Gulf Journal of Mathematics
Vol 20, Issue 2 (2025) 364-386

ISSN: 2309-4966
https://doi.org/10.56947/gjom.v20i2.3248

MONKEYPOX DISEASE WITH SATURATED INCIDENCE RATES: MATHEMATICAL ANALYSIS

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Abstract. In this study, we explore the dynamics of monkeypox disease by developing and examining a deterministic mathematical model that describes the interactions between human and rodent populations. First, we demonstrate the well-posedness of the model. The basic reproduction number is expressed as a function of the human reproduction number and the rodent reproduction number. The problem admits three equilibria, namely the disease-free equilibrium, the human-only endemic equilibrium, and the endemic equilibrium. We then analyze the local and the global stability of those equilibria. To further assess the equilibria stability, different numerical simulations are executed in order to support the theoretical findings and to highlight the impact of different parameters in eradicating the disease.

Keywords. Monkeypox, saturated incidence rate, stability analysis, basic reproduction rate, equilibrium points.

1. Introduction

The monkeypox virus is an orthopoxvirus closely related to the smallpox virus, and it causes monkeypox, a zoonotic infection. Historical records trace the first appearance of human monkeypox to Central Africa in 1970. The disease typically presents with symptoms such as fever, rash, and lymphadenopathy. Studies show that, while vaccines exist, the smallpox vaccine can afford approximately 85% protection against monkeypox [18]. Prior to the identification of human cases, the virus was first detected in monkeys in 1959 by a research facility in Copenhagen, Denmark, where it was recognized as a pox-like illness [12]. Monkeypox can spread in two ways: from human to human or from animal to human. The transmission of the virus can occur from human-to-human through respiratory emissions, contact with infected fluids, environments, or through physical contact with the skin lesions of an infected person [14]. Animal-to-human infection may happen through direct exposure to bodily fluids, blood, or skin and mucosal lesions of an infected animal. Several cases of monkeypox have been reported in

Date: Received: Jul 9, 2025; Accepted: Aug 10, 2025.
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various African animals, such as dormice, tree squirrels, rope squirrels, and Gambian pouched rats, among others. While rats are considered the most likely source of transmission, the definitive natural reservoir of the virus remains unidentified [8]. Compartmental modeling techniques have been extensively used to describe the spread of infectious diseases within populations. The SIR model, introduced by Kermack and McKendrick [10], is the simplest and most traditional approach for modeling disease spread. In the SIR model, people are classified into three epidemiological states: those who are susceptible to the disease (S), those actively infected (I), and those who have recovered (R). The susceptible group (S) includes individuals who are at risk but, have not yet contracted the disease, the infected group (I) consistes of individuals who can transmit the disease, and the recovered group (R) represents individuals who have recovered from the illness. The dynamics of monkeypox transmission have been analyzed using a simplified SIR model [3, 1]. Monkeypox symptoms typically last between two and four weeks, and the incubation period is usually estimated to be around two weeks. To better capture the spread of the disease, an additional compartment for exposed individuals (E) is often included. Several studies have used the $SEIR$ (Susceptible-Exposed-Infected-Recovered) model to investigate monkeypox transmission [17, 21].

Recent research has examined the interaction between infected humans and rodents with monkeypox virus using bilinear infection rates. In [15], researchers developed a model with seven compartments. They studied the stability of the model, and then they established necessary conditions for the existence of optimal control. Including the compartment of exposed rodents E_r a monkeypox model was studied in [14], the human population is divided into four compartments including exposed and quarantine: S_h, E_h, I_h, Q_h and R_h while the rodents are divided to three compartments S_r, E_r and I_r, the authors gave suitable conditions for the local stability of both disease-free equilibrium and endemic equilibrium, they studied the global stability of disease-equilibrium and the existence of the backward bifurcation. Two models describing the transmission of monkeypox dynamics using control measures have been studied in [4], one of them is deterministic and the other is stochastic, each population in this model is divided into four compartments: Susceptible, exposed, infected and removed, the model was analyzed, three equilibrium points were calculated and the global stability of disease-free equilibrium was established. To measure the effectiveness of the vaccine on monkeypox, a compartmental model was established in [16] applying control theory, the human population was divided into susceptible, vaccinated, exposed, infected, and removed, while the rodent population was composed of susceptible and infected. Equilibrium points were calculated, and their global stability was proved under certain circumstances. In [2], a dual-strain SEIR epidemic model was presented; the model introduces four distinct non-monotonic incidence functions to evaluate the transmission dynamics of the Central and West African clades of monkeypox. The model presented in [6] takes into consideration the effect of the interchange between the human and rodent populations, with imperfect vaccination and nonlinear incidence rates. In [19], the authors

proposed a monkeypox model including a level of awareness. The human population is divided into eight compartments, which include vaccinated, quarantine, hospitalized, and infected with severe consequences, and the rodent population is divided into susceptible and infected. The authors of [9] developed a model that considers the impact of contaminated surfaces and provides an optimal control. This study aims to expand on the previous work by incorporating saturated incidence rates instead on the form $\frac{\alpha SI}{1+\nu I}$, where $1+\nu I$ represents the saturation effect. The primary motivation for this adjustment is to account for the effects of a densely crowded infected population. In fact, this type of incidence rates is more realistic when describing the transmission dynamics, for diseases like monkeypox, where public awareness, isolation, and capacity limitations can reduce transmission. The model includes seven dynamic equations that describe the interactions between susceptible and infected humans, as well as susceptible and infected rodents. The mathematical formulation of the model is as follows:

$$\begin{cases} \dfrac{dS_h}{dt} = \Lambda_h - \left(\dfrac{\alpha_h I_h S_h}{1+\nu_h I_h} + \dfrac{\alpha_r I_r S_h}{1+\nu_r I_r} \right) - d_h S_h, \\ \dfrac{dE_h}{dt} = \dfrac{\alpha_h I_h S_h}{1+\nu_h I_h} + \dfrac{\alpha_r I_r S_h}{1+\nu_r I_r} - (\rho + d_h) E_h, \\ \dfrac{dI_h}{dt} = \rho E_h - (d_h + \delta_h + \theta) I_h, \\ \dfrac{dR_h}{dt} = \theta I_h - d_h R_h, \\ \dfrac{dS_r}{dt} = \Lambda_r - \dfrac{\alpha I_r S_r}{1+\beta I_r} - d_r S_r, \\ \dfrac{dE_r}{dt} = \dfrac{\alpha I_r S_r}{1+\beta I_r} - (\tau + d_r) E_r, \\ \dfrac{dI_r}{dt} = \tau E_r - d_r I_r. \end{cases} \tag{1.1}$$

In this model, the variables are defined as follows: S_h represents susceptible humans, E_h represents exposed humans, I_h represents infected humans, R_h represents recovered humans, S_r represents susceptible rodents, E_r represents exposed rodents and I_r represents infected rodents. The parameters in the model are defined as: Λ_h is the human recruitment rate, Λ_r is the rodent recruitment rate, α_h is the human infection rate, ν_h is the human saturated infection rate, α_r is the rodent infection rate, ν_r is the rodent saturated infection rate, d_h is the human natural mortality rate, d_r is the rodent natural mortality rate, ρ is the transfer rate from exposed humans to infected humans, δ_h is the human mortality rate due to monkeypox infection, θ is the human recovery rate, α describes the rodent-rodent infection rate, τ describes the proportion of the exposed rodents becoming infected and β is the rodent-rodent incidence saturation rate.
The transmission dynamics of monkeypox between humans and rodents are illustrated in Fig.1.

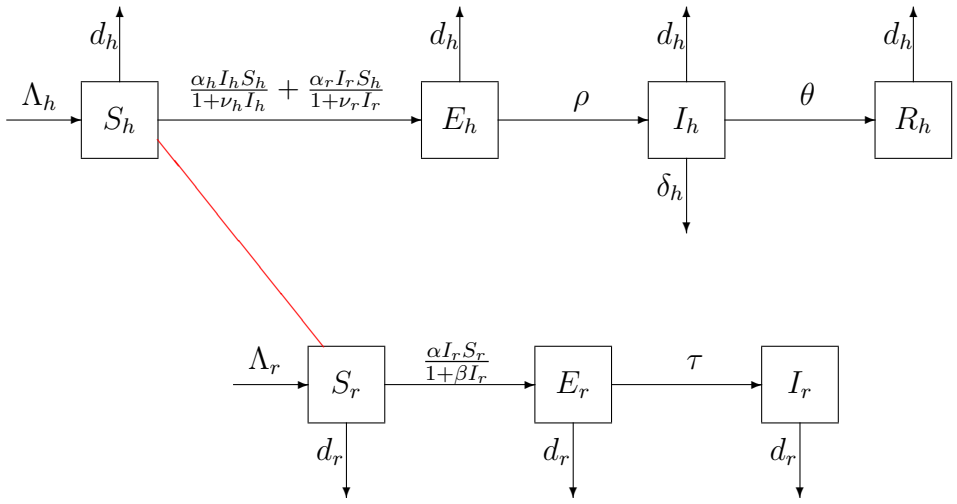

FIGURE 1. Monkeypox infection diagram.

The paper is structured as follows. The problem well-posedness is proved in the next Section 2. The basic reproduction number and the equilibria of the problem are given in Section 3. The local stability study is fulfilled in Section 4. Followed after in Section 5 by the global stability results. Numerical simulations are performed in Section 6. The paper ends with a conclusion in the last section.

2. The problem well-posedness

In this section, we will establish the well-posedness of the model by proving that system (1.1) admits positive and bounded solutions for all initial conditions. The positivity of solutions guarantees that the population variables remain non-negative over time. On the other hand, boundedness ensures that the solutions do not grow without limit. These two properties confirm that the model is mathematically and biologically well-posed.

