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Research Article

Global dynamics of a fractional-order Hantavirus model with awareness-driven protective behavior

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Abstract

In this paper, a new fractional-order model of Hantavirus transmission between humans and rodents is proposed, incorporating the effect of media awareness. The model is formulated with the Caputo fractional derivative, which captures the memory effects inherent in epidemic dynamics. The existence, uniqueness, positivity, and boundedness of the solutions are established. The basic reproduction numbers of humans and rodents are derived using the next-generation matrix approach, and the disease-free and endemic equilibrium points are computed. Their global asymptotic stability is established using Lyapunov theory and LaSalle's invariance principle for fractional-order systems. The sensitivity analysis shows that the transmission and recruitment rates have positive effects on the basic reproduction numbers, whereas the recovery and natural mortality rates have negative effects. The numerical results support the theoretical findings on the global stability of the equilibria and show that increasing the effectiveness of media awareness reduces the infection burden. Moreover, the simulations indicate that decreasing the fractional-order parameter leads to faster convergence toward the equilibrium state. However, variations in the fractional-order parameter do not affect the global stability of the system, while increased media awareness contributes to a reduction in Hantavirus infections.

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1 Introduction

Hantavirus infection is an emerging zoonotic disease primarily associated with rodent reservoirs and poses a serious public health threat in many parts of the world [17]. Hantavirus is maintained in nature by small mammal reservoir hosts and is transmitted to humans primarily through inhalation of aerosolized rodent urine, saliva, and feces [20]. The transmission dynamics of Hantavirus are influenced by the ecology and distribution of reservoir rodents, as well as by human exposure and preventive behaviors, making disease control challenging [5]. Although much has been learned about the epidemiology and pathogenesis of Hantaviruses, no specific treatment or vaccine is yet available [9]. For this reason, reducing human exposure to infected rodents remains the main means of preventing the disease. Thus, a better understanding of the dynamics of Hantavirus transmission will help public health officials enhance their surveillance systems and develop better prevention and control strategies.

Mathematical modeling has become an essential tool for understanding the dynamics of disease transmission and for supporting public health policy [4, 18]. Mathematical epidemic models integrate biological and epidemiological information to analyze infectious disease transmission. Understanding how infectious diseases spread can help predict disease outbreaks and assess the effectiveness of preventive measures [19]. Over the years, a variety of compartmental models have been developed to study Hantavirus epidemiology in humans and rodents. Such work has improved our understanding of host-reservoir interactions, transmission processes, and the effectiveness of different control strategies on disease persistence [21, 24, 27]. Nevertheless, the complex nature of Hantavirus transmission highlights the need for more refined mathematical models that integrate additional biological mechanisms and behavioral factors. Several mathematical models have been proposed to analyze the transmission dynamics of Hantavirus under different epidemiological assumptions. Early studies used deterministic compartmental models to study the relationship between human and rodent populations and to analyze the impact of demographic and epidemiological factors on disease transmission [16, 26]. Building on this approach, subsequent studies incorporated additional mechanisms such as nonlinear incidence rates, ecological transmission, seasonal patterns, spatial heterogeneity, and stochastic effects [29, 3, 22]. Despite these advances, most current models are still based on classical integer-order differential equations. Such models assume that the future evolution of the epidemic depends only on its current state. Although this assumption is reasonable, it may not fully capture the

memory and hereditary effects present in most biological and epidemiological systems. Davarian et al. [10] investigated a multigroup COVID-19 epidemic model in Iran and analyzed the reproduction number and the stability of its equilibrium points. They also considered optimal control strategies aimed at reducing symptomatic and asymptomatic infections.

To overcome the limitations of traditional integer-order models, fractional-order differential equations have attracted increasing attention in epidemiological modeling over the last two decades. Unlike ordinary derivatives, fractional derivatives are nonlocal and can capture the memory and hereditary effects characteristic of biological and epidemiological systems [15]. This property enables fractional-order models to describe infectious disease transmission more realistically, since the current state of an epidemic depends on its past states and on previous interactions [6]. Accordingly, fractional-order epidemic models have been used to study diseases such as COVID-19, influenza, dengue, tuberculosis, and hepatitis. Several studies have shown that these models can describe epidemiological data and make predictions more accurately than classical integer-order models [1, 7, 31, 2, 8, 33].

Compared with the extensive literature on classical models of Hantavirus transmission, studies based on fractional calculus remain scarce. Nevertheless, the ability of fractional calculus to account for memory and hereditary effects may provide a more realistic picture of Hantavirus dynamics. In this context, Rida et al. [25] developed a fractional-order sensitivity index (SI) model of Hantavirus infection in mice to investigate the effects of environmental conditions and habitat variability on disease dynamics. Using the generalized Euler method, they obtained numerical solutions and showed that changes in environmental conditions, represented by the carrying capacity, can substantially influence the persistence and dynamics of the infected mouse population. Yakit Ongun and Arslan [34] developed explicit and implicit numerical schemes for solving fractional-order Hantavirus models and examined the qualitative properties of their solutions. Moustafa et al. [23] considered a fractional-order Hantavirus infection model with logistic growth, describing the transmission dynamics in a population of susceptible and infected mice. They established the existence, uniqueness, nonnegativity, and boundedness of the solutions. Furthermore, they investigated the local and global asymptotic stability of the equilibrium points and derived the basic reproduction number. The effects of the basic reproduction number and the carrying capacity on the stability of the fractional-order system were also examined through theoretical and numerical analyses. Although these studies demonstrated the advantages of fractional-order models over their integer-order counterparts, they mainly focus on the mathematical and numerical aspects of disease dynamics. In particular, the influence of media-driven public awareness, which can induce protective behavioral responses and thereby reduce human exposure, has not yet been investigated within a fractional-order framework for Hantavirus modeling. The aim of the present study is to bridge this gap by developing a fractional-order model

that incorporates media awareness, providing a more realistic description of Hantavirus transmission.

In light of the above observations, this study presents a new fractional-order Hantavirus transmission model that includes the role of media awareness in influencing disease transmission. The proposed model is formulated using the Caputo fractional derivative to incorporate the memory characteristics of the transmission process. We first prove the positivity, existence, uniqueness, and boundedness of the solutions of the model. Then, we identify the equilibrium points, derive the basic reproduction number, and study the global stability of the equilibria. Afterward, numerical simulations are performed to verify the theoretical results and to investigate the effects of the human transmission rate, the rodent transmission rate, media awareness, and the fractional-order parameter on the spread of Hantavirus. A sensitivity analysis is also conducted to determine the parameters that most influence the disease transmission dynamics. The remainder of this paper is organized as follows: Section 2 presents the formulation of the fractional-order Hantavirus model. Section 3 provides proofs of the positivity, existence, uniqueness, and boundedness of the model solutions. Section 4 discusses the existence of the equilibria and the computation of the basic reproduction number, and derives global stability results for the equilibria. Section 5 presents the numerical simulation results. It also discusses how the transmission parameters, media awareness, and the fractional-order parameter affect the dynamics of the model. Section 6 is devoted to the sensitivity analysis. Finally, Section 7 concludes the paper with a brief summary and future directions.

