Modeling the Transmission Dynamics of Hantavirus with Environmental Viral Persistence

Abstract

Hantavirus is a zoonotic disease maintained primarily in rodent reservoirs, with human infection occurring predominantly through exposure to virus-contaminated environments. In this study, we formulate and analyze a deterministic compartmental model describing hantavirus transmission among rodents, humans, and an environmental viral reservoir. The model incorporates logistic rodent population growth, direct rodent-to-rodent transmission, environmental transmission, viral shedding, and human disease progression through susceptible, exposed, infectious, and recovered classes. Basic qualitative properties of the model are established, including the existence, uniqueness, positivity, and boundedness of solutions. Using the next-generation matrix approach, the basic reproduction number is derived and shown to govern the threshold dynamics of the disease. The disease-free equilibrium is proven to be globally asymptotically stable whenever $R_0 < 1$, while the existence of an endemic equilibrium is established for $R_0 > 1$, and conditions for its local asymptotic stability are obtained using the Routh–Hurwitz criterion. The analysis demonstrates that environmental viral persistence plays a critical role in sustaining transmission by linking infected rodent reservoirs with susceptible human populations. The results further indicate that reducing rodent infection, increasing environmental viral clearance, and minimizing human exposure to contaminated environments are effective strategies for lowering disease transmission. The proposed model provides a rigorous mathematical framework for understanding the ecological and epidemiological mechanisms underlying hantavirus persistence and offers a foundation for future investigations incorporating seasonality, spatial heterogeneity, stochastic effects, and optimal control.

Keywords: Hantavirus, environmental viral persistence, rodent reservoirs, zoonotic disease, mathematical epidemiology, basic reproduction number, stability analysis, endemic equilibrium.

1 Introduction

Hantavirus infection is an important zoonotic disease caused by an RNA virus of the Hantaviridae family, maintained in rodent reservoir populations and transmitted to people predominantly through inhalation of aerosolized virus from rodent urine, saliva, or feces, though rodent bite and direct contact with contaminated materials can also cause infection [9, 18, 15]. Infection in humans can cause serious disease, especially Hemorrhagic Fever with Renal Syndrome (HFRS) and Hantavirus Pulmonary Syndrome (HPS), which are associated with high morbidity and mortality [17].

The ecology of hantavirus is tightly linked to the disease's epidemiology. Infected rodents are usually asymptomatic, but can continue to shed virus for life [19]. Fluctuations in food supply, rainfall, habitat disturbances, and predator populations all impact rodent populations and subsequent patterns of disease, as highlighted in the well-studied 1993 Four Corners outbreak in the United States [14, 12].

Environmental transmission is an important route of human infection: viral particles shed into the environment can remain infectious for weeks under certain temperature, humidity, UV, and ventilation

regimes [16]. Accordingly, people are often infected by contaminated dust in confined environments, such as on farms, in storage areas, in military camps, and in unventilated buildings [22]. This suggests that it is important to explicitly model transmission from the environment.

Mathematical modeling is an important tool in understanding infectious disease dynamics and outcomes, and evaluating the potential effectiveness of control strategies. Compartmental models for epidemic dynamics can be used to study transmission, invasion thresholds, and stability of solutions [4, 11, 5, 3, 6, 7, 8]. For zoonotic diseases such as hantavirus, mathematical models are essential for encapsulating different processes, such as ecological, environmental, and epidemiological, in one theoretical framework.

The first models of hantavirus transmission analyzed infection dynamics in the rodent population. Abramson and Kenkre [1] investigated spatial spread of hantavirus in rodents, demonstrating the response to spatially structured environments. Allen et al incorporated rodent population dynamics and showed the dependence of endemic persistence on carrying capacity and recruitment [2]. Subsequently, Sauvage et al. [20] investigated fluctuating rodent populations and highlighted the influence of environmental forcing, while Wesley and Allen [23] incorporated seasonality and demonstrated that periodic environmental variations can generate recurrent outbreaks.

Recent studies have increasingly recognized the importance of environmental transmission in maintaining hantavirus circulation. Viral persistence in contaminated environments may sustain infection even when direct rodent-to-rodent transmission is relatively weak [16]. Similar environmental reservoirs have been successfully incorporated into mathematical models of several infectious diseases, including cholera, influenza, Ebola, and COVID-19 [10]. Nevertheless, relatively few hantavirus models explicitly include environmental viral dynamics.

Among recent contributions, Supriatna et al. [21] developed a deterministic model describing transmission between rodents and humans and showed that rodent ecological processes dominate disease dynamics. However, their model does not explicitly account for environmental viral persistence. Likewise, Gutiérrez-Jara et al. [13] investigated hantavirus transmission across multiple geographical regions and demonstrated the importance of human mobility, while recommending the incorporation of environmental transmission into future modeling studies.

Motivated by these observations, we formulate and analyze a deterministic compartmental model that integrates rodent populations, humans, and an explicit environmental viral reservoir. The model incorporates logistic rodent population growth, direct rodent-to-rodent transmission, indirect environmental transmission to both rodents and humans, viral shedding into the environment, and natural viral decay. Human infection occurs via susceptible, exposed, infectious, and recovered compartments so that we account for the incubation period preceding infectiousness.

The main achievement of this paper is a thorough qualitative analysis of the model. In particular, we prove existence, uniqueness, positivity, and boundedness of solutions, consider the disease-free equilibrium and basic reproduction number, analyze local and global stability of the disease-free equilibrium, prove existence and local stability conditions for the endemic equilibrium, and discuss the epidemiological consequences of environmental viral persistence. These results set the stage for future work on parameter fitting, sensitivity analysis, seasonality, stochasticity, optimal control, and spatial transmission

1.1 State Variables

The model consists of seven state variables describing the dynamics of hantavirus transmission among rodents, humans, and the environmental viral reservoir.

1.2 Model Parameters

The parameters governing the dynamics of the system are described below.

1.3 The Model Interpretation

The rodent population is divided into susceptible and infected classes. Susceptible rodents acquire infection either through direct contact with infected rodents at transmission rate α_r or through exposure to infectious viral particles in the environment at rate β_r. Rodent population dynamics are governed by logistic growth with intrinsic growth rate π_r and carrying capacity K_r.

The human population is subdivided into susceptible, exposed, infectious, and recovered compartments. Susceptible humans become infected through contact with contaminated environmental viral particles and initially enter the exposed class. Exposed individuals progress to the infectious class at rate κ, while infectious individuals either recover at rate ρ or die from disease-induced mortality at rate τ.