Theorem 2.1. *If, at the time $t = 0$, the state variables of the system (1.1) are $S_h(0) > 0$, $E_h(0) > 0$, $I_h(0) > 0$, $R_h(0) > 0$, $S_r(0) > 0$, $E_r(0) > 0$, $I_r(0) > 0$, then $S_h(t)$, $E_h(t)$, $I_h(t)$, $R_h(t)$, $S_r(t)$, $E_r(t)$, and $I_r(t)$ are positive for every $t > 0$. Moreover, all solutions are bounded.*

Proof. Consider the set $T = sup\{\epsilon \geq 0/\forall t \in [0, \epsilon]$ where $S_h(t) \geq 0$, $E_h(t) \geq 0$, $I_h(t) \geq 0$, $R_h(t) \geq 0$, $S_r(t) \geq 0$, $E_r(t) \geq 0$ and $I_r(t) \geq 0\}$.
Now, we prove that $T = +\infty$. We assume that T is finite; by continuity of solutions, we have, $S_h(T) = 0$, $E_h(T) = 0$, $I_h(T) = 0$, $R_h(T) = 0$,
$S_r(T) = 0$, $E_r(T) = 0$, $I_r(T) = 0$.
If $S_h(T) = 0$ before the other variables become zero. Therefore,

$$\frac{dS_h(T)}{dt} = \lim_{t \to T^-} \frac{S_h(T) - S_h(t)}{T - t} = \lim_{t \to T^-} \frac{-S_h(t)}{T - t} \leq 0.$$

From the first equation of system (1.1) we have:

$$\frac{dS_h(T)}{dt} = \Lambda_h - \left(\frac{\alpha_h I_h(T) S_h(T)}{1+\nu_h I_h(T)} + \frac{\alpha_r I_r(T) S_h(T)}{1+\nu_r I_r(T)}\right) - d_h S_h(T),$$

we obtain,

$$\frac{dS_h(T)}{dt} = \Lambda_h > 0,$$

which is a contradiction.
If $E_h(T) = 0$ before the other variables become zero. Therefore,

$$\frac{dE_h(T)}{dt} = \lim_{t\to T^-} \frac{E_h(T) - E_h(t)}{T-t} = \lim_{t\to T^-} \frac{-E_h(t)}{T-t} \leq 0.$$

From the second equation of system (1.1) we have:

$$\frac{dE_h(T)}{dt} = \left(\frac{\alpha_h I_h(T) S_h(T)}{1+\nu_h I_h(T)} + \frac{\alpha_r I_r(T) S_h(T)}{1+\nu_r I_r(T)}\right) - (\rho + d_h) E_h(T),$$

therefore,

$$\frac{dE_h(T)}{dt} = \left(\frac{\alpha_h I_h(T) S_h(T)}{1+\nu_h I_h(T)} + \frac{\alpha_r I_r(T) S_h(T)}{1+\nu_r I_r(T)}\right) > 0,$$

this leads to a contradiction.
If $I_h(T) = 0$ before the other variables become zero. Then,

$$\frac{dI_h(T)}{dt} = \lim_{t\to T^-} \frac{I_h(T) - I_h(t)}{T-t} = \lim_{t\to T^-} \frac{-I_h(t)}{T-t} \leq 0.$$

From the third equation of system (1.1) we have:

$$\frac{dI_h(T)}{dt} = \rho E_h(T) - (d_h + \delta_h + \theta) I_h(T),$$

hence,

$$\frac{dI_h(T)}{dt} = \rho E_h(T) > 0,$$

this represents a contradiction.
If $R_h(T) = 0$ before the other variables become zero. Then we have,

$$\frac{dR_h(T)}{dt} = \lim_{t\to T^-} \frac{R_h(T) - R_h(t)}{T-t} = \lim_{t\to T^-} \frac{-R_h(t)}{T-t} \leq 0.$$

From the fourth equation of system (1.1) we obtain:

$$\frac{dR_h(T)}{dt} = \theta I_h(T) - d_h R_h(T),$$

then,

$$\frac{dR_h(T)}{dt} = \theta I_h(T) > 0,$$

this represents a contradiction.
By the same method we prove the positivity of $S_r(t)$, $E_r(t)$ and $I_r(t)$.
Now, we will prove the boundedness of solutions.
We have,

$$\frac{dN_h}{dt} = \Lambda_h - d_h N_h - \delta_h I_h.$$

Then,

$$\frac{\frac{dN_h}{dt}}{\Lambda_h - d_h N} \leq 1.$$

By integrating, we have:

$$\int_0^t \frac{\frac{dN_h(s)}{ds}}{\Lambda_h - d_h N_h(s)} ds \leq \int_0^t ds.$$

Therefore,

$$ln\left(\Lambda_h - d_h N_h(t)\right) \geq -d_h t + ln\left(\Lambda_h - d_h N_h(0)\right).$$

Which implies that:

$$\Lambda_h - d_h N_h(t) \geq \left(\Lambda_h - d_h N_h(0)\right) e^{-d_h t}.$$

Consequently,

$$N_h(t) \leq \frac{\Lambda_h}{d_h} + N_h(0) e^{-d_h t}.$$

By the same method we will have,

$$N_r(t) \leq \frac{\Lambda_r}{d_r} + N_r(0) e^{-d_r t}.$$

When $t \longrightarrow +\infty$, we will have $N_h(t) \leq \frac{\Lambda_h}{d_h}$ and $N_r(t) \leq \frac{\Lambda_r}{d_r}$.
This proves that N_h and N_r are bounded and so are $S_h,\ E_h,\ I_h,\ R_h,\ S_r,\ E_r$ and I_r. □

3. Analysis of the model

3.1. **The basic reproduction number.** The basic reproduction number, denoted by $\mathcal{R}_0$, is the average number of new infection cases produced in a completely susceptible population, by a typical infectious individual [5]. If $\mathcal{R}_0 < 1$, then the average number of secondary infections generated is less than one, consequently, the infection disappears in the long term; whereas if $\mathcal{R}_0 > 1$, the epidemic will spread in the population.
To determine the disease-free equilibrium, we suppose that there is no infection in the population. The corresponding equilibrium point can then be determined by putting $I_h = I_r = 0$. The disease-free equilibrium is $E_0 = \left(\frac{\Lambda_h}{d_h}, 0, 0, 0, \frac{\Lambda_r}{d_r}, 0, 0\right)$.
We find the expression of $\mathcal{R}_0$ using the method of next generation matrix [5] described by Van den Driessche and Watmoughin in [20].
We use the same notation as in [20], $\mathcal{F}$ is the matrix representing the rate of appearance of new infection cases in the infected compartments, and $\mathcal{V}$ is the matrix

of the rate of the transfer of individuals in and out infected compartments. Since $E_h,\ I_h,\ E_r$, and I_r are the infected compartments, then:

$$\mathcal{F} = \begin{pmatrix} \frac{\alpha_h I_h S_h}{1+\nu_h I_h} + \frac{\alpha_r I_r S_h}{1+\nu_r I_r} \\ 0 \\ \frac{\alpha I_r S_r}{1+\beta I_r} \\ 0 \end{pmatrix} \quad ; \quad \mathcal{V} = \begin{pmatrix} (\rho + d_h)\, E_h \\ -\rho E_h + (d_h + \delta_h + \theta)\, I_h \\ (\tau + d_r)\, E_r \\ -\tau E_r + d_r I_r \end{pmatrix}.$$

Let F and V be the Jacobians of $\mathcal{F}$ and $\mathcal{V}$ at the disease-free equilibrium, respectively, hence,

$$F = \begin{pmatrix} 0 & \frac{\alpha_h \Lambda_h}{d_h} & 0 & \frac{\alpha_r \Lambda_h}{d_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\alpha \Lambda_r}{d_r} \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} \rho + d_h & 0 & 0 & 0 \\ -\rho & d_h + \delta_h + \theta & 0 & 0 \\ 0 & 0 & \tau + d_r & 0 \\ 0 & 0 & -\tau & d_r \end{pmatrix},$$

The next-generation matrix is:

$$FV^{-1} = \begin{pmatrix} \frac{\rho \alpha_h \Lambda_h}{d_h(\rho+d_h)(d_h+\delta_h+\theta)} & \frac{\alpha_h \Lambda_h}{d_h(d_h+\delta_h+\theta)} & \frac{\tau \alpha_r \Lambda_h}{d_h d_r (d_r+\tau)} & \frac{\alpha_h \Lambda_h}{d_h d_r} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\tau \alpha \Lambda_r}{d_r^2 (d_r+\tau)} & \frac{\alpha \Lambda_r}{d_r} \\ 0 & 0 & 0 & 0 \end{pmatrix}.$$

Since $\mathcal{R}_0$ is described as the largest eigenvalue of the characteristic equation $|FV^{-1} - \lambda I_d| = 0$ then $\mathcal{R}_0 = max\,\{\mathcal{R}_h, \mathcal{R}_r\}$.
Where

$$\mathcal{R}_h = \frac{\rho \alpha_h \Lambda_h}{d_h\,(\rho + d_h)\,(d_h + \delta_h + \theta)} \quad ; \quad \mathcal{R}_r = \frac{\tau \alpha \Lambda_r}{d_r^2\,(d_r + \tau)}.$$

Here $\mathcal{R}_h$ stands for the human reproduction number, it is the number of infected humans caused by one infected human in a completely exposed population and $\mathcal{R}_r$ represents the rodent reproduction number, which is the number of infected rodents caused by one infected rodent in a completely exposed population.

