \int_0^t (t-\tau)^{\zeta-1} \mathcal{F}(\tau, \mathcal{X}(\tau)) d\tau.$$

Let $\mathcal{J} = [0, T]$ denote the interval of interest, and define the operator $\mathcal{T} : \mathcal{C}(\mathcal{J}, \mathbb{R}^{10}) \rightarrow \mathcal{C}(\mathcal{J}, \mathbb{R}^{10})$ as follows:

$$(\mathcal{T}\mathcal{X})(t) = \mathcal{X}(0) + \frac{1-\zeta}{\mathcal{B}(\zeta)}\mathcal{F}(t, \mathcal{X}(t)) + \frac{\zeta}{\mathcal{B}(\zeta)\Gamma(\zeta)} \int_0^t (t-\tau)^{\zeta-1} \mathcal{F}(\tau, \mathcal{X}(\tau)) d\tau. \quad (5)$$

Accordingly, Equation (5) can be expressed as follows:

$$\mathcal{X}(t) = (\mathcal{T}\mathcal{X})(t).$$

The supremum norm over the set $\mathcal{C}(\mathcal{J}, \mathbb{R}^{10})$ is denoted by $\|\cdot\|_\infty$ and is defined as follows:

$$\|\mathcal{X}\|_\infty = \sup_{t \in \mathcal{J}} \|\mathcal{X}(t)\|. \quad (6)$$

It can be carefully observed that $\mathcal{C}(\mathcal{J}, \mathbb{R}^{10})$ constitutes a Banach space when equipped with the norm $\|\cdot\|_\infty$. Furthermore, it is clear that:

$$\|(\mathcal{T}\mathcal{X}_1)(t) - (\mathcal{T}\mathcal{X}_2)(t)\| \leq \frac{1-\zeta}{\mathcal{B}(\zeta)} \|\mathcal{F}(t, \mathcal{X}_1) - \mathcal{F}(t, \mathcal{X}_2)\| + \frac{\zeta}{\mathcal{B}(\zeta)\Gamma(\zeta)} \int_0^t (t-\tau)^{\zeta-1} \|\mathcal{F}(\tau, \mathcal{X}_1) - \mathcal{F}(\tau, \mathcal{X}_2)\| d\tau. \quad (7)$$

The functions $\mathcal{X}_1$ and $\mathcal{X}_2$ belong to $\mathcal{C}(\mathcal{J}, \mathbb{R}^{10})$, such that:

$$\|\mathcal{F}(t, \mathcal{X}_1) - \mathcal{F}(t, \mathcal{X}_2)\| \leq L \|\mathcal{X}_1 - \mathcal{X}_2\|. \quad (8)$$

By applying the definition of $\mathcal{T}$ provided in (7), the following result is obtained:

$$\|(\mathcal{T}\mathcal{X}_1) - (\mathcal{T}\mathcal{X}_2)\|_\infty \leq \left(\frac{1-\zeta}{\mathcal{B}(\zeta)} + \frac{\zeta T^\zeta}{\mathcal{B}(\zeta)\Gamma(\zeta+1)} \right) L \|\mathcal{X}_1 - \mathcal{X}_2\|_\infty. \quad (9)$$

Moreover, by applying the Lipschitz condition (8) together with the result from (9), we arrive at the following conclusion:

$$\|\mathcal{T}\mathcal{X}_1 - \mathcal{T}\mathcal{X}_2\|_\infty \leq \kappa \|\mathcal{X}_1 - \mathcal{X}_2\|_\infty,$$

where

$$\kappa = \left(\frac{1-\zeta}{\mathcal{B}(\zeta)} + \frac{\zeta T^\zeta}{\mathcal{B}(\zeta)\Gamma(\zeta+1)} \right) L.$$

Consequently, the following result is obtained, $0 < \kappa < 1$. It is evident that if condition (6) is satisfied, then $\mathcal{T}$ acts as a contraction mapping. Consequently, the model (1) have a unique solution. $\square$

4. Mathematical Analysis of model

This section presents the theoretical analysis of the proposed co-infection model. Specifically, we derive the co-infection-free and endemic equilibrium points, compute the basic reproduction number, and perform sensitivity analysis to identify the key parameters influencing disease transmission dynamics.

4.1. Disease free equilibrium points (DFEP)

Accordingly, the vector of infected human compartments is defined as

$$\mathbf{x} = (\mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c)^T.$$

The (DFEP) associated to the co-infection model (1) is express by

$$\mathcal{E}_0 = \left(\frac{\beta_h}{\mu_h}, 0, 0, 0, 0, 0, 0, \frac{\beta_a}{\mu_a}, 0 \right). \quad (10)$$

Hence,

$$\mathcal{I}_h^0 = \frac{\beta_h}{\mu_h}, \quad \mathcal{I}_a^0 = \frac{\beta_a}{\mu_a}.$$

4.2. Basic reproduction number

The evaluation of the basic reproduction number is determined by the well known method called next-generation matrix. Since the basic reproduction number measures secondary *human* infections generated by infected humans, the mosquito infected compartment $\mathcal{I}_a$ is not included in the infected subsystem. Let assumed,

$$\frac{d\mathcal{X}}{dt} = \mathcal{F}(\mathcal{X}) - \mathcal{V}(\mathcal{X}),$$

where $\mathcal{F}$ characterizes the influx of new infections into the human infectious compartments and $\mathcal{V}$ collects all remaining transition terms. The vector of new infection is given as

$$\mathcal{F} = \begin{pmatrix} k_{ma}\mathcal{I}_h\mathcal{I}_a + k_{mh}\mathcal{I}_h\mathcal{I}_m \\ 0 \\ k_{dh}\mathcal{I}_h\mathcal{I}_d \\ 0 \\ 0 \end{pmatrix},$$

while the transition vector is

$$\mathcal{V} = \begin{pmatrix} (\sigma_m + \mu_h)\mathcal{E}_m \\ (\nu_m + \mu_h)\mathcal{I}_m - \sigma_m\mathcal{E}_m \\ (\sigma_d + \mu_h)\mathcal{E}_d \\ (\nu_d + \mu_h)\mathcal{I}_d - \sigma_d\mathcal{E}_d \\ (\nu_{md} + \delta + \mu_h)\mathcal{I}_c - \eta\mathcal{I}_m\mathcal{I}_d \end{pmatrix}.$$

Evaluating the Jacobians of $\mathcal{F}$ and $\mathcal{V}$ at the DFEP $\mathcal{E}_0$, implies

$$F = \begin{pmatrix} 0 & k_{mh}\mathcal{I}_h^0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & k_{dh}\mathcal{I}_h^0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$V = \begin{pmatrix} \sigma_m + \mu_h & 0 & 0 & 0 & 0 \\ -\sigma_m & v_m + \mu_h & 0 & 0 & 0 \\ 0 & 0 & \sigma_d + \mu_h & 0 & 0 \\ 0 & 0 & -\sigma_d & v_d + \mu_h & 0 \\ 0 & 0 & 0 & 0 & v_{md} + \delta + \mu_h \end{pmatrix}.$$