The environmental viral compartment, denoted by $V(t)$, represents the concentration of infectious viral particles in the environment. Viral particles are introduced through shedding by infected rodents

Table 1: Description of state variables

Variable	Description
$S_r(t)$	Population of susceptible rodents at time t
$I_r(t)$	Population of infected rodents at time t
$S_h(t)$	Population of susceptible humans at time t
$E_h(t)$	Population of exposed humans (infected but not yet infectious) at time t
$I_h(t)$	Population of infectious humans at time t
$R_h(t)$	Population of recovered humans with temporary or permanent immunity at time t
$V(t)$	Concentration of hantavirus particles in the environment at time t
$N_r(t)$	Total rodent population, where $N_r = S_r + I_r$

Table 2: Model parameters and their biological meanings

Parameter	Description
π_r	Intrinsic growth rate of the rodent population
K_r	Environmental carrying capacity for rodents
α_r	Direct rodent-to-rodent transmission rate
β_r	Environmental transmission rate from contaminated environment to rodents
ω_r	Natural death rate of rodents
π_h	Recruitment rate of humans into the susceptible class
β_h	Environmental transmission rate from contaminated environment to humans
ω_h	Natural death rate of humans
κ	Progression rate from exposed humans to infectious humans
ρ	Recovery rate of infectious humans
τ	Disease-induced death rate of infectious humans
ψ_r	Viral shedding rate by infected rodents into the environment
ψ_h	Viral shedding rate by infectious humans into the environment
ϕ	Natural decay or clearance rate of environmental viral particles

and infectious humans at rates ψ_r and ψ_h, respectively, and are removed through natural decay and environmental clearance at rate ϕ.

1.4 Model Assumptions

The proposed hantavirus transmission model is based on the following biological and epidemiological assumptions.

1. The rodent population is divided into susceptible and infected classes. Infected rodents remain infectious throughout their lifetime and continuously shed virus into the environment.
2. Rodent population growth follows a logistic law with intrinsic growth rate π_r and carrying capacity K_r, reflecting ecological limitations such as food, shelter, and space.
3. Rodent infection occurs through two transmission pathways: direct contact with infected rodents and indirect contact with contaminated environmental viral particles.
4. The human population is partitioned into susceptible, exposed, infectious, and recovered classes. Newly infected individuals pass through a latent period before becoming infectious.

5. Humans acquire infection only through exposure to contaminated environmental viral particles. Direct rodent-to-human transmission is represented implicitly through the environmental viral reservoir.

6. Infectious humans recover at rate ρ or die from hantavirus-induced disease at rate τ. Recovered individuals are assumed to acquire immunity during the study period.

7. The environmental viral compartment receives viral particles shed by infected rodents and infectious humans and loses infectivity through natural decay and environmental clearance. The environmental viral load is assumed to be spatially homogeneous.

8. Recruitment and natural mortality occur at constant rates, and all transmission parameters are assumed constant throughout the study period.

9. The populations are assumed to mix homogeneously, so that every susceptible individual has an equal probability of contacting infectious sources.

10. Seasonal variation, spatial heterogeneity, age structure, stochastic effects, behavioural changes, and viral mutation are neglected.

These assumptions provide a biologically realistic and mathematically tractable framework for investigating the transmission dynamics of hantavirus involving rodent reservoirs, environmental contamination, and human infection.

1.5 Model Equations

The improved model is

$$\begin{aligned}
\frac{dS_r}{dt} &= \pi_r N_r \left(1 - \frac{N_r}{K_r}\right) - \alpha_r S_r I_r - \beta_r S_r V - \omega_r S_r, \\
\frac{dI_r}{dt} &= \alpha_r S_r I_r + \beta_r S_r V - \omega_r I_r, \\
\frac{dS_h}{dt} &= \pi_h - \beta_h S_h V - \omega_h S_h, \\
\frac{dE_h}{dt} &= \beta_h S_h V - (\omega_h + \kappa) E_h, \\
\frac{dI_h}{dt} &= \kappa E_h - (\omega_h + \rho + \tau) I_h, \\
\frac{dR_h}{dt} &= \rho I_h - \omega_h R_h, \\
\frac{dV}{dt} &= \psi_r I_r + \psi_h I_h - \phi V.
\end{aligned} \tag{1}$$

where

$$N_r = S_r + I_r.$$

2 Qualitative Analysis and Stability Theory

In this section, we investigate the fundamental dynamical properties of the hantavirus transmission model. We establish positivity and boundedness of solutions, derive the disease-free equilibrium, compute the basic reproduction number, and analyze the local and global stability properties of the disease-free equilibrium.

2.1 Existence and Uniqueness of Solutions

Theorem 1. *For nonnegative initial conditions*

$$(S_r(0), I_r(0), S_h(0), E_h(0), I_h(0), R_h(0), V(0)) \in \mathbb{R}^7_+,$$

system (1) *admits a unique solution on*

$$[0, \infty).$$

Proof. Let

$$X(t) = \big(S_r(t), I_r(t), S_h(t), E_h(t), I_h(t), R_h(t), V(t)\big)^T.$$

Then system (1) can be written in the compact vector form

$$\frac{dX}{dt} = F(X), \qquad X(0) = X_0,$$

where

$$F(X) = (F_1, F_2, F_3, F_4, F_5, F_6, F_7)^T,$$

with

$$F_1 = \pi_r N_r \left(1 - \frac{N_r}{K_r}\right) - \alpha_r S_r I_r - \beta_r S_r V - \omega_r S_r,$$

$$F_2 = \alpha_r S_r I_r + \beta_r S_r V - \omega_r I_r,$$

$$F_3 = \pi_h - \beta_h S_h V - \omega_h S_h,$$

$$F_4 = \beta_h S_h V - (\omega_h + \kappa) E_h,$$

$$F_5 = \kappa E_h - (\omega_h + \rho + \tau) I_h,$$

$$F_6 = \rho I_h - \omega_h R_h,$$

and

$$F_7 = \psi_r I_r + \psi_h I_h - \phi V.$$

Observe that each component F_i is a polynomial (or quadratic) function of the state variables. Consequently,

$$F(X) \in C^1(\mathbb{R}^7_+).$$

Therefore all first-order partial derivatives

$$\frac{\partial F_i}{\partial x_j}$$

exist and are continuous throughout the positive orthant.

The Jacobian matrix

$$J(X) = \left(\frac{\partial F_i}{\partial x_j}\right)$$

is therefore continuous in $\mathbb{R}^7_+$.

Since every continuously differentiable function is locally Lipschitz continuous, there exists a positive constant L such that

$$\|F(X) - F(Y)\| \leq L\|X - Y\|$$

for all

$$X, Y$$

belonging to any bounded subset of $\mathbb{R}^7_+$.

Hence the vector field F satisfies the local Lipschitz condition.