3.2. **Equilibrium points.** In order to determine the equilibrium points of the system (1.1), we set the derivatives of all state variables to zero. We determine the values of the state variables at which the model reaches equilibrium by resolving these equations.

$$\begin{cases} \Lambda_h - \left(\dfrac{\alpha_h I_h S_h}{1+\nu_h I_h} + \dfrac{\alpha_r I_r S_h}{1+\nu_r I_r}\right) - d_h S_h = 0, \\ \dfrac{\alpha_h I_h S_h}{1+\nu_h I_h} + \dfrac{\alpha_r I_r S_h}{1+\nu_r I_r} - (\rho + d_h) E_h = 0, \\ \rho E_h - (d_h + \delta_h + \theta) I_h = 0, \\ \theta I_h - d_h R_h = 0, \\ \Lambda_r - \dfrac{\alpha I_r S_r}{1+\beta I_r} - d_r S_r = 0, \\ \dfrac{\alpha I_r S_r}{1+\beta I_r} - (\tau + d_r) E_r = 0, \\ \tau E_r - d_r I_r = 0. \end{cases} \tag{3.1}$$

Let $E^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_r^*, E_r^*, I_r^*)$ be an arbitrary equilibrium point then :

$$\begin{cases} S_h^* = \dfrac{\Lambda_h}{d_h} - \dfrac{(\rho + d_h)(d_h + \delta_h + \theta)}{\rho d_h} I_h^*, \\ E_h^* = \dfrac{(d_h + \delta_h + \theta)}{\rho} I_h^*, \\ R_h^* = \dfrac{\theta}{d_h} I_h^*, \\ S_r^* = \dfrac{\Lambda_r}{d_r} - \dfrac{(\tau + d_r)}{\tau} I_r^*, \\ E_r^* = \dfrac{d_r}{\tau} I_r^*. \end{cases}$$

We substitute those components in the sixth equation of system (3.1), then we have the two possibilities, $I_r^* = 0$ or $I_r^* = \frac{d_r(\mathcal{R}_r - 1)}{\alpha + \beta d_r}$.

-: If $I_r^* = 0$ then $E_1^* = \left(S_{h1}^*, E_{h1}^*, I_{h1}^*, R_{h1}^*, \frac{\Lambda_r}{d_r}, 0, 0\right)$ is a human-only endemic equilibrium where:

$$\begin{cases} S_{h1}^* = \dfrac{\Lambda_h}{d_h} - \dfrac{(\mathcal{R}_h - 1)(\rho + d_h)(d_h + \delta_h + \theta)}{\rho(\alpha_h + d_h \nu_h)}, \\ E_{h1}^* = \dfrac{d_h(\mathcal{R}_h - 1)(d_h + \delta_h + \theta)}{\rho(\alpha_h + d_h \nu_h)}, \\ I_{h1}^* = \dfrac{d_h(\mathcal{R}_h - 1)}{(\alpha_h + d_h \nu_h)}, \\ R_{h1}^* = \dfrac{\theta(\mathcal{R}_h - 1)}{(\alpha_h + d_h \nu_h)}. \end{cases} \tag{3.2}$$

-: If $I_r^* = \frac{d_r(\mathcal{R}_r - 1)}{\alpha + \beta d_r}$ then $E_2^* = (S_{h2}^*, E_{h2}^*, I_{h2}^*, R_{h2}^*, S_{r2}^*, E_{r2}^*, I_{r2}^*)$ is the endemic equilibrium satisfying system (3.1).

From the above discussion, it is straightforward to state the following result.

Proposition 3.1. *The system (1.1) has three equilibrium points:*

(1) *The disease-free equilibrium* $E_0 = \left(\frac{\Lambda_h}{d_h}, 0, 0, 0, \frac{\Lambda_r}{d_r}, 0, 0\right)$.
(2) *If* $\mathcal{R}_h > 1$ *and* $\mathcal{R}_r < 1$ *there exists a human-only endemic equilibrium* $E_1^* = \left(S_{h1}^*, E_{h1}^*, I_{h1}^*, R_{h1}^*, \frac{\Lambda_r}{d_r}, 0, 0\right)$ *satisfying system (3.2).*
(3) *If* $\mathcal{R}_r > 1$ *there exists a unique endemic equilibrium* $E_2^* = (S_{h2}^*, E_{h2}^*, I_{h2}^*, R_{h2}^*, S_{r2}^*, E_{r2}^*, I_{r2}^*)$ *satisfying:*

$$\begin{cases} S_{h2}^* = \dfrac{\Lambda_h}{d_h} - \dfrac{(\rho + d_h)(d_h + \delta_h + \theta)}{\rho d_h} I_{h2}^*, \\ E_{h2}^* = \dfrac{(d_h + \delta_h + \theta)}{\rho} I_{h2}^*, \\ I_{h2}^* = \dfrac{-B + \sqrt{B^2 - 4AC}}{2A}, \\ R_{h2}^* = \dfrac{\theta}{d_h} I_{h2}^*, \\ S_{r2}^* = \dfrac{\Lambda_r}{d_r} - \dfrac{d_r(\tau + d_r)(\mathcal{R}_r - 1)}{\tau(\alpha + \beta d_r)}, \\ E_{r2}^* = \dfrac{d_r^2(\mathcal{R}_r - 1)}{\tau(\alpha + \beta d_r)}, \\ I_{r2}^* = \dfrac{d_r(\mathcal{R}_r - 1)}{\alpha + \beta d_r}. \end{cases}$$

Where,

$$A = (\rho + d_h)(d_h + \delta_h + \theta)\left[\alpha_h + \frac{\alpha_r \nu_h I_{r2}^*}{1 + \nu_r I_{r2}^*} + d_h \nu_h\right].$$

$$B = -\left[\alpha_h \rho \Lambda_h + \frac{\alpha_r \nu_h \rho I_{r2}^* \Lambda_h}{1 + \nu_r I_{r2}^*} - \frac{\alpha_r I_{r2}^* (\rho + d_h)(d_h + \delta_h + \theta)}{1 + \nu_r I_{r2}^*} - d_h(\rho + d_h)(d_h + \delta_h + \theta)\right].$$

$$C = -\frac{\alpha_r \rho I_{r2}^* \Lambda_h}{(1 + \nu_r I_{r2}^*)}.$$

4. Local stability

In this section, we investigate the local stability using the Routh-Hurwitz criterion [7]. This method consists of analyzing the eigenvalues of the Jacobian matrix of system (1.1) at each equilibrium in order to determine that all eigenvalues have negative real parts, which implies local asymptotic stability. This approach allows us to verify stability without explicitly computing the eigenvalues.

4.1. **Disease-free equilibrium.**

Theorem 4.1. *If $\mathcal{R}_0 < 1$, the disease-free equilibrium E_0 is locally asymptotically stable and it is unstable otherwise.*

Proof. The Jacobian matrix of system (1.1) at an arbitrary point is given by:

$$\mathcal{J} = \begin{pmatrix} -\left(\frac{\alpha_h I_h}{1+\nu_h I_h} + \frac{\alpha_r I_r}{1+\nu_r I_r}\right) - d_h & 0 & -\frac{\alpha_h S_h}{(1+\nu_h I_h)^2} & 0 & 0 & 0 & -\frac{\alpha_r S_h}{(1+\nu_r I_r)^2} \\ \left(\frac{\alpha_h I_h}{1+\nu_h I_h} + \frac{\alpha_r I_r}{1+\nu_r I_r}\right) & -(\rho + d_h) & \frac{\alpha_h S_h}{(1+\nu_h I_h)^2} & 0 & 0 & 0 & \frac{\alpha_r S_h}{(1+\nu_r I_r)^2} \\ 0 & \rho & -(d_h + \delta_h + \theta) & 0 & 0 & 0 & 0 \\ 0 & 0 & \theta & -d_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\frac{\alpha I_r}{1+\beta I_r} - d_r & 0 & -\frac{\alpha S_r}{(1+\beta I_r)^2} \\ 0 & 0 & 0 & 0 & \frac{\alpha I_r}{1+\beta I_r} & -(\tau + d_r) & \frac{\alpha S_r}{(1+\beta I_r)^2} \\ 0 & 0 & 0 & 0 & 0 & \tau & -d_r \end{pmatrix}$$

The corresponding Jacobian matrix at the disease-free equilibrium E_0 is given by:

$$\mathcal{J}_{E_0} = \begin{pmatrix} -d_h & 0 & -\frac{\alpha_h \Lambda_h}{d_h} & 0 & 0 & 0 & -\frac{\alpha_r \Lambda_h}{d_h} \\ 0 & -(\rho + d_h) & \frac{\alpha_h \Lambda_h}{d_h} & 0 & 0 & 0 & \frac{\alpha_r \Lambda_h}{d_h} \\ 0 & \rho & -(d_h + \delta_h + \theta) & 0 & 0 & 0 & 0 \\ 0 & 0 & \theta & -d_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -d_r & 0 & -\frac{\alpha \Lambda_r}{d_r} \\ 0 & 0 & 0 & 0 & 0 & -(\tau + d_r) & \frac{\alpha \Lambda_r}{d_r} \\ 0 & 0 & 0 & 0 & 0 & \tau & -d_r \end{pmatrix}$$

The characteristic equation is obtained by calculating $det\left(\mathcal{J}_{E_0} - \lambda I_d\right) = 0$.
The two obvious eigenvalues are $\lambda_1 = -d_h$ and $\lambda_2 = -d_r$, the other eigenvalues are the roots of the polynomials $P(\lambda) = \lambda^2 + a_1\lambda + a_2$ and $R(\lambda) = \lambda^2 + a_3\lambda + a_4$.
Where:

$$a_1 = (2d_r + \tau) \quad \text{and} \quad a_2 = d_r\left(1 - \mathcal{R}_r\right)\left(d_r + \tau\right).$$

$$a_3 = [(\rho + d_h) + (d_h + \delta_h + \theta)] \quad \text{and} \quad a_4 = \left(1 - \mathcal{R}_h\right)(\rho + d_h)\left(d_h + \delta_h + \theta\right).$$

In order to prove the local stability of E_0, we have to prove that the eigenvalues of the characteristic equation have negative real parts, in other words $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_4 > 0$.
We can remark that:

- $a_1 > 0$ and $a_2 > 0$ if and only if $\mathcal{R}_r < 1$.
- $a_3 > 0$ and $a_4 > 0$ if and only if $\mathcal{R}_h < 1$.

