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Recurrent malaria dynamics: insight from mathematical modelling

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Abstract In this study, a new mathematical model for malaria dynamics featuring all the three categories of recurrent malaria—recrudescence, relapse and re-infection—is presented and rigorously analysed. The formulated model is a nine-dimensional system of ordinary differential equations describing the population dynamics of humans and mosquitoes interaction. The analysis carried out reveals that the model exhibits a backward bifurcation phenomenon in the presence of re-infection, which is the recurrence of malaria symptoms due to infection from new parasites, whenever the associated basic reproduction number is less than unity. However, further investigation shows that the occurrence of backward bifurcation can be successfully ruled out in the absence of re-infection. The global dynamics of the malaria model is established via Lyapunov functions method and the asymptotic behaviour of the system is quantitatively illustrated.

1 Introduction

Malaria is an acute febrile illness caused by protozoan parasites of genus *Plasmodium* spread by infectious female *Anopheles mosquitoes* when they seek blood meal for production of their eggs. There are five species of the parasites that cause malaria in humans. These parasites include: *Plasmodium falciparum*; *Plasmodium vivax*; *Plasmodium malariae*; *Plasmodium ovale*; *Plasmodium knowlesi*, of which *Plasmodium falciparum* and *Plasmodium vivax* pose the greatest threat. Malaria remains one of the major public health challenges and life-threatening diseases worldwide [1]. Currently, an estimated 228 million people is at risk of malaria and mortality rate stands at 405,000 worldwide. The disease is characterized by symptoms such as fever, headache, sweats, muscle aches, chills, tiredness, nausea, vomiting, diarrhea, anaemia, jaundice, seizures [2] and malaria symptoms in individuals usually appear about 10-15 days after the infected mosquito has taken its blood meal [3]. Malaria can be cured, but symptoms may recur if the disease is not efficiently treated and properly controlled.

Recurrent malaria is referred to as the subsequent parasitaemic episodes occurring not later than seven days after receiving anti-malaria treatment. Recurrent malaria constitutes greatest challenge to the total extinction of malaria in our community [4]. Recurrence of malaria can be caused by recrudescence, relapse and re-infection. Recrudescence is the recurrence of malaria symptoms due to the survival of malaria parasites in the blood. Relapse refers to the situation whereby symptoms reappear due to reactivation of dormant hypnozoites in the liver cells after the total clearance of the parasites from the blood. In contrast, re-infection is the reappearance of malaria symptoms due to infection from new mosquito bite [5–8].

A number of contemporary mathematical models have been developed after the pioneer works of Ross [9], Macdonald [10], and Anderson and May [11] to assess the dynamical spread of malaria in human population (see, e.g., [5,12–19] and some of the references therein). In [12], the author studied the bifurcation analysis of a mathematical model for malaria transmission using a system of ordinary differential equation via the modification of models from literature and then incorporated some more realistic features. Anguelov et al. [5] established a nonpolluting technique of curtailing the prevalence of female anopheles mosquito through the release of sterile males using compartmental model. Olaniyi and Obabiyi [17] explored the implication of nonlinear incidence function on the transmission dynamics of malaria in human population. The model incorporated the rate at which recovered humans returned back to the susceptible and their infectious states due to lack of complete acquired immunity. Mbogo et al. [15] proposed a mathematical model which is a modification of [22], where the disease dynamics of deterministic and stochastic models were compared basically to determine the effect of randomness in malaria transmission dynamics. In [16], the authors formulated a mathematical model to investigate the global stability of malaria transmission dynamics with vigilant compartment, where a normalized system of ordinary differential equation with the concept that human may not have equal likelihood of being infected with malaria parasites was considered. Traoré et al. [18] developed a system of retarded functional differential equations to investigate transmission of malaria dynamics with maturation delay of a vector population in a periodic environment.

A limited number of studies have been presented on the mathematical analysis of recurrent malaria featuring relapse and re-infection. Niger and Gumel [20] focused on the mathematical modelling of assessing the role of repeated exposure on the transmission

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dynamics of malaria disease in human population by formulating a comprehensive mathematical model. Li et al. [21] studied the fast and slow dynamics of malaria model with relapse by considering two distinct mathematical models where the first dynamic model was based on constant vector population and the second dynamic model was based on variable vector population sizes. Huo and Qui [22] formulated a mathematical model to assess the stability of malaria transmission with relapse. In a related development, Ghosh et al. [23] explored the analysis of recurrent malaria by considering only two of the phenomena of recurrent malaria representing relapse and re-infection. The model presented in this study extends the features considered in [23]. Till date, and to the best of our knowledge, the mathematical assessment of malaria transmission dynamics detailing all components of recurrent malaria has not been studied in the literature.

Therefore, in this study, recovered human population is stratified into pseudo-recovered humans population with relapse, recovered humans with possibility of re-infection, and recovered humans with recrudescence. Keeping this in mind, a more robust mathematical model of malaria featuring all the classes of recurrent malaria is analysed using stability theory of differential equations. The organization of the remaining aspects of the study is as follows: Sect. 2 presents the mathematical formulation and fundamental properties of the recurrent malaria model. The possibility of existence of bifurcation and global stability analysis of the malaria model around equilibria are presented in Sects. 3 and 4, respectively. Section 5 gives the concluding remarks of the study.

2 Formulation of the model

To develop the transmission dynamics of malaria model with recrudescence, relapse, and re-infection, the following six classes of human population are considered: susceptible human class, denoted by $S_h(t)$; exposed human class, denoted by $E_h(t)$; infectious human class $I_h(t)$; pseudo-recovered human with possible reactivation of infection (relapse), denoted by $R_{1h}(t)$; recovered human with re-infection possibility $R_{2h}(t)$; and recovered human with possibility of recrudescence, denoted by $R_{3h}(t)$. Hence, the total human population at time t, denoted by $N_h(t)$, is given by

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_{1h}(t) + R_{2h}(t) + R_{3h}(t).$$

On the other hand, the mosquito population is stratified into three classes, namely, susceptible $S_m(t)$; exposed $E_m(t)$; and infectious $I_m(t)$, so that the total mosquito population $N_m(t)$ becomes

$$N_m(t) = S_m(t) + E_m(t) + I_m(t).$$

The susceptible human class is increased by the recruitment of humans, assumed susceptible, at a rate given by Λ_h. However, due to effective contact with infectious mosquito $I_m(t)$, the susceptible human class is reduced by the standard incidence of infection, $\beta_h b S_h I_m / N_h$, where β_h and b are infection transmission probability in humans and biting rate of mosquitoes, respectively. The population of susceptible human is further decreased by the natural mortality rate μ_h. Hence, the susceptible human class changing with respect to time is given by

$$\frac{dS_h}{dt} = \Lambda_h - \frac{\beta_h b S_h I_m}{N_h} - \mu_h S_h \tag{1}$$

Following the infection of the susceptible human class and the re-infection of the recovered human class, the population of exposed human class is increased by $\beta_h b S_h I_m / N_h$ and $\varepsilon \beta_h b R_{2h} I_m / N_h$, where $\varepsilon \in (0, 1)$ is the modification parameter responsible for the reduced transmission probability of re-infection, which is due to the prior infection-acquired immunity by recovered individuals [20,23]. Moreover, the population of exposed humans is increased by the relapse of pseudo-recovered human (at a rate θ) and decreased by both progression rate of exposed humans to the infectious class ($I_h(t)$) (at a rate α_h) and natural mortality (at a rate μ_h). Hence, the exposed human class changing with time is given by

$$\frac{dE_h}{dt} = \frac{\beta_h b I_m (S_h + \varepsilon R_{2h})}{N_h} + \theta R_{1h} - (\alpha_h + \mu_h) E_h \tag{2}$$

The infectious human class, ($I_h(t)$) increases following progression of the exposed humans at a rate α_h. This class is further increased due to recrudescence at a rate τ, while the class is reduced due to both recovery from infectious state at *per capita* rate γ and the natural mortality rate μ_h. Therefore, the rate of change of the infectious human class with time is given by

$$\frac{dI_h}{dt} = \alpha_h E_h + \tau R_{3h} - (\gamma + \mu_h) I_h \tag{3}$$

Following the relapse of recovered infectious humans, the population of pseudo-recovered humans with infection at the dormant liver stage is generated by a fraction σ_1 of the *per capita* recovery rate of infectious human. The population is reduced by relapse (at a rate θ) and the natural mortality rate μ_h. Therefore, the rate of change of the pseudo-recovered human class with time is given by

$$\frac{dR_{1h}}{dt} = \sigma_1 \gamma I_h - (\theta + \mu_h) R_{1h} \tag{4}$$

The population of recovered human with re-infection possibility ($R_{2h}(t)$) is increased by a fraction σ_2 of the *per capita* recovery rate of infectious human. Following effective re-infection from infectious mosquito, this population is diminished by the standard incidence $\beta_h b\varepsilon R_{2h}/N_h$ and the natural mortality (at a rate μ_h). Thus, the rate of change of $R_{2h}(t)$ is given by

$$\frac{dR_{2h}}{dt} = \sigma_2\gamma I_h - \frac{\varepsilon\beta_h b R_{2h} I_m}{N_h} - \mu_h R_{2h} \tag{5}$$