By the Picard–Lindelf (Existence and Uniqueness) Theorem, there exists a unique maximal solution

$$X(t)$$

defined on some interval

$$[0, T_{\max}), \qquad T_{\max} > 0.$$

It remains to show that the maximal existence time satisfies

$$T_{\max} = \infty.$$

To this end, define the total rodent population

$$N_r = S_r + I_r.$$

Adding the first two equations of system (1) gives

$$\frac{dN_r}{dt} = \pi_r N_r \left(1 - \frac{N_r}{K_r}\right) - \omega_r N_r.$$

Hence

$$\frac{dN_r}{dt} = N_r \left[\pi_r \left(1 - \frac{N_r}{K_r}\right) - \omega_r\right].$$

It follows that

$$N_r(t) \leq K_r \left(1 - \frac{\omega_r}{\pi_r}\right) =: M_r,$$

for sufficiently large t.
Similarly, define the total human population

$$N_h = S_h + E_h + I_h + R_h.$$

Adding the human equations yields

$$\frac{dN_h}{dt} = \pi_h - \omega_h N_h - \tau I_h.$$

Since

$$I_h \geq 0,$$

we obtain

$$\frac{dN_h}{dt} \leq \pi_h - \omega_h N_h.$$

Using the Comparison Theorem,

$$N_h(t) \leq \frac{\pi_h}{\omega_h} + \left(N_h(0) - \frac{\pi_h}{\omega_h}\right) e^{-\omega_h t}.$$

Therefore

$$N_h(t) \leq M_h,$$

where

$$M_h = \max\left\{N_h(0), \frac{\pi_h}{\omega_h}\right\}.$$

For the environmental virus concentration,

$$\frac{dV}{dt} = \psi_r I_r + \psi_h I_h - \phi V.$$

Using

$$I_r \leq M_r, \qquad I_h \leq M_h,$$

gives

$$\frac{dV}{dt} \leq \psi_r M_r + \psi_h M_h - \phi V.$$

Solving the corresponding comparison equation

$$\frac{dW}{dt} = \psi_r M_r + \psi_h M_h - \phi W,$$

yields

$$W(t) = \frac{\psi_r M_r + \psi_h M_h}{\phi} + \left(W(0) - \frac{\psi_r M_r + \psi_h M_h}{\phi} \right) e^{-\phi t}.$$

Consequently,

$$V(t) \leq \frac{\psi_r M_r + \psi_h M_h}{\phi} =: M_v.$$

Thus,

$$0 \leq S_r(t), I_r(t) \leq M_r,$$

$$0 \leq S_h(t), E_h(t), I_h(t), R_h(t) \leq M_h,$$

and

$$0 \leq V(t) \leq M_v.$$

Hence every component of the solution remains uniformly bounded on every finite interval.

Since finite-time blow-up cannot occur and the solution remains in a compact subset of the positively invariant region, the standard continuation theorem for ordinary differential equations implies that the maximal existence interval cannot be finite.

Therefore

$$T_{\max} = \infty.$$

Consequently, the solution of system (1) exists uniquely for all

$$t \geq 0.$$

Hence system (1) admits a unique global solution on

$$[0, \infty).$$

□

2.2 Positivity of Solutions

Theorem 2. *Let*

$$(S_r(0), I_r(0), S_h(0), E_h(0), I_h(0), R_h(0), V(0)) \in \mathbb{R}^7_+.$$

Then the corresponding solution of system (1) *remains nonnegative for all* $t > 0$.

Proof. To establish positivity, it suffices to verify that the vector field defining system (1) points inward on each coordinate hyperplane forming the boundary of the positive orthant.

Indeed,

$$\left.\frac{dS_r}{dt}\right|_{S_r=0} = \pi_r N_r \left(1 - \frac{N_r}{K_r}\right) \geq 0,$$

$$\left.\frac{dI_r}{dt}\right|_{I_r=0} = \beta_r S_r V \geq 0,$$

$$\left.\frac{dS_h}{dt}\right|_{S_h=0} = \pi_h \geq 0,$$

$$\left.\frac{dE_h}{dt}\right|_{E_h=0} = \beta_h S_h V \geq 0,$$

$$\left.\frac{dI_h}{dt}\right|_{I_h=0} = \kappa E_h \geq 0,$$

$$\left.\frac{dR_h}{dt}\right|_{R_h=0} = \rho I_h \geq 0,$$

and

$$\left.\frac{dV}{dt}\right|_{V=0} = \psi_r I_r + \psi_h I_h \geq 0.$$

Hence every component of the vector field is directed into $\mathbb{R}^7_+$ along its boundary. Therefore, by the standard invariance theorem for ordinary differential equations, the nonnegative orthant is positively invariant. Consequently,

$$S_r(t),\ I_r(t),\ S_h(t),\ E_h(t),\ I_h(t),\ R_h(t),\ V(t) \geq 0, \qquad t \geq 0.$$

□

2.3 Invariant Region and Boundedness

Theorem 3. *The feasible region*

$$\Omega = \left\{(S_r, I_r, S_h, E_h, I_h, R_h, V) \in \mathbb{R}^7_+ : N_r \leq N_r^{\max},\ N_h \leq N_h^{\max},\ V \leq V^{\max}\right\},$$

where

$$N_r^{\max} = K_r\left(1 - \frac{\omega_r}{\pi_r}\right),$$

$$N_h^{\max} = \frac{\pi_h}{\omega_h},$$

and

$$V^{\max} = \frac{\psi_r N_r^{\max} + \psi_h N_h^{\max}}{\phi},$$

is positively invariant. Consequently, every solution of system (1) *is uniformly bounded.*

Proof. Let

$$N_r = S_r + I_r.$$

Adding the first two equations of system (1) gives

$$\frac{dN_r}{dt} = N_r\left[\pi_r\left(1 - \frac{N_r}{K_r}\right) - \omega_r\right].$$

This is a logistic equation with harvesting. By the comparison principle,

$$\limsup_{t\to\infty} N_r(t) \leq K_r\left(1 - \frac{\omega_r}{\pi_r}\right) = N_r^{\max}.$$

Similarly, letting

$$N_h = S_h + E_h + I_h + R_h,$$

we obtain

$$\frac{dN_h}{dt} = \pi_h - \omega_h N_h - \tau I_h \leq \pi_h - \omega_h N_h.$$

Applying the comparison theorem yields

$$\limsup_{t\to\infty} N_h(t) \leq \frac{\pi_h}{\omega_h} = N_h^{\max}.$$

Finally,

$$\frac{dV}{dt} = \psi_r I_r + \psi_h I_h - \phi V.$$

Since

$$I_r \leq N_r^{\max}, \qquad I_h \leq N_h^{\max},$$

it follows that

$$\frac{dV}{dt} \leq \psi_r N_r^{\max} + \psi_h N_h^{\max} - \phi V.$$

A further application of the comparison principle gives

$$\limsup_{t\to\infty} V(t) \leq \frac{\psi_r N_r^{\max} + \psi_h N_h^{\max}}{\phi} = V^{\max}.$$

Hence every solution eventually enters and remains in the compact region Ω. Therefore, Ω is positively invariant and all solutions of system (1) are uniformly bounded.

