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Research article

Global stability for a mosquito-borne disease model with continuous-time age structure in the susceptible and relapsed host classes

Maria Guadalupe Vazquez-Peña[1], Cruz Vargas-De-León[2,3,*] and Jorge Velázquez-Castro[4]

[1] Centro de Investigación en Matemáticas, 36023 Guanajuato, Guanajuato, Mexico
[2] División de Investigación, Hospital Juárez de México, Ciudad de México 07760, Mexico
[3] Sección de Estudios de Posgrado e Investigación, Escuela Superior de Medicina, Instituto Politécnico Nacional, Ciudad de México 11340, Mexico
[4] Facultad de Ciencias Físico-Matemáticas, Benemérita Universidad Autónoma de Puebla, Puebla, Mexico

* **Correspondence:** Email: leoncruz82@yahoo.com.mx.

Abstract: Mosquito-borne infectious diseases represent a significant public health issue. Age has been identified as a key risk factor for these diseases, and another phenomenon reported is relapse, which involves the reappearance of symptoms after a symptom-free period. Recent research indicates that susceptibility to and relapse of mosquito-borne diseases are frequently age-dependent. This paper proposes a new model to better capture the dynamics of mosquito-borne diseases by integrating two age-dependent factors: chronological age and asymptomatic-infection age. Chronological age refers to the time elapsed from the date of birth of the host to the present time. On the other hand, asymptomatic infection age denotes the time elapsed since the host became asymptomatic after the primary infection. The system of integro-differential equations uses flexible, unspecified functions to represent these dependencies, assuming they are integrable. We analyzed the global stability of both the disease-free and endemic equilibrium states using the direct Lyapunov method with Volterra-type Lyapunov functionals. Additionally, the paper explores several special cases involving well-known host-vector models.

Keywords: mosquito-borne infectious diseases; age dependency; susceptibility; relapse; Volterra-type Lyapunov functions

1. Introduction

Mosquito-borne infectious diseases are of global importance. In the last two decades, vector-borne diseases have emerged at a growing rate, and several vector-borne pathogens have colonized new re-

gions [1]. More than 400,000 people die each year from malaria, while dengue, yellow fever, chikungunya, and Zika have caused serious disease outbreaks in many urban areas [2].

Age has been identified as a risk factor for mosquito-borne diseases. In the study by Hasyim et al. [3], age was identified as a risk factor for malaria, with participants older than 35 years being 7.98 times more likely to contract the disease compared to those younger than 35. In a case-control study [4], older patients were found to have a lower risk of contracting dengue compared to younger adults aged 16 to 30 years. In a review [5], they affirmed that the vast majority of dengue cases affect children under 15 years of age. A study from coastal Ecuador [6] reported that dengue seroprevalence was strongly age-dependent, increasing rapidly with age and reaching 97% by age 60. In contrast, chikungunya infection peaked in childhood, with a maximum prevalence of 42% around age 9.

Another phenomenon that has been documented in mosquito-borne diseases is the relapse phenomenon, understood as the reappearance of symptoms after a symptom-free period. In malaria infection, some types of the parasite (such as *Plasmodium vivax* and *Plasmodium ovale*) can cause relapses because they form hypnozoites in the liver that can reactivate and trigger a new phase of the disease [7]. In chikungunya infection, a study of 509 people in France found that 72% of patients experienced relapses, averaging four episodes, and the mean delay between two relapses was 8 weeks [9]. Another study conducted in 2015 on 5870 inhabitants of Acapulco, México, showed that 31% of infected individuals had at least one relapse after being symptom-free for a month [10].

Systems of ordinary differential equations are commonly used to model the propagation of mosquito-borne infectious diseases. We identified some mosquito-borne disease models that consider children and adults as different subpopulations. In dengue and malaria, two age classes have been considered [8, 11–13]. Chamnan et al. [14] developed a three-age-class model for dengue, dividing the population into under 10, 10–44, and over 44 years old. We identified a few mosquito-borne disease models that consider relapses. Huo and Qiu [15] and Ghosh et al. [16] developed malaria models with relapses. More recently, Vázquez-Peña et al. developed a model for chikungunya dynamics with relapse [17].

In mosquito-borne diseases, variability exists in the susceptibility and relapse periods for each infection. Although mathematical models for these diseases address these issues, they often focus on specific scenarios, either using models with two or three age classes [8, 11–14] or assuming constant relapse rates [15–17]. It is well established that compartments without age structure imply that the time distribution of transitions from one stage to another is assumed to be exponential when utilized. For many analyses, this serves as an adequate approximation; however, in other cases, particularly in relapse scenarios, a more specific timing is necessary to model complex behaviors. Recent empirical research indicates that susceptibility and relapse rates are frequently age-dependent. Vargas-De-León et al. [18] introduced and analyzed a host-vector model where the host population is structured by chronological age, with host susceptibility assumed to be age-dependent. Additionally, Vargas-De-León [19] developed a model for non-vector-borne diseases that accounts for variability in relapse periods by incorporating the concept of asymptomatic-infection age. Recent evidence in the literature shows the need for more comprehensive models that integrate both susceptibility and variability in relapse rates to better reflect the complex dynamics of these diseases.

In this paper, we present a model to describe the spread of mosquito-borne diseases, incorporating two different age dependencies: chronological age and asymptomatic-infection age. Chronological age refers to the time elapsed from the date of birth of the host to the present time. Asymptomatic infection

age denotes the time elapsed since the host became asymptomatic after the primary infection. From a biological perspective, the introduction of age dependency implies the possibility of analyzing more complex relationships between the probability of infection and age. For example, age dependence on risk could manifest as a smooth gradient or even a bimodal effect for more susceptible children and older adults. From a biological standpoint, it is crucial to model the effect of a well-defined distribution period during which relapse can occur, rather than relying solely on an information-poor distribution where only the mean is considered, as implied in compartmental models without age structure. Furthermore, the age dependencies in the epidemiological model directly relate to the physiological evolution of the disease within the body, allowing the microscopic and individual-scale dynamics of the disease to be associated with the epidemiological-scale description of a disease.

We use unspecified functions for both dependencies, provided they are integrable. We will analyze the global stability of both the disease-free and endemic equilibrium states using the direct Lyapunov method with the following Volterra-type functional:

$$f(x) = x - 1 - \ln x. \tag{1.1}$$

This analysis enables the characterization of a family of vector-borne disease models as epidemiologically significant, either with or without age structure. The introduction of unspecified functions for the time dependencies of chronological age and symptomatic-infection age facilitates the analysis and identification of emergent and non-trivial complex dynamical behavior of the diseases due to those age structure dependencies. A particularly salient aspect of the model is that it establishes the foundation for analyzing periodic or time-distributed control strategies.

2. The model with continuous-time age structure

We assume that both host and mosquito populations are stratified by their epidemiological stages. The total host population, denoted by N_h, is divided into four classes: susceptible $S_h(t)$, infected $I_h(t)$, asymptomatic $A_h(t)$, and recovered $R_h(t)$. The total mosquito population, denoted by N_m, is divided into two classes: susceptible $S_m(t)$ and infected $I_m(t)$.

The chronological age is denoted by the variable τ. Thus, $s_h(t,\tau)$ represents the number of susceptible hosts aged τ at time t. Therefore, $\int_{\tau_1}^{\tau_2} s_h(t,\tau)d\tau$ gives the number of susceptible hosts whose ages fall between τ_1 and τ_2 at time t. The total number of susceptible individuals is given by $S_h(t) = \int_0^\infty s_h(t,\tau)d\tau$. The chronological age of the infected hosts was not explicitly described because, in contrast to age-dependent susceptibility, age-dependent transmissibility is not readily observable through medical records. This complication makes including this dependence less beneficial, as there is limited data to infer this relationship. Conversely, the importance of explicitly describing age-dependent susceptibility lies in its potential to analyze age-dependent risk and focus control strategies based on age. In contrast, infected individuals are presumed to be readily identifiable, and measures can be implemented irrespective of age.

Similarly, we consider the age of asymptomatic infection, denoted by ω. Here, $a_h(t,\omega)$ denotes the number of individuals in the asymptomatic hosts aged ω at time t. The expression $\int_{\omega_1}^{\omega_2} a_h(t,\omega)d\omega$ represents the number of hosts in the asymptomatic state for a duration between ω_1 and ω_2. $A_h(t) = \int_0^\infty a_h(t,\omega)d\omega$ represents the total number of asymptomatic individuals.