The next-generation matrix is defined by

$$K = FV^{-1}.$$

Since the co-infected class $\mathcal{I}_c$ does not generate new infections at the DFE, the basic reproduction number is determined by the monkeypox and dengue transmission pathways. Therefore,

$$\mathcal{R}_0 = \max \{ \mathcal{R}_m, \mathcal{R}_d \},$$

where

$$\begin{aligned} \mathcal{R}_m &= \frac{k_{mh}\sigma_m\mathcal{I}_h^0}{(\sigma_m + \mu_h)(v_m + \mu_h)} = \frac{k_{mh}\sigma_m\beta_h}{\mu_h(\sigma_m + \mu_h)(v_m + \mu_h)}, \\ \mathcal{R}_d &= \frac{k_{dh}\sigma_d\mathcal{I}_h^0}{(\sigma_d + \mu_h)(v_d + \mu_h)} = \frac{k_{dh}\sigma_d\beta_h}{\mu_h(\sigma_d + \mu_h)(v_d + \mu_h)}. \end{aligned}$$

We conclude that the main result is followed by,

$$\mathcal{R}_0 = \max \left\{ \frac{k_{mh}\sigma_m\beta_h}{\mu_h(\sigma_m + \mu_h)(v_m + \mu_h)}, \frac{k_{rh}\sigma_d\beta_h}{\mu_h(\sigma_d + \mu_h)(v_d + \mu_h)} \right\}. \quad (11)$$

4.3. Sensitivity Analysis

In this part of our work, we investigate how the reproduction numbers $\mathcal{R}_m$ and $\mathcal{R}_d$ respond to small changes in the epidemiological parameters of the model. Sensitivity analysis helps to find the most sensitive variable in disease transmission and control. The sensitivity of $\mathcal{R}$ to the variable p , in normalized forward form, is defined as

$$\Upsilon_p^{\mathcal{R}} = \frac{\partial \mathcal{R}}{\partial p} \times \frac{p}{\mathcal{R}}. \quad (12)$$

The malaria reproduction number is given by

$$\mathcal{R}_m = \frac{k_{mh}\sigma_m\beta_h}{\mu_h(\sigma_m + \mu_h)(v_m + \mu_h)}.$$

We have,

$$\begin{aligned} \Upsilon_{k_{mh}}^{\mathcal{R}_m} &= 1 > 0, \quad \Upsilon_{\beta_h}^{\mathcal{R}_m} = 1 > 0, \quad \Upsilon_{\sigma_m}^{\mathcal{R}_m} = \frac{\mu_h}{\sigma_m + \mu_h} > 0, \\ \Upsilon_{v_m}^{\mathcal{R}_m} &= -\frac{v_m}{v_m + \mu_h} < 0, \quad \Upsilon_{\mu_h}^{\mathcal{R}_m} = -1 - \frac{\mu_h}{\sigma_m + \mu_h} - \frac{\mu_h}{v_m + \mu_h} < 0. \end{aligned}$$

Thus, $\mathcal{R}_m$ increases with k_{mh} , β_h , and σ_m , while it decreases with increasing v_m and μ_h .

Similarly, the dengue reproduction number is

$$\mathcal{R}_d = \frac{k_{rh}\sigma_d\beta_h}{\mu_h(\sigma_d + \mu_h)(v_d + \mu_h)}.$$

The sensitivity indices are given by

$$\begin{aligned} \Upsilon_{k_{rh}}^{\mathcal{R}_d} &= 1 > 0, \quad \Upsilon_{\beta_h}^{\mathcal{R}_d} = 1 > 0, \quad \Upsilon_{\sigma_d}^{\mathcal{R}_d} = \frac{\mu_h}{\sigma_d + \mu_h} > 0, \\ \Upsilon_{v_d}^{\mathcal{R}_d} &= -\frac{v_d}{v_d + \mu_h} < 0, \quad \Upsilon_{\mu_h}^{\mathcal{R}_d} = -1 - \frac{\mu_h}{\sigma_d + \mu_h} - \frac{\mu_h}{v_d + \mu_h} < 0. \end{aligned}$$

Therefore, $\mathcal{R}_d$ increases with k_{rh} , β_h , and σ_d , while it decreases with v_d and μ_h . These results indicate that transmission parameters (k_{mh} , k_{rh} , β_h) have the strongest positive impact on disease spread, whereas recovery and natural death rates (v_m , v_d , μ_h) play significant roles in reducing the reproduction numbers.

5. Hyers-Ulam stability

In this part of our work, we examine the Hyers-Ulam stability of the proposed non-integral co-infection system (1) through by the ABC derivative. We define the required inequalities.

$$\left| {}^{ABC}D_t^\zeta \mathcal{X}(u) - \mathcal{F}(u, \mathcal{X}(u)) \right| \leq \varepsilon, \quad u \in \mathcal{J}. \quad (13)$$

where $\varepsilon = \max(\varepsilon_i)^T$, $i = 1, \dots, 10$.

Definition 5.1. The fractional co-infection model (1) is said to be Hyers-Ulam stable if there exist constants $G_i > 0$, $i = 1, \dots, 10$, such that for every $\varepsilon > 0$ and for any function $\widetilde{\mathcal{X}}$ satisfying inequality (13), there exists a unique solution $\mathcal{X}$ of model (1) satisfying

$$\left\| \widetilde{\mathcal{X}}(u) - \mathcal{X}(u) \right\| \leq G_i \varepsilon, \quad u \in \mathcal{J}.$$

$$\left\| \widetilde{\mathcal{X}}(u) - \mathcal{X}(u) \right\| \leq G_i \varepsilon_i, \quad i = 1, \dots, 10. \quad (14)$$

Remark 5.2. Consider $\widetilde{\mathcal{X}}$ as the solution of inequality (13), and if there exist h_i , $i = 1, \dots, 10$, such that $|h_i(u)| < \varepsilon_i$, we have

$${}^{ABC}D_t^\zeta \widetilde{\mathcal{X}}(u) = \mathcal{F}(u, \widetilde{\mathcal{X}}(u)) + h_i(u), \quad i = 1, \dots, 10. \quad (15)$$

Theorem 5.3. The proposed fractional co-infection model (1) is Hyers-Ulam stable, if condition H_1 is satisfied and

$$\frac{M \mathcal{B}(\zeta)^{-1} T^\zeta}{\Gamma(\zeta + 1)} \Pi_i < 1, \quad i = 1, \dots, 10. \quad (16)$$

Proof. Using the result of (15), we get

$${}^{ABC}D_t^\zeta \widetilde{\mathcal{X}}(u) = \mathcal{F}(u, \widetilde{\mathcal{X}}(u)) + h_i(u),$$

which gives

$$\begin{aligned} \widetilde{\mathcal{X}}(u) &= \widetilde{\mathcal{X}}(0) + \frac{1-\zeta}{\mathcal{B}(\zeta)} \mathcal{F}(u, \widetilde{\mathcal{X}}(u)) + \frac{\zeta}{\mathcal{B}(\zeta)\Gamma(\zeta)} \int_0^u (u-s)^{\zeta-1} \mathcal{F}(s, \widetilde{\mathcal{X}}(s)) ds \\ &\quad + \frac{\zeta}{\mathcal{B}(\zeta)\Gamma(\zeta)} \int_0^u (u-s)^{\zeta-1} h_i(s) ds. \end{aligned}$$