The population of recrudescent human is increased by the remaining fraction $(1-(\sigma_1+\sigma_2))$ of the *per capita* recovery rate of infectious human. Following incomplete treatment of the disease, this population is downsized at per capita recrudescent rate τ, and also reduced by the natural mortality rate μ_h. This translates to the equation specified by

$$\frac{dR_{3h}}{dt} = (1-(\sigma_1+\sigma_2))\gamma I_h - (\tau+\mu_h)R_{3h} \tag{6}$$

Moreover, the susceptible mosquito class is generated by the recruitment rate, Λ_m, assumed susceptible and is decreased due to effective contact with both infectious human and recrudescent human via standard incidences of the form $\beta_m b S_m I_h/N_h$ and $\beta_m b\phi S_m R_{3h}/N_h$, where β_m is the probability of transmission of infection in mosquitoes and $\phi \in (0, 1)$ is the modification parameter reflecting the reduced infectiousness of recrudescent humans when compared to the infectious humans. The susceptible mosquito population is further reduced by natural *per capita* death rate μ_m, so that

$$\frac{dS_m}{dt} = \Lambda_m - \frac{\beta_m b S_m(I_h+\phi R_{3h})}{N_h} - \mu_m S_m \tag{7}$$

Following effective contact with both infectious human and recrudescent human, the class of exposed mosquito is increased by the standard incidences $\beta_m b S_m I_h/N_h$ and $\beta_m b\phi S_m R_{3h}/N_h$. The exposed mosquito population is reduced both at the progression rate α_m and natural *per capita* death rate μ_m. Therefore, the rate of change of the population yields

$$\frac{dE_m}{dt} = \frac{\beta_m b S_m(I_h+\phi R_{3h})}{N_h} - (\alpha_m+\mu_m)E_m \tag{8}$$

The population of infectious mosquito class is increased as a result of progression from the exposed class at the rare α_h. However, the population is downsized at the natural mortality rate μ_m, so that

$$\frac{dI_m}{dt} = \alpha_m E_m - \mu_m I_m \tag{9}$$

In what follows, the mathematical model describing the transmission of malaria with recrudescence, relapse, and re-infection is given by combining all the aforementioned equations to form the following system.

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \frac{\beta_h b S_h I_m}{N_h} - \mu_h S_h \\
\frac{dE_h}{dt} &= \frac{\beta_h b I_m(S_h+\varepsilon R_{2h})}{N_h} + \theta R_{1h} - (\alpha_h+\mu_h)E_h \\
\frac{dI_h}{dt} &= \alpha_h E_h + \tau R_{3h} - (\gamma+\mu_h)I_h \\
\frac{dR_{1h}}{dt} &= \sigma_1\gamma I_h - (\theta+\mu_h)R_{1h} \\
\frac{dR_{2h}}{dt} &= \sigma_2\gamma I_h - \frac{\varepsilon\beta_h b R_{2h} I_m}{N_h} - \mu_h R_{2h} \\
\frac{dR_{3h}}{dt} &= (1-(\sigma_1+\sigma_2))\gamma I_h - (\tau+\mu_h)R_{3h} \\
\frac{dS_m}{dt} &= \Lambda_m - \frac{\beta_m b S_m(I_h+\phi R_{3h})}{N_h} - \mu_m S_m \\
\frac{dE_m}{dt} &= \frac{\beta_m b S_m(I_h+\phi R_{3h})}{N_h} - (\alpha_m+\mu_m)E_m \\
\frac{dI_m}{dt} &= \alpha_m E_m - \mu_m I_m
\end{aligned} \tag{10}$$

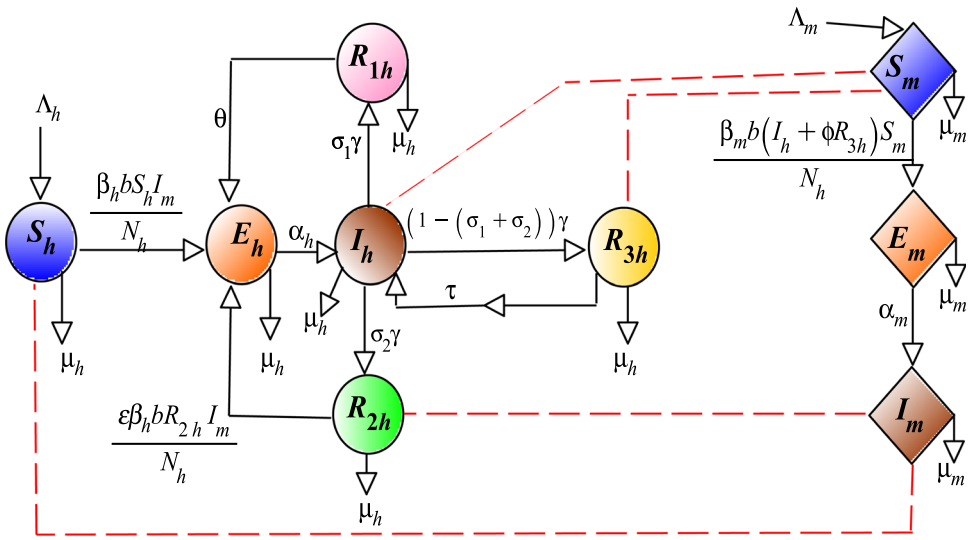

Fig. 1 Schematic diagram for the full recurrent malaria dynamics (10)

Table 1 The description of variables of the malaria model (10)

Variable	Description
$S_h(t)$	Population of susceptible humans
$E_h(t)$	Population of exposed humans
$I_h(t)$	Population of infectious humans
$R_{1h}(t)$	Population of recovered humans with relapse
$R_{2h}(t)$	Population of recovered humans prone to re-infection
$R_{3h}(t)$	Population of recovered humans with possibility of recrudescence
$N_h(t)$	Total human population
$S_m(t)$	Population of susceptible mosquitoes
$E_m(t)$	Population of exposed mosquitoes
$I_m(t)$	Population of infectious mosquitoes
$N_m(t)$	Total mosquito population

A schematic diagram representing the formulation of the model (10) is given in Fig. 1, while the descriptions of the variables and parameters of the model (10) are provided in Tables 1 and 2, respectively.

2.1 Fundamental properties

To begin with, since the parameters of the model governed by system (10) address the interaction between humans and mosquitoes, then, it is worthy of mentioning that, all the associated parameters and state variables of the model are non-negative for all times. Hence, it is cogent to show that all the variables of the model are also non-negative for $t > 0$.

2.1.1 Invariant region and positivity of solutions

Lemma 1 *The feasible region of the malaria model* (10)*, given by* $\mathfrak{D} = \mathfrak{D}_h \times \mathfrak{D}_m \subset \mathbb{R}^6_+ \times \mathbb{R}^3_+$*, where*

$$\mathfrak{D}_h = \left\{(S_h, E_h, I_h, R_{1h}, R_{2h}, R_{3h}) \in \mathbb{R}^6_+ : S_h + E_h + I_h + R_{1h} + R_{2h} + R_{3h} \leq \frac{\Lambda_h}{\mu_h}\right\} \text{ and}$$

$$\mathfrak{D}_m = \left\{(S_m, E_m, I_m) \in \mathbb{R}^3_+ : S_m + E_m + I_m \leq \frac{\Lambda_m}{\mu_m}\right\},$$

is positively-invariant and attracting.

Proof It is straightforward from the rates of change of the total human population $N_h(t)$ and total mosquito population $N_m(t)$, respectively, that

$$N_h(t) \leq N_h(0)\exp(-\mu_h t) + \frac{\Lambda_h}{\mu_h}(1 - \exp(-\mu_h t))$$

Table 2 The description of parameters of the malaria model (10)

Parameter	Description
Λ_h	Recruitment rate into human population
Λ_m	Recruitment rate into mosquito population
β_h	Transmission probability of infection in humans
β_m	Transmission probability of infection in mosquitoes
b	Mosquitoes biting rate
μ_h	Natural mortality rate of humans
μ_m	Natural mortality rate of mosquitoes
α_h	Progression rate of humans from exposed class to the infectious class
α_m	Progression rate of mosquitoes from the exposed class to the infectious class
θ	Relapse rate of recovered humans in $R_{1h}(t)$ class
ε	Modification parameter due to re-infection of humans in $(R_{2h}(t))$ class
τ	Recrudescence rate of humans in $R_{3h}(t)$ class
γ	Recovery rate of infectious humans
σ_1	Fraction of recovered humans that goes to $R_{1h}(t)$ class
σ_2	Fraction of recovered humans that goes to $R_{2h}(t)$ class
$(1-(\sigma_1+\sigma_2))$	Fraction of recovered humans that goes to $R_{3h}(t)$ class
ϕ	Modification parameter due to infectiousness of humans in $R_{3h}(t)$ class

and

$$N_m(t) \leq N_m(0)\exp(-\mu_m t) + \frac{\Lambda_m}{\mu_m}(1-\exp(-\mu_m t)).$$

Therefore, if $N_h(0) \leq \frac{\Lambda_h}{\mu_h}$ and $N_m(0) \leq \frac{\Lambda_m}{\mu_m}$, then, $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ and $N_m(t) \leq \frac{\Lambda_m}{\mu_m}$. Thus, the region $\mathfrak{D}$ is positively-invariant. Further, if $N_h(0) \geq \frac{\Lambda_h}{\mu_h}$ and $N_m(0) \geq \frac{\Lambda_m}{\mu_m}$, then the solution either enters $\mathfrak{D}$ in finite time or both N_h approaches $\frac{\Lambda_h}{\mu_h}$ and N_m approaches $\frac{\Lambda_m}{\mu_m}$ asymptotically as $t \to \infty$. Hence, the region $\mathfrak{D}$ attracts all solutions in $\mathbb{R}^9_+$.