We assume that all hosts are born susceptible at a constant rate Λ, which leads to the boundary condition $s_h(t,0) = \Lambda$. When a host is bitten by an infected mosquito, there is a probability of contracting the pathogen that depends on the age of the host, denoted by $\beta_h(\tau)$. Therefore, the number of infections is proportional to this parameter and the number of bites. If b is the average number of bites per mosquito per unit time, each individual receives b/N_h bites, resulting in a transmission rate of $\beta_h(\tau)b/N_h$. Thus, the term $b\beta_h(\tau)s_h(t,\tau)I_m(t)/N_h$ stands for the rate of infections of susceptible hosts of age τ by the infected mosquitoes I_m. Because there is no distinction between infected individuals due to their age, the infections should be integrated over all ages in the corresponding equation, resulting in the term $\frac{b}{N_h}\int_0^\infty b\beta_h(\tau)s_h(t,\tau)I_m(t)d\tau$.

We also assume that the host population decreases due to natural mortality at a constant rate μ_h. Additionally, we introduce the parameter $\rho(\tau)$ to represent an infection prevention strategy, such as vaccination, that is applied only to specific age groups. Consequently, the rate of removing individuals from the susceptible class is given by $\varepsilon_h(\tau) = \mu_h + \rho(\tau)$. Thus, the term $\varepsilon(\tau)s_h(t,\tau)$ stands for the total rate of removal from the group of the susceptible hosts aged τ.

The parameter γ denotes the rate at which symptomatology disappears from infected hosts, leading to the term $(\mu_h + \gamma)I_h(t)$ that represents the removal rate from the infected compartment due to natural per-capita mortality μ_h plus symptom disappearance. The parameter p represents the fraction of infected individuals leaving the infected class γI_h, who do not fully recover but instead enter an asymptomatic phase for a period of time ω. Thus, this leads to the boundary condition $a_h(t,0) = p\gamma I_h(t)$. When symptoms reappear, they return to the infected stage according to a relapse rate dependent on the asymptomatic infection age, $\delta_h(\omega)$. The integral in the infected class equation $\int_0^\infty \delta_h(\omega)a_h(t,\omega)d\omega$ stands for the rate of transition of all asymptomatic hosts. A fraction $(1-p)$ of infected hosts is assumed to undergo full recovery with permanent immunity, and $(1-p)\gamma$ represents the recovery rate of the infected hosts.

On the other hand, mosquitoes are assumed to be born and die at a constant rate μ_m, as is commonly proposed to describe a stationary population. The constant vector population assumption implies that the vectors are continuously reproducing, and the system has reached a state where the population death rate equates to its population birth rate.

A susceptible mosquito becomes infected only if it bites an infected host and the bite effectively transmits the pathogen. Similar to host transmission, the probability that a mosquito will contract the pathogen after biting an infected host is denoted by β_m, and the transmission rate in mosquitoes depends on the bite rate b. We introduce the parameter θ to account for any potential differences in transmission probability between infected and asymptomatic hosts, leading to the term $\theta\frac{\beta_m b}{N_h}S_m(t)\int_0^\infty a_h(t,\omega)d\omega$ representing the mosquito infections due to asymptomatic hosts.

The dynamics described above translate into the following system of integro–differential equations:

$$\begin{aligned}
\frac{\partial s_h(t,\tau)}{\partial t} + \frac{\partial s_h(t,\tau)}{\partial \tau} &= -\frac{b}{N_h}\beta_h(\tau)s_h(t,\tau)I_m(t) - \varepsilon_h(\tau)s_h(t,\tau),\\
s_h(t,0) &= \Lambda,\\
\frac{dI_h(t)}{dt} &= \frac{b}{N_h}\int_0^\infty \beta_h(\tau)s_h(t,\tau)I_m(t)d\tau - (\mu_h+\gamma)I_h(t) + \int_0^\infty \delta_h(\omega)a_h(t,\omega)d\omega,\\
\frac{\partial a_h(t,\omega)}{\partial t} + \frac{\partial a_h(t,\omega)}{\partial \omega} &= -(\mu_h + \delta_h(\omega))a_h(t,\omega),
\end{aligned} \tag{2.1}$$

$$
\begin{aligned}
a_h(t,0) &= p\gamma I_h(t),\\
\frac{dR_h(t)}{dt} &= (1-p)\gamma I_h(t) + \int_0^\infty \rho(\tau)s_h(t,\tau)d\tau - \mu_h R_h(t),\\
\frac{dS_m(t)}{dt} &= \mu_m N_m - \frac{\beta_m b}{N_h} S_m(t) I_h(t) - \theta\frac{\beta_m b}{N_h} S_m(t)\int_0^\infty a_h(t,\omega)d\omega - \mu_m S_m(t),\\
\frac{dI_m(t)}{dt} &= \frac{\beta_m b}{N_h} S_m(t) I_h(t) + \theta\frac{\beta_m b}{N_h} S_m(t)\int_0^\infty a_h(t,\omega)d\omega - \mu_m I_m(t).
\end{aligned}
$$

Considering that the mosquito population is $N_m = S_m(t) + I_m(t)$, and the equation corresponding to the recovery class is decoupled from the rest of the equations of the system, it could be reduce to:

$$
\begin{aligned}
\frac{\partial s_h(t,\tau)}{\partial t} + \frac{\partial s_h(t,\tau)}{\partial \tau} &= -\frac{b}{N_h}\beta_h(\tau)s_h(t,\tau)I_m(t) - \varepsilon_h(\tau)s_h(t,\tau),\\
s_h(t,0) &= \Lambda,\\
\frac{dI_h(t)}{dt} &= \frac{b}{N_h}\int_0^\infty \beta_h(\tau)s_h(t,\tau)I_m(t)d\tau - (\mu_h+\gamma)I_h(t) + \int_0^\infty \delta_h(\omega)a_h(t,\omega)d\omega,\\
\frac{\partial a_h(t,\omega)}{\partial t} + \frac{\partial a_h(t,\omega)}{\partial \omega} &= -\alpha(\omega)a_h(t,\omega), \text{ with } \alpha(\omega) = \mu_h + \delta_h(\omega),\\
a_h(t,0) &= p\gamma I_h(t),\\
\frac{dI_m(t)}{dt} &= \frac{\beta_m b}{N_h}(N_m - I_m(t))\left(I_h(t) + \theta\int_0^\infty a_h(t,\omega)d\omega\right) - \mu_m I_m(t),
\end{aligned}
\tag{2.2}
$$

with the initial conditions given by

$$
s_h(0,\tau) = s_{h0}(\tau),\ \ I_h(0) = I_{h0},\ \ a_h(0,\omega) = a_{h0}(\omega),\ \ I_v(0) = I_{v0}.
$$

$s_{h0}(\tau)$ and $a_{h0}(\omega)$ are the initial distribution of the susceptible host and asymptomatic host with chronological age and asymptomatic-infection age, respectively. I_{h0} and I_{v0} are the initial infected hosts and infected mosquitoes, respectively. In what follows, we also have assumed that the initial conditions are in the invariant subspace where $N_h = \Lambda/\mu$ is constant. By adding the host equations, it can be verified that $\frac{dN_h}{dt} = \Lambda - \mu N_h$ and thus $N_h = \Lambda/\mu$ is also a stable subspace.

3. Existence of equilibrium states and their global stability

The basic reproductive number of the system (2.2) is given by:

$$
R_0 = \frac{\eta p\gamma N_h + D\beta_m b N_m}{N_h(\mu_h+\gamma)},
$$

where D and η are defined by

$$
D = \frac{b}{\mu_m N_h}\int_0^\infty s_h^0(\tau)\beta_h(\tau)d\tau, \tag{3.1}
$$

$$
\eta = \int_0^\infty [\delta_h(v)+k]\exp\left(-\int_0^v \alpha(\phi)d\phi\right)dv, \tag{3.2}
$$

with

$$k = \frac{\beta_m b^2 N_m \theta}{N_h^2 \mu_m} \int_0^\infty s_h^0(\tau)\beta_h(\tau)d\tau. \tag{3.3}$$

The parameter R_0 has significant epidemiological meaning; it represents the average number of secondary cases generated by a primary case introduced into a fully susceptible host population. This includes disease transmission from asymptomatic and infected hosts to susceptible mosquitoes, as well as from infected mosquitoes to susceptible hosts.