Let $\mathcal{X}(u)$ be the unique solution of the co-infection system, then

$$\mathcal{X}(u) = \mathcal{X}(0) + \frac{1-\zeta}{\mathcal{B}(\zeta)} \mathcal{F}(u, \mathcal{X}(u)) + \frac{\zeta}{\mathcal{B}(\zeta)\Gamma(\zeta)} \int_0^u (u-s)^{\zeta-1} \mathcal{F}(s, \mathcal{X}(s)) ds.$$

Hence,

$$\begin{aligned} \|\widetilde{\mathcal{X}} - \mathcal{X}\| &\leq \frac{\zeta}{\mathcal{B}(\zeta)\Gamma(\zeta)} \int_0^u (u-s)^{\zeta-1} \|\mathcal{F}(s, \widetilde{\mathcal{X}}(s)) - \mathcal{F}(s, \mathcal{X}(s))\| ds \\ &\quad + \frac{\zeta}{\mathcal{B}(\zeta)\Gamma(\zeta)} \int_0^u (u-s)^{\zeta-1} |h_i(s)| ds \\ &\leq \frac{M\mathcal{B}(\zeta)^{-1}T^\zeta}{\Gamma(\zeta+1)} \Pi_i \|\widetilde{\mathcal{X}} - \mathcal{X}\| + \frac{M\mathcal{B}(\zeta)^{-1}T^\zeta}{\Gamma(\zeta+1)} \varepsilon_i. \end{aligned}$$

Thus,

$$\|\widetilde{\mathcal{X}} - \mathcal{X}\| \leq \frac{\frac{M\mathcal{B}(\zeta)^{-1}T^\zeta}{\Gamma(\zeta+1)}}{1 - \frac{M\mathcal{B}(\zeta)^{-1}T^\zeta}{\Gamma(\zeta+1)} \Pi_i} \varepsilon_i.$$

Hence, the proposed fractional co-infection model is Hyers-Ulam stable. $\square$

6. Optimal Control Analysis

In order to understand and slow the development of co-infectious diseases, research has used fractional-order mathematical models with optimal control strategies [21]. In order to lower disease transmission and enhance public health outcomes, these studies highlight the beneficial effect of combining preventative strategies, such as awareness campaigns, with treatment regimens. In this section, we improve our model by adding four time-dependent control measures, we extend the model to control model by introducing the control parameters:

- $u_1(t)$: malaria prevention control,
- $u_2(t)$: dengue prevention control,
- $u_3(t)$: treatment control for co-infected individuals,
- $u_4(t)$: mosquito/vector control.

The controls satisfy

$$0 \leq u_i(t) \leq 1, \quad i = 1, 2, 3, 4.$$

The transmission parameters are modified as

$$\begin{aligned} k_{ma} &\rightarrow (1 - u_1)k_{ma}, & k_{mh} &\rightarrow (1 - u_1)k_{mh}, & k_{dh} &\rightarrow (1 - u_2)k_{dh}, \\ k_a &\rightarrow (1 - u_4)k_a, & v_{md} &\rightarrow v_{md} + u_3. \end{aligned}$$

The control co-infection model as

$$\begin{aligned} {}^{ABC}D_t^\zeta \mathcal{S}_h &= \beta_h - (1 - u_1)(k_{ma}\mathcal{S}_h\mathcal{I}_a + k_{mh}\mathcal{S}_h\mathcal{I}_m) - (1 - u_2)k_{dh}\mathcal{S}_h\mathcal{I}_d - \mu_h\mathcal{S}_h, \\ {}^{ABC}D_t^\zeta \mathcal{E}_m &= (1 - u_1)(k_{ma}\mathcal{S}_h\mathcal{I}_a + k_{mh}\mathcal{S}_h\mathcal{I}_m) - (\sigma_m + \mu_h)\mathcal{E}_m, \\ {}^{ABC}D_t^\zeta \mathcal{I}_m &= \sigma_m\mathcal{E}_m - (\eta\mathcal{I}_d + v_m + \mu_h)\mathcal{I}_m, \\ {}^{ABC}D_t^\zeta \mathcal{E}_d &= (1 - u_2)k_{dh}\mathcal{S}_h\mathcal{I}_d - (\sigma_d + \mu_h)\mathcal{E}_d, \\ {}^{ABC}D_t^\zeta \mathcal{I}_d &= \sigma_d\mathcal{E}_d - (\eta\mathcal{I}_m + v_d + \mu_h)\mathcal{I}_d, \\ {}^{ABC}D_t^\zeta \mathcal{I}_c &= \eta\mathcal{I}_m\mathcal{I}_d - (v_{md} + u_3 + \delta + \mu_h)\mathcal{I}_c, \\ {}^{ABC}D_t^\zeta \mathcal{H}_h &= \delta\mathcal{I}_c - (v_q + \mu_h)\mathcal{H}_h, \\ {}^{ABC}D_t^\zeta \mathcal{R}_h &= v_m\mathcal{I}_m + v_d\mathcal{I}_d + (v_{md} + u_3)\mathcal{I}_c + v_q\mathcal{H}_h - \mu_h\mathcal{R}_h, \\ {}^{ABC}D_t^\zeta \mathcal{S}_a &= \beta_a - (1 - u_4)k_a\mathcal{S}_a\mathcal{I}_a - \mu_a\mathcal{S}_a, \\ {}^{ABC}D_t^\zeta \mathcal{I}_a &= (1 - u_4)k_a\mathcal{S}_a\mathcal{I}_a - \mu_a\mathcal{I}_a. \end{aligned} \tag{17}$$

We minimize infected individuals and implementation related costs:

$$J(u_1, u_2, u_3, u_4) = \int_0^T \left[A_1\mathcal{I}_m + A_2\mathcal{I}_d + A_3\mathcal{I}_c + \frac{1}{2} (B_1u_1^2 + B_2u_2^2 + B_3u_3^2 + B_4u_4^2) \right] dt. \tag{18}$$

Let $\lambda_i(t)$ be associated parameters corresponding to each main compartment variable.

The Hamiltonian is defined as

$$\begin{aligned} \mathcal{H} &= A_1\mathcal{I}_m + A_2\mathcal{I}_d + A_3\mathcal{I}_c + \frac{1}{2} (B_1u_1^2 + B_2u_2^2 + B_3u_3^2 + B_4u_4^2) \\ &\quad + \lambda_{\mathcal{S}_h} \frac{d\mathcal{S}_h}{dt} + \lambda_{\mathcal{E}_m} \frac{d\mathcal{E}_m}{dt} + \lambda_{\mathcal{I}_m} \frac{d\mathcal{I}_m}{dt} + \lambda_{\mathcal{E}_d} \frac{d\mathcal{E}_d}{dt} \\ &\quad + \lambda_{\mathcal{I}_d} \frac{d\mathcal{I}_d}{dt} + \lambda_{\mathcal{I}_c} \frac{d\mathcal{I}_c}{dt} + \lambda_{\mathcal{Q}_h} \frac{d\mathcal{Q}_h}{dt} + \lambda_{\mathcal{R}_h} \frac{d\mathcal{R}_h}{dt} + \lambda_{\mathcal{S}_a} \frac{d\mathcal{S}_a}{dt} + \lambda_{\mathcal{I}_a} \frac{d\mathcal{I}_a}{dt}. \end{aligned} \tag{19}$$

By Pontryagin's Maximum Principle:

$$\frac{d\lambda_i}{dt} = -\frac{\partial \mathcal{H}}{\partial x_i}, \quad \lambda_i(T) = 0.$$