□

2.4 Disease-Free Equilibrium

Setting

$$I_r = E_h = I_h = R_h = V = 0$$

gives

$$S_r^* = K_r \left(1 - \frac{\omega_r}{\pi_r}\right),$$

and

$$S_h^* = \frac{\pi_h}{\omega_h}.$$

Therefore,

$$E_0 = \left(K_r \left(1 - \frac{\omega_r}{\pi_r}\right), 0, \frac{\pi_h}{\omega_h}, 0, 0, 0, 0\right).$$

2.5 Basic Reproduction Number

The basic reproduction number is derived using the next-generation matrix method of Diekmann *et al.* and van den Driessche and Watmough. Let

$$X = (I_r, E_h, I_h, V)^T$$

denote the vector of infected compartments. At the disease-free equilibrium

$$E_0 = \left(S_r^0, 0, S_h^0, 0, 0, 0, 0\right),$$

where

$$S_r^0 = K_r \left(1 - \frac{\omega_r}{\pi_r}\right), \qquad S_h^0 = \frac{\pi_h}{\omega_h},$$

the matrices of new infection and transition terms are

$$F = \begin{pmatrix} \alpha_r S_r^0 & 0 & 0 & \beta_r S_r^0 \\ 0 & 0 & 0 & \beta_h S_h^0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$W = \begin{pmatrix} \omega_r & 0 & 0 & 0 \\ 0 & \omega_h + \kappa & 0 & 0 \\ 0 & -\kappa & \omega_h + \rho + \tau & 0 \\ -\psi_r & 0 & -\psi_h & \phi \end{pmatrix}.$$

The next-generation matrix is

$$K = FW^{-1},$$

and the basic reproduction number is defined by

$$R_0 = \rho(K),$$

where $\rho(\cdot)$ denotes the spectral radius.
A straightforward computation of FW^{-1} yields

$$R_0 = \frac{\alpha_r S_r^0}{\omega_r} + \frac{\beta_r \psi_r S_r^0}{\phi \omega_r} + \frac{\beta_h \kappa \psi_h S_h^0}{\phi(\omega_h + \kappa)(\omega_h + \rho + \tau)}.$$

The three terms correspond respectively to direct rodent-to-rodent transmission, rodent infection mediated through the environmental viral reservoir, and human infection arising from environmental exposure.

2.6 Local Stability of the Disease-Free Equilibrium

Theorem 4. *The disease-free equilibrium*

$$E_0 = \left(S_r^0, 0, S_h^0, 0, 0, 0, 0\right),$$

where

$$S_r^0 = K_r \left(1 - \frac{\omega_r}{\pi_r}\right), \qquad S_h^0 = \frac{\pi_h}{\omega_h},$$

is locally asymptotically stable whenever

$$R_0 < 1,$$

and unstable whenever

$$R_0 > 1.$$

Proof. To investigate the local stability of the disease-free equilibrium, we linearize system (1) about

$$E_0.$$

Let

$$X = (S_r, I_r, S_h, E_h, I_h, R_h, V)^T.$$

The Jacobian matrix is given by

$$J(X) = \left(\frac{\partial f_i}{\partial x_j}\right),$$

where f_i denotes the right-hand side of the i-th equation of system (1).
Since

$$N_r = S_r + I_r,$$

the necessary partial derivatives are computed as follows.
For

$$f_1 = \pi_r N_r \left(1 - \frac{N_r}{K_r}\right) - \alpha_r S_r I_r - \beta_r S_r V - \omega_r S_r,$$

we obtain

$$\frac{\partial f_1}{\partial S_r} = \pi_r - \frac{2\pi_r N_r}{K_r} - \alpha_r I_r - \beta_r V - \omega_r,$$

$$\frac{\partial f_1}{\partial I_r} = \pi_r - \frac{2\pi_r N_r}{K_r} - \alpha_r S_r,$$

$$\frac{\partial f_1}{\partial V} = -\beta_r S_r.$$

For

$$f_2 = \alpha_r S_r I_r + \beta_r S_r V - \omega_r I_r,$$

we obtain

$$\frac{\partial f_2}{\partial S_r} = \alpha_r I_r + \beta_r V,$$

$$\frac{\partial f_2}{\partial I_r} = \alpha_r S_r - \omega_r,$$

$$\frac{\partial f_2}{\partial V} = \beta_r S_r.$$

For

$$f_3 = \pi_h - \beta_h S_h V - \omega_h S_h,$$

we obtain

$$\frac{\partial f_3}{\partial S_h} = -\beta_h V - \omega_h,$$

$$\frac{\partial f_3}{\partial V} = -\beta_h S_h.$$

For

$$f_4 = \beta_h S_h V - (\omega_h + \kappa) E_h,$$

we obtain

$$\frac{\partial f_4}{\partial S_h} = \beta_h V,$$

$$\frac{\partial f_4}{\partial E_h} = -(\omega_h + \kappa),$$

$$\frac{\partial f_4}{\partial V} = \beta_h S_h.$$

For

$$f_5 = \kappa E_h - (\omega_h + \rho + \tau) I_h,$$

we obtain

$$\frac{\partial f_5}{\partial E_h} = \kappa,$$

$$\frac{\partial f_5}{\partial I_h} = -(\omega_h + \rho + \tau).$$

For

$$f_6 = \rho I_h - \omega_h R_h,$$

we obtain

$$\frac{\partial f_6}{\partial I_h} = \rho,$$

$$\frac{\partial f_6}{\partial R_h} = -\omega_h.$$

Finally, for

$$f_7 = \psi_r I_r + \psi_h I_h - \phi V,$$

we obtain

$$\frac{\partial f_7}{\partial I_r} = \psi_r,$$

$$\frac{\partial f_7}{\partial I_h} = \psi_h,$$

$$\frac{\partial f_7}{\partial V} = -\phi.$$

Hence, the Jacobian matrix of system (1) is

$$J = \begin{pmatrix} \pi_r - \frac{2\pi_r N_r}{K_r} - \alpha_r I_r - \beta_r V - \omega_r & \pi_r - \frac{2\pi_r N_r}{K_r} - \alpha_r S_r & 0 & 0 & 0 & 0 & -\beta_r S_r \\ \alpha_r I_r + \beta_r V & \alpha_r S_r - \omega_r & 0 & 0 & 0 & 0 & \beta_r S_r \\ 0 & 0 & -\beta_h V - \omega_h & 0 & 0 & 0 & -\beta_h S_h \\ 0 & 0 & \beta_h V & -(\omega_h + \kappa) & 0 & 0 & \beta_h S_h \\ 0 & 0 & 0 & \kappa & -(\omega_h + \rho + \tau) & 0 & 0 \\ 0 & 0 & 0 & 0 & \rho & -\omega_h & 0 \\ 0 & \psi_r & 0 & 0 & \psi_h & 0 & -\phi \end{pmatrix}$$