Consequently all eigenvalues of $\mathcal{J}_{E_0}$ have negative real parts hence, if $\mathcal{R}_h < 1$ and $\mathcal{R}_r < 1$ then E_0 is locally asymptotically stable.

□

4.2. **Human-only endemic equilibrium.**

Theorem 4.2. *If $\mathcal{R}_h > 1$ and $\mathcal{R}_r < 1$, then the human-only endemic equilibrium E_1^* is locally asymptotically stable.*

Proof. The Jacobian matrix of system (1.1) at the human-only endemic equilibrium E_1^* is given by:

$$\mathcal{J}_{E_1^*} = \begin{pmatrix} -\frac{\alpha_h I_{h1}^*}{1+\nu_h I_{h1}^*} - d_h & 0 & -\frac{\alpha_h S_{h1}^*}{\left(1+\nu_h I_{h1}^*\right)^2} & 0 & 0 & 0 & -\alpha_r S_{h1}^* \\ \frac{\alpha_h I_{h1}^*}{1+\nu_h I_{h1}^*} & -(\rho+d_h) & \frac{\alpha_h S_{h1}^*}{\left(1+\nu_h I_{h1}^*\right)^2} & 0 & 0 & 0 & \alpha_r S_{h1}^* \\ 0 & \rho & -(d_h+\delta_h+\theta) & 0 & 0 & 0 & 0 \\ 0 & 0 & \theta & -d_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -d_r & 0 & -\alpha\frac{\Lambda_r}{d_r} \\ 0 & 0 & 0 & 0 & 0 & -(\tau+d_r) & \alpha\frac{\Lambda_r}{d_r} \\ 0 & 0 & 0 & 0 & 0 & \tau & -d_r \end{pmatrix}$$

Let λ be an eigenvalue of $\mathcal{J}_{E_1^*}$, the characteristic equation is obtained by calculating $det\left(\mathcal{J}_{E_1^*} - \lambda I_d\right) = 0$.
We can remark that we have two obvious eigenvalues $\lambda_1 = -d_h$ and $\lambda_2 = -d_r$, the other eigenvalues are the roots of the polynomials $P(\lambda)$ and $R'(\lambda) = \lambda^3 + b_1\lambda^2 + b_2\lambda + b_3$.
Where:

- $b_1 = \frac{d_h\mathcal{R}_h}{1+\nu_h I_{h1}^*} + (\rho+d_h) + (d_h+\delta_h+\theta)$.
- $b_2 = \frac{d_h\mathcal{R}_h}{1+\nu_h I_{h1}^*}(\rho+d_h) + \frac{d_h\mathcal{R}_h(d_h+\delta_h+\theta)}{1+\nu_h I_{h1}^*} + (\rho+d_h)(d_h+\delta_h+\theta) - \rho\frac{\alpha_h S_{h1}^*}{\left(1+\nu_h I_{h1}^*\right)^2}$.
- $b_3 = \frac{d_h\mathcal{R}_h(\rho+d_h)(d_h+\delta_h+\theta)}{1+\nu_h I_{h1}^*} - \rho\frac{\alpha_h d_h S_{h1}^*}{\left(1+\nu_h I_{h1}^*\right)^2}$.

To prove the local stability of E_1^*, we should prove that $b_1 > 0$, $b_3 > 0$ and $b_1b_2 - b_3 > 0$.
It's obvious that $b_1 > 0$. From system (3.1) we have:

$$(\rho+d_h)(d_h+\delta_h+\theta) = \frac{\rho\alpha_h S_{h1}^*}{(1+\nu_h I_{h1}^*)}. \tag{4.1}$$

This proves that if $\mathcal{R}_h > 1$ then $b_3 > 0$.
Now we calculate $b_1b_2 - b_3$,

$$\begin{aligned} b_1b_2 - b_3 = &\left[\frac{d_h\mathcal{R}_h}{1+\nu_h I_{h1}^*} + (\rho+d_h) + (d_h+\delta_h+\theta)\right]\left[\frac{d_h\mathcal{R}_h}{1+\nu_h I_{h1}^*}(\rho+d_h) + \frac{d_h\mathcal{R}_h(d_h+\delta_h+\theta)}{1+\nu_h I_{h1}^*}\right] \\ &+ \left[\frac{d_h\mathcal{R}_h}{1+\nu_h I_{h1}^*} + (\rho+d_h) + (d_h+\delta_h+\theta)\right][(\rho+d_h)(d_h+\delta_h+\theta) \\ &-\rho\frac{\alpha_h S_{h1}^*}{\left(1+\nu_h I_{h1}^*\right)^2}\Bigg] - \frac{d_h\mathcal{R}_h(\rho+d_h)(d_h+\delta_h+\theta)}{1+\nu_h I_{h1}^*} + \rho\frac{\alpha_h d_h S_{h1}^*}{\left(1+\nu_h I_{h1}^*\right)^2}. \end{aligned}$$

Using equation (4.1) we can easily show that if $\mathcal{R}_h > 1$ then $b_1b_2 - b_3 > 0$, and we showed above that if $\mathcal{R}_r < 1$, the roots of $P(\lambda)$ have negative real parts, therefore by Routh-Hurwitz criterion if $\mathcal{R}_h > 1$ and $\mathcal{R}_r < 1$, all eigenvalues of $\mathcal{J}_{E_1^*}$ have negative real parts so E_1^* is locally asymptotically stable. □

4.3. **Endemic equilibrium.**

Theorem 4.3. *If $\mathcal{R}_0 > 1$ the endemic equilibrium E_2^* is locally asymptotically stable.*

Proof. The Jacobian matrix of system (1.1) at E_2^* is given by:

$$\mathcal{J}_{E_2^*} = \begin{pmatrix} -\left(\frac{\alpha_h I_{h2}^*}{1+\nu_h I_{h2}^*} + \frac{\alpha_r I_{r2}^*}{1+\nu_r I_{r2}^*}\right) - d_h & 0 & -\frac{\alpha_h S_{h2}^*}{\left(1+\nu_h I_{h2}^*\right)^2} & 0 & 0 & 0 & -\frac{\alpha_r S_{h2}^*}{\left(1+\nu_r I_{r2}^*\right)^2} \\ \left(\frac{\alpha_h I_{h2}^*}{1+\nu_h I_{h2}^*} + \frac{\alpha_r I_{r2}^*}{1+\nu_r I_{r2}^*}\right) & -(\rho+d_h) & \frac{\alpha_h S_{h2}^*}{\left(1+\nu_h I_{h2}^*\right)^2} & 0 & 0 & 0 & \frac{\alpha_r S_{h2}^*}{\left(1+\nu_{r2} I_{r2}^*\right)^2} \\ 0 & \rho & -(d_h+\delta_h+\theta) & 0 & 0 & 0 & 0 \\ 0 & 0 & \theta & -d_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\frac{\alpha I_{r2}^*}{1+\beta I_{r2}^*} - d_r & 0 & -\frac{\alpha S_{r2}^*}{\left(1+\beta I_{r2}^*\right)^2} \\ 0 & 0 & 0 & 0 & \frac{\alpha I_{r2}^*}{1+\beta I_{r2}^*} & -(\tau+d_r) & \frac{\alpha S_{r2}^*}{\left(1+\beta I_{r2}^*\right)^2} \\ 0 & 0 & 0 & 0 & 0 & \tau & -d_r \end{pmatrix}$$

Here, the characteristic equation is obtained by calculating $det\left(\mathcal{J}_{E_2^*} - \lambda I_d\right) = 0$. We have an obvious eigenvalue $\lambda_1 = -d_h$, the other eigenvalues are the roots of the polynomials $Q(\lambda) = \lambda^3 + c_1\lambda^2 + c_2\lambda + c_3$ and $Q'(\lambda) = \lambda^3 + c_4\lambda^2 + c_5\lambda + c_6$. Where:

- $c_1 = \frac{\alpha I_{r2}^*}{1+\beta I_{r2}^*} + 3d_r + \tau.$
- $c_2 = \left(\frac{\alpha I_{r2}^*}{1+\beta I_{r2}^*} + d_r\right)(d_r+\tau) + d_r(d_r+\tau) - \frac{\tau\alpha S_{r2}^*}{\left(1+\beta I_{r2}^*\right)^2}.$
- $c_3 = d_r(d_r+\tau)\left(\frac{\alpha I_{r2}^*}{1+\beta I_{r2}^*} + d_r\right) - \frac{\tau\alpha S_{r2}^*}{\left(1+\beta I_{r2}^*\right)^2}.$
- $c_4 = \left(\frac{\alpha_h I_{h2}^*}{1+\nu_h I_{h2}^*} + \frac{\alpha_r I_{r2}^*}{1+\nu_r I_{r2}^*} + d_h\right) + (\rho+d_h) + (d_h+\delta_h+\theta).$
- $c_5 = \left(\frac{\alpha_h I_{h2}^*}{1+\nu_h I_{h2}^*} + \frac{\alpha_r I_{r2}^*}{1+\nu_r I_{r2}^*} + d_h\right)(\rho+d_h) + \left(\frac{\alpha_h I_{h2}^*}{1+\nu_h I_{h2}^*} + \frac{\alpha_r I_{r2}^*}{1+\nu_r I_{r2}^*} + d_h\right)(d_h+\delta_h+\theta) + (\rho+d_h)(d_h+\delta_h+\theta) - \frac{\rho\alpha_h S_{h2}^*}{\left(1+\nu_h I_{h2}^*\right)^2}.$
- $c_6 = \left(\frac{\alpha_h I_{h2}^*}{1+\nu_h I_{h2}^*} + \frac{\alpha_r I_{r2}^*}{1+\nu_r I_{r2}^*} + d_h\right)(\rho+d_h)(d_h+\delta_h+\theta) - \frac{\rho\alpha_h d_h S_{h2}^*}{\left(1+\nu_h I_{h2}^*\right)^2}.$