Extract all terms containing $\mathcal{I}_m$:

$$\frac{\partial \mathcal{H}}{\partial \mathcal{I}_m} = A_1 + \lambda_{\mathcal{S}_h}(-(1 - u_1)k_{mh}\mathcal{S}_h) + \lambda_{\mathcal{E}_m}((1 - u_1)k_{mh}\mathcal{S}_h)$$

$$+ \lambda_{\mathcal{I}_m}(-\eta \mathcal{I}_d - v_m - \mu_h) + \lambda_{\mathcal{I}_c}(\eta \mathcal{I}_d) + \lambda_{\mathcal{R}_h}(v_m). \quad (20)$$

Hence,

$$\begin{aligned} \frac{d\lambda_{\mathcal{I}_m}}{dt} = & - \left[A_1 - \lambda_{\mathcal{I}_h}(1 - u_1)k_{mh}\mathcal{I}_h + \lambda_{\mathcal{E}_m}(1 - u_1)k_{mh}\mathcal{I}_h \right. \\ & \left. - \lambda_{\mathcal{I}_m}(\eta \mathcal{I}_d + v_m + \mu_h) + \lambda_{\mathcal{I}_c}\eta \mathcal{I}_d + \lambda_{\mathcal{R}_h}v_m \right]. \end{aligned} \quad (21)$$

All other adjoint equations are computed similarly.

We compute

$$\frac{\partial \mathcal{H}}{\partial u_i} = 0.$$

Thus,

$$\begin{aligned} u_1^* &= \min \left\{ 1, \max \left(0, \frac{(\lambda_{\mathcal{I}_h} - \lambda_{\mathcal{E}_m})(k_{ma}\mathcal{I}_h\mathcal{I}_a + k_{mh}\mathcal{I}_h\mathcal{I}_m)}{B_1} \right) \right\}, \\ u_2^* &= \min \left\{ 1, \max \left(0, \frac{(\lambda_{\mathcal{I}_h} - \lambda_{\mathcal{E}_d})k_{dh}\mathcal{I}_h\mathcal{I}_d}{B_2} \right) \right\}, \\ u_3^* &= \min \left\{ 1, \max \left(0, \frac{\lambda_{\mathcal{I}_c}\mathcal{I}_c}{B_3} \right) \right\}, \\ u_4^* &= \min \left\{ 1, \max \left(0, \frac{(\lambda_{\mathcal{I}_a} - \lambda_{\mathcal{I}_d})k_a\mathcal{I}_a\mathcal{I}_d}{B_4} \right) \right\}. \end{aligned}$$

7. Numerical Analysis of co-infection model of Dengue and Malaria

The MEM is especially appealing in the larger setting of fractional-order models since it eliminates multi-step predictor-corrector iterations, which makes it simpler to construct for large-scale simulations and more computationally efficient. The approach is generally resilient for moderate step sizes, however it is conditionally stable. For sensitivity and subjective research, the MEM offers a respectable level of accuracy. Overall, the MEM strikes a balance between dependability and efficiency, which makes it ideal for variables sensitivity calculations and numerical experimentation in fractional models of epidemiology [43]. In this part, we presents the general solutions and numerical models of the suggested system using the MEM approach.

7.1. General solution of the co-infection system

In this section, an approximate solution to the problem under study is presented. To achieve this, the modified Euler method (MEM) is employed as the numerical scheme.

$$\left\{ \begin{array}{l} {}^{ABC}D_t^\zeta \mathcal{S}_h(u) = B_1(u, \mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a), \\ {}^{ABC}D_t^\zeta \mathcal{E}_m(u) = B_2(u, \mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a), \\ {}^{ABC}D_t^\zeta \mathcal{I}_m(u) = B_3(u, \mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a), \\ {}^{ABC}D_t^\zeta \mathcal{E}_d(u) = B_4(u, \mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a), \\ {}^{ABC}D_t^\zeta \mathcal{I}_d(u) = B_5(u, \mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a), \\ {}^{ABC}D_t^\zeta \mathcal{I}_c(u) = B_6(u, \mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a), \\ {}^{ABC}D_t^\zeta \mathcal{H}_h(u) = B_7(u, \mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a), \\ {}^{ABC}D_t^\zeta \mathcal{R}_h(u) = B_8(u, \mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a), \\ {}^{ABC}D_t^\zeta \mathcal{S}_a(u) = B_9(u, \mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a), \\ {}^{ABC}D_t^\zeta \mathcal{I}_a(u) = B_{10}(u, \mathcal{S}_h, \mathcal{E}_m, \mathcal{I}_m, \mathcal{E}_d, \mathcal{I}_d, \mathcal{I}_c, \mathcal{H}_h, \mathcal{R}_h, \mathcal{S}_a, \mathcal{I}_a). \end{array} \right. \quad (10.1)$$

Let the interval be subdivided as $[0, T]$ into m subintervals $u \in [u_j, u_{j+1}]$ with same width $h = \frac{T}{m}$ by utilizing the grid point $u_j = jh$ for $j = 0, 1, \dots, m$. Assumed all state variables and their corresponding ABC fractional derivatives are continuous on the interval. We expand the solutions about $u = u_0 = 0$ by MEM. The system for u_1 is followed by,

$$\left\{ \begin{array}{l} \mathcal{J}_h(u_1) = \mathcal{J}_h(u_0) + {}^{ABC}D_t^\zeta \mathcal{J}_h(u_0) \frac{h^\zeta}{\Gamma(\zeta+1)} + {}^{ABC}D_t^{2\zeta} \mathcal{J}_h(u_0) \frac{h^{2\zeta}}{\Gamma(2\zeta+1)}, \\ \mathcal{E}_m(u_1) = \mathcal{E}_m(u_0) + {}^{ABC}D_t^\zeta \mathcal{E}_m(u_0) \frac{h^\zeta}{\Gamma(\zeta+1)} + {}^{ABC}D_t^{2\zeta} \mathcal{E}_m(u_0) \frac{h^{2\zeta}}{\Gamma(2\zeta+1)}, \\ \mathcal{J}_m(u_1) = \mathcal{J}_m(u_0) + {}^{ABC}D_t^\zeta \mathcal{J}_m(u_0) \frac{h^\zeta}{\Gamma(\zeta+1)} + {}^{ABC}D_t^{2\zeta} \mathcal{J}_m(u_0) \frac{h^{2\zeta}}{\Gamma(2\zeta+1)}, \\ \mathcal{E}_d(u_1) = \mathcal{E}_d(u_0) + {}^{ABC}D_t^\zeta \mathcal{E}_d(u_0) \frac{h^\zeta}{\Gamma(\zeta+1)} + {}^{ABC}D_t^{2\zeta} \mathcal{E}_d(u_0) \frac{h^{2\zeta}}{\Gamma(2\zeta+1)}, \\ \mathcal{J}_d(u_1) = \mathcal{J}_d(u_0) + {}^{ABC}D_t^\zeta \mathcal{J}_d(u_0) \frac{h^\zeta}{\Gamma(\zeta+1)} + {}^{ABC}D_t^{2\zeta} \mathcal{J}_d(u_0) \frac{h^{2\zeta}}{\Gamma(2\zeta+1)}, \\ \mathcal{J}_c(u_1) = \mathcal{J}_c(u_0) + {}^{ABC}D_t^\zeta \mathcal{J}_c(u_0) \frac{h^\zeta}{\Gamma(\zeta+1)} + {}^{ABC}D_t^{2\zeta} \mathcal{J}_c(u_0) \frac{h^{2\zeta}}{\Gamma(2\zeta+1)}, \\ \mathcal{H}_h(u_1) = \mathcal{H}_h(u_0) + {}^{ABC}D_t^\zeta \mathcal{H}_h(u_0) \frac{h^\zeta}{\Gamma(\zeta+1)} + {}^{ABC}D_t^{2\zeta} \mathcal{H}_h(u_0) \frac{h^{2\zeta}}{\Gamma(2\zeta+1)}, \\ \mathcal{R}_h(u_1) = \mathcal{R}_h(u_0) + {}^{ABC}D_t^\zeta \mathcal{R}_h(u_0) \frac{h^\zeta}{\Gamma(\zeta+1)} + {}^{ABC}D_t^{2\zeta} \mathcal{R}_h(u_0) \frac{h^{2\zeta}}{\Gamma(2\zeta+1)}, \\ \mathcal{S}_a(u_1) = \mathcal{S}_a(u_0) + {}^{ABC}D_t^\zeta \mathcal{S}_a(u_0) \frac{h^\zeta}{\Gamma(\zeta+1)} + {}^{ABC}D_t^{2\zeta} \mathcal{S}_a(u_0) \frac{h^{2\zeta}}{\Gamma(2\zeta+1)}, \\ \mathcal{I}_a(u_1) = \mathcal{I}_a(u_0) + {}^{ABC}D_t^\zeta \mathcal{I}_a(u_0) \frac{h^\zeta}{\Gamma(\zeta+1)} + {}^{ABC}D_t^{2\zeta} \mathcal{I}_a(u_0) \frac{h^{2\zeta}}{\Gamma(2\zeta+1)}. \end{array} \right.$$