2 Model formulation

Motivated by the need for a better understanding of the role of public awareness in Hantavirus transmission, we propose a new fractional-order epidemic model in the Caputo sense. The model incorporates the role of media through a compartment of protected humans and assumes that greater public awareness reduces the effectiveness of disease transmission, since the public is encouraged to adopt preventive measures. The total human population is divided into four compartments: Susceptible S_h, protected P_h, infectious I_h, and recovered R_h, such that

$$N_h(t) = S_h(t) + P_h(t) + I_h(t) + R_h(t). \tag{1}$$

The rodent population consists of susceptible S_r and infected I_r rodents, satisfying

$$N_r(t) = S_r(t) + I_r(t). \tag{2}$$

Humans acquire infection through effective contact with infected rodents or infectious humans. The parameter η represents the relative contribution of human-to-human transmission. The level of media awareness, denoted by $M(t)$, is generated by infectious individuals. As awareness increases, susceptible individuals adopt precautions and move to the protected class at the rate $\tau M(t)$. Moreover, media awareness further reduces the force of infection through the factor $\frac{1}{1+\kappa M}$. Protected individuals gradually lose their protection and return to the susceptible class at the rate ξ, while recovered individuals lose their temporary immunity at the rate ω. Under these assumptions, the dynamics of Hantavirus transmission are governed by the following system of Caputo fractional differential equations:

$$\begin{cases} D^{\alpha}S_h = \Lambda_h + \omega R_h - \tau M S_h - \dfrac{\beta_h S_h(I_r + \eta I_h)}{1+\kappa M} - \mu_h S_h + \xi P_h, \\ D^{\alpha}P_h = \tau M S_h - (\xi + \mu_h)P_h, \\ D^{\alpha}I_h = \dfrac{\beta_h S_h(I_r + \eta I_h)}{1+\kappa M} - (\gamma_h + \mu_h)I_h, \\ D^{\alpha}R_h = \gamma_h I_h - (\omega + \mu_h)R_h, \\ D^{\alpha}S_r = \Lambda_r - \beta_r S_r I_r - \mu_r S_r, \\ D^{\alpha}I_r = \beta_r S_r I_r - \mu_r I_r, \\ D^{\alpha}M = aI_h - bM. \end{cases} \tag{3}$$

The corresponding initial conditions are $S_h(0) > 0$, $P_h(0) \geq 0$, $I_h(0) \geq 0$, $R_h(0) \geq 0$, $S_r(0) > 0$, $I_r(0) \geq 0$, and $M(0) \geq 0$. The Caputo fractional derivative of order $\alpha \in (0, 1]$ is defined by

$$D^{\alpha}f(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t \frac{f'(s)}{(t-s)^{\alpha}}\, ds,$$

where $\Gamma(\cdot)$ denotes the Gamma function. The biological meanings of the state variables and model parameters are summarized in Tables 1 and 2. The schematic diagram of the proposed model is shown in Figure 1.

Table 1: Description of the state variables.

Variable	**Description**
$S_h(t)$	Susceptible human population.
$P_h(t)$	Protected human population due to media awareness.
$I_h(t)$	Infectious human population.
$R_h(t)$	Recovered human population with temporary immunity.
$S_r(t)$	Susceptible rodent population.
$I_r(t)$	Infected rodent population.
$M(t)$	Media awareness level.

Table 2: Description of the model parameters.

Parameter	**Description**	**Unit**
Λ_h	Recruitment rate of the human population.	Individuals/day
Λ_r	Recruitment rate of the rodent population.	Rodents/day
μ_h	Natural death rate of humans.	Day^{-1}
μ_r	Natural death rate of rodents.	Day^{-1}
β_h	Effective transmission rate from infected rodents and infectious humans to susceptible humans.	Day^{-1}
β_r	Transmission rate among rodents.	Day^{-1}
η	Modification parameter representing the relative contribution of human-to-human transmission.	–
τ	Protection rate induced by media awareness.	Day^{-1}
κ	Media effectiveness parameter reducing disease transmission.	–
ξ	Rate at which protected individuals return to the susceptible class.	Day^{-1}
γ_h	Recovery rate of infectious humans.	Day^{-1}
ω	Loss of immunity rate of recovered humans.	Day^{-1}
a	Media awareness generation rate.	Day^{-1}
b	Natural decay rate of media awareness.	Day^{-1}
α	Fractional-order parameter, $0 < \alpha \leq 1$.	–

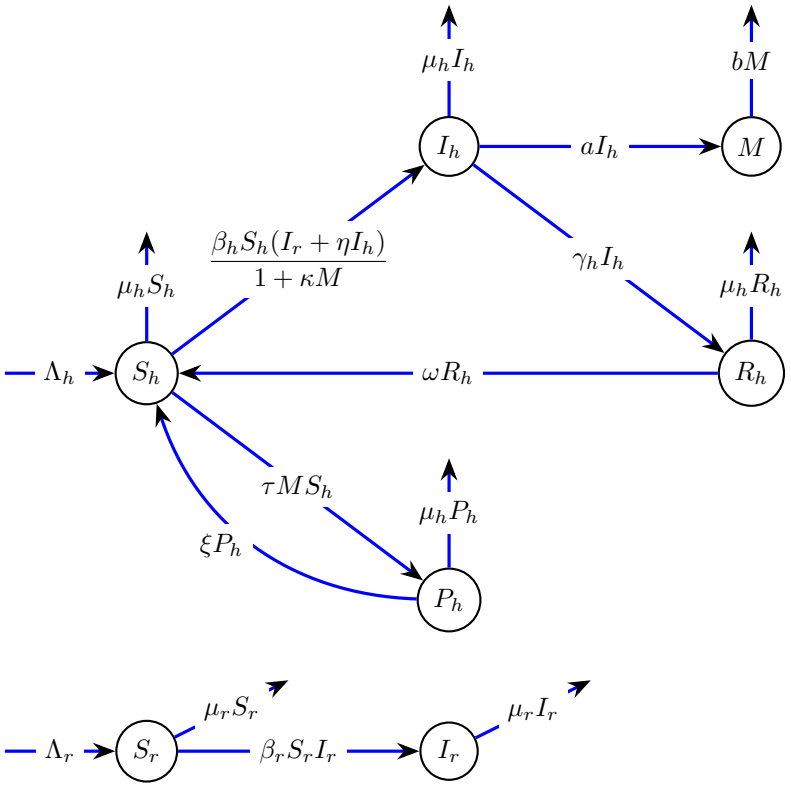


Figure 1: Flow diagram of the fractional-order Hantavirus transmission model with awareness dynamics.

3 Mathematical well-posedness of the model

In this section, we verify the well-posedness of system (3). Specifically, we prove the existence and uniqueness of a positive solution of the fractional-order model and show that the state variables remain bounded for all $t > 0$.

Proposition 1. For any positive initial conditions, the variables of system (3), namely $S_h(t)$, $P_h(t)$, $I_h(t)$, $R_h(t)$, $S_r(t)$, $I_r(t)$, and $M(t)$, remain positive for all $t > 0$.

Proof. From the different components of system (3), we have

$$\begin{cases} D^{\alpha} S_h \mid_{S_h=0} = \Lambda_h + \omega R_h + \xi P_h \geq 0, \\ D^{\alpha} P_h \mid_{P_h=0} = \tau M S_h \geq 0, \\ D^{\alpha} I_h \mid_{I_h=0} \geq 0, \\ D^{\alpha} R_h \mid_{R_h=0} = \gamma_h I_h \geq 0, \\ D^{\alpha} S_r \mid_{S_r=0} = \Lambda_r \geq 0, \\ D^{\alpha} I_r \mid_{I_r=0} = 0, \\ D^{\alpha} M \mid_{M=0} = a I_h \geq 0. \end{cases} \tag{4}$$

By applying [1, Lemmas 2 and 3], it follows that every solution of system (3) with positive initial conditions remains positive for all $t > 0$. Hence, the proposed model preserves the positivity of its solutions, which ensures that it is both biologically meaningful and mathematically well-defined. □

Next, we investigate the boundedness of the state variables of system (3). The following proposition establishes that all solutions of the system remain bounded.

Proposition 2. The biologically feasible region

$$H = \Big\{ (S_h, P_h, I_h, R_h, S_r, I_r, M) \in \mathbb{R}_+^7 : \\ S_h + P_h + I_h + R_h \leq \frac{\Lambda_h}{\mu_h}, \quad S_r + I_r \leq \frac{\Lambda_r}{\mu_r}, \quad M \leq \frac{a\Lambda_h}{b\mu_h} \Big\}$$

is positively invariant.