On this note, it is therefore suffices to consider the dynamics of malaria transmission governed by a system of differential equation (10) in the biologically feasible region $\mathfrak{D}$, where the model is epidemiologically and mathematically well posed. □

Theorem 1 *The solution set,* $\{(S_h, E_h, I_h, R_{1h}, R_{2h}, R_{3h}, S_m, E_m, I_m)\}$ *of the malaria model* (10) *with positive initial data* $S_h(0)$, $E_h(0)$, $I_h(0)$, $R_{1h}(0)$, $R_{2h}(0)$, $R_{3h}(0)$, $S_m(0)$, $E_m(0)$, *and* $I_m(0)$) *in* $\mathfrak{D}$, *remain positive in* $\mathfrak{D}$ *for all time,* $t > 0$.

Proof The first equation of the model (10) gives rise to the following differential inequality

$$\frac{dS_h}{dt} \geq -\left(\frac{\beta_h b I_m}{N_h} + \mu_h\right) S_h.$$

so that,

$$\frac{d}{dt}\left[S_h(t)\exp\left(-\int_0^t \frac{\beta_h b I_m(\psi)}{N_h(\psi)}d\psi + \mu_h t\right)\right] > 0$$

It therefore follows that,

$$S_h(t) \geq S_h(0)\exp\left[-\int_0^t \frac{\beta_h b I_m(\psi)}{N_h(\psi)}d\psi + \mu_h t\right] > 0, \quad \text{for all} \quad t > 0.$$

The remaining state variables E_h, I_h, R_{1h}, R_{2h}, R_{3h}, S_m, E_m, I_m can also be shown in a similar manner. Hence, the solution set $(S_h, E_h, I_h, R_{1h}, R_{2h}, R_{3h}, S_m, E_m, I_m)$ is non-negative $\forall\, t > 0$. □

3 Equilibrium points and bifurcation analysis

This section explores the existence of equilibrium points and the type of bifurcation the model (10) exhibits.

3.1 Disease-free equilibrium

At malaria-free equilibrium, it is assumed that there is no infection, such that, all the infection related variables in the malaria model (10) are set to zero. Therefore, the disease-free equilibrium of the malaria model (10), designated by $\mathcal{E}_0$, is given by

$$\mathcal{E}_0 = (S_h^*, E_h^*, I_h^*, R_{1h}^*, R_{2h}^*, R_{3h}^*, S_m^*, E_m^*, I_m^*) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0\right) \tag{11}$$

The basic reproduction number, $\mathcal{R}_0$, of the malaria model (10) can be obtained using the well-known next generation operator approach as described in [24]. Evaluating the matrices F (of the new infection terms) and V (of the transition terms) at the given point $\mathcal{E}_0$ (11), respectively, gives

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & \beta_h b \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_m b \Lambda_m \mu_h}{\Lambda_h \mu_m} & 0 & \frac{\beta_m b \phi \Lambda_m \mu_h}{\Lambda_h \mu_m} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} k_1 & 0 & -\theta & 0 & 0 & 0 \\ -\alpha_h & k_2 & 0 & -\tau & 0 & 0 \\ 0 & -\sigma_1\gamma & k_3 & 0 & 0 & 0 \\ 0 & x & 0 & k_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & k_5 & 0 \\ 0 & 0 & 0 & 0 & -\alpha_m & \mu_m \end{pmatrix},$$

where $k_1 = (\alpha_h + \mu_h)$, $k_2 = (\gamma + \mu_h)$, $k_3 = (\theta + \mu_h)$, $k_4 = (\tau + \mu_h)$, $k_5 = (\alpha_m + \mu_m)$ and $x = [1 - (\sigma_1 + \sigma_2)]\gamma$. It follows that the spectral radius of FV^{-1}, denoted by $\rho(FV^{-1})$, is the basic reproduction number given by

$$\mathcal{R}_0 = \sqrt{\frac{\beta_h b^2 \alpha_m \beta_m \mu_h \Lambda_m \alpha_h k_3 (k_4 + \phi x)}{\mu_m^2 \Lambda_h k_5 [k_1 k_3 (k_2 k_4 - \tau x) - \theta \alpha_h \sigma_1 \gamma k_4]}}. \tag{12}$$

Keeping in mind that the denominator in (12) is positive by algebraic simplification. Thus, it follows that

$$\begin{aligned} \mu_m^2 \Lambda_h k_5 [k_1 k_3 (k_2 k_4 - \tau x) - \theta \alpha_h \sigma_1 \gamma k_4] = \mu_m^2 \Lambda_h k_5 [\mu_h k_3 [\tau k_1 + \mu_h k_2 + \tau(\sigma_1 + \sigma_2)\gamma] \\ + \alpha_h \mu_h [\mu_h (\theta + k_2) + \tau(\sigma_1 + \sigma_2)\gamma] + \theta \alpha_h [\mu_h (1 - \sigma_1) + \tau \sigma_2]] \end{aligned} \tag{13}$$

The basic reproduction number, $\mathcal{R}_0$, of the malaria model (10) is the threshold parameter that determines the spread potential of the disease when an infectious individual enters a completely susceptible population.

The local asymptotic stability of the disease-free equilibrium (DFE) (11) follows from Theorem 2 in [24]. Hence, the following result is claimed.

Lemma 2 *The DFE, denoted by $\mathcal{E}_0$, of the malaria model (10) is locally asymptotically stable in $\mathfrak{D}$ if $\mathcal{R}_0 < 1$ and unstable otherwise.*

The epidemiological implication of Lemma 2 is that malaria can be effectively curtailed in the population if the initial sizes of the infected individuals (humans and mosquitoes) of the malaria model (10) are in the basin of attraction of the DFE, such that $\mathcal{R}_0 < 1$.

3.2 Endemic equilibrium

The endemic equilibrium point of the malaria model (10) is a non-trivial steady state where the disease is present in the population. Due to the complexity of the model which incorporates the features of recurrent malaria, the analytical expression of the endemic equilibrium point is not shown, as multiple endemic equilibria could be obtained. This possibility leads to the investigation of bifurcation property of the malaria model (10) in the next section.

3.3 Bifurcation property

The center manifold theory made popular by Castillo-Chavez and Song [25] is employed to show the type of bifurcation that the malaria model (10) will exhibit. To apply this theory, it is convenient to perform the following changes of variables.

Let the malaria model (10) be written in the vector form $dX/dt = F(X)$, where $X = (x_1, x_2, \ldots, x_9)^\intercal$ and $F = (f_1, f_2, \ldots, f_9)^\intercal$, so that $S_h = x_1$, $E_h = x_2$, $I_h = x_3$, $R_{1h} = x_4$, $R_{2h} = x_5$, $R_{3h} = x_6$, $S_m = x_7$, $E_m = x_8$, $I_m = x_9$. Then, the malaria model (10) becomes:

$$\begin{aligned}
\frac{dx_1}{dt} &\equiv f_1 = \Lambda_h - \frac{\beta_h b x_1 x_9}{\sum_{i=1}^{6} x_i} - \mu_h x_1 \\
\frac{dx_2}{dt} &\equiv f_2 = \frac{\beta_h b x_9 (x_1 + \varepsilon x_5)}{\sum_{i=1}^{6} x_i} + \theta x_4 - (\alpha_h + \mu_h) x_2 \\
\frac{dx_3}{dt} &\equiv f_3 = \alpha_h x_2 + \tau x_6 - (\gamma + \mu_h) x_3 \\
\frac{dx_4}{dt} &\equiv f_4 = \sigma_1 \gamma x_3 - (\theta + \mu_h) x_4 \\
\frac{dx_5}{dt} &\equiv f_5 = \sigma_2 \gamma x_3 - \frac{\varepsilon \beta_h b x_5 x_9}{\sum_{i=1}^{6} x_i} - \mu_h x_5 \\
\frac{dx_6}{dt} &\equiv f_6 = (1 - (\sigma_1 + \sigma_2))\gamma x_3 - (\tau + \mu_h) x_6 \\
\frac{dx_7}{dt} &\equiv f_7 = \Lambda_m - \frac{\beta_m b x_7 (x_3 + \phi x_6)}{\sum_{i=1}^{6} x_i} - \mu_m x_7 \\
\frac{dx_8}{dt} &\equiv f_8 = \frac{\beta_m b x_7 (x_3 + \phi x_6)}{\sum_{i=1}^{6} x_i} - (\alpha_m + \mu_m) x_8 \\
\frac{dx_9}{dt} &\equiv f_9 = \alpha_m x_8 - \mu_m x_9
\end{aligned} \tag{14}$$