To prove the local stability of E_2^*, we should prove that the coefficients $c_1,\ c_3,\ c_4$, and c_6 are positive and the same for $c_1c_2 - c_3$ and $c_4c_5 - c_6$.
From system (3.1) we have:

$$d_r(d_r+\tau) = \frac{\tau\alpha S_{r2}^*}{1+\beta I_{r2}^*}.$$

$$(\rho+d_h)(d_h+\delta_h+\theta) = \frac{\rho S_{h2}^*}{I_{h2}^*}\left(\frac{\alpha_h I_{h2}^*}{1+\nu_h I_{h2}^*} + \frac{\alpha_r I_{r2}^*}{1+\nu_r I_{r2}^*}\right).$$

Then we find that:

$$c_1 > 0, \quad c_3 > 0 \quad \text{and} \quad c_1c_2 - c_3 > 0.$$
$$c_4 > 0, \quad c_6 > 0 \quad \text{and} \quad c_4c_5 - c_6 > 0.$$

Hence, E_2^* is locally asymptotically stable if $\mathcal{R}_r > 1$. □

5. Global stability

In this section, we establish the global stability of the equilibrium points by constructing suitable Lyapunov functions and applying LaSalle's Invariance Principle. This method requires identifying a function that is positive definite and whose time derivative is non positive.
In each Lyapunov function we introduce some coefficients, to simplify terms so that helps the derivative of the function to be non positive.

5.1. **Disease-free equilibrium.**

Theorem 5.1. *If $\mathcal{R}_0 < 1$, the disease-free equilibrium E_0 is globally asymptotically stable.*

Proof. We consider the following Lyapunov function:
$V(X) = V(S_h, E_h, I_h, S_r, E_r, I_r) = \frac{d_h}{\alpha_r \Lambda_h} V_1 + \frac{1}{d_r(1-\mathcal{R}_r)} V_2$.
With,
$V_1 = S_h - \frac{\Lambda_h}{d_h} - \frac{\Lambda_h}{d_h} ln\left(\frac{d_h S_h}{\Lambda_h}\right) + E_h + \frac{(\rho+d_h)}{\rho} I_h$.
$V_2 = S_r - \frac{\Lambda_r}{d_r} - \frac{\Lambda_r}{d_r} ln\left(\frac{d_r S_r}{\Lambda_r}\right) + E_r + \frac{(\tau+d_r)}{\tau} I_r$.
We introduce the coefficients $\frac{d_h}{\alpha_r \Lambda_h}$ and $\frac{1}{d_r(1-\mathcal{R}_r)}$, to simplify terms and that helps the derivative of the function V to be non positive.
Since $lnx \leq x-1$ then $ln(\frac{d_h S_h}{\Lambda_h}) \leq \frac{d_h S_h}{\Lambda_h} - 1$ and $ln(\frac{d_r S_r}{\Lambda_r}) \leq \frac{d_r S_r}{\Lambda_r} - 1$ consequently $S_h - \frac{\Lambda_h}{d_h} - \frac{\Lambda_h}{d_h} ln(\frac{d_h S_h}{\Lambda_h}) \geq 0$ and $S_r - \frac{\Lambda_r}{d_r} - \frac{\Lambda_r}{d_r} ln(\frac{d_r S_r}{\Lambda_r}) \geq 0$.
We conclude that V is a positive-definite function.
Notice that:

$$
\begin{aligned}
\frac{dV_1}{dt} &= \frac{dS_h}{dt} - \frac{\Lambda_h}{d_h} \frac{\frac{dS_h}{dt}}{S_h} + \frac{dE_h}{dt} + \frac{(\rho+d_h)}{\rho} \frac{dI_h}{dt}. \\
&= \frac{dS_h}{dt}\left(1 - \frac{\Lambda_h}{d_h S_h}\right) + \frac{dE_h}{dt} + \frac{(\rho+d_h)}{\rho} \frac{dI_h}{dt}. \\
&= \left(1 - \frac{\Lambda_h}{d_h S_h}\right)\left(\Lambda_h - \left(\frac{\alpha_h I_h S_h}{1+\nu_h I_h} + \frac{\alpha_r I_r S_h}{1+\nu_r I_r}\right) - d_h S_h\right) + \left(\frac{\alpha_h I_h S_h}{1+\nu_h I_h}\right. \\
&\left. + \frac{\alpha_r I_r S_h}{1+\nu_r I_r}\right) - (\rho+d_h) E_h + (\rho+d_h) E_h - \frac{(\rho+d_h)(d_h+\delta_h+\theta)}{\rho} I_h. \\
&= -d_h S_h \left(1 - \frac{\Lambda_h}{d_h S_h}\right)^2 - \left(1 - \frac{\Lambda_h}{d_h S_h}\right)\left(\frac{\alpha_h I_h S_h}{1+\nu_h I_h} + \frac{\alpha_r I_r S_h}{1+\nu_r I_r}\right) \\
&+ \left(\frac{\alpha_h I_h S_h}{1+\nu_h I_h} + \frac{\alpha_r I_r S_h}{1+\nu_r I_r}\right) - \frac{(\rho+d_h)(d_h+\delta_h+\theta)}{\rho} I_h. \\
&\leq -d_h S_h \left(1 - \frac{\Lambda_h}{d_h S_h}\right)^2 + \frac{\alpha_h I_h \Lambda_h}{d_h} + \frac{\alpha_r I_r \Lambda_h}{d_h} - \frac{(\rho+d_h)(d_h+\delta_h+\theta)}{\rho} I_h. \\
&\leq -d_h S_h \left(1 - \frac{\Lambda_h}{d_h S_h}\right)^2 + \frac{(\mathcal{R}_h - 1)(\rho+d_h)(d_h+\delta_h+\theta)}{\rho} I_h + \frac{\alpha_r I_r \Lambda_h}{d_h}.
\end{aligned}
$$

And,

$$\begin{aligned}\frac{dV_2}{dt} &= \frac{dS_r}{dt} - \frac{\Lambda_r}{d_r}\frac{\frac{dS_r}{dt}}{S_r} + \frac{dE_r}{dt} + \frac{(\tau+d_r)}{\tau}\frac{dI_r}{dt}.\\ &= -d_r S_r\left(1-\frac{\Lambda_r}{d_r S_r}\right)^2 + \frac{\alpha I_r \Lambda_r}{d_r(1+\beta I_r)} - \frac{d_r(\tau+d_r)}{\tau}I_r.\\ &\leq -d_r S_r\left(1-\frac{\Lambda_r}{d_r S_r}\right)^2 + \frac{d_r(\mathcal{R}_r-1)(\tau+d_r)}{\tau}I_r.\end{aligned}$$

We conclude that if $\mathcal{R}_h < 1$ and $\mathcal{R}_r < 1$ then $\frac{dV}{dt} \leq 0$.
We can remark that $\frac{dV}{dt}(X) = 0$ if and only if $X = E_0$, then by applying LaSalle's invariance argument, we confirm that E_0 attracts all solutions in the domain, indicating global asymptotic stability.

□

5.2. **Human-only endemic equilibrium.**

Theorem 5.2. *If $\mathcal{R}_h > 1$ and $\mathcal{R}_r < 1$, then the human-only endemic equilibrium E_1^* is globally asymptotically stable.*

Proof. We consider the following Lyapunov function:
$\widehat{V}(X) = \widehat{V}(S_h, E_h, I_h, S_r, E_r, I_r) = \frac{1}{\alpha_r S_{h1}^*}\widehat{V}_1 + \frac{1}{d_r(1-\mathcal{R}_r)}\widehat{V}_2$.
With,

$$\begin{aligned}\widehat{V}_1 &= S_h - S_{h1}^* - S_{h1}^* ln\left(\frac{S_h}{S_{h1}^*}\right) + E_h - E_{h1}^* - E_{h1}^* ln\left(\frac{E_h}{E_{h1}^*}\right) + \frac{(\rho+d_h)}{\rho}\left(I_h - I_{h1}^*\right.\\ &\quad \left. - I_{h1}^* ln\left(\frac{I_h}{I_{h1}^*}\right)\right).\\ \widehat{V}_2 &= V_2.\end{aligned}$$

$\widehat{V}$ is a positive-definite function.
Notice that:

$$\begin{aligned}\frac{d\widehat{V}_1}{dt} &= \frac{dS_h}{dt}\left(1-\frac{S_{h1}^*}{S_h}\right) + \frac{dE_h}{dt}\left(1-\frac{E_{h1}^*}{E_h}\right) + \frac{(\rho+d_h)}{\rho}\frac{dI_h}{dt}\left(1-\frac{I_{h1}^*}{I_h}\right).\\ &= \left(1-\frac{S_{h1}^*}{S_h}\right)\left(\Lambda_h - \frac{\alpha_h I_h S_h}{1+\nu_h I_h} - \frac{\alpha_r I_r S_h}{1+\nu_r I_r} - d_h S_h\right) + \left(1-\frac{E_{h1}^*}{E_h}\right)\left(\frac{\alpha_h I_h S_h}{1+\nu_h I_h}\right.\\ &\quad \left. + \frac{\alpha_r I_r S_h}{1+\nu_r I_r} - (\rho+d_h)E_h\right) + \frac{(\rho+d_h)}{\rho}\left(1-\frac{I_{h1}^*}{I_h}\right)\left(\rho E_h - (d_h+\delta_h+\theta)I_h\right).\end{aligned}$$