We define a positive auxiliary function [18, 19],

$$g(\omega) = \int_\omega^\infty [\delta_h(v) + c] \exp\left(-\int_\omega^v \alpha(\phi)d\phi\right) dv, \quad c > 0, \tag{3.4}$$

that we will use for the construction of Lyapunov functionals in the proof of global stability of the equilibrium state for the system (2.2) by the direct Lyapunov method. Observe that $g(\omega) > 0$ for any $w \geq 0$ and it verifies $g(0) = \eta$ when we consider $c = k$, where k is given in (3.3).

3.1. Disease-free equilibrium state

Through direct calculations, the disease-free equilibrium state E^0 is obtained taking $I_h(t) = 0$, $a_h(t, \omega) = 0$, and $I_m(t) = 0$, and then $\frac{ds_h(\tau)}{d\tau} = -\varepsilon_h(\tau)s_h(\tau)$. Solving this differential equation, we have that the disease-free equilibrium state is $E^0 = (s_h^0(\tau), 0, 0, 0)$ where

$$s_h^0(\tau) = \Lambda \exp\left(-\int_0^\tau \varepsilon_h(\phi)d\phi\right). \tag{3.5}$$

To prove its global stability, we consider a Lyapunov functional, which is the linear combination of Volterra-type and linear functions:

$$W(t) = \int_0^\infty s_h^0(\tau) f\left(\frac{s_h(t,\tau)}{s_h^0(\tau)}\right) d\tau + I_h(t) + \int_0^\infty g(\omega)a(t,\omega)d\omega + \frac{b}{\mu_m N_h} I_m(t) \int_0^\infty s_h^0(\tau)\beta_h(\tau)d\tau,$$

where $f(x)$ is given by (1.1) and $g(\omega)$ by (3.4) with the constant c equal to k defined in (3.3). $W(t)$ satisfies

$$\frac{dW}{dt} = \int_0^\infty \left(1 - \frac{s_h^0(\tau)}{s_h(t,\tau)}\right) \frac{\partial s_h(t,\tau)}{\partial t} d\tau + \frac{dI_h(t)}{dt} + \int_0^\infty g(\omega)\frac{\partial a(t,\omega)}{\partial t} d\omega + \frac{b}{\mu_m N_h}\left(\int_0^\infty s_h^0(\tau)\beta_h(\tau)d\tau\right)\frac{dI_m(t)}{dt}.$$

Using the equations of the system (2.2),

$$\begin{aligned}\frac{dW}{dt} =& -\int_0^\infty \left(1-\frac{s_h^0(\tau)}{s_h(t,\tau)}\right)\left(\frac{\partial s_h(t,\tau)}{\partial \tau}+\frac{b}{N_h}\beta_h(\tau)s_h(t,\tau)I_m(t)+\varepsilon_h(\tau)s_h(t,\tau)\right)d\tau\\ &+\frac{b}{N_h}\int_0^\infty \beta_h(\tau)s_h(t,\tau)I_m(t)d\tau-(\mu_h+\gamma)I_h(t)+\int_0^\infty \delta_h(\omega)a_h(t,\omega)d\omega\\ &-\int_0^\infty g(\omega)\left[\frac{\partial a_h(t,\omega)}{\partial \omega}+\alpha(\omega)a_h(t,\omega)\right]d\omega+D\frac{\beta_m b}{N_h}(N_m-I_m(t))\left(I_h(t)+\theta\int_0^\infty a_h(t,\omega)d\omega\right)\\ &-D\mu_m I_m(t).\\ =& -\int_0^\infty s_h^0(\tau)\left(\frac{s_h(t,\tau)}{s_h^0(\tau)}-1\right)\left(\frac{\frac{\partial s_h(t,\tau)}{\partial \tau}}{s_h(t,\tau)}+\varepsilon_h(\tau)\right)d\tau+\frac{b}{N_h}\int_0^\infty s_h^0(\tau)\beta_h(\tau)I_m(t)d\tau-(\mu_h+\gamma)I_h(t)\\ &-\int_0^\infty\left[g(\omega)\alpha(\omega)-\delta_h(\omega)-\frac{\beta_m b}{N_h}DN_m\theta\right]a_h(t,\omega)d\omega-\int_0^\infty g(\omega)\frac{\partial a_h(t,\omega)}{\partial \omega}d\omega-D\mu_m I_m(t)\\ &+\frac{\beta_m bN_m}{N_h}DI_h(t)-\frac{\beta_m b}{N_h}DI_m(t)I_h(t)-\frac{\theta\beta_m b}{N_h}DI_m(t)\int_0^\infty a_h(t,\omega)d\omega,\end{aligned}$$

with D given by (3.1).

By direct calculations can be shown as

$$\frac{\partial}{\partial \tau}f\left(\frac{s_h(t,\tau)}{s_h^0(\tau)}\right)=\left(\frac{s_h(t,\tau)}{s_h^0(\tau)}-1\right)\left(\frac{\frac{\partial s_h(t,\tau)}{\partial \tau}}{s_h(t,\tau)}+\varepsilon_h(\tau)\right),$$

which makes it possible to simplify the first term of dW/dt integrating by parts:

$$\int_0^\infty s_h^0(\tau)\frac{\partial}{\partial \tau}f\left(\frac{s_h(t,\tau)}{s_h^0(\tau)}\right)d\tau=\left[s_h^0(\tau)f\left(\frac{s_h(t,\tau)}{s_h^0(\tau)}\right)\right]_\infty+\int_0^\infty \varepsilon_h(\tau)s_h^0(\tau)f\left(\frac{s_h(t,\tau)}{s_h^0(\tau)}\right)d\tau. \tag{3.6}$$

Applying integration by parts over another term of dW/dt with the boundary condition $a_h(t,0)=p\gamma I_h(t)$ results in

$$\int_0^\infty g(\omega)\frac{\partial a_h(t,\omega)}{\partial \omega}d\omega=[g(\omega)a_h(t,\omega)]_\infty-\eta p\gamma I_h(t)-\int_0^\infty\left[g(\omega)\alpha(\omega)-\delta_h(\omega)-k\right]a_h(t,\omega)d\omega. \tag{3.7}$$

Using Eqs (3.6) and (3.7) results in:

$$\begin{aligned}\frac{dW}{dt} =& -\left[s_h^0(\tau)f\left(\frac{s_h(t,\tau)}{s_h^0(\tau)}\right)+g(\omega)a_h(t,\omega)\right]_\infty-\int_0^\infty \varepsilon_h(\tau)s_h^0(\tau)f\left(\frac{s_h(t,\tau)}{s_h^0(\tau)}\right)d\tau\\ &-D\frac{\beta_m b}{N_h}I_m(t)I_h(t)-D\frac{\theta\beta_m b}{N_h}I_m(t)\int_0^\infty a_h(t,\omega)d\omega-I_h(t)(\mu_h+\gamma)\left[1-R_0\right].\end{aligned}$$

Therefore, $W(t)$ is a positive definite function whose derivative dW/dt is negative provided that $R_0<1$. Moreover, the only positively invariant set where $dW/dt=0$ is $\{E^0\}$. By Theorem 2.53 of Smith and Thieme [20], the disease-free equilibrium state is globally asymptotically stable when $R_0\le 1$. We arrived at the following results:

Theorem 3.1. *The disease-free equilibrium state E^0 of the system (2.2) is globally asymptotically stable if $R_0\le 1$.*

3.2. Endemic equilibrium state

The equilibrium state $E^* = (s_h^*(\tau), I_h^*, a_h^*(\omega), I_m^*)$ must satisfy the system of equations:

$$\frac{ds_h^*(\tau)}{d\tau} = -\frac{b}{N_h}\beta_h(\tau)s_h^*(\tau)I_m^* - \varepsilon_h(\tau)s_h^*(\tau), \tag{3.8}$$

$$s_h^*(0) = \Lambda, \tag{3.9}$$

$$0 = \frac{b}{N_h}\int_0^\infty \beta_h(\tau)s_h^*(\tau)I_m^* d\tau - (\mu_h + \gamma)I_h^* + \int_0^\infty \delta_h(\omega)a_h^*(\omega)d\omega, \tag{3.10}$$

$$\frac{da_h^*(\omega)}{d\omega} = -\alpha(\omega)a_h^*(\omega), \tag{3.11}$$

$$a_h^*(0) = p\gamma I_h^*, \tag{3.12}$$

$$0 = \frac{\beta_m b}{N_h}(N_m - I_m^*)\left(I_h^* + \theta\int_0^\infty a_h^*(\omega)d\omega\right) - \mu_m I_m^*. \tag{3.13}$$

Although the system of Eqs (3.8)–(3.13) will not be solved explicitly, but we can prove the existence of the endemic equilibrium state.