Consider the case when $\beta_h = \beta_h^*$ is chosen as the bifurcation parameter. Solving for $\beta_h = \beta_h^*$ at $\mathcal{R}_0 = 1$ in (12) yields

$$\beta_h^* = \frac{\mu_m^2 \Lambda_h k_5 [k_1 k_3 (k_2 k_4 - \tau x) - \theta \alpha_h \sigma_1 \gamma k_4]}{b^2 \alpha_m \beta_m \mu_h \Lambda_m \alpha_h k_3 (k_4 + \phi[1 - (\sigma_1 + \sigma_2)]\gamma)}$$

The Jacobian matrix of (14) evaluated at disease-free equilibrium ($\mathcal{E}_0$) with $\beta_h = \beta_h^{**}$, is given by

$$\mathcal{J}(\mathcal{E}_0)\,|_{\beta_h^*} = \begin{pmatrix}
-\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\beta_h^* b \\
0 & -k_1 & 0 & \theta & 0 & 0 & 0 & 0 & \beta_h^* b \\
0 & \alpha_h & -k_2 & 0 & 0 & \tau & 0 & 0 & 0 \\
0 & 0 & \sigma_1 \gamma & -k_3 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \sigma_2 \gamma & 0 & -\mu_h & 0 & 0 & 0 & 0 \\
0 & 0 & x & 0 & 0 & -k_4 & 0 & 0 & 0 \\
0 & 0 & -y & 0 & 0 & -z & -\mu_m & 0 & 0 \\
0 & 0 & y & 0 & 0 & z & 0 & -k_5 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha_m & -\mu_m
\end{pmatrix}, \tag{15}$$

where $k_1 = (\alpha_h + \mu_h), k_2 = (\gamma + \mu_h), k_3 = (\theta + \mu_h), k_4 = (\tau + \mu_h), k_5 = (\alpha_m + \mu_m), x = [1 - (\sigma_1 + \sigma_2)]\gamma, y = (\beta_m b \mu_h \Lambda_m)/\Lambda_h \mu_m$, $z = (\phi \beta_m b \mu_h \Lambda_m)/\Lambda_h \mu_m$.

Solving for the eigenvalues of the Jacobian matrix (15) gives simple zero eigenvalue and the other eight eigenvalues having negative real part. It follows that a right eigenvector corresponding to the simple zero eigenvalue is given by $\mathbf{w} = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9)^\intercal$, where

$$\begin{aligned}
w_1 &= -\left[\frac{\beta_h^* b^2 \alpha_m \beta_m \mu_h \Lambda_m (k_4 + \phi[1 - (\sigma_1 + \sigma_2)]\gamma)}{\Lambda_h \mu_h \mu_m^2 k_4 k_5}\right] w_3, \\
w_2 &= \frac{\theta \sigma_1 \gamma \Lambda_h \mu_m^2 k_4 k_5 + \alpha_m \beta_h^* b^2 \beta_m \mu_h \Lambda_m (k_4 + \phi[1 - (\sigma_1 + \sigma_2)]\gamma)}{\Lambda_h \mu_m^2 k_1 k_3 k_4 k_5} w_3, \\
w_3 &= w_3 > 0, \qquad w_4 = \frac{\sigma_1 \gamma}{k_3} w_3,
\end{aligned}$$

$$
\begin{aligned}
w_5 &= \frac{\sigma_2\gamma}{\mu_h}w_3 \qquad w_6 = \frac{[1-(\sigma_1+\sigma_2)]\gamma}{k_4}w_3,\\
w_7 &= -\left[\frac{\beta_m b\mu_h\Lambda_m(k_4+\phi[1-(\sigma_1+\sigma_2)]\gamma)}{\Lambda_h\mu_m^2k_4}\right]w_3,\\
w_8 &= \frac{\beta_m b\mu_h\Lambda_m(k_4+\phi[1-(\sigma_1+\sigma_2)]\gamma)}{\Lambda_h\mu_m k_4k_5}w_3,\\
w_9 &= \frac{\alpha_m\beta_m b\mu_h\Lambda_m(k_4+\phi[1-(\sigma_1+\sigma_2)]\gamma)}{\Lambda_h\mu_m^2k_4k_5}w_3.
\end{aligned}
\tag{16}
$$

In addition to the right eigenvectors, the Jacobian matrix has a left eigenvector $\mathbf{v} = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9)^\intercal$, where

$$
\begin{aligned}
&v_1 = 0, \qquad v_2 = \frac{\alpha_h}{k_1}v_3,\\
&v_3 = v_3 > 0, \qquad v_4 = \frac{\theta\alpha_h}{k_1k_3}v_3, \qquad v_5 = 0,\\
&v_6 = \frac{\tau\Lambda_h\mu_m^2k_1k_5+\phi\alpha_h\beta_h^*\alpha_m\beta_m b^2\mu_h\Lambda_m}{\Lambda_h\mu_m^2k_1k_4k_5}v_3,\\
&v_7 = 0, \qquad v_8 = \frac{\alpha_h\alpha_m\beta_h^*b}{\mu_m k_1k_5}v_3,\\
&v_9 = \frac{\alpha_h\beta_h^*b}{\mu_m k_1}v_3.
\end{aligned}
\tag{17}
$$

Noting that $\mathbf{v.w} = 1$ as required in [25]. The non-varnishing partial derivatives (evaluated at $\mathcal{E}_0$ with $\beta_h = \beta_h^*$) of the right hand side of the system (14) are given by

$$
\begin{aligned}
&\frac{\partial^2 f_2}{\partial x_5\partial x_9} = \frac{\varepsilon\beta_h^*b\mu_h}{\Lambda_h}, \qquad \frac{\partial^2 f_5}{\partial x_5\partial x_9} = -\frac{\varepsilon\beta_h^*b\mu_h}{\Lambda_h},\\
&\frac{\partial^2 f_7}{\partial x_3\partial x_7} = -\frac{\beta_m b\mu_h}{\Lambda_h}, \qquad \frac{\partial^2 f_7}{\partial x_6\partial x_7} = -\frac{\phi\beta_m b\mu_h}{\Lambda_h},\\
&\frac{\partial^2 f_8}{\partial x_3\partial x_7} = \frac{\beta_m b\mu_h}{\Lambda_h}, \qquad \frac{\partial^2 f_8}{\partial x_6\partial x_7} = \frac{\phi\beta_m b\mu_h}{\Lambda_h},\\
&\frac{\partial^2 f_1}{\partial x_9\partial\beta_h} = -b, \qquad \frac{\partial^2 f_2}{\partial x_9\partial\beta_h} = b.
\end{aligned}
\tag{18}
$$

Using the partial derivatives in (18), the bifurcation coefficient **a** is computed as follows

$$
\begin{aligned}
\mathbf{a} &= \sum_{k,i,j=1}^{9} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i\partial x_j}(\mathcal{E}_0)\\
&= \frac{\alpha_h\sigma_2\gamma\varepsilon\beta_h^*b\mu_h A}{\Lambda_h^2\mu_h\mu_m^2k_1k_4k_5}v_3w_3^2 - \frac{\alpha_h\beta_h^*b^2\beta_m\mu_h A}{\Lambda_h^2\mu_m^3k_1k_4}(1+\phi x k_4)v_3w_3^2,
\end{aligned}
\tag{19}
$$

where $A = \alpha_m\beta_m b\mu_h\Lambda_m(k_4+\phi x)$ and $x = [1-(\sigma_1+\sigma_2)]\gamma$. Further, the bifurcation coefficient **b** is obtained by

$$
\begin{aligned}
\mathbf{b} &= \sum_{k,i=1}^{9} v_k w_i \frac{\partial^2 f_k}{\partial x_i\partial\beta_h}(\mathcal{E}_0)\\
&= \left[\frac{\alpha_h\alpha_m\beta_m b^2\mu_h\Lambda_m(k_4+\phi x)}{\Lambda_h\mu_m^2k_1k_4k_5}\right]v_3w_3.
\end{aligned}
\tag{20}
$$

Since the parameters of the model are positive, then it is expected that the bifurcation coefficient **b** is positive. Therefore, the sign of the bifurcation coefficient **a** determines the type of bifurcation to be exhibited by the malaria model (10). The following results is then established using the Theorem 4.1 in [25].

Theorem 2 *The malaria model* (10) *exhibits backward bifurcation as* $\mathcal{R}_0$ *crosses unity whenever the bifurcation coefficient* $\boldsymbol{a}$ *in* (19) *is positive. That is,*

$$\frac{\alpha_h \sigma_2 \gamma \varepsilon \beta_h^* b \mu_h A}{\Lambda_h^2 \mu_h \mu_m^2 k_1 k_4 k_5} v_3 w_3^2 > \frac{\alpha_h \beta_h^* b^2 \beta_m \mu_h A}{\Lambda_h^2 \mu_m^3 k_1 k_4} (1 + \phi x k_4) v_3 w_3^2. \tag{21}$$

The occurrence of backward bifurcation in epidemiological models makes the disease control difficult. In other words, the necessary requirement of having $\mathcal{R}_0 < 1$, when backward bifurcation occurs, is not sufficient to control malaria effectively in the community (see, [20,23]). Thus, the implication of Theorem 2 is that the possibility of controlling malaria when $\mathcal{R}_0 < 1$ would be dependent on the initial sizes of the infected sub-populations of the malaria model (10). Now, to rule out the occurrence of this backward bifurcation, the next analysis is carried out.