Proof. Let

$$N_h(t) = S_h(t) + P_h(t) + I_h(t) + R_h(t).$$

By summing the first four equations of system (3), we obtain

$$\begin{aligned} D^{\alpha} N_h(t) &= D^{\alpha} S_h + D^{\alpha} P_h + D^{\alpha} I_h + D^{\alpha} R_h \\ &\leq \Lambda_h - \mu_h S_h - \mu_h P_h - \mu_h I_h - \mu_h R_h \\ &\leq \Lambda_h - \mu_h \big(S_h + P_h + I_h + R_h \big). \end{aligned}$$

Thus,

$$D^{\alpha} N_h(t) \leq \Lambda_h - \mu_h N_h(t).$$

Therefore, we have

$$\limsup_{t\to+\infty} N_h(t) \leq \frac{\Lambda_h}{\mu_h}.$$

Similarly, let

$$N_r(t) = S_r(t) + I_r(t).$$

By summing the rodent equations of system (3), we have

$$\begin{aligned} D^\alpha N_r(t) &= D^\alpha S_r + D^\alpha I_r \\ &\leq \Lambda_r - \mu_r S_r - \mu_r I_r. \end{aligned}$$

Hence,

$$D^\alpha N_r(t) \leq \Lambda_r - \mu_r N_r(t),$$

which implies

$$\limsup_{t\to+\infty} N_r(t) \leq \frac{\Lambda_r}{\mu_r}.$$

Now, from the last equation of system (3), we obtain

$$D^\alpha M = aI_h - bM.$$

Since

$$I_h \leq N_h(t) \leq \frac{\Lambda_h}{\mu_h},$$

we have

$$D^\alpha M \leq \frac{a\Lambda_h}{\mu_h} - bM.$$

Therefore,

$$\limsup_{t\to+\infty} M(t) \leq \frac{a\Lambda_h}{b\mu_h}.$$

Consequently, the region H is positively invariant. □

Theorem 1. For every initial condition in H, system (3) has a unique solution.

Proof. First, system (3) can be rewritten as

$$D^{\alpha}X(t) = F(t, X(t)),$$

where

$$X(t) = \left(S_h(t), P_h(t), I_h(t), R_h(t), S_r(t), I_r(t), M(t)\right),$$

and

$$F(t, X(t)) = \begin{pmatrix} \Lambda_h + \omega R_h - \tau M S_h - \dfrac{\beta_h S_h(I_r + \eta I_h)}{1 + \kappa M} - \mu_h S_h + \xi P_h \\ \tau M S_h - (\xi + \mu_h) P_h \\ \dfrac{\beta_h S_h(I_r + \eta I_h)}{1 + \kappa M} - (\gamma_h + \mu_h) I_h \\ \gamma_h I_h - (\omega + \mu_h) R_h \\ \Lambda_r - \beta_r S_r I_r - \mu_r S_r \\ \beta_r S_r I_r - \mu_r I_r \\ a I_h - b M \end{pmatrix}.$$

The function $F(t, X(t))$ satisfies the first condition of [32, Proposition 4]. To verify the second condition of this proposition, we use the boundedness of the solutions established above, which gives

$$\begin{aligned} \|F(t, X(t))\|_1 \leq & \Lambda_h + \Lambda_r + \left(\frac{2a\tau\Lambda_h}{b\mu_h} + \frac{2b\beta_h\mu_h(\Lambda_r\mu_h + \eta\Lambda_h\mu_r)}{b\mu_h + \kappa a\Lambda_h} + \mu_h \right) \|S_h(t)\|_1 \\ & + (2\xi + \mu_h) \|P_h(t)\|_1 + (2\gamma_h + a + \mu_h) \|I_h(t)\|_1 \\ & + (2\omega + \mu_h) \|R_h(t)\|_1 + \left(\frac{2\beta_r\Lambda_r}{\mu_r} + \mu_r \right) \|S_r(t)\|_1 + \mu_r \|I_r(t)\|_1 \\ & + b\|M\|_1 \leq \Lambda_T + \Theta \|X(t)\|_1, \end{aligned}$$

with

$$\Lambda_T = \Lambda_h + \Lambda_r$$

and

$$\Theta = \max\left\{ \frac{2a\tau\Lambda_h}{b\mu_h} + \frac{2b\beta_h\mu_h(\Lambda_r\mu_h + \eta\Lambda_h\mu_r)}{b\mu_h + \kappa a\Lambda_h} + \mu_h,\ 2\xi + \mu_h, \right.$$
$$\left. 2\gamma_h + a + \mu_h,\ 2\omega + \mu_h,\ \frac{2\beta_r\Lambda_r}{\mu_r} + \mu_r,\ \mu_r,\ b \right\}.$$

Hence, $F(t, X(t))$ satisfies both conditions of [32, Proposition 4]. Consequently, the fractional system (3) admits a unique solution in the region H. This completes the proof. □

4 Global stability analysis of the equilibria

This section analyzes the existence of the equilibrium points of the model, namely the disease-free equilibrium E_0, the human-only equilibrium E_h, the rodent-only equilibrium E_r, and the endemic equilibrium E^*. In addition, the basic reproduction number R_0 is computed using the next-generation matrix technique, and the conditions for the existence of each equilibrium point are derived. Moreover, using Lyapunov functions together with LaSalle's invariance principle, we prove the global stability of these equilibrium points. We begin with the derivation of the basic reproduction number associated with system (3).

4.1 Computation of the basic reproduction number

The basic reproduction number, denoted by R_0, is defined as the average number of secondary infections produced by a single infected individual introduced into a fully susceptible population [28]. We use the next-generation matrix method as in [30] to calculate R_0, which is given by

$$R_0 = \rho(FV^{-1}),$$

where F is the nonnegative matrix of new infection terms, V is the matrix of the remaining transition terms in system (3), and ρ denotes the spectral radius of the matrix FV^{-1}. Let

$$F = \begin{pmatrix} \dfrac{\beta_h\eta\Lambda_h}{\mu_h} & \dfrac{\beta_h\Lambda_h}{\mu_h} \\ 0 & \dfrac{\beta_r\Lambda_r}{\mu_r} \end{pmatrix}, \quad V = \begin{pmatrix} \gamma_h + \mu_h & 0 \\ 0 & \mu_r \end{pmatrix}.$$

Therefore, by computing the matrix FV^{-1}, we obtain

$$R_0 = \max\left\{R_0^h, R_0^r\right\},$$

where

$$\begin{cases} R_0^h = \dfrac{\beta_h \eta \Lambda_h}{\mu_h(\gamma_h + \mu_h)}, \\ R_0^r = \dfrac{\beta_r \Lambda_r}{\mu_r^2}. \end{cases}$$

Here,

- R_0^h represents the human-related basic reproduction number associated with the human-to-human transmission pathway;
- R_0^r denotes the rodent-related basic reproduction number associated with the transmission dynamics among rodents.

Consequently, the overall basic reproduction number R_0 is determined by the dominant transmission mechanism between the human and rodent populations.

4.2 Determination of steady states

Model (3) admits four equilibrium points: The disease-free equilibrium E_0, the human-only equilibrium E_h, the rodent-only equilibrium E_r, and the endemic equilibrium E^*. These equilibrium states are given as follows:

1. **Disease-free equilibrium point.** The disease-free equilibrium point is given by

$$E_0 = \left(S_h^0,\, P_h^0,\, I_h^0,\, R_h^0,\, S_r^0,\, I_r^0,\, M^0\right),$$

where

$$S_h^0 = \frac{\Lambda_h}{\mu_h}, \quad S_r^0 = \frac{\Lambda_r}{\mu_r}, \quad P_h^0 = I_h^0 = R_h^0 = I_r^0 = M^0 = 0.$$

The disease-free equilibrium point E_0 always exists, since all its components are nonnegative.

2. **Human-only equilibrium point.** The human-only equilibrium point, denoted by E_h, represents a state in which the infection persists exclusively within the human population, while it is absent from the rodent population. It is given by

$$E_h = (S_h^h, P_h^h, I_h^h, R_h^h, S_r^h, 0, M^h),$$

where

$$S_r^h = \frac{\Lambda_r}{\mu_r}, \quad M^h = \frac{a}{b} I_h^h, \quad R_h^h = \frac{\gamma_h}{\omega + \mu_h} I_h^h, \quad P_h^h = \frac{\tau M^h S_h^h}{\xi + \mu_h},$$

and

$$S_h^h = \frac{(\gamma_h + \mu_h)(1 + \kappa M^h)}{\beta_h \eta}.$$

Furthermore, I_h^h is obtained as a positive solution of the corresponding equilibrium equation. Since $I_h^h > 0$, the infected human equilibrium equation is given by

$$\frac{\beta_h \eta S_h^h}{1 + \kappa M^h} = \gamma_h + \mu_h.$$

Therefore, a positive human-only equilibrium exists whenever

$$R_0^h = \frac{\beta_h \eta \Lambda_h}{\mu_h(\gamma_h + \mu_h)} > 1.$$

3. **Rodent-only equilibrium point.** The rodent-only equilibrium point, denoted by E_r, corresponds to the persistence of the infection in the rodent population while the human population remains disease-free. It is given by

$$E_r = (S_h^r, 0, 0, 0, S_r^r, I_r^r, 0),$$

where

$$S_h^r = \frac{\Lambda_h}{\mu_h}, \quad S_r^r = \frac{\mu_r}{\beta_r}, \quad I_r^r = \frac{\Lambda_r \beta_r - \mu_r^2}{\beta_r \mu_r}.$$