Since the step size h is sufficiently small, we neglects the second-order expression having $h^{2\zeta}$. Hence, we get

$$\left\{ \begin{array}{l}
\mathcal{J}_h(u_1) = \mathcal{J}_h(u_0) + B_1(u_0, \mathbb{X}(u_0)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\
\mathcal{E}_m(u_1) = \mathcal{E}_m(u_0) + B_2(u_0, \mathbb{X}(u_0)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\
\mathcal{J}_m(u_1) = \mathcal{J}_m(u_0) + B_3(u_0, \mathbb{X}(u_0)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\
\mathcal{E}_d(u_1) = \mathcal{E}_d(u_0) + B_4(u_0, \mathbb{X}(u_0)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\
\mathcal{J}_d(u_1) = \mathcal{J}_d(u_0) + B_5(u_0, \mathbb{X}(u_0)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\
\mathcal{J}_c(u_1) = \mathcal{J}_c(u_0) + B_6(u_0, \mathbb{X}(u_0)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\
\mathcal{H}_h(u_1) = \mathcal{H}_h(u_0) + B_7(u_0, \mathbb{X}(u_0)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\
\mathcal{B}_h(u_1) = \mathcal{B}_h(u_0) + B_8(u_0, \mathbb{X}(u_0)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\
\mathcal{S}_a(u_1) = \mathcal{S}_a(u_0) + B_9(u_0, \mathbb{X}(u_0)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\
\mathcal{J}_a(u_1) = \mathcal{J}_a(u_0) + B_{10}(u_0, \mathbb{X}(u_0)) \frac{h^\zeta}{\Gamma(\zeta + 1)}.
\end{array} \right. \quad 5 \quad (10.2)$$

Furthermore, the subsequent terms are

$$\left\{ \begin{array}{l} \mathcal{S}_h(u_2) = \mathcal{S}_h(u_1) + B_1(u_1, \mathbb{X}(u_1)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{E}_m(u_2) = \mathcal{E}_m(u_1) + B_2(u_1, \mathbb{X}(u_1)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{J}_m(u_2) = \mathcal{J}_m(u_1) + B_3(u_1, \mathbb{X}(u_1)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{E}_d(u_2) = \mathcal{E}_d(u_1) + B_4(u_1, \mathbb{X}(u_1)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{J}_d(u_2) = \mathcal{J}_d(u_1) + B_5(u_1, \mathbb{X}(u_1)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{J}_c(u_2) = \mathcal{J}_c(u_1) + B_6(u_1, \mathbb{X}(u_1)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{H}_h(u_2) = \mathcal{H}_h(u_1) + B_7(u_1, \mathbb{X}(u_1)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{R}_h(u_2) = \mathcal{R}_h(u_1) + B_8(u_1, \mathbb{X}(u_1)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{S}_a(u_2) = \mathcal{S}_a(u_1) + B_9(u_1, \mathbb{X}(u_1)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{J}_a(u_2) = \mathcal{J}_a(u_1) + B_{10}(u_1, \mathbb{X}(u_1)) \frac{h^\zeta}{\Gamma(\zeta + 1)}. \end{array} \right.$$

At $u_{j+1} = u_j + h$, $j = 0, 1, \dots, m$, we obtain

$$\left\{ \begin{array}{l} \mathcal{S}_h(u_{j+1}) = \mathcal{S}_h(u_j) + B_1(u_j, \mathbb{X}(u_j)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{E}_m(u_{j+1}) = \mathcal{E}_m(u_j) + B_2(u_j, \mathbb{X}(u_j)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{I}_m(u_{j+1}) = \mathcal{I}_m(u_j) + B_3(u_j, \mathbb{X}(u_j)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{E}_d(u_{j+1}) = \mathcal{E}_d(u_j) + B_4(u_j, \mathbb{X}(u_j)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{I}_d(u_{j+1}) = \mathcal{I}_d(u_j) + B_5(u_j, \mathbb{X}(u_j)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{I}_c(u_{j+1}) = \mathcal{I}_c(u_j) + B_6(u_j, \mathbb{X}(u_j)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{H}_h(u_{j+1}) = \mathcal{H}_h(u_j) + B_7(u_j, \mathbb{X}(u_j)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{R}_h(u_{j+1}) = \mathcal{R}_h(u_j) + B_8(u_j, \mathbb{X}(u_j)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{S}_a(u_{j+1}) = \mathcal{S}_a(u_j) + B_9(u_j, \mathbb{X}(u_j)) \frac{h^\zeta}{\Gamma(\zeta + 1)}, \\ \mathcal{I}_a(u_{j+1}) = \mathcal{I}_a(u_j) + B_{10}(u_j, \mathbb{X}(u_j)) \frac{h^\zeta}{\Gamma(\zeta + 1)}. \end{array} \right.$$

After simplification, we obtain the general approximate solution of the proposed fractional co-infection model.