3.3.1 Non-existence of backward bifurcation

The malaria model (10) may not undergo a backward bifurcation property if an endemic equilibrium does not exist at $\mathcal{R}_0 < 1$. To ensure the non-existence of endemic equilibrium when $\mathcal{R}_0 < 1$, let the modification parameter due to re-infection, denoted by ε, be set to zero, i.e. $\varepsilon = 0$. Thus, the malaria model (10) with $\varepsilon = 0$ will possess unique endemic equilibrium when $\mathcal{R}_0 > 1$. This result is summarized as follows.

Theorem 3 *The malaria model (10) in the absence of re-infection* ($\varepsilon = 0$) *has no endemic equilibrium whenever* $\mathcal{R}_0 < 1$*, and a unique endemic equilibrium point exists whenever* $\mathcal{R}_0 > 1$.

It is worthy of note that the presence or absence of both relapse rate, θ, and recrudescence rate, τ, does not affect the backward bifurcation property of the malaria model (10). Thus, Theorem 3 indicates that the possibility of backward bifurcation can only be ruled out when re-infection seizes to occur. This result is further confirmed by setting $\varepsilon = 0$ in (19), so that the bifurcation coefficient **a** becomes

$$\mathbf{a} = -\frac{\alpha_h \beta_h^* b^2 \beta_m \mu_h A}{\Lambda_h^2 \mu_m^3 k_1 k_4} (1 + \phi x k_4) v_3 w_3^2 < 0. \tag{22}$$

Since $\mathbf{a} < 0$, it follows that the malaria model (10) with $\varepsilon = 0$ will not undergo backward bifurcation property at $\mathcal{R}_0 = 1$. Hence, the following result, which is equivalent to Theorem 3 is claimed.

Theorem 4 *The malaria model (10) in the absence of re-infection rate, (i.e., when* $\varepsilon = 0$*), exhibits forward bifurcation as* $\mathcal{R}_0$ *crosses unity.*

The epidemiological implication of Theorem 4 is that the disease will only persist at the basic reproduction number above the unity. It also implies that the unique endemic equilibrium of the malaria model is locally asymptotically stable, meaning that a small influx of infected humans or mosquitoes will cause malaria to persist in the population. This endemism is, however, dependent on the initial sizes of the infected individuals in the population.

4 Global asymptotic dynamics of the model

In this section, the global asymptotic dynamics of the malaria model (10) around the disease-free equilibrium ($\mathcal{E}_0$) and endemic equilibrium (ε_e) are explored to show the behaviour of the model regardless of the initial sizes of the infected individuals in the population.

4.1 Global asymptotic stability of $\mathcal{E}_0$

Theorem 5 *The disease-free equilibrium point* $\mathcal{E}_0$*, given in* (11)*, of the model (10) in the absence of re-infection rate* ε*, is globally asymptotically stable if the basic reproduction* $\mathcal{R}_0$ *is less than one.*

Proof Consider the continuously differentiable linear Lyapunov function [26–29] given by

$$V = v_1 E_h + v_2 I_h + v_3 R_{1h} + v_4 R_{3h} + v_5 E_m + v_6 I_m, \tag{23}$$

where

$$v_1 = \frac{\alpha_h k_3(k_4+\phi x)}{B},\ v_2 = \frac{k_1 k_3(k_4+\phi x)}{B},\ v_3 = \frac{\theta\alpha_h(k_4+\phi x)}{B},$$

$$v_4 = \frac{\tau k_1 k_3 + \phi(k_4+\phi x-\theta\alpha_h\sigma_1\gamma)}{B},\ v_5 = \sqrt{\frac{\Lambda_h\beta_h\alpha_m\alpha_h k_3(k_4+\phi x)}{\beta_m\mu_h\Lambda_m k_5 B}},$$

$$v_6 = \sqrt{\frac{\Lambda_h\beta_h\alpha_h k_3 k_5(k_4+\phi x)}{\alpha_m\beta_m\mu_h\Lambda_m B}},$$

and where $B = [k_1k_3(k_2k_4-\tau x)-\theta\alpha_h\sigma_1\gamma k_4] > 0$ as shown in (13). The time derivative of the Lyapunov function (23), denoted by $\dot{V}$, along the solution path of (10) with $\varepsilon = 0$, is given by

$$\begin{aligned}\dot{V} =& \frac{\alpha_h k_3(k_4+\phi x)}{B}\left[\frac{\beta_h b I_m S_h}{N_h} + \theta R_{1h} - (\alpha_h+\mu_h)E_h\right] + \frac{k_1k_3(k_4+\phi x)}{B}\\ &\times\left[\alpha_h E_h + \tau R_{3h} - (\gamma+\mu_h)I_h\right] + \frac{\theta\alpha_h(k_4+\phi x)}{B}\left[\sigma_1\gamma I_h - (\theta+\mu_h)R_{1h}\right]\\ &+ \frac{\tau k_1k_3+\phi(k_4+\phi x-\theta\alpha_h\sigma_1\gamma)}{B}\left[(1-(\sigma_1+\sigma_2))\gamma I_h - (\tau+\mu_h)R_{3h}\right]\\ &+ \sqrt{\frac{\Lambda_h\beta_h\alpha_m\alpha_h k_3(k_4+\phi x)}{\beta_m\mu_h\Lambda_m k_5 B}}\left[\frac{\beta_m b S_m(I_h+\phi R_{3h})}{N_h} - (\alpha_m+\mu_m)E_m\right]\\ &+ \sqrt{\frac{\Lambda_h\beta_h\alpha_h k_3 k_5(k_4+\phi x)}{\alpha_m\beta_m\mu_h\Lambda_m B}}\left[\alpha_m E_m - \mu_m I_m\right]\end{aligned} \tag{24}$$

Algebraic simplification of (24) gives

$$\begin{aligned}\dot{V} =& \left(\frac{\alpha_h\beta_h b k_3(k_4+\phi x)S_h}{BN_h} - \sqrt{\frac{\mu_m^2\Lambda_h\beta_h\alpha_h k_3k_5(k_4+\phi x)}{\alpha_m\beta_m\mu_h\Lambda_m B}}\right)I_m - I_h - \phi R_{3h}\\ &+ \frac{\beta_m b S_m}{N_h}\sqrt{\frac{\Lambda_h\beta_h\alpha_m\alpha_h k_3(k_4+\phi x)}{\beta_m\mu_h\Lambda_m k_5 B}}(I_h+\phi R_{3h}),\end{aligned}$$

which, after substituting the limiting value $N_h = \Lambda_h/\mu_h$ together with the bounds $S_h \le \Lambda_h/\mu_h$ and $S_m \le \Lambda_m/\mu_m$ in $\mathfrak{D}$, becomes

$$\begin{aligned}\dot{V} \le& \left(\frac{\alpha_h\beta_h b k_3(k_4+\phi x)}{B} - \sqrt{\frac{\mu_m^2\Lambda_h\beta_h\alpha_h k_3k_5(k_4+\phi x)}{\alpha_m\beta_m\mu_h\Lambda_m B}}\right)I_m\\ &+ \left(\frac{\beta_m b\Lambda_m\mu_m}{\mu_m\Lambda_h}\sqrt{\frac{\Lambda_h\beta_h\alpha_m\alpha_h k_3(k_4+\phi x)}{\beta_m\mu_h\Lambda_m k_5 B}} - 1\right)I_h\\ &+ \left(\frac{\beta_m b\Lambda_m\mu_m}{\mu_m\Lambda_h}\sqrt{\frac{\Lambda_h\beta_h\alpha_m\alpha_h k_3(k_4+\phi x)}{\beta_m\mu_h\Lambda_m k_5 B}} - 1\right)\phi R_{3h}\\ =& \left(\sqrt{\frac{\mu_m^2\Lambda_h\beta_h\alpha_h k_3k_5(k_4+\phi x)}{\alpha_m\beta_m\mu_h\Lambda_m B}}(\mathcal{R}_0-1)\right)I_m + (\mathcal{R}_0-1)I_h + \phi(\mathcal{R}_0-1)R_{3h}\end{aligned}$$

This shows that $\dot{V} < 0$ for $\mathcal{R}_0 < 1$, and $\dot{V} = 0$ provided that $I_h = I_m = R_{3h} = 0$. Then, it follows that $(S_h^*, E_h^*, I_h^*, R_{1h}^*, R_{2h}^*, R_{3h}^*, S_m^*, E_m^*, I_m^*) = (\Lambda_h/\mu_h, 0, 0, 0, 0, 0, \Lambda_m/\mu_m, 0, 0)$ as $t \to \infty$. Accordingly, by LaSalle's invariance

principle [30], the largest compact invariant set for which $\dot{V} = 0$ is the singleton $\{\mathcal{E}_0\}$. Hence, the disease-free equilibrium, $\mathcal{E}_0$, of the model (10) is globally-asymptotically stable. □

The asymptotic behaviour of the model (10) is quantitatively illustrated at different initial sizes of the sub-population of the model (see, Fig. 2).