Furthermore, I_r^r is obtained as a positive solution of the corresponding equilibrium equation. Since $I_r^r > 0$, the infected rodent equilibrium equation is given by

$$\beta_r S_r^r = \mu_r.$$

Substituting $S_r^r = \dfrac{\mu_r}{\beta_r}$ into the susceptible rodent equilibrium equation gives

$$I_r^r = \frac{\Lambda_r \beta_r - \mu_r^2}{\beta_r \mu_r}.$$

Therefore, a positive rodent-only equilibrium exists whenever

$$\beta_r \Lambda_r - \mu_r^2 > 0.$$

Hence, the rodent-only equilibrium point E_r exists if and only if

$$R_0^r = \frac{\beta_r \Lambda_r}{\mu_r^2} > 1.$$

4. **Endemic equilibrium point.** The endemic equilibrium point, denoted by E^*, represents a steady state in which the infection persists in both the human and rodent populations. It is given by

$$E^* = (S_h^*, P_h^*, I_h^*, R_h^*, S_r^*, I_r^*, M^*),$$

where

$$S_r^* = \frac{\mu_r}{\beta_r}, \quad I_r^* = \frac{\Lambda_r \beta_r - \mu_r^2}{\beta_r \mu_r}, \quad M^* = \frac{a}{b} I_h^*,$$
$$R_h^* = \frac{\gamma_h}{\omega + \mu_h} I_h^*, \quad P_h^* = \frac{\tau M^* S_h^*}{\xi + \mu_h},$$

and

$$S_h^* = \frac{(\gamma_h + \mu_h) I_h^* (1 + \kappa M^*)}{\beta_h (I_r^* + \eta I_h^*)}.$$

The quantity I_h^* is determined as a positive solution of the corresponding equilibrium equation. For the endemic equilibrium to be biologically meaningful, all state variables must be positive. In particular, since $I_r^* > 0$, we require

$$\Lambda_r \beta_r - \mu_r^2 > 0,$$

which can be rewritten as

$$R_0^r = \frac{\beta_r \Lambda_r}{\mu_r^2} > 1.$$

Similarly, persistence of the disease in the human population requires a positive infected class, $I_h^* > 0$. This condition is satisfied whenever

$$R_0^h = \frac{\beta_h \eta \Lambda_h}{\mu_h (\gamma_h + \mu_h)} > 1.$$

Therefore, the endemic equilibrium E^* exists whenever both the rodent and human basic reproduction numbers exceed unity, that is,

$$R_0^h > 1 \qquad \text{and} \qquad R_0^r > 1.$$

The existence conditions of the equilibrium points of system (3) are summarized in Table 3.

Table 3: Summary of the existence conditions for the equilibrium points of system (3).

Equilibrium Point	**Description**	**Existence Condition**
E_0	Disease-free equilibrium	Always exists
E_h	Human-only equilibrium	$R_0^h > 1$
E_r	Rodent-only equilibrium	$R_0^r > 1$
E^*	Endemic equilibrium	$R_0^h > 1$ and $R_0^r > 1$

Next, we investigate the global stability properties of the equilibrium points of system (3).

4.3 Global stability

This subsection investigates the global stability of the equilibrium points of system (3) using Lyapunov's direct method and LaSalle's invariance principle [12]. Although a human-only equilibrium E_h may exist when $R_0^h > 1$, it is of limited relevance to Hantavirus transmission, because rodents are the natural reservoir of the virus and the long-term persistence of the disease depends on the survival and proliferation of infected rodent populations. Thus, the equilibrium point E_h, which excludes the persistence of infection in rodents, does not accurately reflect the key dynamics of the coupled human-rodent transmission system. Similarly, the rodent-only equilibrium E_r, in which rodents remain infected while humans are free of infection, is not the primary focus of this study. This equilibrium may be biologically feasible, but it provides limited insight into the public health consequences of Hantavirus infection. Since our main aim is to determine the mechanisms governing disease spread and persistence in both host populations, the global stability analysis is restricted to the disease-free equilibrium E_0 and the endemic equilibrium E^*. These equilibria represent the most epidemiologically significant states of the system and are crucial in determining the threshold dynamics and the long-term behavior of the Hantavirus transmission system.

Theorem 2. If $R_0 = \max\{R_0^h, R_0^r\} \leq 1$, then the disease-free equilibrium point E_0 of system (3) is globally asymptotically stable in the feasible region H.

Proof. Consider the following function:

$$V_0 = \frac{I_h}{\gamma_h + \mu_h} + \frac{I_r}{\mu_r}.$$

Clearly, V_0 is continuous and nonnegative throughout the feasible region H. Since $I_h \geq 0$ and $I_r \geq 0$, it follows directly that $V_0 \geq 0$. Moreover, $V_0 = 0$ if and only if $I_h = I_r = 0$, which implies that V_0 is positive definite with respect to the disease-free equilibrium E_0. Consequently, V_0 is a suitable Lyapunov function candidate for analyzing the global stability of E_0. By applying the properties of the Caputo fractional derivative, we obtain

$$D^\alpha V_0 \leq \frac{1}{\gamma_h + \mu_h} D^\alpha I_h + \frac{1}{\mu_r} D^\alpha I_r.$$

Substituting the equations of system (3), we get

$$D^\alpha V_0 \leq \frac{1}{\gamma_h + \mu_h} \left(\frac{\beta_h S_h (I_r + \eta I_h)}{1 + \kappa M} - (\gamma_h + \mu_h) I_h \right) + \frac{1}{\mu_r} \left(\beta_r S_r I_r - \mu_r I_r \right).$$

Since

$$\frac{1}{1 + \kappa M} \leq 1, \qquad S_h \leq \frac{\Lambda_h}{\mu_h}, \qquad S_r \leq \frac{\Lambda_r}{\mu_r},$$

it follows that

$$D^\alpha V_0 \leq \frac{\beta_h \Lambda_h}{\mu_h(\gamma_h + \mu_h)} I_r + \left(\frac{\beta_h \eta \Lambda_h}{\mu_h(\gamma_h + \mu_h)} - 1 \right) I_h + \left(\frac{\beta_r \Lambda_r}{\mu_r^2} - 1 \right) I_r.$$

Using the definitions

$$R_0^h = \frac{\beta_h \eta \Lambda_h}{\mu_h(\gamma_h + \mu_h)} \quad \text{and} \quad R_0^r = \frac{\beta_r \Lambda_r}{\mu_r^2},$$

we obtain

$$D^\alpha V_0 \leq (R_0^h - 1) I_h + (R_0^r - 1) I_r.$$

Since $R_0^h \leq 1$ and $R_0^r \leq 1$, it follows that $D^\alpha V_0 \leq 0$. Moreover, $D^\alpha V_0 = 0$ if and only if $I_h = 0$ and $I_r = 0$. From the system equations, when $I_h = I_r = 0$, we necessarily obtain $M = 0$, $P_h = 0$, and $R_h = 0$, while $S_h = \frac{\Lambda_h}{\mu_h}$ and $S_r = \frac{\Lambda_r}{\mu_r}$. Therefore, the largest invariant set contained

in $\{(I_h, I_r) : D^\alpha V_0 = 0\}$ is the singleton $\{E_0\}$. Consequently, by LaSalle's invariance principle for fractional-order systems, the disease-free equilibrium E_0 is globally asymptotically stable in H. □

Theorem 3. If $R_0^h > 1$ and $R_0^r > 1$, then the endemic equilibrium point E^* is globally asymptotically stable in the feasible region H.