$$\left\{ \begin{aligned}
\mathcal{S}_h(u_j) &= \mathcal{S}_h(0) + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \left[(j-1)^{\zeta+1} - (j-\zeta-1)j^\zeta \right] B_1(u_0, \mathbb{X}(u_0)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \sum_{i=1}^{j-1} \left[(j-i+1)^{\zeta+1} - 2(j-i)^{\zeta+1} + (j-i-1)^{\zeta+1} \right] B_1(u_i, \mathbb{X}(u_i)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} B_1(u_{j-1}, \mathbb{X}(u_{j-1})) + \frac{\zeta h^\zeta}{\Gamma(\zeta+1)} B_1(u_{j-1}, \mathbb{X}(u_{j-1})), \\
\mathcal{E}_m(u_j) &= \mathcal{E}_m(0) + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \left[(j-1)^{\zeta+1} - (j-\zeta-1)j^\zeta \right] B_2(u_0, \mathbb{X}(u_0)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \sum_{i=1}^{j-1} \left[(j-i+1)^{\zeta+1} - 2(j-i)^{\zeta+1} + (j-i-1)^{\zeta+1} \right] B_2(u_i, \mathbb{X}(u_i)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} B_2(u_{j-1}, \mathbb{X}(u_{j-1})) + \frac{\zeta h^\zeta}{\Gamma(\zeta+1)} B_2(u_{j-1}, \mathbb{X}(u_{j-1})), \\
\mathcal{J}_m(u_j) &= \mathcal{J}_m(0) + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \left[(j-1)^{\zeta+1} - (j-\zeta-1)j^\zeta \right] B_3(u_0, \mathbb{X}(u_0)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \sum_{i=1}^{j-1} \left[(j-i+1)^{\zeta+1} - 2(j-i)^{\zeta+1} + (j-i-1)^{\zeta+1} \right] B_3(u_i, \mathbb{X}(u_i)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} B_3(u_{j-1}, \mathbb{X}(u_{j-1})) + \frac{\zeta h^\zeta}{\Gamma(\zeta+1)} B_3(u_{j-1}, \mathbb{X}(u_{j-1})), \\
\mathcal{E}_d(u_j) &= \mathcal{E}_d(0) + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \left[(j-1)^{\zeta+1} - (j-\zeta-1)j^\zeta \right] B_4(u_0, \mathbb{X}(u_0)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \sum_{i=1}^{j-1} \left[(j-i+1)^{\zeta+1} - 2(j-i)^{\zeta+1} + (j-i-1)^{\zeta+1} \right] B_4(u_i, \mathbb{X}(u_i)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} B_4(u_{j-1}, \mathbb{X}(u_{j-1})) + \frac{\zeta h^\zeta}{\Gamma(\zeta+1)} B_4(u_{j-1}, \mathbb{X}(u_{j-1})), \\
\mathcal{J}_d(u_j) &= \mathcal{J}_d(0) + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \left[(j-1)^{\zeta+1} - (j-\zeta-1)j^\zeta \right] B_5(u_0, \mathbb{X}(u_0)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \sum_{i=1}^{j-1} \left[(j-i+1)^{\zeta+1} - 2(j-i)^{\zeta+1} + (j-i-1)^{\zeta+1} \right] B_5(u_i, \mathbb{X}(u_i)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} B_5(u_{j-1}, \mathbb{X}(u_{j-1})) + \frac{\zeta h^\zeta}{\Gamma(\zeta+1)} B_5(u_{j-1}, \mathbb{X}(u_{j-1})), \\
\mathcal{J}_c(u_j) &= \mathcal{J}_c(0) + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \left[(j-1)^{\zeta+1} - (j-\zeta-1)j^\zeta \right] B_6(u_0, \mathbb{X}(u_0)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \sum_{i=1}^{j-1} \left[(j-i+1)^{\zeta+1} - 2(j-i)^{\zeta+1} + (j-i-1)^{\zeta+1} \right] B_6(u_i, \mathbb{X}(u_i)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} B_6(u_{j-1}, \mathbb{X}(u_{j-1})) + \frac{\zeta h^\zeta}{\Gamma(\zeta+1)} B_6(u_{j-1}, \mathbb{X}(u_{j-1})),
\end{aligned} \right. \tag{22}$$

$$\left\{ \begin{aligned}
\mathcal{H}_h(u_j) &= \mathcal{H}_h(0) + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \left[(j-1)^{\zeta+1} - (j-\zeta-1)j^\zeta \right] B_7(u_0, \mathbb{X}(u_0)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \sum_{i=1}^{j-1} \left[(j-i+1)^{\zeta+1} - 2(j-i)^{\zeta+1} + (j-i-1)^{\zeta+1} \right] B_7(u_i, \mathbb{X}(u_i)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} B_7(u_{j-1}, \mathbb{X}(u_{j-1})) + \frac{\zeta h^\zeta}{\Gamma(\zeta+1)} B_7(u_{j-1}, \mathbb{X}(u_{j-1})), \\
\mathcal{R}_h(u_j) &= \mathcal{R}_h(0) + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \left[(j-1)^{\zeta+1} - (j-\zeta-1)j^\zeta \right] B_8(u_0, \mathbb{X}(u_0)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \sum_{i=1}^{j-1} \left[(j-i+1)^{\zeta+1} - 2(j-i)^{\zeta+1} + (j-i-1)^{\zeta+1} \right] B_8(u_i, \mathbb{X}(u_i)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} B_8(u_{j-1}, \mathbb{X}(u_{j-1})) + \frac{\zeta h^\zeta}{\Gamma(\zeta+1)} B_8(u_{j-1}, \mathbb{X}(u_{j-1})), \\
\mathcal{S}_a(u_j) &= \mathcal{S}_a(0) + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \left[(j-1)^{\zeta+1} - (j-\zeta-1)j^\zeta \right] B_9(u_0, \mathbb{X}(u_0)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \sum_{i=1}^{j-1} \left[(j-i+1)^{\zeta+1} - 2(j-i)^{\zeta+1} + (j-i-1)^{\zeta+1} \right] B_9(u_i, \mathbb{X}(u_i)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} B_9(u_{j-1}, \mathbb{X}(u_{j-1})) + \frac{\zeta h^\zeta}{\Gamma(\zeta+1)} B_9(u_{j-1}, \mathbb{X}(u_{j-1})), \\
\mathcal{S}_a(u_j) &= \mathcal{S}_a(0) + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \left[(j-1)^{\zeta+1} - (j-\zeta-1)j^\zeta \right] B_{10}(u_0, \mathbb{X}(u_0)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} \sum_{i=1}^{j-1} \left[(j-i+1)^{\zeta+1} - 2(j-i)^{\zeta+1} + (j-i-1)^{\zeta+1} \right] B_{10}(u_i, \mathbb{X}(u_i)) \\
&\quad + \frac{\zeta h^\zeta}{\Gamma(\zeta+2)} B_{10}(u_{j-1}, \mathbb{X}(u_{j-1})) + \frac{\zeta h^\zeta}{\Gamma(\zeta+1)} B_{10}(u_{j-1}, \mathbb{X}(u_{j-1})).
\end{aligned} \right. \tag{23}$$

7.2. Numerical simulation of co-infection model of Dengue and Malaria

In this section, we conduct numerical simulations to analyze the dynamic behavior of the proposed model of Dengue-Malaria co-infection within the structure of the fractal-fractional Caputo operator using the MEM numerical scheme for real data of Thailand. In 2025 in Thailand, 1194 case was report as Malaria while in 2026 also 1503 case report up to 28 march 2026 [44]. Dengue case in 2025 in thailand reported 60,000 while in 2026 3795 up to today 28 march [45]. Using this data from [15, 44, 45], we plots the numerical results for our model. The simulations are performed for different fractional orders $\zeta = 1, 0.95, 0.85$, and 0.75 to examine the influence of memory effects on disease transmission dynamics. The results presented in Figure (2) show that all trajectories corresponding to fractional orders less than one gradually converge to the ODE case $\zeta = 1$ as time progresses, confirming the consistency and stability of the model. The fractional order plays a significant role in capturing memory effects, where smaller values of ζ indicate stronger dependence on past states of the system, thereby affecting the rate of disease progression and recovery.