4.2 Global asymptotic stability of endemic equilibrium

Following the non-existence of backward bifurcation of the model (10) without re-infection as shown in Theorem 3 and Theorem 4, a special case of the malaria model (10), in the absence of the re-infection compartment R_{2h}, is hereby presented as follows.

$$\frac{dS_h}{dt} = \Lambda_h - \frac{\beta_h b S_h I_m}{N_h} - \mu_h S_h$$

$$\frac{dE_h}{dt} = \frac{\beta_h b I_m S_h}{N_h} + \theta R_{1h} - (\alpha_h + \mu_h) E_h$$

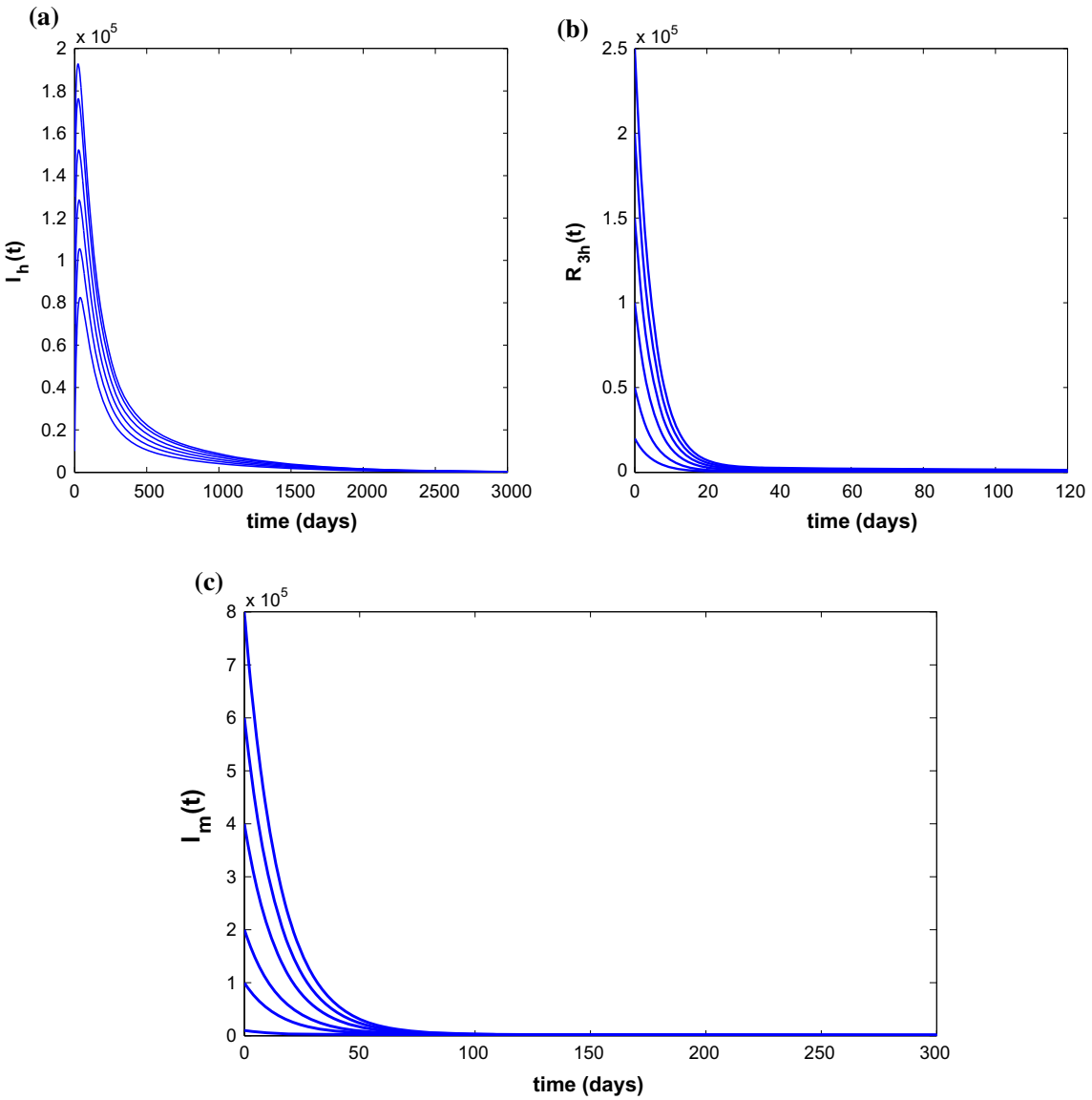

Fig. 2 Simulations showing global asymptotic behaviour of the recurrent malaria model (25) with different initial conditions. Parameter values used include $b = 0.3$, $\beta_h = 0.05$, $\beta_m = 0.15$, $\Lambda_h = 2.5$, $\Lambda_m = 1000$, $\mu_h = 0.0000548$, $\mu_m = 0.066$, $\sigma_1 = 0.25$, $\sigma_2 = 0.5$, $\varepsilon = 0$, $\theta = 0.0028$, $\gamma = 0.01$, $\phi = 0.75$, $\alpha_m = 1/18$, $\alpha_h = 1/17$, $\tau = 0.2$, so that $\mathcal{R}_0 = 0.5537 < 1$

$$\begin{aligned}
\frac{dI_h}{dt} &= \alpha_h E_h + \tau R_{3h} - (\gamma + \mu_h) I_h \\
\frac{dR_{1h}}{dt} &= \sigma_1 \gamma I_h - (\theta + \mu_h) R_{1h} \\
\frac{dR_{3h}}{dt} &= (1-\sigma_1)\gamma I_h - (\tau + \mu_h) R_{3h} \\
\frac{dS_m}{dt} &= \Lambda_m - \frac{\beta_m b S_m (I_h + \phi R_{3h})}{N_h} - \mu_m S_m \\
\frac{dE_m}{dt} &= \frac{\beta_m b S_m (I_h + \phi R_{3h})}{N_h} - (\alpha_m + \mu_m) E_m \\
\frac{dI_m}{dt} &= \alpha_m E_m - \mu_m I_m
\end{aligned} \tag{25}$$

Let the unique endemic equilibrium of the model (25) be arbitrarily given by

$$\varepsilon_e = (S_h^{**}, E_h^{**}, I_h^{**}, R_{1h}^{**}, R_{3h}^{**}, S_m^{**}, E_m^{**}, I_m^{**}).$$

Since the model (25) is obtained by setting σ_2 and ε to zero, the corresponding basic reproduction number for the special case model (25) is now given by

$$\mathcal{R}_0|_{\sigma_2=0} = \sqrt{\frac{\beta_h b^2 \alpha_m \beta_m \mu_h \Lambda_m \alpha_h k_3 (k_4 + \phi(1-\sigma_1)\gamma)}{\mu_m^2 \Lambda_h k_5 [k_1 k_3 (k_2 k_4 - \tau(1-\sigma_1)\gamma)) - \theta \alpha_h \sigma_1 \gamma k_4]}} \tag{26}$$

Therefore, the global asymptotic stability of the model (25) around the endemic equilibrium ε_e is investigated next.

Theorem 6 *The endemic equilibrium point of system (25), denoted by ε_e, is globally-asymptotically stable when $\mathcal{R}_0|_{\sigma_2=0} > 1$ with $S_m^{**} E_m \le S_m E_m^{**}$ and $R_{1h} \le R_{1h}^{**}$.*

Proof Consider the following nonlinear Lyapunov function of Goh-Volterra type [31–34]

$$\begin{aligned}
D = {} & d_1 \left(S_h - S_h^{**} - S_h^{**} \ln \frac{S_h}{S_h^{**}} \right) + d_2 \left(E_h - E_h^{**} - E_h^{**} \ln \frac{E_h}{E_h^{**}} \right) \\
& + d_3 \left(I_h - I_h^{**} - I_h^{**} \ln \frac{I_h}{I_h^{**}} \right) + d_4 \left(R_{1h} - R_{1h}^{**} - R_{1h}^{**} \ln \frac{R_{1h}}{R_{1h}^{**}} \right) \\
& + d_5 \left(R_{3h} - R_{3h}^{**} - R_{3h}^{**} \ln \frac{R_{3h}}{R_{3h}^{**}} \right) + d_6 \left(S_m - S_m^{**} - S_m^{**} \ln \frac{S_m}{S_m^{**}} \right) \\
& + d_7 \left(E_m - E_m^{**} - E_m^{**} \ln \frac{E_m}{E_m^{**}} \right) + d_8 \left(I_m - I_m^{**} - I_m^{**} \ln \frac{I_m}{I_m^{**}} \right),
\end{aligned} \tag{27}$$

where $d_1 = d_2 = \frac{\beta_m S_m^{**} (I_h^{**} + \phi R_{3h}^{**})}{\beta_h S_h^{**} I_m^{**}}$, $\quad d_3 = \frac{\beta_m S_m^{**} (I_h^{**} + \phi R_{3h}^{**})}{\alpha_h E_h^{**}} \left[\frac{b\mu_h}{\Lambda_h} + \frac{\theta R_{1h}^{**}}{\beta_h S_h^{**} I_m^{**}} \right]$,

$d_4 = \frac{\beta_m S_m^{**} (I_h^{**} + \phi R_{3h}^{**})}{\sigma_1 \gamma I_h^{**} \alpha_h E_h^{**}} \left[\frac{b\mu_h}{\Lambda_h} + \frac{\theta R_{1h}^{**}}{\beta_h S_h^{**} I_m^{**}} \right] \tau R_{3h}^{**} + \frac{\beta_m b \mu_h S_m^{**} (I_h^{**} + \phi R_{3h}^{**})}{2\sigma_1 \gamma I_h^{**} \Lambda_h} + \frac{\beta_m S_m^{**} (I_h^{**} + \phi R_{3h}^{**}) \theta R_{1h}^{**}}{\sigma_1 \gamma I_h^{**} \beta_h S_h^{**} I_m^{**}}$,

$d_5 = \frac{\beta_m S_m^{**} (I_h^{**} + \phi R_{3h}^{**})}{(1-\sigma_1) \gamma I_h^{**} \alpha_h E_h^{**}} \left[\frac{b\mu_h}{\Lambda_h} + \frac{\theta R_{1h}^{**}}{\beta_h S_h^{**} I_m^{**}} \right] \tau R_{3h}^{**} + \frac{\beta_m b \mu_h S_m^{**} (I_h^{**} + \phi R_{3h}^{**})}{2(1-\sigma_1) \gamma I_h^{**} \Lambda_h}, \quad d_6 = d_7 = 1,$

$d_8 = \frac{\beta_m b \mu_h S_m^{**} (I_h^{**} + \phi R_{3h}^{**})}{\alpha_m E_m^{**} \Lambda_h}$.