Proof. Consider the following function:

$$\begin{aligned} V^* = {} & S_h^* \left(\frac{S_h}{S_h^*} - \ln \frac{S_h}{S_h^*} - 1 \right) + P_h^* \left(\frac{P_h}{P_h^*} - \ln \frac{P_h}{P_h^*} - 1 \right) + I_h^* \left(\frac{I_h}{I_h^*} - \ln \frac{I_h}{I_h^*} - 1 \right) \\ & + R_h^* \left(\frac{R_h}{R_h^*} - \ln \frac{R_h}{R_h^*} - 1 \right) + S_r^* \left(\frac{S_r}{S_r^*} - \ln \frac{S_r}{S_r^*} - 1 \right) \\ & + I_r^* \left(\frac{I_r}{I_r^*} - \ln \frac{I_r}{I_r^*} - 1 \right) + M^* \left(\frac{M}{M^*} - \ln \frac{M}{M^*} - 1 \right). \end{aligned}$$

Clearly, V^* is continuous and nonnegative throughout the feasible region H. Indeed, for every positive variable x, the inequality

$$x - \ln(x) - 1 \geq 0$$

holds, with equality if and only if $x = 1$. Since each component of V^* is of this form, every term is nonnegative, implying that

$$V^* \geq 0 \quad \text{for all } (S_h, P_h, I_h, R_h, S_r, I_r, M) \in H.$$

Moreover, $V^* = 0$ if and only if $S_h = S_h^*$, $P_h = P_h^*$, $I_h = I_h^*$, $R_h = R_h^*$, $S_r = S_r^*$, $I_r = I_r^*$, $M = M^*$, which is equivalent to

$$(S_h, P_h, I_h, R_h, S_r, I_r, M) = E^*.$$

Therefore, V^* vanishes only at the endemic equilibrium E^* and remains strictly positive elsewhere in H. Hence, V^* is positive definite with respect to E^*, which makes it an appropriate Lyapunov function candidate for establishing the global asymptotic stability of the endemic equilibrium point E^*. By [1, Lemma 4], we obtain

$$\begin{aligned} D^\alpha V^* \leq {} & \left(1 - \frac{S_h^*}{S_h}\right) D^\alpha S_h + \left(1 - \frac{P_h^*}{P_h}\right) D^\alpha P_h + \left(1 - \frac{I_h^*}{I_h}\right) D^\alpha I_h \\ & + \left(1 - \frac{R_h^*}{R_h}\right) D^\alpha R_h + \left(1 - \frac{S_r^*}{S_r}\right) D^\alpha S_r + \left(1 - \frac{I_r^*}{I_r}\right) D^\alpha I_r \end{aligned}$$

$$+\left(1-\frac{M^*}{M}\right)D^\alpha M.$$

Substituting the model equations into the above inequality yields

$$\begin{aligned}
D^\alpha V^* \leq & \left(1-\frac{S_h^*}{S_h}\right)\left(\Lambda_h+\omega R_h-\tau M S_h-\frac{\beta_h S_h(I_r+\eta I_h)}{1+\kappa M}-\mu_h S_h+\xi P_h\right)\\
&+\left(1-\frac{P_h^*}{P_h}\right)(\tau M S_h-(\xi+\mu_h)P_h)\\
&+\left(1-\frac{I_h^*}{I_h}\right)\left(\frac{\beta_h S_h(I_r+\eta I_h)}{1+\kappa M}-(\gamma_h+\mu_h)I_h\right)\\
&+\left(1-\frac{R_h^*}{R_h}\right)(\gamma_h I_h-(\omega+\mu_h)R_h)\\
&+\left(1-\frac{S_r^*}{S_r}\right)(\Lambda_r-\beta_r S_r I_r-\mu_r S_r)\\
&+\left(1-\frac{I_r^*}{I_r}\right)(\beta_r S_r I_r-\mu_r I_r)\\
&+\frac{\gamma_h+\mu_h}{a}\left(1-\frac{M^*}{M}\right)(aI_h-bM).
\end{aligned}$$

Using the equilibrium relations

$$\begin{cases}
\tau M^* S_h^* = (\xi+\mu_h)P_h^*,\\
\dfrac{\beta_h S_h^*(I_r^*+\eta I_h^*)}{1+\kappa M^*} = (\gamma_h+\mu_h)I_h^*,\\
\gamma_h I_h^* = (\omega+\mu_h)R_h^*,\\
\beta_r S_r^* I_r^* = \mu_r I_r^*,\\
aI_h^* = bM^*,
\end{cases} \tag{5}$$

and after straightforward calculations, we obtain

$$D^\alpha V^* \leq -F_h^*\Phi_1 - G_h^*\Phi_2 - \gamma_h I_h^*\Phi_3 - F_r^*\Phi_4 - bM^*\Phi_5,$$

where

$$\begin{cases}
F_h^* = \dfrac{\beta_h S_h^*(I_r^*+\eta I_h^*)}{1+\kappa M^*},\\
G_h^* = \tau M^* S_h^*,\\
F_r^* = \beta_r S_r^* I_r^*,
\end{cases} \tag{6}$$

and

$$\begin{cases} \Phi_1 = 3 - \dfrac{S_h^*}{S_h} - \dfrac{S_h(I_r+\eta I_h)(1+\kappa M^*)I_h^*}{S_h^*(I_r^*+\eta I_h^*)(1+\kappa M)I_h} - \dfrac{I_h S_h^*(I_r^*+\eta I_h^*)(1+\kappa M)}{I_h^* S_h(I_r+\eta I_h)(1+\kappa M^*)}, \\ \Phi_2 = 3 - \dfrac{S_h^*}{S_h} - \dfrac{M S_h P_h^*}{M^* S_h^* P_h} - \dfrac{P_h M^* S_h^*}{P_h^* M S_h}, \\ \Phi_3 = 2 - \dfrac{I_h R_h^*}{I_h^* R_h} - \dfrac{R_h I_h^*}{R_h^* I_h}, \\ \Phi_4 = 2 - \dfrac{S_r^*}{S_r} - \dfrac{S_r I_r^*}{S_r^* I_r}, \\ \Phi_5 = 2 - \dfrac{I_h M^*}{I_h^* M} - \dfrac{M I_h^*}{M^* I_h}. \end{cases} \tag{7}$$

By the arithmetic-geometric mean inequality,

$$\Phi_i \geq 0, \qquad i = 1, \ldots, 5.$$

Therefore,

$$D^\alpha V^* \leq 0 \quad \text{for all } (S_h, P_h, I_h, R_h, S_r, I_r, M) \in H.$$

Moreover, $D^\alpha V^* = 0$ if and only if $S_h = S_h^*$, $P_h = P_h^*$, $I_h = I_h^*$, $R_h = R_h^*$, $S_r = S_r^*$, $I_r = I_r^*$, and $M = M^*$. Therefore, every state variable coincides with its corresponding endemic equilibrium value, which implies that the largest invariant set contained in $\{D^\alpha V^* = 0\}$ is the singleton $\{E^*\}$. Consequently, by LaSalle's invariance principle for fractional-order systems, every solution of system (3) with initial conditions in H approaches E^* as t tends to infinity. Hence, the endemic equilibrium point E^* is globally asymptotically stable in H. □

5 Numerical simulations

In this section, a series of numerical simulations is carried out to investigate the influence of the fractional order and of the transmission and awareness parameters on the infection dynamics. All simulations were performed using MATLAB. To obtain approximate solutions of the proposed fractional-order model, we employed the numerical scheme introduced in [14]. The discretization formula is given by

$$\begin{aligned} Y(t_i) = Y(t_0) &+ \frac{h^\alpha}{\Gamma(\alpha+2)}\left[(i-1)^{\alpha+1} - (i-\alpha-1)i^\alpha\right] G\big(Y(t_0)\big) \\ &+ \frac{h^\alpha}{\Gamma(\alpha+2)} \sum_{j=1}^{i-1} \left[(i-j+1)^{\alpha+1} - 2(i-j)^{\alpha+1} + (i-j-1)^{\alpha+1}\right] G\big(Y(t_j)\big) \end{aligned}$$

$$+\frac{h^{\alpha}}{\Gamma(\alpha+2)}G\left(Y(t_{i-1})+\frac{h^{\alpha}}{\Gamma(\alpha+1)}F\big(Y(t_{i-1})\big)\right), \tag{8}$$

where the discrete time nodes are defined by

$$t_i = t_0 + ih, \qquad i = 0, 1, \ldots, N-1,$$

with h denoting the time-step size. The initial condition is

$$Y_0 = (50,\ 30,\ 10,\ 5,\ 30,\ 7,\ 15).$$

Unless otherwise specified, the parameter values used in the numerical simulations are listed in Table 4. These values were used to generate all the figures presented in this section.