From system (3.1) we have:

$$\begin{cases} \Lambda_h = \dfrac{\alpha_h I_h^* S_h^*}{1+\nu_h I_h^*} + \dfrac{\alpha_r I_r^* S_h^*}{1+\nu_r I_r^*} + d_h S_h^*, \\ (\rho + d_h)\, E_h^* = \dfrac{\alpha_h I_h^* S_h^*}{1+\nu_h I_h^*} + \dfrac{\alpha_r I_r^* S_h^*}{1+\nu_r I_r^*}, \\ \dfrac{(d_h+\delta_h+\theta)}{\rho} = \dfrac{E_h^*}{I_h^*}. \end{cases} \tag{5.1}$$

Then:

$$\begin{aligned} \frac{d\widehat{V}_1}{dt} &= -d_h S_h \left(1 - \frac{S_{h1}^*}{S_h}\right)^2 + 3\frac{\alpha_h I_{h1}^* S_{h1}^*}{1+\nu_h I_{h1}^*} - \frac{\alpha_h I_{h1}^* S_{h1}^{*2}}{S_h\,(1+\nu_h I_{h1}^*)} + \frac{\alpha_h I_h S_{h1}^*}{1+\nu_h I_h} + \frac{\alpha_r I_r S_{h1}^*}{1+\nu_r I_r} \\ &\quad - \frac{\alpha_h I_h S_h E_{h1}^*}{E_h\,(1+\nu_h I_h)} - \frac{\alpha_r I_r S_h E_{h1}^*}{E_h\,(1+\nu_r I_r)} - (\rho+d_h)\left(\frac{I_h E_{h1}^*}{I_{h1}^*} + \frac{I_{h1}^* E_h}{I_h}\right). \\ &= -d_h S_h \left(1 - \frac{S_{h1}^*}{S_h}\right)^2 + \frac{\alpha_h I_{h1}^* S_{h1}^*}{1+\nu_h I_{h1}^*}\left[4 - \frac{S_{h1}^*}{S_h} - \frac{I_h S_h E_{h1}^*}{E_h I_{h1}^* S_{h1}^*} \times \frac{1+\nu_h I_{h1}^*}{1+\nu_h I_h} - \frac{I_{h1}^* E_h}{I_h E_{h1}^*}\right. \\ &\quad \left. - \frac{1+\nu_h I_h}{1+\nu_h I_{h1}^*}\right] - \frac{\alpha_h I_{h1}^* S_{h1}^*}{1+\nu_h I_{h1}^*} + \frac{\alpha_h I_{h1}^* S_{h1}^*\,(1+\nu_h I_h)}{(1+\nu_h I_{h1}^*)^2} + \frac{\alpha_h I_h S_{h1}^*}{1+\nu_h I_h} - \frac{\alpha_h I_h S_{h1}^*}{1+\nu_h I_{h1}^*} \\ &\quad - \frac{\alpha_r I_r S_h E_{h1}^*}{E_h\,(1+\nu_r I_r)} + \frac{\alpha_r I_r S_{h1}^*}{1+\nu_r I_r}. \end{aligned}$$

The property of arithmetic geometric mean leads to:

$$\left[4 - \frac{S_{h1}^*}{S_h} - \frac{I_h S_h E_{h1}^*}{E_h I_{h1}^* S_{h1}^*} \times \frac{1+\nu_h I_{h1}^*}{1+\nu_h I_h} - \frac{I_{h1}^* E_h}{I_h E_{h1}^*} - \frac{1+\nu_h I_h}{1+\nu_h I_{h1}^*}\right] \leq 0.$$

Since

$$-\frac{\alpha_h I_{h1}^* S_{h1}^*}{1+\nu_h I_{h1}^*} + \frac{\alpha_h I_{h1}^* S_{h1}^*\,(1+\nu_h I_h)}{(1+\nu_h I_{h1}^*)^2} + \frac{\alpha_h I_h S_{h1}^*}{1+\nu_h I_h} - \frac{\alpha_h I_h S_{h1}^*}{1+\nu_h I_{h1}^*} \leq 0.$$

Therefore,

$$\frac{d\widehat{V}_1}{dt} \leq \alpha_r I_r S_{h1}^*.$$

It leads to,

$$\begin{aligned} \frac{d\widehat{V}}{dt}(S_h, E_h, I_h, S_r, E_r, I_r) &\leq I_r - \frac{\tau + d_r}{\tau} I_r. \\ &\leq 0. \end{aligned}$$

We can see that $\frac{d\widehat{V}}{dt}(X) = 0$ if and only if $X = E_1^*$ then by Lasalle's invariance principle, if $\mathcal{R}_h > 1$ and $\mathcal{R}_r < 1$ then E_1^* is globally asymptotically stable . $\square$

5.3. **Endemic equilibrium.**

Theorem 5.3. *If $\mathcal{R}_r > 1$ then the endemic equilibrium E_2^* is globally asymptotically stable.*

Proof. We consider the following Lyapunov function:
$\widetilde{V}(X) = \widetilde{V}(S_h, E_h, I_h, S_r, E_r, I_r) = \frac{d_r\psi}{\alpha_r S_{h2}^*(d_r+\Lambda_r)}\widetilde{V}_1 + \widetilde{V}_2$.
With ψ a positive constant and,
$\widetilde{V}_1 = S_h - S_{h2}^* - S_{h2}^* ln\left(\frac{S_h}{S_{h2}^*}\right) + E_h - E_{h2}^* - E_{h2}^* ln\left(\frac{E_h}{E_{h2}^*}\right) + \frac{(\rho+d_h)}{\rho}\left(I_h - I_{h2}^*\right.$
$\left. - I_{h2}^* ln\left(\frac{I_h}{I_{h2}^*}\right)\right)$.
$\widetilde{V}_2 = S_r - S_{r2}^* - S_{r2}^* ln\left(\frac{S_r}{S_{r2}^*}\right) + E_r - E_{r2}^* - E_{r2}^* ln\left(\frac{E_r}{E_{r2}^*}\right) + \frac{(\tau+d_r)}{\tau}\left(I_r - I_{r2}^* - I_{r2}^* ln\left(\frac{I_r}{I_{r2}^*}\right)\right)$.
$\widetilde{V}$ is a positive-definite function.
Notice that:

$$\begin{aligned}\frac{d\widetilde{V}_1}{dt} &= \frac{dS_h}{dt}\left(1 - \frac{S_{h2}^*}{S_h}\right) + \frac{dE_h}{dt}\left(1 - \frac{E_{h2}^*}{E_h}\right) + \frac{(\rho+d_h)}{\rho}\frac{dI_h}{dt}\left(1 - \frac{I_{h2}^*}{I_h}\right).\\ &= \left(1 - \frac{S_{h2}^*}{S_h}\right)\left(\Lambda_h - \frac{\alpha_h I_h S_h}{1+\nu_h I_h} - \frac{\alpha_r I_r S_h}{1+\nu_r I_r} - d_h S_h\right) + \left(1 - \frac{E_{h2}^*}{E_h}\right)\left(\frac{\alpha_h I_h S_h}{1+\nu_h I_h}\right.\\ &\left. + \frac{\alpha_r I_r S_h}{1+\nu_r I_r} - (\rho+d_h)E_h\right) + \frac{(\rho+d_h)}{\rho}\left(1 - \frac{I_{h2}^*}{I_h}\right)\left(\rho E_h - (d_h+\delta_h+\theta)I_h\right).\end{aligned}$$

From system (5.1) we have:

$$\begin{aligned}\frac{d\widetilde{V}_1}{dt} &= -d_h S_h\left(1 - \frac{S_{h2}^*}{S_h}\right)^2 + 3\frac{\alpha_h I_{h2}^* S_{h2}^*}{1+\nu_h I_{h2}^*} + 3\frac{\alpha_r I_{r2}^* S_{h2}^*}{1+\nu_r I_{r2}^*} - \frac{\alpha_h I_{h2}^* S_{h2}^{*2}}{S_h\left(1+\nu_h I_{h2}^*\right)}\\ &- \frac{\alpha_h I_{r2}^* S_{h2}^{*2}}{S_h\left(1+\nu_h I_{h2}^*\right)} + \frac{\alpha_h I_h S_{h2}^*}{1+\nu_h I_h} + \frac{\alpha_r I_r S_{h2}^*}{1+\nu_r I_r} - \frac{\alpha_h I_h S_h E_{h2}^*}{E_h\left(1+\nu_h I_h\right)} - \frac{\alpha_r I_r S_h E_{h2}^*}{E_h\left(1+\nu_r I_r\right)}\\ &- (\rho+d_h)\left(\frac{I_h E_{h2}^*}{I_{h2}^*} + \frac{I_{h2}^* E_h}{I_h}\right).\\ &= -d_h S_h\left(1 - \frac{S_{h2}^*}{S_h}\right)^2 + \frac{\alpha_h I_{h2}^* S_{h2}^*}{1+\nu_h I_{h2}^*}\left[4 - \frac{S_{h2}^*}{S_h} - \frac{I_h S_h E_{h2}^*}{E_h I_{h2}^* S_{h2}^*} \times \frac{1+\nu_h I_{h2}^*}{1+\nu_h I_h}\right.\\ &\left. - \frac{I_{h2}^* E_h}{I_h E_{h2}^*} - \frac{1+\nu_h I_h}{1+\nu_h I_{h2}^*}\right] - \frac{\alpha_h I_{h2}^* S_{h2}^*}{1+\nu_h I_{h2}^*} + \frac{\alpha_h I_{h2}^* S_{h2}^*\left(1+\nu_h I_h\right)}{\left(1+\nu_h I_{h2}^*\right)^2} + \frac{\alpha_h I_h S_{h2}^*}{1+\nu_h I_h}\\ &- \frac{\alpha_h I_h S_{h2}^*}{1+\nu_h I_{h2}^*} - \frac{\alpha_r I_r S_h E_{h2}^*}{E_h\left(1+\nu_r I_r\right)} + \frac{\alpha_r I_r S_{h2}^*}{1+\nu_r I_r} + \frac{\alpha_r I_{r2}^* S_{h2}^*}{1+\nu_r I_{r2}^*}\left[5 - \frac{S_{h2}^*}{S_h} - \frac{I_h}{I_{h2}^*}\right.\\ &\left. - \frac{I_r S_h E_{h2}^*}{E_h I_{r2}^* S_{h2}^*} \times \frac{1+\nu_r I_{r2}^*}{1+\nu_r I_r} - \frac{I_{h2}^* E_h}{I_h E_{h2}^*} - \frac{I_{r2}^*\left(1+\nu_r I_r\right)}{I_r\left(1+\nu_r I_{r2}^*\right)}\right] - 2\frac{\alpha_r I_{r2}^* S_{h2}^*}{1+\nu_r I_{r2}^*}\\ &+ \frac{\alpha_r I_{r2}^{*2} S_{h2}^*\left(1+\nu_r I_r\right)}{I_r\left(1+\nu_r I_{r2}^*\right)^2}.\end{aligned}$$