The time derivative of D in (27) is given by

$$\begin{aligned}
\frac{dD}{dt} = {} & d_1 \left(1 - \frac{S_h^{**}}{S_h} \right) \frac{dS_h}{dt} + d_2 \left(1 - \frac{E_h^{**}}{E_h} \right) \frac{dE_h}{dt} + d_3 \left(1 - \frac{I_h^{**}}{I_h} \right) \frac{dI_h}{dt} \\
& + d_4 \left(1 - \frac{R_{1h}^{**}}{R_{1h}} \right) \frac{dR_{1h}}{dt} + d_5 \left(1 - \frac{R_{3h}^{**}}{R_{3h}} \right) \frac{dR_{3h}}{dt} + d_6 \left(1 - \frac{S_m^{**}}{S_m} \right) \frac{dS_m}{dt} \\
& + d_7 \left(1 - \frac{E_m^{**}}{E_m} \right) \frac{dE_m}{dt} + d_8 \left(1 - \frac{I_m^{**}}{I_m} \right) \frac{dI_m}{dt}
\end{aligned} \tag{28}$$

Substituting the expressions on the right hand sides of the model (25) into (28) gives

$$\begin{aligned}\frac{dD}{dt} &= d_1\left(1-\frac{S_h^{**}}{S_h}\right)\left(\Lambda_h-\frac{\beta_h b S_h I_m}{N_h}-\mu_h S_h\right)+d_2\left(1-\frac{E_h^{**}}{E_h}\right)\\ &\times\left(\frac{\beta_h b I_m S_h}{N_h}+\theta R_{1h}-(\alpha_h+\mu_h)E_h\right)+d_3\left(1-\frac{I_h^{**}}{I_h}\right)\\ &\times(\alpha_h E_h+\tau R_{3h}-(\gamma+\mu_h)I_h)+d_4\left(1-\frac{R_{1h}^{**}}{R_{1h}}\right)(\sigma_1\gamma I_h-(\theta+\mu_h)R_{1h})\\ &+d_5\left(1-\frac{R_{3h}^{**}}{R_{3h}}\right)((1-\sigma_1)\gamma I_h-(\tau+\mu_h)R_{3h})\\ &+d_6\left(1-\frac{S_m^{**}}{S_m}\right)\left(\Lambda_m-\frac{\beta_m b S_m(I_h+\phi R_{3h})}{N_h}-\mu_m S_m\right)+d_7\left(1-\frac{E_m^{**}}{E_m}\right)\\ &\times\left(\frac{\beta_m b S_m(I_h+\phi R_{3h})}{N_h}-(\alpha_m+\mu_m)E_m\right)+d_8\left(1-\frac{I_m^{**}}{I_m}\right)(\alpha_m E_m-\mu_m I_m)\end{aligned}\tag{29}$$

Clearly, it can be shown from (25) that the following equilibrium relations hold.

$$\begin{aligned}&\Lambda_h=\frac{\beta_h b\mu_h S_h^{**}I_m^{**}}{\Lambda_h}+\mu_h S_h^{**},\qquad(\alpha_h+\mu_h)=\frac{\beta_h b\mu_h I_m^{**}S_h^{**}}{\Lambda_h E_h^{**}}+\frac{\theta R_{1h}^{**}}{E_h^{**}},\\ &(\gamma+\mu_h)=\frac{\alpha_h E_h^{**}}{I_h^{**}}+\frac{\tau R_{3h}^{**}}{I_h^{**}},\qquad(\theta+\mu_h)=\frac{\sigma_1\gamma I_h^{**}}{R_{1h}^{**}},\\ &(\tau+\mu_h)=\frac{(1-\sigma_1)\gamma I_h^{**}}{R_{3h}^{**}},\qquad\Lambda_m=\frac{\beta_m b\mu_h S_m^{**}(I_h^{**}+\phi R_{3h}^{**})}{\Lambda_h}+\mu_m S_m^{**},\\ &(\alpha_m+\mu_m)=\frac{\beta_m b\mu_h S_m^{**}(I_h^{**}+\phi R_{3h}^{**})}{\Lambda_h E_m^{**}},\qquad\mu_m=\frac{\alpha_m E_m^{**}}{I_m^{**}}.\end{aligned}\tag{30}$$

Further algebraic simplification of (29) using (30) yields

$$\begin{aligned}\frac{dD}{dt} &= u_1\left(8-\frac{S_h^{**}}{S_h}-\frac{S_h I_m E_h^{**}}{S_h^{**}I_m^{**}E_h}+\frac{I_h}{I_h^{**}}-\frac{I_h^{**}E_h}{I_h E_h^{**}}-\frac{S_m^{**}}{S_m}+\frac{I_h+\phi R_{3h}}{I_h^{**}+\phi R_{3h}^{**}}\left(1-\frac{S_m E_m^{**}}{S_m^{**}E_m}\right)\right.\\ &\left.-\frac{I_m^{**}E_m}{I_m E_m^{**}}-\frac{R_{1h}}{R_{1h}^{**}}-\frac{R_{1h}^{**}I_h}{R_{1h}I_h}-\frac{R_{3h}}{R_{3h}^{**}}-\frac{R_{3h}^{**}I_h}{R_{3h}I_h^{**}}\right)\\ &+u_2\left(2-\frac{S_h}{S_h^{**}}-\frac{S_h^{**}}{S_h}\right)+u_3\left(3-\frac{R_{1h}E_h^{**}}{R_{1h}^{**}E_h}-\frac{I_h^{**}E_h}{I_h E_h^{**}}-\frac{R_{1h}^{**}I_h}{R_{1h}I_h^{**}}\right)\\ &+u_4\left(3-\frac{I_h^{**}R_{3h}}{I_h R_{3h}^{**}}-\frac{R_{3h}^{**}I_h}{R_{3h}I_h^{**}}+\frac{I_h}{I_h^{**}}-\frac{R_{1h}}{R_{1h}^{**}}-\frac{R_{1h}^{**}I_h}{R_{1h}I_h^{**}}\right)\\ &+u_5\left(3-\frac{I_h^{**}R_{3h}}{I_h R_{3h}^{**}}-\frac{R_{3h}^{**}I_h}{R_{3h}I_h^{**}}+\frac{I_h}{I_h^{**}}-\frac{R_{1h}}{R_{1h}^{**}}-\frac{R_{1h}^{**}I_h}{R_{1h}I_h^{**}}\right)+\mu_m S_m^{**}\left(2-\frac{S_m}{S_m^{**}}-\frac{S_m^{**}}{S_m}\right),\end{aligned}\tag{31}$$

where

$$u_1=\frac{\beta_m b\mu_h S_m^{**}(I_h^{**}+\phi R_{3h}^{**})}{\Lambda_h},\quad u_2=\frac{\beta_m S_m^{**}(I_h^{**}+\phi R_{3h}^{**})}{\beta_h I_m^{**}},\quad u_3=\frac{\beta_m S_m^{**}(I_h^{**}+\phi R_{3h}^{**})\theta R_{1h}^{**}}{\beta_h S_h^{**}I_m^{**}},$$

$$u_4=\frac{\beta_m b\mu_h S_m^{**}(I_h^{**}+\phi R_{3h}^{**})\tau R_{3h}^{**}}{\Lambda_h\alpha_h E_h^{**}},\quad u_5=\frac{\beta_m S_m^{**}(I_h^{**}+\phi R_{3h}^{**})\theta R_{1h}^{**}\tau R_{3h}^{**}}{\alpha_h E_h^{**}\beta_h S_h^{**}I_m^{**}}.$$