Table 4: Parameter values used in the numerical simulations.

Parameters	Figure 2	Figure 3	Figure 4	Figure 5	Figure 6
Λ_h	1	1	1	1	1
μ_h	0.05	0.1	0.1	0.1	0.1
β_h	0.0002	0.25	-	0.25	0.25
η	0.1	0.8	0.8	0.8	0.8
κ	0.1	0.5	0.5	0.5	-
τ	0.02	0.002	0.002	0.002	0.002
ξ	0.01	0.05	0.05	0.05	0.05
γ_h	0.3	0.08	0.08	0.08	0.08
ω	0.05	0.05	0.05	0.05	0.05
Λ_r	1	1	1	1	1
β_r	0.0003	0.2	0.2	-	0.2
μ_r	0.1	0.25	0.25	0.25	0.25
a	0.05	0.02	0.02	0.02	0.02
b	0.3	0.20	0.20	0.20	0.20
R_0^h	0.00114	11.11	-	-	-
R_0^r	0.03	3.20	-	-	-
Theorems	Theorem 2	Theorem 3	-	-	-

5.1 Global stability and effect of the fractional order

To illustrate the global stability of the equilibria and the effect of the fractional order on the infection dynamics, we used scheme (8), with the fractional order ranging between 0.55 and 1. Figure 2 illustrates the time evolution of the Hantavirus model variables, namely S_h, P_h, I_h, R_h, S_r, I_r, and M, for different values of the fractional-order parameter α. As shown, all state trajectories converge to the disease-free equilibrium $E_0 = (20, 0, 0, 0, 10, 0, 0)$ for all values of α considered. This behavior indicates the complete elimination of the infection from both the human and rodent populations, with only the susceptible classes remaining positive at equilibrium. These numerical results are fully consistent with Theorem 2, which guarantees the global asymptotic stability of the disease-free equilibrium. Furthermore, the basic reproduction numbers reported in Table 4, $R_0^h = 0.00114 < 1$ and $R_0^r = 0.03 < 1$, satisfy the conditions of Theorem 2.

Consequently, the infection cannot persist in either the human or the rodent population, and the disease-free state is globally attractive. The simulations also show that the fractional-order parameter has a significant impact on the transient dynamics of the system. In particular, lower values of α lead to faster convergence to the equilibrium, whereas higher values lead to slower convergence. This phenomenon is due to the memory effect characteristic of fractional-order derivatives: The higher the fractional order, the stronger the memory of the system and the longer the transient response before the steady state is reached. It should be noted that changing the fractional-order parameter alters neither the location nor the stability of the equilibrium point. In other words, the fractional order only influences the convergence speed of the trajectories, while the long-term qualitative behavior remains unchanged. Figure 3 illustrates the time evolution of the Hantavirus model variables for different values of α. The numerical results show that, irrespective of the chosen fractional order, all solution trajectories converge to the endemic equilibrium point $E^* = (0.820, 0.009, 5.983, 3.191, 1.250, 2.750, 0.873)$. This convergence demonstrates the long-term persistence of the disease in both the human and rodent populations. In particular, the infected human and rodent compartments approach positive equilibrium values, confirming that the infection remains endemic. Furthermore, the protected human compartment P_h stabilizes at a very small positive level, which can be attributed to the relatively low awareness rate τ. These numerical observations are fully consistent with Theorem 3, which establishes the global asymptotic stability of the endemic equilibrium. Moreover, as reported in Table 4, the basic reproduction numbers satisfy $R_0^h = 11.11 > 1$ and $R_0^r = 3.20 > 1$, thereby fulfilling the conditions of Theorem 3.

Consequently, the endemic equilibrium exists and is globally asymptotically stable. The simulations also emphasize the role of the fractional-order parameter in the transient behavior

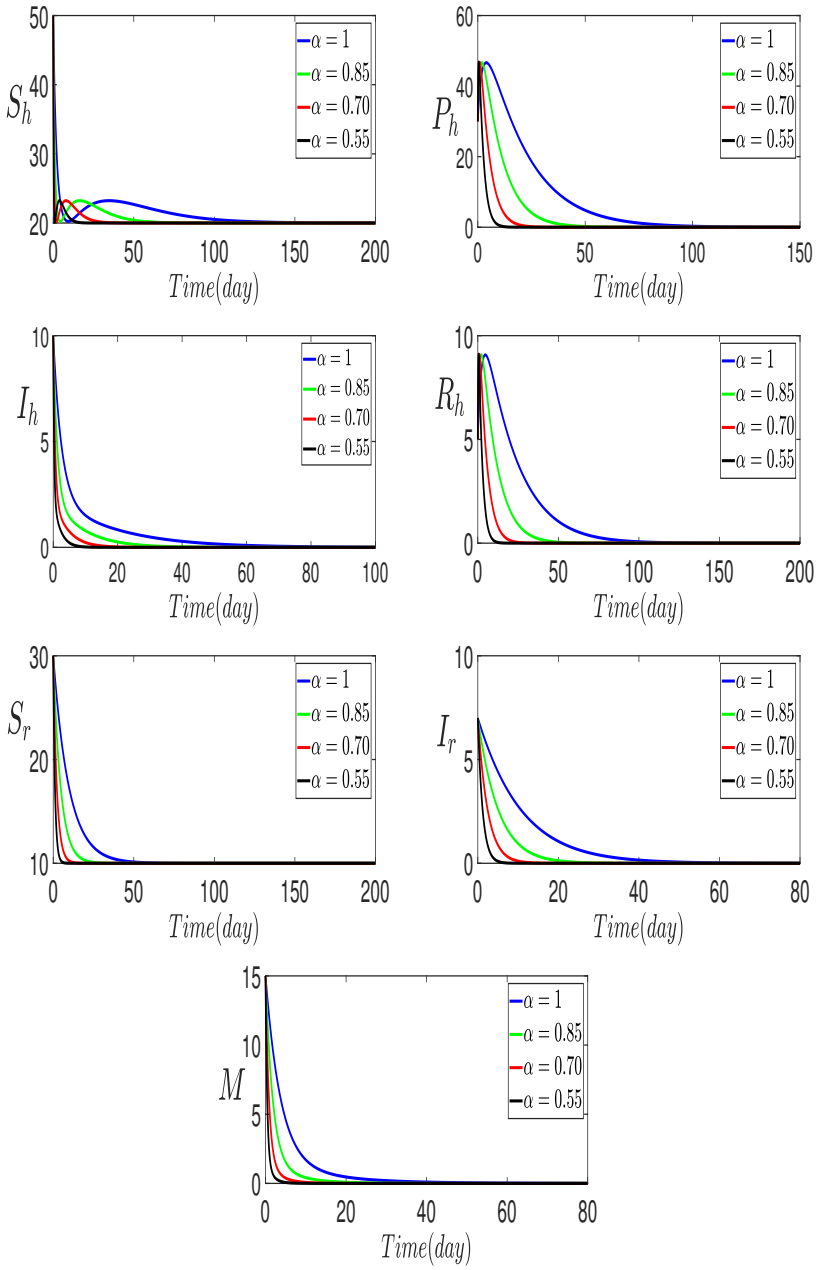


Figure 2: Stability of the disease-free equilibrium point E_0 for different values of the fractional-order parameter α.

of the system. In particular, lower fractional-order values result in faster convergence to the endemic equilibrium, while higher values lead to a longer transient phase. This behavior is typical