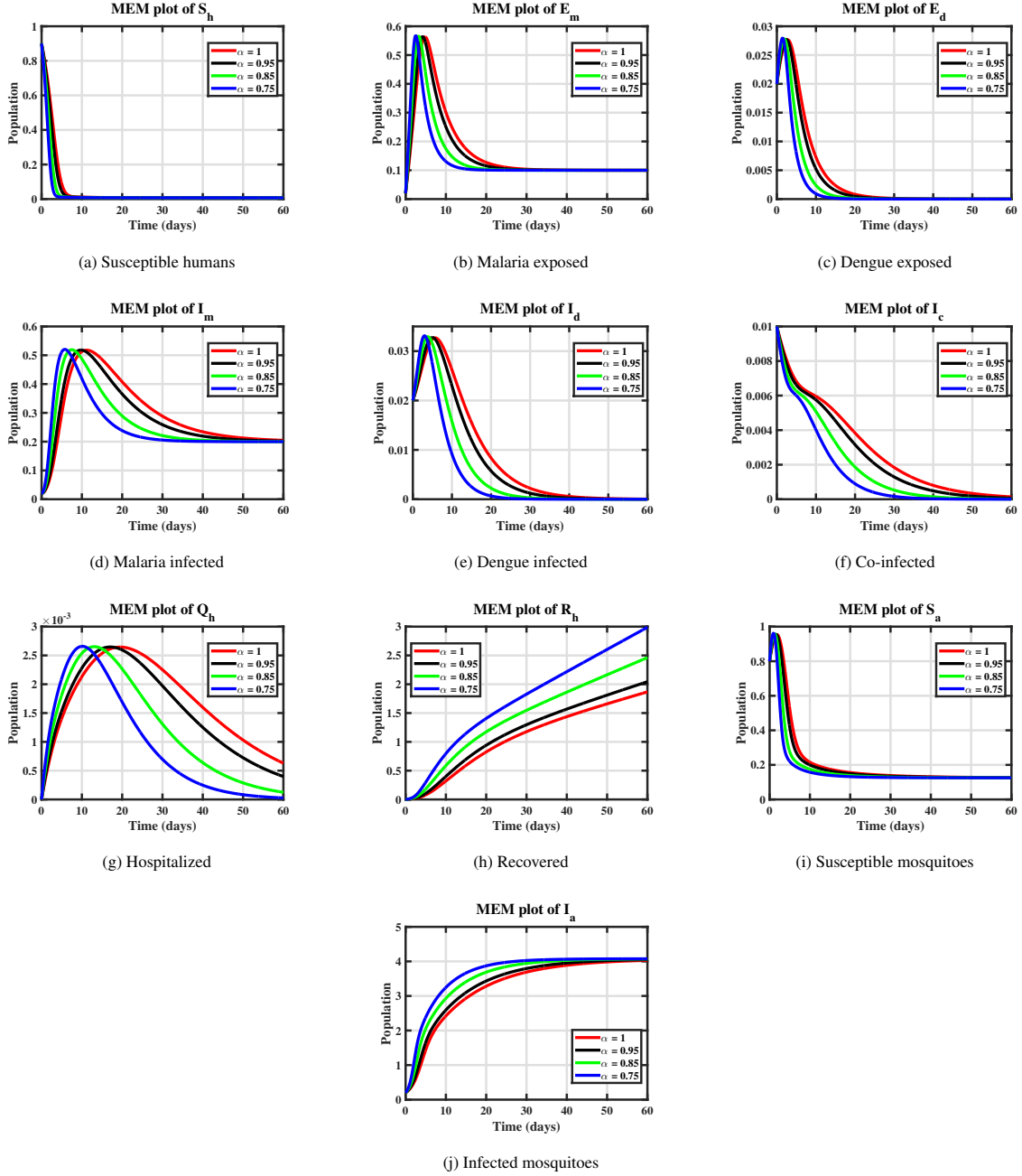


Figure 2: Numerical simulations of human and mosquito populations in the dengue-malaria co-infection model.

From the plots numerical results, it is seen that the susceptible human density $\mathcal{S}_h$ (Fig.2a) exponentially decreases for all order, going to stability within a short time interval, also the small order the decline go faster due to stronger mummy influence. The exposed compartment $\mathcal{E}_m$ (Fig.2b) of Malaria and $\mathcal{E}_d$ (Fig.2c) Dengue show increased in starting, and go to a peak, and after that come back to stability with slowly decrease. The fractional order small the dhow best dynamic of the class. The infected human compartments from Malaria $\mathcal{I}_m$ (Fig.2d) and $\mathcal{I}_d$ (Fig.2e) Dengue show exponential growth initially and then start decrease

to the equilibrium point, where again the smaller fractional order give a faster growth and faster decline, in the result we get a small period of the infection. The co-infection compartment $\mathcal{I}_c$ (Fig.2f), show a exponential decrease with faster decay which suggest that the co-infection rate to hospitalization or recovery is good where treatment is need to the co-infected persons. In (Fig.2g), the hospitalized population dynamic is present in different fractional order, in which the lower order show faster increased and then decrease with continues decay and go to stability. On the other hand the recovered compartment of the human population (Fig.2h), show the monotonic increased over the time with different fractional level, and the smaller order show fast recovery of the population which is the evidence of the treatment in hospital.

On the other hand, the mosquito density the susceptible compartment $\mathcal{S}_a$ (Fig.2i), show the continues decay and become stable to a lower level. The infected mosquito compartment $\mathcal{I}_a$ (Fig.2j) show exponential growth of the density and goes to a saturation level, hence the lower order of fractional order show faster increased and convergence to the stability. All theses dissuasions show that the memory effects has important influence on vector dynamic of the co-infection of Malaria and Dengue. The numerical results verify our theoretical results the stability of the model is gained through real data of thailand.