Solving the differential equation (3.11) with initial condition (3.12), we have that:

$$a_h^*(\omega) = p\gamma I_h^* \exp\left(-\int_0^\omega \alpha(\phi)d\phi\right), \tag{3.14}$$

and replacing it in Eq (3.13), we obtain the following expression:

$$I_h^* = \frac{\mu_m N_h I_m^*}{\beta_m b(N_m - I_m^*)\left[1 + \theta p\gamma\int_0^\infty \exp\left(-\int_0^\omega \alpha(\phi)d\phi\right)d\omega\right]}. \tag{3.15}$$

In addition,

$$s_h^*(\tau) = s_h^0(\tau)\exp\left(-\frac{b}{N_h}I_m^*\int_0^\tau \beta(\phi)d\phi\right) \tag{3.16}$$

is the particular solution of (3.8) under the initial condition (3.9) and $s_h^0(\tau)$ has been defined in (3.5).

Replacing the Eqs (3.15), (3.14), and (3.16) in (3.10) we get:

$$\begin{aligned} 0 = I_m^*\Bigg(&\frac{(\mu_h+\gamma)\mu_m N_h - p\gamma\mu_m N_h\int_0^\infty \delta_h(\omega)\exp\left(-\int_0^\omega \alpha(\phi)d\phi\right)d\omega}{\beta_m b(N_m - I_m^*)\left[1 + \theta p\gamma\int_0^\infty \exp\left(-\int_0^\omega \alpha(\phi)d\phi\right)d\omega\right]}\Bigg)\cdot \\ &\left(\frac{\beta_m b^2(N_m - I_m^*)\left[1 + \theta p\gamma\int_0^\infty \exp\left(-\int_0^\omega \alpha(\phi)d\phi\right)d\omega\right]}{N_h\left[(\mu_h+\gamma)\mu_m N_h - p\gamma\mu_m N_h\int_0^\infty \delta_h(\omega)\exp\left(-\int_0^\omega \alpha(\phi)d\phi\right)d\omega\right]\int_0^\infty \beta_h(\tau)s_h^0(\tau)\exp\left(-\frac{b}{N_h}I_m^*\int_0^\tau \beta(\phi)d\phi\right)d\tau} - 1\right). \end{aligned} \tag{3.17}$$

The case $I_m^* = 0$ reduces to the disease-free equilibrium state. The second term in (3.17) is always positive because

$$\int_0^\infty \delta_h(\omega)\exp\left(-\int_0^\omega \alpha(\phi)d\phi\right)d\omega \le \int_0^\infty \delta_h(\omega)\exp\left(-\int_0^\omega \delta_h(\phi)d\phi\right)d\omega = 1 - \exp\left(-\int_0^\infty \delta_h(\phi)d\phi\right), \tag{3.18}$$

so the equality in (3.17) only can be satisfied when the last term in the product of the right member of (3.17) is equal to 0. To prove that, we define the function $h(I_m^*)$ given by:

$$h(I_m^*) = \frac{\beta_m b^2 \left[1 + \theta p\gamma \int_0^\infty \exp\left(-\int_0^\omega \alpha(\phi)d\phi\right)d\omega\right](N_m - I_m^*)}{N_h\left[(\mu_h+\gamma)\mu_m N_h - p\gamma\mu_m N_h \int_0^\infty \delta_h(\omega)\exp\left(-\int_0^\omega \alpha(\phi)d\phi\right)d\omega\right]} \int_0^\infty \beta_h(\tau)s_h^0(\tau)\exp\left(-\frac{b}{N_h}I_m^*\int_0^\tau \beta(\phi)d\phi\right)d\tau,$$

which satisfies that $h(N_m) = 0$ and $h(0) = \dfrac{R_0 N_h^2\mu_m(\mu_h+\gamma) - p\gamma\int_0^\infty N_h^2\mu_m\delta_h(v)\exp\left(-\int_0^v \alpha(\phi)d\phi\right)}{N_h^2\mu_m(\mu_h+\gamma) - p\gamma\int_0^\infty N_h^2\mu_m\delta_h(v)\exp\left(-\int_0^v \alpha(\phi)d\phi\right)}$.

It is clear that $h(0) > 1$ when $R_0 > 1$.

In order to analyse the behavior of the function $h(I_m^*)$ in the interval $(0, N_m)$, the first derivative test is applied. By simple calculations, we obtain

$$h'(I_m^*) = \frac{-\beta_m b^2\left[1 + \theta p\gamma\int_0^\infty \exp\left(-\int_0^\omega \alpha(\phi)d\phi\right)d\omega\right]\Delta}{N_h^2\mu_m\left[\mu_h + \gamma\left(1 - p\int_0^\infty \delta_h(\omega)\exp\left(-\int_0^\omega \alpha(\phi)d\phi\right)d\omega\right)\right]}, \tag{3.19}$$

with

$$\Delta = \int_0^\infty \beta_h(\tau)s_h^0(\tau)\left(1 + \frac{b}{Nh}(N_m - I_m^*)\int_0^\tau \beta(\phi)d\phi\right)\exp\left(-\frac{b}{N_h}I_m^*\int_0^\tau \beta(\phi)d\phi\right)d\tau.$$

Using (3.18), we can note that the denominator of (3.19) is positive, so that $h'(I_m^*) < 0$. Hence, $h(I_m^*)$ is a decreasing monotonic function that satisfies $h(N_m) = 0$ and $h(0) > 1$ when $R_0 > 1$, so there exists one and only $I_m^* \in (0, N_m)$ such that $h(I_m^*) = 1$, being only determined by $s_h(\tau)$, I_h^* and $a_h^*(\omega)$ in Eqs (3.16), (3.15), and (3.14), respectively. We arrived at the following results:

Theorem 3.2. *The system (2.2) exhibits an endemic equilibrium state when $R_0 > 1$.*

Now, we proove the global stability of the endemic equilibrium state using the direct Lyapunov method. We consider the Volterra-type Lyapunov functional:

$$\begin{aligned} L(t) = {} & \frac{\mu_m N_h}{I_m^* b}\int_0^\infty s_h^*(\tau) f\left(\frac{s_h(t,\tau)}{s_h^*(\tau)}\right)d\tau + \frac{I_h^*\mu_m N_h}{I_m^* b} f\left(\frac{I_h(t)}{I_h^*}\right) \\ & + \frac{\mu_m N_h}{I_m^* b}\int_0^\infty a_h^*(\omega)g(\omega) f\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}\right)d\omega + f\left(\frac{I_m(t)}{I_m^*}\right)\int_0^\infty \beta_h(\tau)s_h^*(\tau)d\tau, \end{aligned}$$

where $g(\omega)$ is defined in (3.4) with the constant $c = \left(\frac{b}{N_h}\right)^2 \frac{\beta_m}{\mu_m}(N_m - I_m^*)\theta\int_0^\infty \beta_h(\tau)s_h^*(\tau)d\tau$.