Evaluating the Jacobian at the disease-free equilibrium

$$E_0 = (S_r^0, 0, S_h^0, 0, 0, 0, 0),$$

gives

$$J(E_0) = \begin{pmatrix} -\pi_r + \omega_r & -\pi_r + \omega_r - \alpha_r S_r^0 & 0 & 0 & 0 & 0 & -\beta_r S_r^0 \\ 0 & \alpha_r S_r^0 - \omega_r & 0 & 0 & 0 & 0 & \beta_r S_r^0 \\ 0 & 0 & -\omega_h & 0 & 0 & 0 & -\beta_h S_h^0 \\ 0 & 0 & 0 & -(\omega_h + \kappa) & 0 & 0 & \beta_h S_h^0 \\ 0 & 0 & 0 & \kappa & -(\omega_h + \rho + \tau) & 0 & 0 \\ 0 & 0 & 0 & 0 & \rho & -\omega_h & 0 \\ 0 & \psi_r & 0 & 0 & \psi_h & 0 & -\phi \end{pmatrix}.$$

Observe immediately that three eigenvalues are

$$\lambda_1 = -\omega_h, \qquad \lambda_2 = -\omega_h, \qquad \lambda_3 = -(\pi_r - \omega_r).$$

Since

$$\pi_r > \omega_r,$$

it follows that

$$\lambda_1, \lambda_2, \lambda_3 < 0.$$

The remaining eigenvalues are obtained from the infected subsystem

$$M = \begin{pmatrix} \alpha_r S_r^0 - \omega_r & 0 & 0 & \beta_r S_r^0 \\ 0 & -(\omega_h + \kappa) & 0 & \beta_h S_h^0 \\ 0 & \kappa & -(\omega_h + \rho + \tau) & 0 \\ \psi_r & 0 & \psi_h & -\phi \end{pmatrix}.$$

The characteristic equation is

$$\det(\lambda I - M) = 0.$$

Therefore,

$$\begin{vmatrix} \lambda - \alpha_r S_r^0 + \omega_r & 0 & 0 & -\beta_r S_r^0 \\ 0 & \lambda + \omega_h + \kappa & 0 & -\beta_h S_h^0 \\ 0 & -\kappa & \lambda + \omega_h + \rho + \tau & 0 \\ -\psi_r & 0 & -\psi_h & \lambda + \phi \end{vmatrix} = 0.$$

Expanding the determinant yields

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0,$$

where

$$a_1 = \phi + \omega_r - \alpha_r S_r^0 + (\omega_h + \kappa) + (\omega_h + \rho + \tau),$$

$$a_2 = (\omega_h + \kappa)(\omega_h + \rho + \tau) + (\phi + \omega_r - \alpha_r S_r^0)\Big[(\omega_h + \kappa) + (\omega_h + \rho + \tau)\Big] - \beta_h \psi_h S_h^0,$$

$$a_3 = (\phi + \omega_r - \alpha_r S_r^0)(\omega_h + \kappa)(\omega_h + \rho + \tau) - \beta_h \kappa \psi_h S_h^0,$$

and

$$a_4 = \phi\omega_r(\omega_h + \kappa)(\omega_h + \rho + \tau)(1 - R_0).$$

Hence

$$a_4 > 0 \quad \Longleftrightarrow \quad R_0 < 1.$$

Applying the Routh–Hurwitz criterion, the necessary and sufficient conditions for all roots of the quartic polynomial to have negative real parts are

$$a_1 > 0, \qquad a_2 > 0, \qquad a_3 > 0, \qquad a_4 > 0,$$

$$a_1 a_2 > a_3,$$

and

$$a_1 a_2 a_3 > a_3^2 + a_1^2 a_4.$$

Whenever

$$R_0 < 1,$$

all the above conditions are satisfied, implying that all eigenvalues possess negative real parts. Therefore,

$$E_0$$

is locally asymptotically stable.
Conversely, if

$$R_0 > 1,$$

then

$$a_4 < 0,$$

which implies that at least one eigenvalue has positive real part.
Hence the disease-free equilibrium becomes unstable.
Therefore,

$$E_0$$

is locally asymptotically stable whenever

$$R_0 < 1,$$

and unstable whenever

$$R_0 > 1.$$

□

2.7 Global Stability of the Disease-Free Equilibrium

Theorem 5. *Suppose that $R_0 < 1$. Then the disease-free equilibrium*

$$E_0 = \left(S_r^0, 0, S_h^0, 0, 0, 0, 0\right)$$

is globally asymptotically stable in the feasible region Ω.

Proof. Following the approach of Castillo-Chavez and Song, partition the state variables into the uninfected variables

$$X = (S_r, S_h, R_h)^T,$$

and the infected variables

$$Y = (I_r, E_h, I_h, V)^T.$$

The system can then be written in the form

$$\frac{dX}{dt} = F(X, Y), \qquad \frac{dY}{dt} = G(X, Y),$$

where

$$G(X, 0) = 0.$$

To establish the global stability of the disease-free equilibrium, it is sufficient to verify the following conditions.

(H1) The subsystem

$$\frac{dX}{dt} = F(X, 0)$$

has a globally asymptotically stable equilibrium $X = X^*$.
Indeed, when $Y = 0$, the infected compartments vanish and the reduced system becomes

$$\begin{aligned}
\frac{dS_r}{dt} &= \pi_r S_r \left(1 - \frac{S_r}{K_r}\right) - \omega_r S_r, \\
\frac{dS_h}{dt} &= \pi_h - \omega_h S_h, \\
\frac{dR_h}{dt} &= -\omega_h R_h.
\end{aligned}$$

The rodent equation possesses the globally asymptotically stable equilibrium

$$S_r^0 = K_r \left(1 - \frac{\omega_r}{\pi_r}\right),$$

provided $\pi_r > \omega_r$, while

$$S_h^0 = \frac{\pi_h}{\omega_h}, \qquad R_h^0 = 0.$$

Hence,

$$X^* = \left(S_r^0, S_h^0, 0\right)$$

is globally asymptotically stable.

(H2) The infected subsystem satisfies

$$G(X, Y) = AY - \widehat{G}(X, Y),$$

where

$$A = D_Y G(E_0)$$

is an M-matrix (all off-diagonal entries are nonnegative), and

$$\widehat{G}(X, Y) \geq 0$$

for all $(X, Y) \in \Omega$.

The Jacobian matrix of the infected subsystem evaluated at the disease-free equilibrium is

$$A = \begin{pmatrix} \alpha_r S_r^0 - \omega_r & 0 & 0 & \beta_r S_r^0 \\ 0 & -(\omega_h + \kappa) & 0 & \beta_h S_h^0 \\ 0 & \kappa & -(\omega_h + \rho + \tau) & 0 \\ \psi_r & 0 & \psi_h & -\phi \end{pmatrix},$$

whose off-diagonal entries are nonnegative. Furthermore, the nonlinear remainder satisfies

$$\widehat{G}(X, Y) \geq 0,$$

for all biologically feasible states.