The property of arithmetic geometric mean leads to:

$$\left[4-\frac{S_{h2}^*}{S_h}-\frac{I_hS_hE_{h2}^*}{E_hI_{h2}^*S_{h2}^*}\times\frac{1+\nu_hI_{h2}^*}{1+\nu_hI_h}-\frac{I_{h2}^*E_h}{I_hE_{h2}^*}-\frac{1+\nu_hI_h}{1+\nu_hI_{h2}^*}\right]\leq 0,$$

and

$$\left[5-\frac{S_{h2}^*}{S_h}-\frac{I_h}{I_{h2}^*}-\frac{I_rS_hE_{h2}^*}{E_hI_{r2}^*S_{h2}^*}\times\frac{1+\nu_rI_{r2}^*}{1+\nu_rI_r}-\frac{I_{h2}^*E_h}{I_hE_{h2}^*}-\frac{I_{r2}^*\left(1+\nu_rI_r\right)}{I_r\left(1+\nu_rI_{r2}^*\right)}\right]\leq 0.$$

Then

$$\begin{aligned}\frac{d\widetilde{V}_1}{dt}&\leq\frac{\alpha_rI_rS_{h2}^*}{1+\nu_rI_r}+\frac{\alpha_rI_{r2}^{*^2}S_{h2}^*}{I_r\left(1+\nu_rI_{r2}^*\right)^2}.\\&\leq\frac{\alpha_rS_{h2}^*\left(\Lambda_r+d_r\right)}{d_r}.\end{aligned}$$

We have:

$$\begin{aligned}\frac{d\widetilde{V}_2}{dt}&=\frac{dS_r}{dt}\left(1-\frac{S_{r2}^*}{S_r}\right)+\frac{dE_r}{dt}\left(1-\frac{E_{r2}^*}{E_r}\right)+\frac{\left(\tau+d_r\right)}{\tau}\frac{dI_r}{dt}\left(1-\frac{I_{r2}^*}{I_r}\right).\\&=\left(1-\frac{S_{r2}^*}{S_r}\right)\left(\Lambda_r-\frac{\alpha I_rS_r}{1+\beta I_r}-d_rS_r\right)+\left(1-\frac{E_{r2}^*}{E_r}\right)\left(\frac{\alpha_rI_rS_r}{1+\beta I_r}\right.\\&\left.-\left(\tau+d_r\right)E_r\right)+\frac{\left(\tau+d_r\right)}{\tau}\left(1-\frac{I_{r2}^*}{I_r}\right)\left(\tau E_r-d_rI_r\right).\end{aligned}$$

From system (3.1) we have:

$$\begin{cases}\Lambda_r=\dfrac{\alpha I_r^*S_r^*}{1+\beta I_r^*}+d_rS_r^*,\\\left(d_r+\tau\right)E_r^*=\dfrac{\alpha I_r^*S_r^*}{1+\beta I_r^*},\\\dfrac{d_r}{\tau}=\dfrac{E_r^*}{I_r^*}.\end{cases}\tag{5.2}$$

Then:

$$\begin{aligned}\frac{d\widetilde{V}_2}{dt}&=-d_rS_r\left(1-\frac{S_{r2}^*}{S_r}\right)^2+3\frac{\alpha I_{r2}^*S_{r2}^*}{1+\beta I_{r2}^*}-\frac{\alpha I_{r2}^*S_{r2}^{*^2}}{S_r\left(1+\beta I_{r2}^*\right)}+\frac{\alpha I_rS_{r2}^*}{1+\beta I_r}-\frac{\alpha I_rS_rE_{r2}^*}{E_r\left(1+\beta I_r\right)}\\&-\left(\tau+d_r\right)\left(\frac{I_rE_{r2}^*}{I_{r2}^*}+\frac{I_{r2}^*E_r}{I_r}\right).\\&=-d_rS_r\left(1-\frac{S_{r2}^*}{S_r}\right)^2+\frac{\alpha I_{r2}^*S_{r2}^*}{1+\beta I_{r2}^*}\left[4-\frac{S_{r2}^*}{S_r}-\frac{I_rS_rE_{r2}^*}{E_rI_{r2}^*S_{r2}^*}\times\frac{1+\beta I_{r2}^*}{1+\beta I_h}-\frac{I_{r2}^*E_r}{I_rE_{r2}^*}\right.\\&\left.-\frac{1+\beta I_r}{1+\beta I_{r2}^*}\right]-\frac{\alpha I_{r2}^*S_{r2}^*}{1+\beta I_{r2}^*}+\frac{\alpha I_{r2}^*S_{r2}^*\left(1+\beta I_h\right)}{\left(1+\beta I_{r2}^*\right)^2}-\frac{\alpha I_rS_{r2}^*}{1+\beta I_{r2}^*}+\frac{\alpha I_rS_{r2}^*}{1+\beta I_r}.\end{aligned}$$

The property of arithmetic geometric mean leads to:

$$\left[4-\frac{S_{r2}^*}{S_r}-\frac{I_rS_rE_{r2}^*}{E_rI_{r2}^*S_{r2}^*}\times\frac{1+\beta I_{r2}^*}{1+\beta I_r}-\frac{I_{r2}^*E_r}{I_rE_{r2}^*}-\frac{1+\beta I_r}{1+\beta I_{r2}^*}\right]\leq 0.$$

As,

$$-\frac{\alpha I_{r2}^* S_{r2}^*}{1+\beta I_{r2}^*}+\frac{\alpha I_{r2}^* S_{r2}^*\left(1+\beta I_r\right)}{\left(1+\beta I_{r2}^*\right)^2}+\frac{\alpha I_r S_{r2}^*}{1+\beta I_r}-\frac{\alpha I_r S_{r2}^*}{1+\beta I_{r2}^*} \leq 0.$$

Then,

$$\frac{d\widetilde{V}_2}{dt} \leq 0.$$

Consequently,

$$\frac{d\widetilde{V}}{dt}(S_h, E_h, I_h, S_r, E_r, I_r) \leq \psi + \widetilde{V}_2.$$

Let $0 < \psi < \left|\widetilde{V}_2\right|$.
Then,

$$\widetilde{V} \leq 0.$$

Since $\frac{d\widetilde{V}}{dt}(X) = 0$ if and only if $X = E_2^*$ then by Lasalle's invariance principle, if $\mathcal{R}_r > 1$, E_2^* is globally asymptotically stable . □

6. Numerical simulation

To assert the theoretical results obtained in the previous sections, we execute numerical simulations using the parameter values listed in Table 1. These simulations aim to illustrate the system's dynamics and to affirm the stability properties predicted by the analytical analysis.

Parameters	Figure 2	Figure 3	Figure 4	Figure 5	Source
Λ_h	0.8	0.3	0.3	0.8	Assumed
α_h	0.00006	0.06	0.06	0.00006	[13]
α_r	0.09	0.09	0.09	0.09	[11]
ν_h	0.05	0.05	0.05	0.05	Assumed
ν_r	0.03	0.03	0.03	0.03	Assumed
d_h	0.01794	0.01794	0.01794	0.01794	[13]
δ_h	0.0247	0.0247	0.0247	0.0247	[13]
ρ	0.14	0.14	0.14	0.14	[11]
θ	0.01	0.01	0.01	0.01	[11]
Λ_r	0.2	0.2	0.2	0.2	Assumed
α	0.027	0.027	0.027	0.027	[11]
d_r	0.03	0.03	0.03	0.03	[11]
τ	0.002	0.002	0.3	0.3	Assumed/ [13]
β	0.2	0.2	0.2	0.2	Assumed

Table 1. Table of parameters.

6.1. **Disease-free equilibium.** In this subsection, we present the numerical simulation setup corresponding to the human-only equilibrium E_0, based on the model equations and the second column of Table 1.

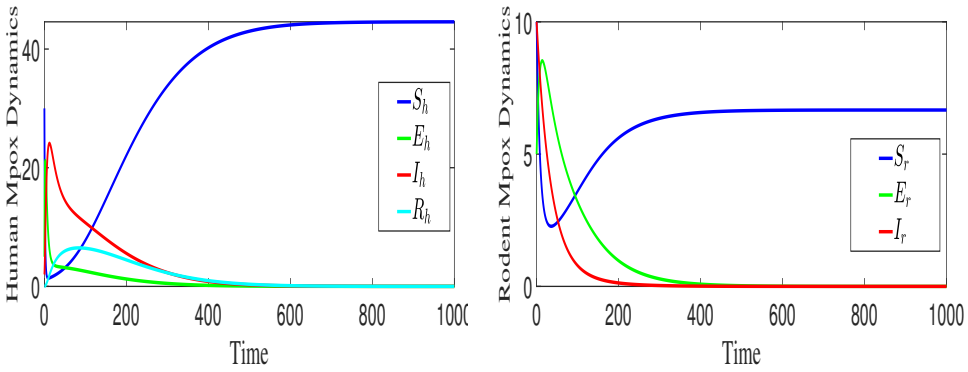

FIGURE 2. The disease-free equilibrium stability: The human monkeypox dynamics (left) and the rodent dynamics (right) (Case: $\mathcal{R}_h < 1$ and $\mathcal{R}_r < 1$).