After calculating the derivative with respect to time and using the equations of the model (2.2), it results in:

$$\begin{aligned} \frac{dL}{dt} = {} & -\frac{\mu_m N_h}{I_m^* b}\int_0^\infty \left(1 - \frac{s_h^*(\tau)}{s_h(t,\tau)}\right)\left(\frac{\partial s_h(t,\tau)}{\partial\tau} + \frac{b}{N_h}\beta_h(\tau)s_h(t,\tau)I_m(t) + \varepsilon_h(\tau)s_h(t,\tau)\right)d\tau \\ & + \frac{\mu_m N_h}{I_m^* b}\left(1 - \frac{I_h^*}{I_h(t)}\right)\left(I_m(t)\frac{b}{N_h}\int_0^\infty \beta_h(\tau)s_h(t,\tau)d\tau - (\mu_h+\gamma)I_h(t) + \int_0^\infty \delta_h(\omega)a_h(t,\omega)d\omega\right) \\ & - \frac{\mu_m N_h}{I_m^* b}\int_0^\infty g(\omega)\left(1 - \frac{a_h^*(\omega)}{a_h(t,\omega)}\right)\left(\frac{\partial a_h(t,\omega)}{\partial\omega} + \alpha(\omega)a_h(t,\omega)\right)d\omega \\ & + \frac{1}{I_m^*}\int_0^\infty \beta_h(\tau)s_h^*(\tau)d\tau\left(1 - \frac{I_m^*}{I_m(t)}\right)\left(\frac{\beta_m b}{N_h}(N_m - I_m(t))\left(I_h(t) + \theta\int_0^\infty a_h(t,\omega)d\omega\right) - \mu_m I_m(t)\right). \end{aligned} \tag{3.20}$$

Using Eq (3.11), it can be directly shown that

$$\frac{\partial}{\partial\omega}f\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}\right)=\frac{1}{a_h^*(\omega)}\left(1-\frac{a_h^*(\omega)}{a_h(t,\omega)}\right)\left(\frac{\partial a_h(t,\omega)}{\partial\omega}+\alpha(\omega)a_h(t,\omega)\right).$$

After integrating by parts with the boundary condition $a_h(t,0)=p\gamma I_h(t)$ and Eq (3.12), we have

$$\begin{aligned}\int_0^\infty g(\omega)a_h^*(\omega)\frac{\partial}{\partial\omega}f\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}\right)d\omega=&\left[g(\omega)a_h^*(\omega)f\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}\right)\right]_{\omega=\infty}-\eta p\gamma I_h^*f\left(\frac{I_h(t)}{I_h^*}\right)\\&+\int_0^\infty(\delta_h(\omega)+k)a_h^*(\omega)f\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}\right)d\omega.\end{aligned}\tag{3.21}$$

Similarly, by Eq (3.8), it is proved that

$$\frac{\partial}{\partial\tau}f\left(\frac{s_h(t,\tau)}{s_h^*(\tau)}\right)=\left(\frac{s_h(t,\tau)}{s_h^*(\tau)}-1\right)\left(\frac{\frac{\partial s_h(t,\tau)}{\partial\tau}}{s_h(t,\tau)}+\frac{b}{N_h}I_m^*\beta_h(\tau)+\varepsilon_h(\tau)\right),$$

and integrating by parts again, we have

$$\int_0^\infty s_h^*(\tau)\frac{\partial}{\partial\tau}f\left(\frac{s_h(t,\tau)}{s_h^*(\tau)}\right)d\tau=\left[s_h^*(\tau)f\left(\frac{s_h(t,\tau)}{s_h^*(\tau)}\right)\right]_{\tau=\infty}-\int_0^\infty\frac{\partial s_h^*(\tau)}{\partial\tau}f\left(\frac{s_h(t,\tau)}{s_h^*(\tau)}\right)d\tau.\tag{3.22}$$

After several calculations making use of (3.8) and the result in (3.22) to simplify the first term of (3.20), rewriting the identity (3.10) as $\mu_h+\gamma=\left(N_hI_h^*\right)^{-1}bI_v^*\int_0^\infty\beta_h(\tau)s_h^*(\tau)d\tau+\left(I_h^*\right)^{-1}\int_0^\infty\delta_h(\omega)a_h^*(\omega)d\omega$ for the second term, using the result of the integral (3.21) for the third term, and rephrasing Eq (3.13) as $\mu_mI_v^*=\beta_mbN_h^{-1}(N_v-I_v^*)\left(I_h^*+\theta\int_0^\infty a_h^*(\omega)d\omega\right)$ for the fourth term of the derivative of L, this reduces to

$$\begin{aligned}\frac{dL}{dt}=&-\frac{\mu_mN_h}{I_m^*b}\int_0^\infty s_h^*(\tau)\left(\frac{S_h(t,\tau)}{s_h^*(\tau)}-1\right)\left(\frac{\frac{\partial S_h(t,\tau)}{\partial\tau}}{S_h(t,\tau)}+\frac{b}{N_h}I_m^*\beta_h(\tau)+\kappa_h(\tau)\right)d\tau\\&+\mu_m\int_0^\infty s_h^*(\tau)\beta_h(\tau)\left(\frac{S_h(t,\tau)}{s_h^*(\tau)}-\frac{S_h(t,\tau)I_m(t)}{s_h^*(\tau)I_m^*}+\frac{I_m(t)}{I_m^*}-1\right)d\tau\\&+\mu_m\int_0^\infty\beta_h(\tau)s_h^*(\tau)\left(\frac{S_h(t,\tau)I_m(t)}{s_h^*(\tau)I_m^*}-\frac{I_h(t)}{I_h^*}-\frac{S_h(t,\tau)I_h^*I_m(t)}{s_h^*(\tau)I_h(t)I_m^*}+1\right)d\tau\\&+\frac{\mu_mN_h}{I_m^*b}\int_0^\infty\delta_h(\omega)a_h^*(\omega)\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}-\frac{I_h(t)}{I_h^*}-\frac{I_h^*a_h(t,\omega)}{I_h(t)a_h^*(\omega)}+1\right)d\omega-\frac{\mu_mN_h}{I_m^*b}\left[g(\omega)a_h^*(\omega)f\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}\right)\right]_{\omega=\infty}\\&+\frac{\mu_mN_h}{I_m^*b}\int_0^\infty\delta_h(\omega)a_h^*(\omega)\left(f\left(\frac{I_h(t)}{I_h^*}\right)-f\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}\right)\right)d\omega\\&+\frac{\beta_mbN_m\theta}{I_m^*N_h}\int_0^\infty s_h^*(\tau)\beta_h(\tau)d\tau\int_0^\infty a_h^*(\omega)\left(f\left(\frac{I_h(t)}{I_h^*}\right)-f\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}\right)\right)d\omega\\&+\frac{\beta_mb^2(N_m-I_m^*)\theta}{I_m^*N_h^2\mu_m}\int_0^\infty s_h^*(\tau)\beta_h(\tau)d\tau\int_0^\infty a_h^*(\omega)\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}-\frac{I_m(t)}{I_m^*}-\frac{a_h(t,\omega)I_m^*}{a_h^*(\omega)I_m(t)}+1\right)d\omega\\&+\frac{\beta_mb^2(N_m-I_m^*)I_h^*}{I_m^*N_h^2\mu_m}\int_0^\infty s_h^*(\tau)\beta_h(\tau)d\tau\left(\frac{I_h(t)}{I_h^*}-\frac{I_m(t)}{I_m^*}-\frac{I_h(t)I_m^*}{I_h^*I_m^(t)}+1\right)\\&+\frac{\beta_mb^2}{N_h^2\mu_m}\int_0^\infty s_h^*(\tau)\beta_h(\tau)d\tau\left(2-\frac{I_m^*}{I_m(t)}-\frac{I_m(t)}{I_m^*}\right)\left(I_h(t)+\theta\int_0^\infty a_h(t,\omega)d\omega\right).\end{aligned}$$