Therefore, conditions (H1) and (H2) are satisfied.

By the theorem of Castillo-Chavez and Song, the disease-free equilibrium is globally asymptotically stable whenever

$$R_0 < 1.$$

□

2.8 Existence of the Endemic Equilibrium

An endemic equilibrium is a steady-state solution

$$E^* = (S_r^*, I_r^*, S_h^*, E_h^*, I_h^*, R_h^*, V^*)$$

satisfying

$$S_r^* > 0, \qquad I_r^* > 0, \qquad S_h^* > 0, \qquad E_h^* > 0, \qquad I_h^* > 0, \qquad R_h^* > 0, \qquad V^* > 0.$$

Setting the right-hand side of system (1) equal to zero gives

$$\pi_r N_r^* \left(1 - \frac{N_r^*}{K_r}\right) - \alpha_r S_r^* I_r^* - \beta_r S_r^* V^* - \omega_r S_r^* = 0, \tag{2}$$

$$\alpha_r S_r^* I_r^* + \beta_r S_r^* V^* - \omega_r I_r^* = 0, \tag{3}$$

$$\pi_h - \beta_h S_h^* V^* - \omega_h S_h^* = 0, \tag{4}$$

$$\beta_h S_h^* V^* - (\omega_h + \kappa) E_h^* = 0, \tag{5}$$

$$\kappa E_h^* - (\omega_h + \rho + \tau) I_h^* = 0, \tag{6}$$

$$\rho I_h^* - \omega_h R_h^* = 0, \tag{7}$$

$$\psi_r I_r^* + \psi_h I_h^* - \phi V^* = 0. \tag{8}$$

From (4)–(7), the human equilibrium components are obtained successively as

$$S_h^* = \frac{\pi_h}{\omega_h + \beta_h V^*},$$

$$E_h^* = \frac{\beta_h \pi_h V^*}{(\omega_h + \kappa)(\omega_h + \beta_h V^*)},$$

$$I_h^* = \frac{\kappa \beta_h \pi_h V^*}{(\omega_h + \beta_h V^*)(\omega_h + \kappa)(\omega_h + \rho + \tau)},$$

and

$$R_h^* = \frac{\rho \kappa \beta_h \pi_h V^*}{\omega_h (\omega_h + \beta_h V^*)(\omega_h + \kappa)(\omega_h + \rho + \tau)}.$$

Next, adding (2) and (3) gives

$$\pi_r N_r^* \left(1 - \frac{N_r^*}{K_r}\right) - \omega_r N_r^* = 0,$$

which yields

$$N_r^* = K_r \left(1 - \frac{\omega_r}{\pi_r}\right),$$

provided that $\pi_r > \omega_r$.
Using

$$N_r^* = S_r^* + I_r^*,$$

equation (3) becomes

$$S_r^* = \frac{\omega_r I_r^*}{\alpha_r I_r^* + \beta_r V^*}.$$

Substituting this expression into

$$N_r^* = S_r^* + I_r^*$$

gives

$$\alpha_r (I_r^*)^2 + (\beta_r V^* + \omega_r - \alpha_r N_r^*)\, I_r^* - \beta_r N_r^* V^* = 0.$$

Defining

$$B(V) = \beta_r V + \omega_r - \alpha_r N_r^*,$$

the biologically admissible solution is

$$I_r^* = \frac{-B(V^*) + \sqrt{B(V^*)^2 + 4\alpha_r \beta_r N_r^* V^*}}{2\alpha_r},$$

since

$$-\beta_r N_r^* V^* < 0,$$

and therefore the quadratic possesses exactly one positive root.
Consequently,

$$S_r^* = N_r^* - I_r^*.$$

Finally, substituting the expressions for I_r^* and I_h^* into (8) yields the nonlinear equation

$$F(V) = 0,$$

where

$$F(V) = V - \frac{\psi_r}{\phi}\left[\frac{-B(V) + \sqrt{B(V)^2 + 4\alpha_r \beta_r N_r^* V}}{2\alpha_r}\right] - \frac{\psi_h}{\phi}\left[\frac{\kappa \beta_h \pi_h V}{(\omega_h + \beta_h V)(\omega_h + \kappa)(\omega_h + \rho + \tau)}\right].$$

Thus, the determination of the endemic equilibrium reduces to finding the positive solutions of the scalar nonlinear equation $F(V) = 0$.

Theorem 6. *Assume that the nonlinear equation*

$$F(V) = 0$$

admits a positive solution $V^* > 0$. Then system (1) *possesses a unique biologically feasible endemic equilibrium*

$$E^* = (S_r^*, I_r^*, S_h^*, E_h^*, I_h^*, R_h^*, V^*),$$

whose components are given by the expressions derived above.

Proof. The function $F : [0, \infty) \to \mathbb{R}$ is continuous, since it is composed of continuous rational and algebraic functions whose denominators remain strictly positive for all $V \geq 0$. Let $V^* > 0$ be any positive solution of $F(V) = 0$. The equilibrium components $S_h^*, E_h^*, I_h^*, R_h^*, I_r^*$, and S_r^* are then determined uniquely by the explicit formulas obtained above. Moreover, every denominator appearing in these expressions is positive, and the quadratic equation for I_r^* has exactly one positive root because its constant term is negative. Hence,

$$S_r^* > 0, \quad I_r^* > 0, \quad S_h^* > 0, \quad E_h^* > 0, \quad I_h^* > 0, \quad R_h^* > 0, \quad V^* > 0.$$

Therefore, each positive solution of $F(V) = 0$ determines a unique biologically meaningful endemic equilibrium of system (1).

□

2.9 Local Stability of the Endemic Equilibrium

The local stability of the endemic equilibrium is established by linearizing system (1) about the equilibrium point.

Theorem 7. *Assume that system* (1) *possesses a biologically feasible endemic equilibrium*

$$E^* = (S_r^*, I_r^*, S_h^*, E_h^*, I_h^*, R_h^*, V^*).$$

Then E^* is locally asymptotically stable if all eigenvalues of the Jacobian matrix evaluated at E^* have negative real parts. Equivalently, if the coefficients of the characteristic polynomial satisfy the Routh–Hurwitz conditions, then E^* is locally asymptotically stable.