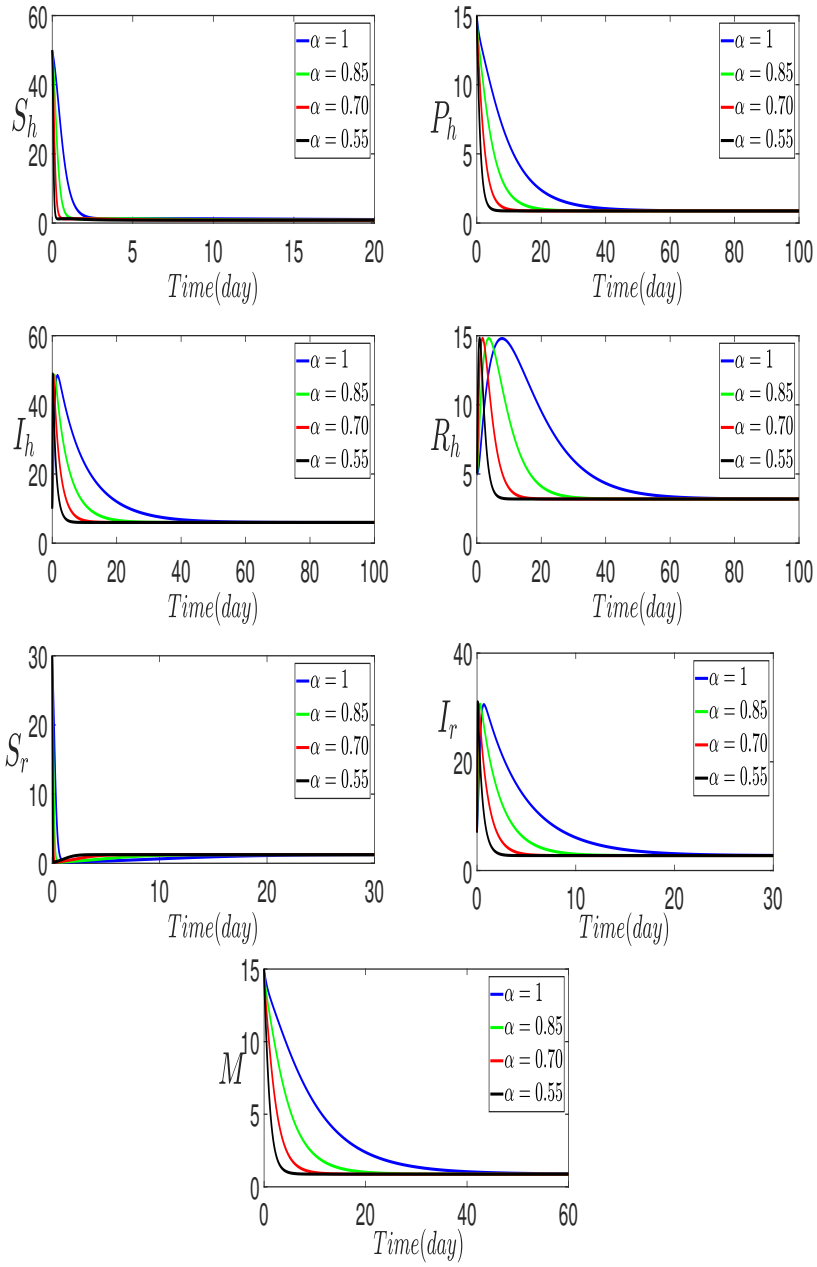


Figure 3: Stability of the endemic equilibrium point E^* for different values of the fractional-order parameter α.

of fractional-order dynamical systems, in which memory effects arise from the Caputo fractional derivative. As the order increases, the memory effect becomes more significant, which translates

into a longer transient period before the system reaches equilibrium. However, changing the value of the fractional-order parameter alters neither the position nor the stability of the endemic equilibrium; it only affects the rate at which the solution trajectories converge to it.

5.2 Effects of the transmission rates

In this subsection, we study how the transmission rates affect the dynamics of the Hantavirus model. The transmission rates play a key role in the spread of the disease among humans and rodents. We analyze the impact of varying these parameters on the evolution of the model variables while keeping the other parameters unchanged.

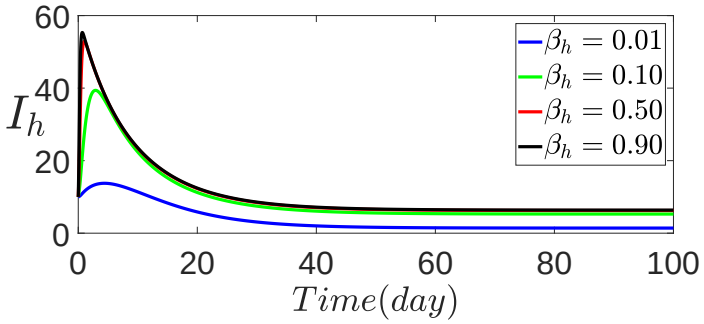


Figure 4: Influence of the human transmission rate β_h on the infected human population I_h.

Figure 4 highlights the impact of the human transmission rate β_h on the infected human population I_h. The results show that, as β_h increases, the number of infected individuals increases throughout the simulation. For small values of β_h, the number of infections decreases quickly, leading to a low endemic level. In contrast, larger values of β_h result in a high infection peak and a high endemic equilibrium. These findings demonstrate that reducing the human transmission rate is an effective means of preventing the spread of Hantavirus and reducing its burden on the human population over time. Figure 5 presents the impact of the rodent transmission rate β_r on the infected rodent population. It can be seen that increasing β_r increases the number of infected rodents throughout the simulation, whereas decreasing this parameter substantially reduces the infection level among rodents. Although all trajectories eventually converge to an equilibrium, the transient dynamics are considerably affected by the value of β_r. For instance, for larger values of β_r, the infection level is higher and persists longer before the steady state is reached. Thus, this figure illustrates the importance of the rodent transmission rate in maintaining and propagating Hantavirus.

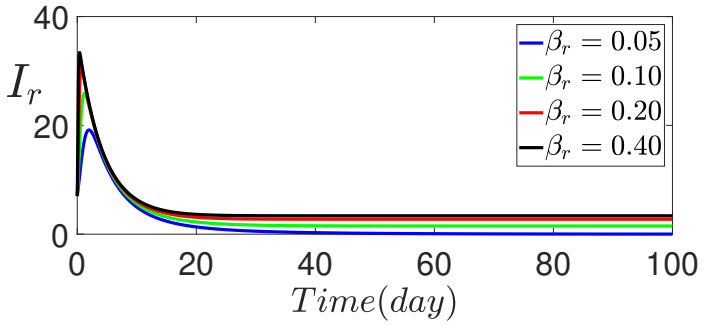


Figure 5: Impact of the rodent transmission rate β_r on the infected rodent population I_r.

5.3 Effect of media awareness

In this subsection, we analyze the effect of media awareness, which encourages individuals to adopt preventive behaviors and thereby reduces the infection rate. By varying the media awareness parameter while keeping all other parameters unchanged, we assess its effect on the infected population.

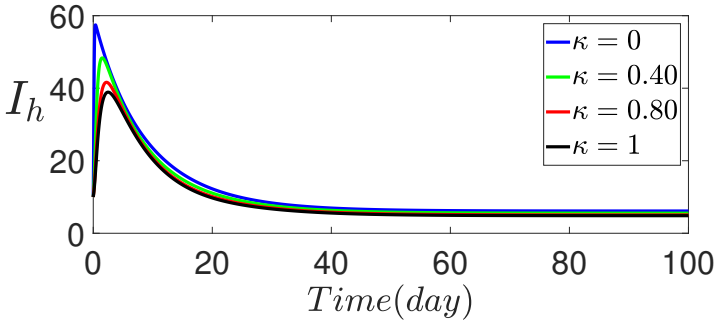


Figure 6: Influence of the media awareness parameter κ on the infected human population I_h.

Figure 6 illustrates the effect of media awareness on the infected human population I_h for different values of the media awareness effectiveness parameter κ. In the proposed model, media awareness acts through two mechanisms. First, the awareness level $M(t)$ promotes protective behavior by transferring susceptible individuals from S_h to the protected class P_h at the rate $\tau M S_h$. Second, it reduces the force of infection through the factor $\frac{1}{1+\kappa M}$. Therefore, increasing κ strengthens the effect of media awareness on reducing disease transmission. The numerical simulations consider $\kappa = 0$, 0.40, 0.80, and 1, while keeping the other parameters unchanged. As shown in Figure 6, increasing κ progressively reduces the number of infected individuals. When $\kappa = 0$, the infected population remains at a higher level, whereas larger values of κ lead to a

lower infection peak and a lower infection level over time. These results show numerically that increasing the effectiveness of media awareness can reduce the infection burden by promoting protective behavior and decreasing effective transmission. However, under the endemic conditions considered here, media awareness reduces the infection level but does not necessarily lead to complete disease elimination. Thus, media awareness represents an important behavioral control mechanism for reducing Hantavirus transmission.