As a result of various calculations, we have

$$\frac{dL}{dt} = -\frac{\mu_m N_h}{I_m^* b}\left[s_h^*(\tau) f\left(\frac{s_h(t,\tau)}{s_h^*(\tau)}\right)\right]_{\tau=\infty} - \frac{\mu_m N_h}{I_m^* b}\int_0^\infty \varepsilon_h(\tau) s_h^*(\tau) f\left(\frac{s_h(t,\tau)}{s_h^*(\tau)}\right) d\tau$$
$$+ \frac{\beta_m b}{N_h}\int_0^\infty \beta_h(\tau) s_h^*(\tau) d\tau \left(2 - \frac{I_m^*}{I_m(t)} - \frac{I_m(t)}{I_m^*}\right)\left(I_h(t) + \theta\int_0^\infty a_h(t,\omega) d\omega\right) - \left[\frac{\mu_m N_h}{I_m^* b} g(\omega) a_h^*(\omega) f\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}\right)\right]_{\omega=\infty}$$
$$+ \frac{\beta_m b I_h^*}{N_h I_m^*}(N_m - I_m^*)\int_0^\infty \beta_h(\tau) s_h^*(\tau)\left(\ln\left(\frac{s_h(t,\tau) I_h^* I_m(t)}{s_h^*(\tau) I_h(t) I_m^*}\right) + 1 - \frac{s_h(t,\tau) I_h^* I_m(t)}{s_h^*(\tau) I_h(t) I_m^*} + \ln\left(\frac{I_h(t) I_m^*}{I_h^* I_m(t)}\right) + 1 - \frac{I_h(t) I_m^*}{I_h^* I_m(t)}\right) d\tau$$
$$+ \frac{\beta_m b\theta}{N_h I_m^*}(N_m - I_m^*)\int_0^\infty\int_0^\infty a_h^*(\omega)\beta_h(\tau) s_h^*(\tau)\left(\ln\left(\frac{s_h(t,\tau)}{s_h^*(\tau)}\right) - \frac{s_h(t,\tau) I_h^* I_m(t)}{s_h^*(\tau) I_h(t) I_m^*} - \frac{a_h(t,\omega) I_m^*}{a_h^*(\omega) I_m(t)}\right) d\tau d\omega$$
$$+ \frac{\beta_m b\theta}{N_h I_m^*}(N_m - I_m^*)\int_0^\infty\int_0^\infty a_h^*(\omega)\beta_h(\tau) s_h^*(\tau)\left(2 - \ln\left(\frac{I_h(t)}{I_h^*}\right) + \ln\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}\right)\right) d\tau d\omega$$
$$+ \frac{\mu_m N_h}{I_m^* b}\int_0^\infty \delta_h(\omega) a_h^*(\omega)\left(-\frac{I_h^* a_h(t,\omega)}{I_h(t) a_h^*(\omega)} + 1 - \ln\left(\frac{I_h(t)}{I_h^*}\right) + \ln\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}\right)\right) d\omega$$
$$= -\frac{\mu_m N_h}{I_m^* b}\left[s_h^*(\tau) f\left(\frac{s_h(t,\tau)}{s_h^*(\tau)}\right)\right]_{\tau=\infty} - \frac{\mu_m N_h}{I_m^* b}\int_0^\infty \varepsilon_h(\tau) s_h^*(\tau) f\left(\frac{s_h(t,\tau)}{s_h^*(\tau)}\right) d\tau - \left[\frac{\mu_m N_h}{I_m^* b} g(\omega) a_h^*(\omega) f\left(\frac{a_h(t,\omega)}{a_h^*(\omega)}\right)\right]_{\omega=\infty}$$
$$- \frac{\beta_m b}{N_h}\int_o^\infty \beta_h(\tau) s_h^*(\tau) d\tau \left(I_h(t) + \theta\int_0^\infty a_h(t,\omega) d\omega\right)\left(f\left(\frac{I_m^*}{I_m(t)}\right) + f\left(\frac{I_m(t)}{I_m^*}\right)\right)$$
$$- \frac{\beta_m b}{N_h}(N_m - I_m^*)\frac{I_h^*}{I_m^*}\int_0^\infty \beta_h(\tau) s_h^*(\tau)\left(f\left(\frac{s_h(t,\tau) I_h^* I_m(t)}{s_h^*(\tau) I_h(t) I_m^*}\right) + f\left(\frac{I_h(t) I_m^*}{I_h^* I_m(t)}\right)\right) d\tau$$
$$- \frac{\beta_m b}{N_h I_m^*}(N_m - I_m^*)\theta\int_0^\infty\int_0^\infty a_h^*(\omega)\beta_h(\tau) s_h^*(\tau)\left(f\left(\frac{s_h(t,\tau) I_h^* I_m(t)}{s_h^*(\tau) I_h(t) I_m^*}\right) + f\left(\frac{a_h(t,\omega) I_m^*}{a_h^*(\omega) I_m(t)}\right)\right) d\tau d\omega$$
$$- \frac{\mu_m N_h}{I_m^* b}\int_0^\infty \delta_h(\omega) a_h^*(\omega)\left(f\left(\frac{I_h^* a_h(t,\omega)}{I_h(t) a_h^*(\omega)}\right)\right) d\omega.$$

Hence, $R_0 > 1$ guarantees that the endemic equilibrium state exists and $L(t)$ is a positive definite functional such that $dL/dt \geq 0$ and $\{E^*\}$ is the maximum invariant set where $dL/dt = 0$. If $R_0 > 1$, by Theorem 2.53 of Smith and Thieme [20], the endemic equilibrium E^* is globally asymptotically stable. We arrived at the following results:

Theorem 3.3. *The endemic equilibrium state E^* of the system (2.2) is globally asymptotically stable if $R_0 > 1$.*

4. Particular cases of the model

4.1. Host-vector model with asymptomatic-infection age for the relapse rate

We consider the change of variable $S_h(t) = \int_0^\infty s_h(t,\tau) d\tau$, which represents the total number of susceptible hosts. By defining the functions $\beta_h(\tau) = \beta_h$ and $\varepsilon_h(\tau) = \varepsilon_h$, we integrate the first equation of the system (2.2) using the boundary condition given in the second expression. This yields:

$$\frac{dS_h}{dt} = \Lambda - \beta_h \frac{b}{N_h} S_h(t) I_m(t) - \rho_h S_h(t),$$
$$\frac{dI_h(t)}{dt} = \beta_h \frac{b}{N_h} S_h(t) I_m(t) d\tau - (\mu_h + \gamma) I_h(t) + \int_0^\infty \delta_h(\omega) a_h(t,\omega) d\omega,$$
$$\frac{\partial a_h(t,\omega)}{\partial t} + \frac{\partial a_h(t,\omega)}{\partial \omega} = -(\mu_h + \delta_h(\omega)) a_h(t,\omega),$$

$$a_h(t,0) = p\gamma I_h(t),$$
$$\frac{dI_m(t)}{dt} = \frac{\beta_m b}{N_h}(N_m - I_m(t))\left(I_h(t) + \theta\int_0^\infty a_h(t,\omega)d\omega\right) - \mu_m I_m(t).$$

The model is now independent of the age of susceptible hosts and depends only on the asymptomatic-infection age.

4.2. Host-vector model with chronological age of susceptible hosts

To reduce the original model (2.2) to one that depends only on the age of susceptible hosts, we define $A(t) = \int_0^\infty a(t,\omega)d\omega$ as the total number of asymptomatic hosts and consider the function to be a constant $\delta_h(\omega) = \delta_h$. By integrating the fourth equation of (2.2) with the boundary condition given in the fifth expression, we obtain:

$$\frac{\partial s_h(t,\tau)}{\partial t} + \frac{\partial s_h(t,\tau)}{\partial \tau} = -\beta_h(\tau)\frac{b}{N_h}s_h(t,\tau)I_m(t) - \rho_h(\tau)s_h(t,\tau), \tag{4.1}$$
$$s_h(t,0) = \Lambda, \tag{4.2}$$
$$\frac{dI_h(t)}{dt} = \int_0^\infty \beta_h(\tau)\frac{b}{N_h}s_h(t,\tau)I_m(t)d\tau - (\mu_h+\gamma)I_h(t) + \delta_h A_h(t), \tag{4.3}$$
$$\frac{dA_h(t)}{dt} = p\gamma I_h(t) - (\mu_h+\delta_h)A_h(t), \tag{4.4}$$
$$\frac{dI_m(t)}{dt} = \frac{\beta_m b}{N_h}(N_m - I_m(t))\,(I_h(t) + \theta A_h(t)) - \mu_m I_m(t). \tag{4.5}$$

We have an age-independent model of relapse that depends only on the chronological age. In the special case where $\delta_h = \theta = p = 0$, the model reduces to an SIR-SI host-vector model with chronological age of susceptible hosts [18].