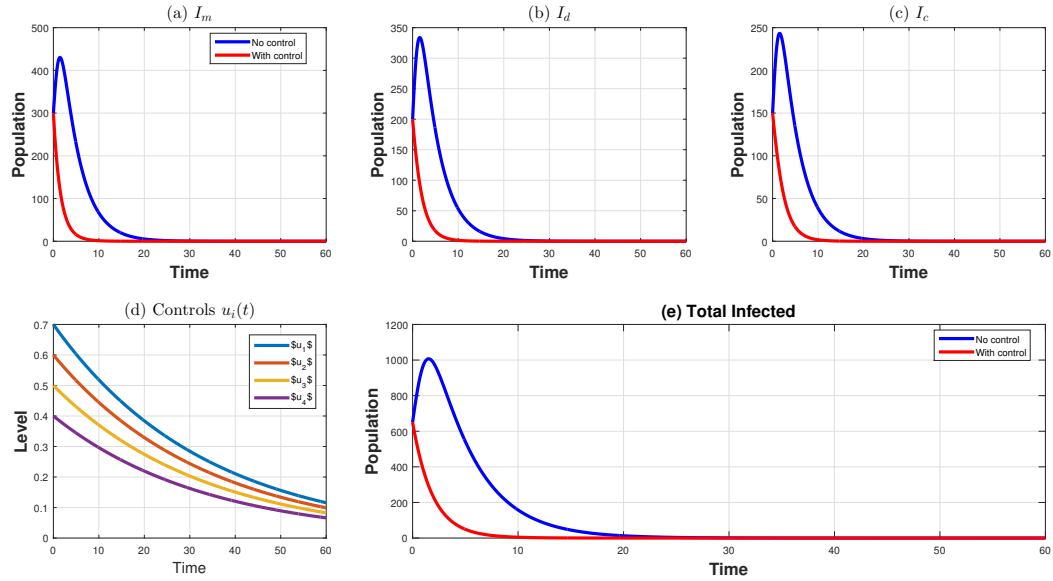


Figure 3: Control plots

In figure (Fig.3), we present the impact of the optimal control of the co-infection model of Malaria and Dengue. We see that the without control the infection of Malaria $\mathcal{I}_m$, infection of Dengue $\mathcal{I}_d$, and co-infection $\mathcal{I}_c$ show exponential increase initially and slower decline of the infection to stability. Perhaps, apply the optimal control strategies present in the work, the infection show faster decline and become control and stable. Hence, the implementation of the the prevention of malaria $u_1(t)$, prevention of the dengue $u_2(t)$, treatment of the co-infection $u_3(t)$, and control of the mosquito $u_4(t)$ the infection is control well. The

control profile of the infected compartment initial high intervention level which decrease exponentially over time, highlights the strategy define importance of the control measure early stage of the disease. Finally the optimal policy define in this work indicate that the presentation and treatment approach is highly recommend for reducing the intensity and period of the infection.

7.3. Model Limitations

Although national surveillance data from Thailand were incorporated into the study, detailed monthly compartment-specific observations were not available. Consequently, the model parameters were not estimated through a formal calibration procedure. Instead, the parameter values were selected from the epidemiological literature and adjusted within biologically realistic ranges to investigate the qualitative dynamics of dengue–malaria co-infection.

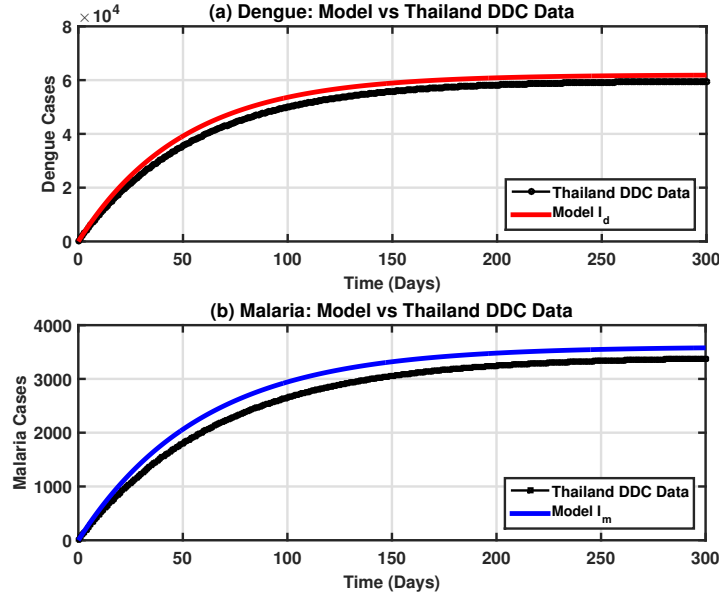


Figure 4: Comparison of real data Thailand and model

The performance of the proposed model was evaluated using the root mean square error (RMSE) and the coefficient of determination (R^2). The dengue component yielded an RMSE of 3023.62 and an (R^2) value of 0.9590, indicating that approximately 95.9% of the variability in the reported dengue cases was captured by the model. Similarly, the malaria component produced an RMSE of 242.94 with an (R^2) value of 0.9227, demonstrating that about 92.3% of the variability in the malaria data was explained by the model. These results suggest a strong agreement between the model predictions and the observed epidemiological data.

8. Conclusion

In this research work, we present a fractional-order mathematical system to investigate the spreading dynamics of dengue and malaria co-infection using the Atangana–Baleanu–Caputo (ABC) fractional derivative

for real data of thailand. The incorporation of the fractional operator enables the system to capture memory effects and hereditary properties inherent in epidemiological procedures, thereby providing a more accurate description of disease transmission. A rigorous qualitative study of the system was carried out, containing the investigation of existence and uniqueness of solutions, as well as the derivation of the basic reproduction numbers associated with malaria and dengue infections. Moreover, sensitivity analysis was performed to find out the sensitive variable affecting the spreading dynamics. Furthermore, the system was reformulate by introducing four control parameters: malaria prevention (u_1), dengue prevention (u_2), treatment of co-infected individuals (u_3), and vector control (u_4). The uses of Pontryagin's Maximum Principle enabled the characterization of optimal control profiles that reduced both the number of infected individuals and the associated implementation costs. Numerical analysis were conducted to illustrate the impact of the proposed control strategies. The results demonstrated that the combined implementation of preventive, treatment, and vector control measures significantly reduces the prevalence of both single and co-infections, as well as shortens the duration of the epidemic. In particular, early and intensive intervention was shown to be highly effective in suppressing disease transmission. Overall, this work highlights the importance of fractional-order modeling in capturing the complex dynamics of co-infectious diseases and yields important insights for developing effective public health strategies. Future research directions may include the incorporation of stochastic effects, the exploration of different fractional operators, and the application of the model to real epidemiological data for validation and policy-making purposes. We will include rainfall factor to better understand disease transmission dynamics, as well as include environmental factors across different regions of Thailand for a more detailed study.

Data availability

The epidemiological data used in this study were obtained from publicly available, aggregated surveillance sources. Malaria surveillance data for Thailand were obtained from the Malaria Division, Department of Disease Control, Ministry of Public Health, Thailand [44], and dengue surveillance data were obtained from the publicly available dengue surveillance dashboard [45]. No individual-level, personally identifiable, or confidential patient data were used or generated in this study.

Ethics approval

This study did not involve human participants, animals, human tissues, or biological specimens. It was based on mathematical modelling and publicly available, aggregated and non-identifiable epidemiological surveillance data. Therefore, ethics committee or institutional review board approval was not required.

Consent to participate

Not applicable. No human participants were recruited or directly involved in this study, and no identifiable individual-level data were used.

Consent for publication

Not applicable. No identifiable personal information, clinical images, or other information that could identify an individual was used.

Competing interests

The authors declare no competing interests.

Authors contributions Conceptualization; R.U.D., A.U., P.P.
Methodology.; R.U.D., A.U., P.P.
Mathematical modeling formal analysis; R.U.D., A.U., P.P.
Investigation and numerical simulation; R.U.D., A.U., P.P.
Visualization, and writing original draft; R.U.D., A.U., P.P.
Writing review and editing; R.U.D., A.U., P.P.
Supervision; R.U.D., A.U., P.P.
Methodology; R.U.D., A.U., P.P.
Validation; R.U.D., A.U., P.P.
Project administration; R.U.D., A.U., P.P.
Writing review and editing; R.U.D., A.U., P.P.

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