We have the $\mathcal{R}_h = 0.0466 < 1$ and $\mathcal{R}_r = 0.3750 < 1$, which implies the stability of the disease-free equilibrium. Indeed, from Fig. 2, we can see that the exposed populations and infected populations for both rodent and human vanish. However, the susceptible populations tend to their maximal levels for both human and rodent. The convergence towards the disease-free equilibrium $E_0 = (44.5931, 0, 0, 0, 6.6667, 0, 0)$ is observed, which is consistent with the theoretical predictions, demonstrated in Theorem 4.1 concerning the local stability result and Theorem 5.1 concerning the global stability result.

6.2. **Human-only endemic equilibrium.** In this subsection, we present the numerical simulation setup corresponding to the human-only equilibrium E_1^*, based on the model equations and the third column of Table 1.

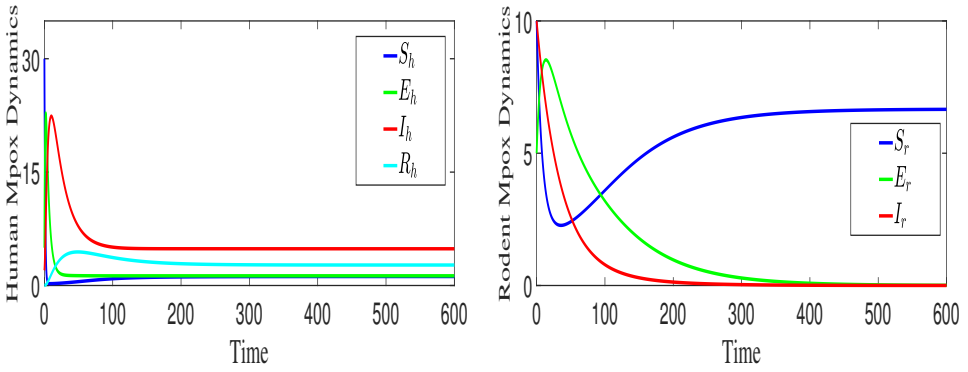

FIGURE 3. Human-only endemic Equilibrium stability: The human monkeypox dynamics (left) and the rodent dynamics (right) (Case: $\mathcal{R}_h > 1$ and $\mathcal{R}_r < 1$).

We have the human reproduction number $\mathcal{R}_h = 17.4915 > 1$ and $\mathcal{R}_r = 0.3750 < 1$, which predicts the stability of the human-only endemic equilibrium. Indeed, from Fig. 3, we can see that the number of infected rodent population tend to zero, similarly the number of exposed rodent population vanish, while the number of susceptible rodent tends to the maximal level. However, the infected human tend to the maximal level and the susceptible tends to zero. The convergence towards the human-only endemic equilibrium $E_1^* = (1.1883, 1.2787, 4.8583, 2.7081, 6.6667, 0, 0)$ is observed which is in agreement with the theoretical findings, demonstrated in Theorem 4.2 concerning the local stability result and Theorem 5.2 concerning the global stability result.

6.3. **Endemic equilibrium.** In this subsection, we present the numerical simulation setup corresponding to the human-only equilibrium E_2^*, based on the model equations and the fourth and fifth column of Table 1.

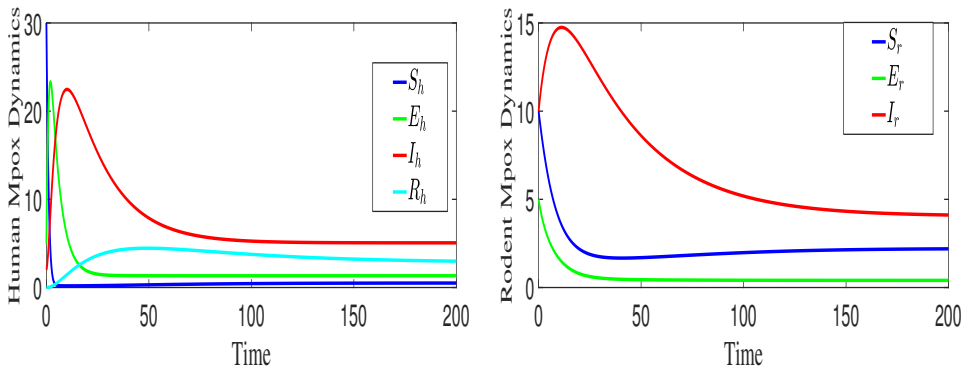

FIGURE 4. Disease endemic equilibrium stability: The human monkeypox dynamics (left) and the rodent dynamics (right) (Case: $\mathcal{R}_h > 1$ and $\mathcal{R}_r > 1$).

According to the fourth column of Table 1, we have the human reproduction number $\mathcal{R}_h = 17.4915 > 1$ and $\mathcal{R}_r = 5.4545 > 1$, which predicts the stability of the endemic equilibrium. Indeed, from Fig. 4, all the model components remain at a strictly positive level, and we can see that the susceptible populations for both rodent and human tend to zero. However, the infected populations tend to their maximal levels for both human and rodent. The convergence towards the endemic equilibrium $E_2^* = (0.5123, 1.3344, 5.0697, 2.8259, 2.2121, 0.4050, 4.0496)$ is observed which is in agreement with the theoretical findings, demonstrated in Theorem 4.3 concerning the local stability result and Theorem 5.3 concerning the global stability result.

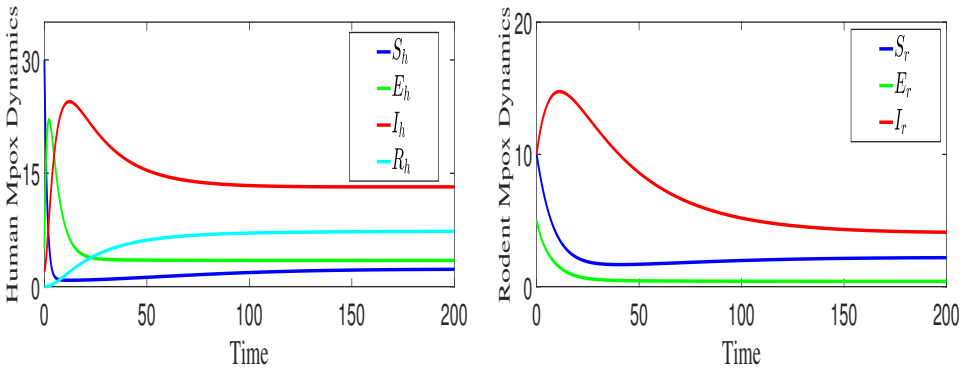

FIGURE 5. Disease endemic equilibrium stability: The human monkeypox dynamics (left) and the rodent dynamics (right) (Case: $\mathcal{R}_h < 1$ and $\mathcal{R}_r > 1$).

According to the fifth column of Table 1, we have the human reproduction number $\mathcal{R}_h = 0.0466 < 1$ and $\mathcal{R}_r = 5.4545 > 1$, which predicts the stability of the endemic equilibrium. Indeed, from Fig. 5, all the model components remain at a strictly positive level, and we can see that the susceptible populations for both rodent and human vanish. However, the infected populations tend to their maximal levels for both human and rodent. The convergence towards the endemic equilibrium $E_2^* = (2.3297, 3.4790, 13.218, 7.3679, 2.2121, 0.4050, 4.0496)$ is observed which is in agreement with the theoretical findings, demonstrated in Theorem 4.3 concerning the local stability result and Theorem 5.3 concerning the global stability result.
It can be seen from figures 4 and 5 that if $\mathcal{R}_r > 1$ the endemic equilibrium point E_2^* is asymptotically stable and even if $\mathcal{R}_h < 1$ the disease continue to spread in human population which means that $\mathcal{R}_r > 1$ has an impact not only on rodents population but also in humans population.

7. CONCLUSION

In this paper, we have studied a two-group monkeypox model including a saturated incidence rate. We investigated the basic reproduction number $\mathcal{R}_0$, which is the maximum of the human reproduction number and the rodent reproduction number, denoted as $\mathcal{R}_h$ and $\mathcal{R}_r$, respectively. We calculated the equilibrium points and established their stability. We found that if $\mathcal{R}_0 < 1$, i.e, $\mathcal{R}_h < 1$ and $\mathcal{R}_r < 1$ there exists a unique equilibrium point, which is the disease-free equilibrium E_0 and it is globally asymptotically stable; if $\mathcal{R}_h > 1$ and $\mathcal{R}_r < 1$, there exist two equilibrium points, the disease-free equilibrium E_0 which is unstable and the human-only endemic equilibrium E_1^* which is globally asymptotically stable and if $\mathcal{R}_r > 1$ the disease-free equilibrium E_0 is unstable and there exists an endemic equilibrium E_2^* which is globally asymptotically stable. The results of this study show that the transmission of monkeypox in the human population is affected by the human infection rate and the rodent infection rate, i.e, the disease in the

population of rodents has an impact on the behavior of the disease in the human population, while the transmission of monkeypox in the population of rodents is only affected by the rodent-rodent infection rate because there is transmission of the disease from rodents to humans but there is no transmission of the disease from humans to rodents. We showed that the existence and stability of the endemic equilibrium depend just on $\mathcal{R}_r$ and presented numerical simulations which support our theoretical results.

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