4.3. Age-independent model

Now, applying both transformations $S_h(t) = \int_0^\infty s_h(t,\tau)d\tau$ and $A(t) = \int_0^\infty a(t,\omega)d\omega$ simultaneously, and defining the constant functions $\beta_h(\tau) = \beta_h$, $\rho_h(\tau) = \mu_h$, $\delta_h(\omega) = \delta_h$, and $\Lambda = \mu_h N_h$, we integrate the first equation in (2.2) with respect to τ and the fourth equation with respect to ω, applying their respective boundary conditions. This reduction yields:

$$\frac{dS_h}{dt} = \mu_h N_h - \frac{\beta_h b}{N_h}S_h(t)I_m(t) - \mu_h S_h(t),$$
$$\frac{dI_h}{dt} = \frac{\beta_h b}{N_h}S_h(t)I_m(t) - (\mu_h+\gamma)I_h(t) + \delta_h A_h(t),$$
$$\frac{dA_h}{dt} = p\gamma I_h(t) - (\mu_h+\delta_h)A_h(t),$$
$$\frac{dI_m}{dt} = \frac{\beta_m b}{N_h}(N_m - I_m(t))(I_h(t) + \theta A_h(t)) - \mu_m I_m(t).$$

This model is independent of both the age of susceptible hosts and the relapse age. It is isomorphic to the malaria model without temporary immunity [15] and the same for the chikungunya model [17].

In the special case where $\delta_h = \theta = p = 0$, the age-independent model reduces to a classical dengue model [21]. In [17], this model was fitted to a chikungunya outbreak in Mexico. It was shown that relapse has important effects on the dynamics of the disease; however, the age-dependent model can describe a more detailed and complex dynamic as the reduced model, as we will see in the next section of numerical simulations.

5. Numerical simulations

Numerical simulations were performed to test the effect of chronological age and asymptomatic-infection age. We used the method of lines with a first-order upwind scheme for derivatives concerning the chronological age τ and the age of asymptomatic infection ω [22]. For integration in time t, the *odeint* Python function that employs the *lsoda* algorithm was used. We used the following function: $\beta_h(\tau)$

$$\beta_h(\tau) = \begin{cases} \beta_1, & \text{if } 0 < \tau \leq \tau^{\beta}_{min} \\ \beta_2, & \text{if } \tau^{\beta}_{min} < \tau \leq \tau^{\beta}_{med} \\ \beta_3, & \text{if } \tau^{\beta}_{med} < \tau, \end{cases}$$

where τ^{β}_{min}, τ^{β}_{med}, and τ^{β}_{max} are chronological ages, and β_1, β_2, and β_3 , are positive constants. This function represents three age groups: young people, adults, and older adults, where the probability of contracting the pathogen of an infected mosquito in young people is greater than in adults ($\beta_1 > \beta_2$), and the probability of contagion in adults is greater than in older adults ($\beta_2 > \beta_3$). First we made a simulation of the model without asymptomatic age-dependence relapse. Then we compared with the case of age of asymptomatic infection dependence. We employed the following relapse rate $\delta(\omega)$ reported in [19]:

$$\delta(\omega) = \begin{cases} \delta_1, & \text{if } 0 < \omega \leq \omega^{\delta}_{min} \\ \delta_2, & \text{if } \omega^{\delta}_{min} < \omega \leq \omega^{\delta}_{med} \\ \delta_3, & \text{if } \omega^{\delta}_{med} < \omega, \end{cases}$$

where ω^{δ}_{min}, ω^{δ}_{med}, and ω^{δ}_{max} are asymptomatic-infection ages, and δ_1, δ_2, and δ_3 , are positive constants.

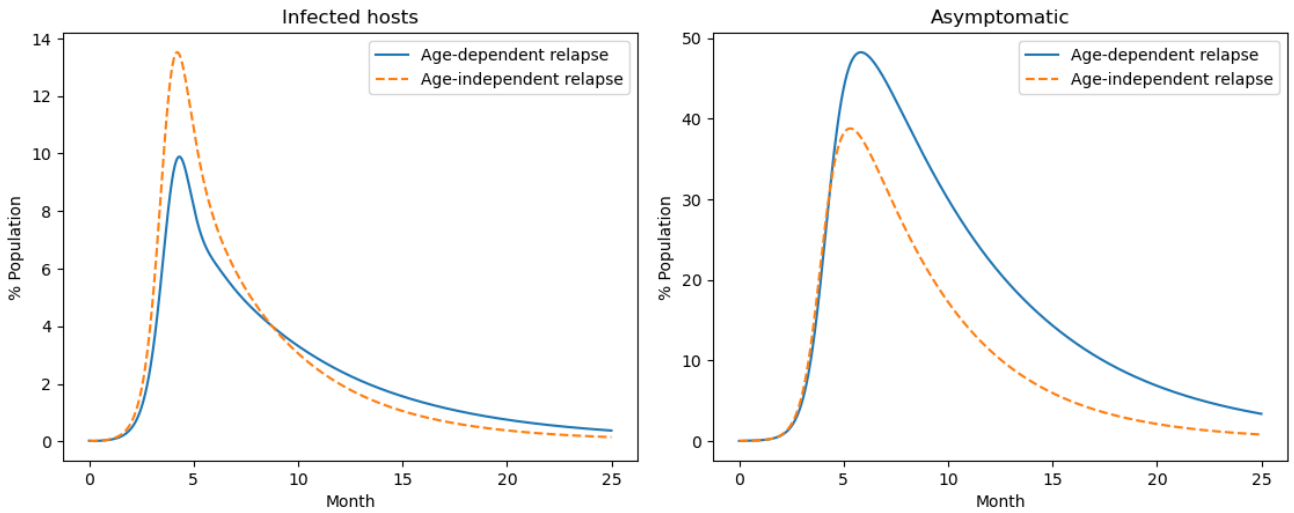

Figure 1. Effect of age dependence on relapse in infected and asymptomatic hosts.

We observe that asymptomatic age dependence leads to a prolonged period of infection following the primary outbreak and is caused by the relapse. Consequently, the duration of the infection is extended, and the asymptomatic cases are amplified. The unconventional behavior observed in Figure 1 (right) can only be achieved by considering the relapse rate dependence on the asymptomatic age, in contrast to the common behavior shown by traditional compartmental models, as shown in Figure 1 (left).

We also compare a simulation of an age-independent vaccination strategy with a more realistic age-dependence vaccination strategy. We used a specific form of the age-dependent vaccination rate $\rho(\tau)$:

$$\rho(\tau) = \begin{cases} 0, & \text{if } 0 < \tau \leq \tau^{\rho}_{min} \\ \rho_0, & \text{if } \tau^{\rho}_{min} < \tau \leq \tau^{\rho}_{med} \\ 0, & \text{if } \tau^{\rho}_{med} < \tau, \end{cases}$$

where ρ_0 is a positive constant. The age group of those under τ^{ρ}_{min} and over τ_{med} represents those children and older adults for whom the vaccine is not approved. The vaccination is applied for the age group $\tau^{\rho}_{min} < \tau \leq \tau^{\rho}_{med}$ with the highest risk of mosquito-borne infection.

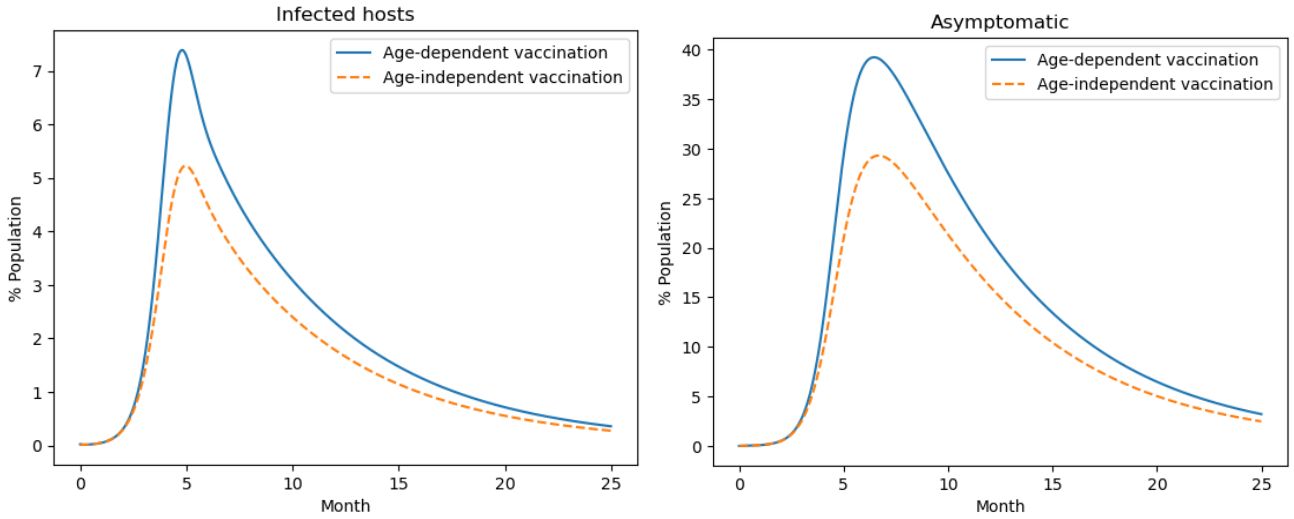

Figure 2. Effect of age-dependent vaccination strategies on infected and asymptomatic hosts.