Thus, using the given inequality conditions $S_m^{**}E_m \le S_m E_m^{**}$ and $R_{1h} \le R_{1h}^{**}$, then, $\left(1-\frac{S_m E_m^{**}}{S_m^{**}E_m}\right) \le 0$ and $\left(1-\frac{R_{1h}^{**}}{R_{1h}}\right) \le 0$ with equalities if and only if $\frac{S_m^{**}}{S_m}=\frac{E_m^{**}}{E_m}$ and $R_{1h}=R_{1h}^{**}$. As a consequence,

$$\begin{aligned}\frac{dD}{dt} &= u_1\left(7-\frac{S_h^{**}}{S_h}-\frac{S_h I_m E_h^{**}}{S_h^{**}I_m^{**}E_h}-\frac{I_h^{**}E_h}{I_h E_h^{**}}-\frac{S_m^{**}}{S_m}-\frac{R_{3h}}{R_{3h}^{**}}-\frac{R_{3h}^{**}I_h}{R_{3h}I_h^{**}}-\frac{I_m^{**}E_m}{I_m E_m^{**}}\right)\\ &+u_2\left(2-\frac{S_h}{S_h^{**}}-\frac{S_h^{**}}{S_h}\right)+u_3\left(3-\frac{E_h^{**}}{E_h}-\frac{I_h^{**}E_h}{I_h E_h^{**}}-\frac{I_h}{I_h^{**}}\right)\\ &+u_4\left(2-\frac{I_h^{**}R_{3h}}{I_h R_{3h}^{**}}-\frac{R_{3h}^{**}I_h}{R_{3h}I_h^{**}}\right)\end{aligned}$$

$$+u_5\left(2-\frac{I_h^{**}R_{3h}}{I_hR_{3h}^{**}}-\frac{R_{3h}^{**}I_h}{R_{3h}I_h^{**}}\right)+\mu_mS_m^{**}\left(2-\frac{S_m}{S_m^{**}}-\frac{S_m^{**}}{S_m}\right) \tag{32}$$

By AM-GM inequality: the arithmetic mean is greater than or equal to the geometric mean, it follows that

$$\begin{aligned}
&\left(7-\frac{S_h^{**}}{S_h}-\frac{S_hI_mE_h^{**}}{S_h^{**}I_m^{**}E_h}-\frac{I_h^{**}E_h}{I_hE_h^{**}}-\frac{S_m^{**}}{S_m}-\frac{R_{3h}}{R_{3h}^{**}}-\frac{R_{3h}^{**}I_h}{R_{3h}I_h^{**}}-\frac{I_m^{**}E_m}{I_mE_m^{**}}\right)\le 0,\\
&\left(2-\frac{S_h}{S_h^{**}}-\frac{S_h^{**}}{S_h}\right)\le 0,\quad \left(3-\frac{E_h^{**}}{E_h}-\frac{I_h^{**}E_h}{I_hE_h^{**}}-\frac{I_h}{I_h^{**}}\right)\le 0,\\
&\left(2-\frac{I_h^{**}R_{3h}}{I_hR_{3h}^{**}}-\frac{R_{3h}^{**}I_h}{R_{3h}I_h^{**}}\right)\le 0,\quad \left(2-\frac{S_m}{S_m^{**}}-\frac{S_m^{**}}{S_m}\right)\le 0.
\end{aligned} \tag{33}$$

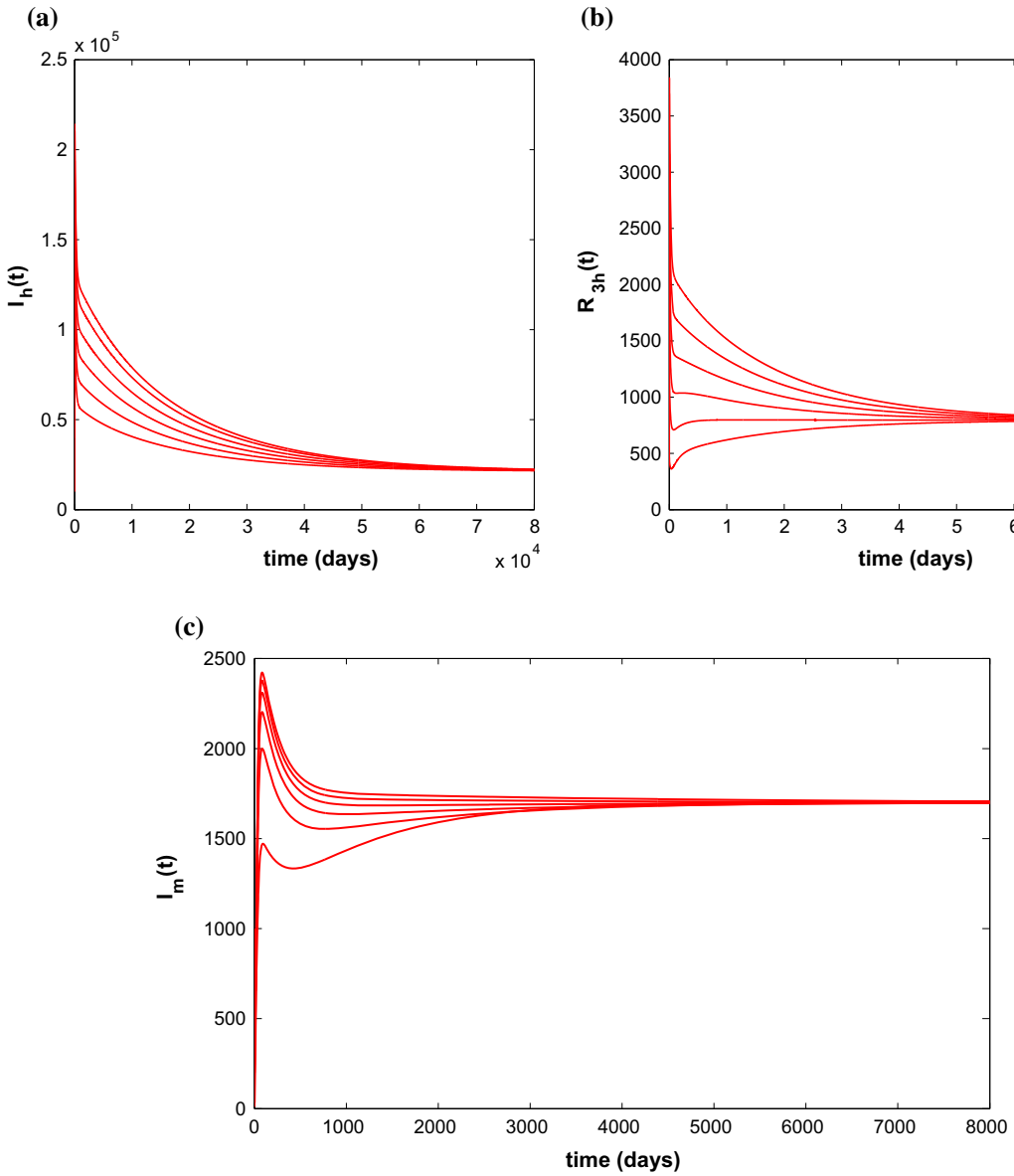

Fig. 3 Simulations showing global asymptotic behaviour of the recurrent malaria model (25) with different initial conditions. The parameter values used are the same as in Fig. 2, except that $\sigma_2 = 0$, so that $\mathcal{R}_0 = 3.8581 > 1$

Hence, $dD/dt \leq 0$ with $dD/dt = 0$ if and only if $S_h = S_h^{**}$, $E_h = E_h^{**}$, $I_h = I_h^{**}$, $R_{1h} = R_{1h}^{**}$, $R_{3h} = R_{3h}^{**}$, $S_m = S_m^{**}$, $E_m = E_m^{**}$, $I_m = I_m^{**}$, which indicates that

$$(S_h, E_h, I_h, R_{1h}, R_{3h}, S_m, E_m, I_m) \to (S_h^{**}, E_h^{**}, I_h^{**}, R_{1h}^{**}, R_{3h}^{**}, S_m^{**}, E_m^{**}, I_m^{**})$$

as $t \to \infty$. Thus, by Lasalle's invariance principle [30], the endemic equilibrium of the system (25) is globally-asymptotically stable. □

The quantitative illustration of Theorem 6 at various initial sizes of the sub-populations of the system (25) is given in Fig. 3.

5 Concluding remarks

A mathematical model representing the time-evolution of malaria spread with a nine-dimensional system of ordinary differential equations is presented. The model takes into account all the three classes of recurrent malaria, which are relapse, re-infection and recrudescence. Theoretical analysis of the model is performed to understand the behaviour of the system, and the following major findings are obtained.

i. The model reveals backward bifurcation property, which is due to the existence of re-infection of recovered humans in the population. However, the presence of both relapse and recrudescence in malaria dynamics do not cause the phenomenon of backward bifurcation. Hence, the possibility of backward bifurcation can only be ruled out when re-infection seizes to occur.
ii. The global asymptotic dynamics of the model without re-infection is established; showing that the resulting model has a globally-asymptotically stable disease-free equilibrium point whenever the basic reproduction number is below one, and a globally-asymptotically stable unique endemic equilibrium point if the basic reproduction number is above one.
iii. Simulations of the recurrent malaria model are done to support the theoretical results; showing the convergence of solutions at various initial sizes of sub-populations of the model to the equilibrium points.

Since the occurrence of backward bifurcation driven by re-infection shows how hard it is to control or eliminate the transmission of malaria even when the basic reproduction is below one, it is, therefore, imperative to scale up efforts or measures toward interrupting malaria re-infection in the community. More attention should be given to the control of recurrent malaria in order to achieve a malaria-free community.

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Data availability This manuscript has no associated data in a data repository. The data supporting the findings of this study are included within the article.

Declarations

Conflict of interest The authors declare no conflict of interest.

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