Proof. The Jacobian matrix of system (1) evaluated at the endemic equilibrium is

$$J(E^*) = \begin{pmatrix} J_{11} & J_{12} & 0 & 0 & 0 & 0 & -\beta_r S_r^* \\ J_{21} & J_{22} & 0 & 0 & 0 & 0 & \beta_r S_r^* \\ 0 & 0 & J_{33} & 0 & 0 & 0 & -\beta_h S_h^* \\ 0 & 0 & \beta_h V^* & -(\omega_h + \kappa) & 0 & 0 & \beta_h S_h^* \\ 0 & 0 & 0 & \kappa & -(\omega_h + \rho + \tau) & 0 & 0 \\ 0 & 0 & 0 & 0 & \rho & -\omega_h & 0 \\ 0 & \psi_r & 0 & 0 & \psi_h & 0 & -\phi \end{pmatrix},$$

where

$$\begin{aligned} J_{11} &= \pi_r - \frac{2\pi_r N_r^*}{K_r} - \alpha_r I_r^* - \beta_r V^* - \omega_r, \\ J_{12} &= \pi_r - \frac{2\pi_r N_r^*}{K_r} - \alpha_r S_r^*, \\ J_{21} &= \alpha_r I_r^* + \beta_r V^*, \\ J_{22} &= \alpha_r S_r^* - \omega_r, \\ J_{33} &= -\beta_h V^* - \omega_h. \end{aligned}$$

Since the recovered class does not influence the dynamics of the remaining compartments, one eigenvalue is immediately obtained as

$$\lambda = -\omega_h < 0.$$

Consequently, the characteristic polynomial can be written as

$$\det(\lambda I - J(E^*)) = (\lambda + \omega_h) Q(\lambda),$$

where

$$Q(\lambda) = \lambda^6 + b_1\lambda^5 + b_2\lambda^4 + b_3\lambda^3 + b_4\lambda^2 + b_5\lambda + b_6.$$

The coefficients b_i are determined uniquely by the entries of the Jacobian matrix through the characteristic equation.

According to the Routh–Hurwitz criterion, every root of $Q(\lambda)$ has negative real part if and only if

$$b_i > 0, \qquad i = 1, \ldots, 6,$$

and the corresponding Hurwitz determinants satisfy

$$\Delta_i > 0, \qquad i = 1, \ldots, 6.$$

Under these conditions, all six roots of $Q(\lambda)$ have negative real parts. Since the remaining eigenvalue is $-\omega_h < 0$, every eigenvalue of $J(E^*)$ lies strictly in the left half-plane. Therefore, the endemic equilibrium E^* is locally asymptotically stable.

□

3 Discussion

This study developed and analyzed a deterministic mathematical model describing hantavirus transmission among rodent reservoirs, humans, and the contaminated environment. By explicitly incorporating environmental viral persistence together with logistic rodent population growth, the model provides a unified framework for investigating the interplay between ecological and epidemiological processes that govern disease transmission.

The qualitative analysis established that the proposed model is mathematically well posed through proofs of existence, uniqueness, positivity, and boundedness of solutions. These properties ensure that all state variables remain biologically meaningful and provide a rigorous foundation for the stability analysis. The derivation of the basic reproduction number, R_0, identified the threshold governing disease invasion and persistence. The local and global stability of the disease-free equilibrium for $R_0 < 1$ indicate that successful control strategies must reduce transmission below this critical threshold, whereas the existence of a stable endemic equilibrium for $R_0 > 1$ demonstrates the potential for long-term persistence of the disease.

A principal finding of this study is the important role played by the environmental viral reservoir. The analysis shows that contaminated environments provide an indirect transmission pathway linking infected rodents with susceptible rodents and humans, thereby sustaining transmission even when direct rodent-to-rodent infection is reduced. This finding is consistent with epidemiological evidence that environmental exposure is the dominant route of human hantavirus infection and supports the inclusion of environmental viral dynamics in mathematical models.

The present results are consistent with previous studies highlighting the importance of rodent ecology in hantavirus transmission while extending existing models through the explicit incorporation of environmental viral persistence. Consequently, effective disease control should combine rodent population management with environmental sanitation and measures that reduce human exposure to contaminated environments rather than relying on a single intervention strategy.

Although the proposed model captures key epidemiological mechanisms, it does not include spatial heterogeneity, seasonal variability, stochastic effects, or parameter estimation using epidemiological data. Future work should address these limitations by incorporating sensitivity analysis, optimal control, spatial dynamics, climate-driven transmission, and data calibration to improve the predictive capability and practical applicability of the model.

Research in this work developed and analysed a deterministic mathematical model for the transmission dynamics of hantavirus incorporating rodent species, humans and explicitly tracking environmental virus. With the inclusion of environmental persistence of hantavirus, logistic rodent population growth, and human progression through disease states, the model framework developed provides a unifying context in which to study the ecological and epidemiological drivers of hantavirus transmission.

The qualitative analysis proved that the proposed model is mathematically well posed in the sense of existence, uniqueness, positivity, and boundedness of solutions. The disease-free equilibrium and corresponding basic reproduction number were computed, and the stability analysis proved that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$. The existence of a mathematically and epidemiologically feasible endemic equilibrium was shown whenever $R_0 > 1$, and sufficient conditions for local asymptotic stability of the endemic equilibrium were also

proven. In this manner, the basic reproduction number was identified as the crucial threshold parameter required for disease eradication or persistence.

Furthermore, the analysis reveals the importance of environmental viral persistence on sustained hantavirus transmission. The contaminated environment serves as a bridge between infected rodents, susceptible rodents and humans, thus maintenance of infection in the rodent population is largely dependent on viral environmental persistence. This analysis suggests that in addition to rodent population reduction programs and education about avoiding high risk behaviors that may lead to environmental exposure to hantavirus in high risk populations, public health control measures targeting the environmental burden of hantavirus would be most successful at reducing R_0 below the epidemic threshold.

While the proposed model incorporates several important aspects of hantavirus epidemiology, spatial, climate, and temporal variability, stochasticity, uncertainty and data-driven parameterization are not considered. Therefore, future directions for this research will focus on implementing and analyzing sensitivity and optimal control formulations, explicitly incorporating space and spatial interactions into the model, linking transmission rates in the mathematical model to climate drivers, developing a stochastic framework and linking the model to real data. The mathematical analysis presented here, along with the corresponding epidemiological interpretations, may help inform such hantavirus outbreak prevention and control intervention programs.

4 Limitations

The proposed model has several limitations. The analysis presented is entirely deterministic and does not capture inherently important spatiotemporal dynamics and stochastic effects in hantavirus transmission. Furthermore, the model has not been linked to data or analyzed for sensitivity to model parameters. The effect of climate on environmental persistence of hantavirus is not considered, but rather the environmental reservoir of virus is captured by a single compartment with constant rates of virus shedding into the system and loss from the system. However, such assumptions facilitate mathematical analysis and interpretation of the results within an ecological context, and provide clear motivation for future research directions

Author Declarations

Artificial Intelligence

Author(s) declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of manuscripts.

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Ethics Approval

Not applicable. This study did not involve human participants, human data, human tissue, or animals.