Figure 2 shows the importance of considering age dependence in control strategies. If not, the effectiveness of a strategy can be overestimated, as shown in Figure 2 (left). This overestimation also occurs in the effect of the control in asymptomatic cases. In realistic scenarios, vaccination is focused on an age group, as shown in Figure 2 (right) (vaccination between 12 and 22 years), because of vaccine specifications or economic constraints. The values for the simulations were taken from [17], and are shown in Tables 1 and 2.

Table 1. Parameter values used for the simulations.

Parameter	Value
β_h	0.7379
β_v	0.7570
b	3.2158
μ_v	3.3510
γ	4.5821
p	0.6536
δ_h	0.7506
σ	0.2558
N_v	19,440.1813

Table 2. Parameter values used for the age-dependent rates.

parameter	β_1	β_2	β_3	τ^{β}_{min}	τ^{β}_{med}
value	$1.5\beta_2$	0.7379	$0.5\beta_2$	12 years	60 years
parameter	δ_1	δ_2	δ_3	ω^{δ}_{min}	ω^{δ}_{med}
value	0	0.7506	$0.5\delta_2$	1 week	2 months
parameter		ρ_0		τ^{ρ}_{min}	τ^{ρ}_{med}
values		$100/(\tau^{\rho}_{med} - \tau^{\rho}_{min})$		12 years	22 years

6. Concluding remarks

In this study, we have developed a mathematical model to describe the dynamics of mosquito-borne diseases by incorporating both chronological age and asymptomatic-infection age. Chronological age accounts for the time since birth, affecting susceptibility and exposure, while asymptomatic-infection age reflects the duration of the asymptomatic phase before relapse. By allowing both dependencies to be functions of ages τ and ω, our model provides a more complete view of mosquito-borne disease dynamics, addressing variability in susceptibility and relapse rates, which have been ignored in other models.

We have analytically demonstrated the global stability of the system using the direct Lyapunov method with a Volterra-type functional. Our results confirm that the system (2.2) behaves as follows: If $R_0 \leq 1$, the disease-free equilibrium state E^0 is globally asymptotically stable. Conversely, if $R_0 > 1$, the endemic equilibrium state E^* exists and is also globally asymptotically stable.

It is noteworthy that we only impose integrability conditions on the age-specific probability of becoming an infected host through mosquito bites $\beta_h(\omega)$, age-dependent vaccination rate $\rho(\tau)$, and age-dependent relapse rate $\delta_h(\omega)$ in the model, and hence, the global stability results derived are robust and do not depend on specific forms of this function.

Some forms of the functions $\rho(\tau)$, $\beta_h(\tau)$, and $\delta(\omega)$ have been proposed in [19, 23]. The mosquito-host transmission function $\beta_h(\tau) = \beta_0\tau \exp[-\beta_1\tau]$ is reported in [23], with $\beta_0 > 0, \beta_1 > 0$, and $\tau > 0$ as

the chronological age. We proposed the following function $\beta_h(\tau)$:

$$\beta_h(\tau) = \begin{cases} \beta_1, & \text{if } 0 < \tau \leq \tau^{\beta}_{min} \\ \beta_2, & \text{if } \tau^{\beta}_{min} < \tau \leq \tau^{\beta}_{med} \\ \beta_3, & \text{if } \tau^{\beta}_{med} < \tau < \tau^{\beta}_{max}, \end{cases}$$

where τ^{β}_{min}, τ^{β}_{med}, and τ^{β}_{max} are chronological ages, and β_1, β_2, and β_3 , are positive constants. This function represents three age groups: young people, adults, and older adults, where the probability of contracting the pathogen of an infected mosquito in young people is greater than in adults ($\beta_1 > \beta_2$), and the probability of contagion in adults is greater than in older adults ($\beta_2 > \beta_3$).

We proposed a specific form of the age-dependent vaccination rate $\rho(\tau)$:

$$\rho(\tau) = \begin{cases} 0, & \text{if } 0 < \tau \leq \tau^{\rho}_{min} \\ \rho_0 \tau \exp\left[-\rho_1 \tau\right], & \text{if } \tau^{\rho}_{min} < \tau \leq \tau^{\rho}_{med} \\ 0, & \text{if } \tau^{\rho}_{med} < \tau < \tau^{\rho}_{max}, \end{cases}$$

where ρ_0 and ρ_1 are positive constants. The age group of those under τ^{ρ}_{min} and over τ_{med} represents those children and older adults for whom the vaccine is not approved. $\rho_0 \tau \exp\left[-\rho_1 \tau\right]$ is the vaccination strategy for the age group $\tau^{\rho}_{min} < \tau \leq \tau^{\rho}_{med}$ with the highest risk of mosquito-borne infection.

The following relapse rate $\delta(\omega)$ is reported in [19]:

$$\delta(\omega) = \begin{cases} \delta_1, & \text{if } 0 < \omega \leq \omega^{\delta}_{min} \\ \delta_2, & \text{if } \omega^{\delta}_{min} < \omega \leq \omega^{\delta}_{med} \\ \delta_3, & \text{if } \omega^{\delta}_{med} < \omega < \omega^{\delta}_{max}, \end{cases}$$

where ω^{δ}_{min}, ω^{δ}_{med}, and ω^{δ}_{max} are asymptomatic-infection ages, and δ_1, δ_2, and δ_3 are positive constants. The relapse rate and the duration of an asymptomatic infection are monitored in this function using the variable asymptomatic-infection age. Considering the fact that relapses decrease in frequency over time, $1/\delta_2 < 1/\delta_3$.

We further explored specific cases of the model to understand the impact of different assumptions on its dynamics. By focusing on asymptomatic-infection age and simplifying the model to be independent of the chronological age of susceptible hosts, we derived a model that emphasizes the influence of the variability of relapse periods. Additionally, by concentrating on chronological age and simplifying the model to be independent of the asymptomatic-infection age, the model reduces to the SIR-SI model with the chronological age of susceptible hosts [18]. By applying transformations to eliminate dependencies on both the chronological age of susceptible hosts and the asymptomatic-infection age, we obtained a model that simplifies to forms isomorphic to existing malaria, chikungunya, and dengue models [15, 17, 21]. These examples demonstrate the versatility of the model and its applicability to a range of mosquito-borne diseases. In [17], a simplified version of the model presented in this manuscript without age-dependent factors is fitted to an actual chikungunya outbreak in Acapulco, Mexico. The model gave a good general description of the outbreak, although the outbreak data showed a fat tail in the infected class. On the other hand, the numerical simulations in this manuscript showed that the age-dependent factors in the proposed model led to a fat tail epidemic curve, making this model a plausible explanation of this observed phenomenon. Models with age structure have significant potential to describe complex dynamics resulting from non-trivial relationships between

age-related transitions between compartments. For instance, it has been observed that Zika exhibits vertical transmission in addition to the presence of asymptomatic Zika [24, 25]. The effect of these phenomena should be analyzed using an age-structured model in future research.

Finally, we suggest that the age-dependent functions $\rho(\tau)$, $\beta_h(\tau)$, and $\delta(\omega)$ for the basic reproductive number can offer useful guidance for control and prevention strategies. These models with continuous-time age structure can be applied to the dynamics of mosquito-borne diseases.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

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Conflict of interest

The authors declare there is no conflict of interest.

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