6 Sensitivity analysis of the basic reproduction number

This section is devoted to the sensitivity analysis of the model. The main objective is to determine the parameters that most strongly influence the dynamics of disease transmission and the basic reproduction number R_0. The following definition is required for the subsequent analysis.

Definition 1. [13] The normalized forward SI of the basic reproduction number R_0 with respect to a parameter θ measures the relative variation in R_0 resulting from a relative change in θ. It is defined by

$$S_\theta^{R_0} = \frac{\partial R_0}{\partial \theta}\frac{\theta}{R_0}.$$

Since $R_0 = \max\left\{R_0^h, R_0^r\right\}$, the sensitivity analysis is conducted separately for the two threshold quantities R_0^h and R_0^r. Specifically, we first calculate the normalized forward SIs of R_0^h with respect to the human-related parameters β_h, η, Λ_h, μ_h, and γ_h. We then determine the SIs of R_0^r with respect to the rodent-related parameters β_r, Λ_r, and μ_r.

The SIs of R_0^h are given by

$$\begin{cases} S_{\beta_h}^{R_0^h} = 1, \\ S_{\eta}^{R_0^h} = 1, \\ S_{\Lambda_h}^{R_0^h} = 1, \\ S_{\gamma_h}^{R_0^h} = -\dfrac{\gamma_h}{\gamma_h + \mu_h}, \\ S_{\mu_h}^{R_0^h} = -\dfrac{\gamma_h + 2\mu_h}{\gamma_h + \mu_h}. \end{cases} \tag{9}$$

The SIs of R_0^r are given by

$$\begin{cases} S_{\beta_r}^{R_0^r} = 1, \\ S_{\Lambda_r}^{R_0^r} = 1, \\ S_{\mu_r}^{R_0^r} = -2. \end{cases} \tag{10}$$

The computed SIs of the model parameters with respect to R_0^h and R_0^r are reported in Table 5 and illustrated in Figure 7.

Table 5: SIs of the model parameters associated with the basic reproduction numbers R_0^h and R_0^r.

Parameter	Value	SI for R_0^h	SI for R_0^r
Λ_h	1	1	–
β_h	0.25	1	–
η	0.80	1	–
γ_h	0.08	-0.4444	–
μ_h	0.10	-1.5556	–
Λ_r	1	–	1
β_r	0.20	–	1
μ_r	0.25	–	-2

Figure 7 and Table 5 show the normalized forward SIs of the model parameters with respect to the basic reproduction numbers R_0^h and R_0^r. In general, a positive SI indicates that an increase in the corresponding parameter leads to an increase in the basic reproduction number, whereas a negative SI indicates that increasing the parameter decreases the reproduction number. Furthermore, the magnitude of the SI reflects the relative importance of each parameter in influencing disease transmission. For the human basic reproduction number R_0^h, the parameters Λ_h, β_h, and η all have SIs equal to $+1$. Consequently, a 10% increase (or decrease) in any of these parameters results in a 10% increase (or decrease) in the value of R_0^h. This shows that the human recruitment rate, the human transmission rate, and the modification parameter have the largest positive impact on disease transmission within the human population. On the other hand, the SIs of the recovery rate γ_h and the natural death rate μ_h are both negative, equal to -0.4444 and -1.5556, respectively. Consequently, increasing either of these parameters decreases the reproduction number R_0^h, whereas decreasing them favors the spread of the disease. Similarly, for the rodent basic reproduction number R_0^r, the recruitment rate Λ_r and the transmission rate β_r have SIs of $+1$, indicating that a 10% increase in either parameter increases R_0^r by 10%. On the other hand, the natural death rate μ_r has an SI of -2, which means that a 10% increase in μ_r reduces R_0^r by approximately 20%. Among all rodent-related parameters,

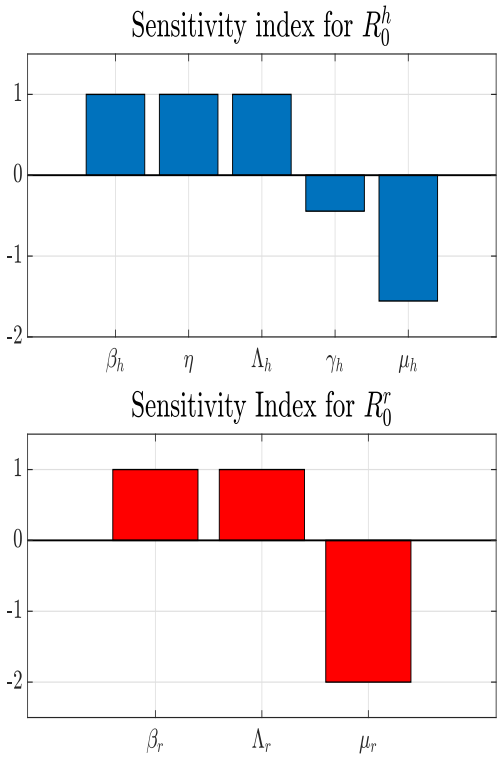


Figure 7: Normalized forward SIs of the parameters associated with R_0^h and R_0^r.

μ_r has the largest SI in absolute value and is therefore the most significant factor determining Hantavirus persistence in the rodent population. In summary, the sensitivity analysis shows that the transmission parameters β_h and β_r drive the spread of the disease, while the recovery rate γ_h and the natural death rates μ_h and μ_r help limit its spread. For this reason, reducing transmission through preventive actions, combined with measures aimed at speeding up recovery and shortening the infectious period of humans, is expected to be the most effective way to bring the basic reproduction numbers below one and thus limit the spread of Hantavirus among humans and rodents.

7 Conclusion

This study proposed a novel fractional-order Hantavirus transmission model based on the Caputo fractional derivative that takes into account media awareness and the protective behavior of humans. By accounting for memory effects, the model better represents disease dynamics and

captures the complex interactions between humans and rodents. The mathematical analysis established the existence, uniqueness, positivity, and boundedness of the solutions, confirming that the model is mathematically well-posed and biologically meaningful. Moreover, the basic reproduction numbers were computed, and they determine the conditions for disease extinction or persistence.

The global stability analysis focused on two epidemiologically significant equilibria: The disease-free equilibrium E_0 and the endemic equilibrium E^*. While the human-only equilibrium E_h may exist when $R_0^h > 1$, it is of limited epidemiological significance, since Hantavirus is mainly sustained in infected rodent populations, the natural reservoir of the virus. Therefore, E_h does not properly describe the long-term dynamics of the coupled human-rodent transmission system. Similarly, the rodent-only equilibrium E_r, although biologically possible under certain circumstances, is not very informative about the public health impact of the disease because human infections are not involved. For this reason, a global stability analysis of E_h and E_r was not undertaken. Instead, only the disease-free and fully endemic equilibria were examined, since they determine the threshold behavior and the long-term dynamics of the complete transmission system. Specifically, it was shown that the disease-free equilibrium E_0 is globally asymptotically stable whenever $R_0 = \max\{R_0^h, R_0^r\} \leq 1$, whereas the endemic equilibrium E^* is globally asymptotically stable whenever $R_0^h > 1$ and $R_0^r > 1$. These global stability results were established using Lyapunov functions and LaSalle's invariance principle for fractional-order systems.

The simulations confirmed the theoretical results and illustrated the importance of the fractional-order parameter in the epidemic dynamics. In particular, reducing the fractional order accelerated convergence to the equilibrium without affecting its asymptotic stability. The simulations also showed that an increase in the transmission rates resulted in a higher number of infected individuals, whereas increasing the effectiveness of media awareness reduced the number of infections through changes in protective behavior. According to the sensitivity analysis, the transmission and recruitment parameters are the main contributors to the spread of the disease, while higher recovery and natural mortality rates are associated with a decrease in disease transmission. These findings highlight the importance of combining transmission-reduction measures, such as limiting contact between humans and infected rodents, with effective public awareness and appropriate protective behaviors as part of a comprehensive strategy for controlling Hantavirus transmission.

The proposed fractional-order framework provides a useful mathematical approach for investigating the combined effects of memory, media awareness, and behavioral responses on Hantavirus transmission dynamics. Future work could extend the present model to a multi-group setting to account for population heterogeneity and investigate optimal control strategies for reducing Hantavirus transmission [11].

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Conflicts of Interest

The author declares no conflicts of interest.

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