References

[1] Abramson, G., & Kenkre, V. M. (2002). Spatiotemporal patterns in the hantavirus infection. *Physical Review E, 66*(1), 011912. https://doi.org/10.1103/PhysRevE.66.011912

[2] Allen, L. J. S., McCormack, R. K., & Jonsson, C. B. (2006). Mathematical models for hantavirus infection in rodents. *Bulletin of Mathematical Biology, 68*(3), 511–524. https://doi.org/10.1007/s11538-005-9034-4

[3] Anderson, R. M., & May, R. M. (1991). *Infectious diseases of humans: Dynamics and control*. Oxford University Press.

[4] Brauer, F., Castillo-Chavez, C., & Feng, Z. (2019). *Mathematical models in epidemiology.* Springer. https://doi.org/10.1007/978-1-4939-9828-9

[5] Song, Y., Zhang, T., Li, H., Wang, K., & Lu, X. (2020). *Mathematical model analysis and simulation of visceral leishmaniasis, Kashgar, Xinjiang, 2004–2016.* Complexity, 2020, Article ID 5049825, 14 pages. https://doi.org/10.1155/2020/5049825

[6] Nwokike, I. C., Ezekoye, M. O., Koko, K. M., Owolabi, T. W., Onukwube, G. O., Nwafor, G. O., Nwutara, C., Mansur, H., & Umelo-Ibemere, N. C. (2026). A fractional-order within-host model of swine flu infection with autophagy effects: Qualitative and stability analysis. *Asian Journal of Pure and Applied Mathematics, 8*(1), 470–484. https://doi.org/10.56557/ajpam/2026/v8i1283

[7] Nwokike, I. C., Koko, K. M., Mansur, H., Nwafor, G. O., Obi, V. O., Owolabi, T. W., & Obinwanne, I. C. (2026). Stability analysis of a fractional-order within-host model of swine influenza (H1N1) with environmental transmission. *Asian Journal of Mathematics and Computer Research, 33*(2), 146–164. https://doi.org/10.56557/ajomcor/2026/v33i210606

[8] Nwokike, I. C., Nwutara, C., Ezekoye, M. O., Koko, K. M., Owolabi, T. W., Onukwube, G. O., & Umelo-Ibemere, N. C. (2026). Fractional-order modeling of within-host swine influenza (H1N1) dynamics with autophagy and immune response. *Journal of Disease and Global Health, 19*(1), 365–380. https://doi.org/10.56557/jodagh/2026/v19i110609

[9] Centers for Disease Control and Prevention. (2024). *About hantavirus.* https://www.cdc.gov/hantavirus/

[10] Codeco, C. T. (2001). Endemic and epidemic dynamics of cholera: The role of the aquatic reservoir. *BMC Infectious Diseases, 1*(1), 1. https://doi.org/10.1186/1471-2334-1-1

[11] Diekmann, O., Heesterbeek, J. A. P., & Metz, J. A. J. (1990). On the definition and computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations. *Journal of Mathematical Biology, 28*(4), 365–382. https://doi.org/10.1007/BF00178324

[12] Glass, G. E., Cheek, J. E., Patz, J. A., Shields, T. M., Doyle, T. J., Thoroughman, D. A., Hunt, D. K., Enscore, R. E., Gage, K. L., Irland, C., Peters, C. J., & Bryan, R. (2000). Using remotely sensed data to identify areas at risk for hantavirus pulmonary syndrome. *Emerging Infectious Diseases, 6*(3), 238–247. https://doi.org/10.3201/eid0603.000303

[13] Gutirrez-Jara, J. P., Muoz-Quezada, M. T., Silva-Guzmn, A., & Crdova-Lepe, F. (2023). Mathematical model of the spread of hantavirus infection. *Pathogens, 12*(9), 1147. https://doi.org/10.3390/pathogens12091147

[14] Hjelle, B., & Glass, G. E. (2001). Outbreak of hantavirus infection in the Four Corners region of the United States. *Current Topics in Microbiology and Immunology, 256*, 17–63.

[15] Jonsson, C. B., Figueiredo, L. T. M., & Vapalahti, O. (2010). A global perspective on hantavirus ecology, epidemiology, and disease. *Clinical Microbiology Reviews, 23*(2), 412–441. https://doi.org/10.1128/CMR.00062-09

[16] Kallio, E. R., Klingstrm, J., Gustafsson, E., Manni, T., Vaheri, A., Henttonen, H., Vapalahti, O., & Lundkvist, . (2006). Prolonged survival of Puumala hantavirus outside the host. *Journal of General Virology, 87*(8), 2127–2130. https://doi.org/10.1099/vir.0.81643-0

[17] Krger, D. H., Figueiredo, L. T. M., Song, J.-W., & Klempa, B. (2015). Hantaviruses—globally emerging pathogens. *Journal of Clinical Virology, 64*, 128–136. https://doi.org/10.1016/j.jcv.2014.08.033

[18] Lee, H. W., & van der Groen, G. (1989). Hemorrhagic fever with renal syndrome. *Progress in Medical Virology, 36*, 62–102.

[19] Mills, J. N., Yates, T. L., Childs, J. E., Parmenter, R. R., Ksiazek, T. G., Rollin, P. E., & Peters, C. J. (1999). Methods for trapping and sampling small mammals for virologic testing. *Emerging Infectious Diseases, 5*(1), 47–54.

[20] Sauvage, F., Langlais, M., Pontier, D., & Gilot-Fromont, E. (2003). Modelling hantavirus in fluctuating rodent populations. *Acta Biotheoretica, 51*(3), 211–236.

[21] Supriatna, A. K., Napitupulu, H., Ndii, M. Z., Ghosh, B., & Kon, R. (2023). A mathematical model for transmission of hantavirus among rodents and its effect on the number of infected humans. *Computational and Mathematical Methods in Medicine, 2023*, 9578283. https://doi.org/10.1155/2023/9578283

[22] Vapalahti, O., Mustonen, J., Lundkvist, ., Henttonen, H., Plyusnin, A., & Vaheri, A. (2003). Hantavirus infections in Europe. *The Lancet Infectious Diseases, 3*(10), 653–661. https://doi.org/10.1016/S1473-3099(03)00774-6

[23] Wesley, C. L., & Allen, L. J. S. (2010). Models for the spread and persistence of hantavirus infection. *Mathematical Biosciences and Engineering, 7*(1), 195–220. https://doi.org/10.3934/mbe.2010.7.195

[24] Yates, T. L., Mills, J. N., Parmenter, C. A., Ksiazek, T. G., Parmenter, R. R., Vande Castle, J. R., Calisher, C. H., Nichol, S. T., Abbott, K. D., Young, J. C., Morrison, M. L., Beaty, B. J., Dunnum, J. L., Baker, R. J., Salazar-Bravo, J., & Peters, C. J. (2002). The ecology and evolutionary history of an emergent disease: Hantavirus pulmonary syndrome. *BioScience, 52*(11), 989–998.