Modeling the Transmission Dynamics of Hantavirus with Environmental Viral Persistence

Abstract

Hantavirus is a zoonosis maintained in a reservoir population of rodents and transmitted to humans through contaminated environment. Elucidating the role of hosts, environment and human in disease transmission is necessary for designing an effective control strategy against hantavirus. In this paper, a mathematical model for the transmission dynamics of hantavirus with environmental viral persistence is formulated and then analysed. The model consists of susceptible and infected rodents, susceptible, exposed, infected and recovered humans, and an environmental viral compartment which accounts for indirect transmission through contaminated environment. The rodent population grows logistically. Basic dynamical properties are established: we show existence and uniqueness as well as positivity and boundedness of solutions. A compact positively invariant feasible region is derived and the disease-free equilibrium exists. The basic reproduction number R_0 is obtained using the next-generation matrix method, and is shown to be the sum of direct reproduction by horizontal transmission between rodents, horizontal transmission through environment in rodent population, and transmission from the environment to humans. We show that the disease-free equilibrium is locally as well as globally asymptotically stable whenever $R_0 < 1$ and the disease persists in the system whenever $R_0 > 1$. Existence of a biologically meaningful endemic equilibrium is proved and criteria for its stability are determined using the Routh, Hurwitz condition. The analysis shows that the persistence of virus in the environment favours the persistence of hantavirus in nature; high rates of viral release into environment by infected rodents, a high value of the natural survival time of viral particles in the environment, and efficient environmental transmission all greatly increase disease persistence, whereas high clearance of virus from the environment and few infected rodents decrease the amount of transmission. The important role of the rodent reservoir population in producing new infections is highlighted. The work indicates that the best strategy to control hantavirus infection is through controlling the rodent population size, hygiene and removal of virus from human contact and the environment. This work lays a firm mathematical foundation for future investigation of the role of seasonality, stochasticity, optimal or impulsive control, spatial spread and ecological drivers of hantavirus transmission in nature.

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

1 Introduction

Hantavirus infection is a significant zoonotic disease caused by RNA viruses belonging to the *Hantaviridae* family. It is transmitted by infected rodents and their environments, and remains one of the most important emerging viral diseases with wildlife reservoirs [8, 17]. Transmission to humans usually occurs after inhalation of aerosolized virus from rodent urine, saliva, or feces; though contact with rodent urine, saliva, and feces and rodent bites may also transmit the disease [14]. Hantavirus infections can cause severe disease in the form of Hemorrhagic Fever with Renal Syndrome (HFRS) and Hantavirus Pulmonary Syndrome (HPS) with considerable associated mortality [16].

The occurrence of hantavirus outbreaks are highly dependent on ecological and environmental conditions. Hantavirus rodent reservoir population are the main vertebrate hosts for hantavirus, and an infected rodent may carry the virus for life asymptomatically [18]. These populations are prone to rapid growth when conditions such as abundance of food, rainfall, and lack of predators are favorable; and climate variability and disturbance of habitats typically trigger changes in population density and composition [11]. One of the most famous hantavirus outbreaks was that of 1993 in the Four Corners region of the United States, and many ecologic studies of this event have been performed [13].

Beyond rodent contact, epidemiologically environmental transmission plays a large role in infection. Viral particles shed in the environment may remain infectious for hours or more depending on humidity conditions, temperature, ventilation, and exposure to UV light [15]. Humans are often infected via this transmission route, by inhalation of dust in closed environments such as farms, storage sheds, army camps, and poorly ventilated houses [21]. Environmental transmission is an important epidemiologic mechanism to include explicitly in mathematical models of hantavirus infection.

Mathematical modeling is essential for studying infectious disease dynamics and designing control strategies. Compartmental epidemic models facilitate the study of disease transmission, conditions for threshold behavior, and stability of equilibria for predictions of long-term epidemiological trends [4, 5, 10, 6, 3, 7]. In the case of zoonotic diseases like hantavirus infection, mathematical models are especially useful because the different population and environmental processes driving transmission can be considered together.

Early mathematical models of hantavirus focused on rodent reservoir dynamics. One of the first mathematical studies of hantavirus infection in general was performed by physicists Abramson and Kenkre, who studied the spatial dynamics of hantavirus infection in rodents [1]. They showed that spatial structure and regional movement of rodents can affect persistence of endemic infection, and induce different transmission dynamics in disparate locations. Several deterministic models of hantavirus transmission among rodent hosts were developed by Allen and colleagues [2]. They studied the effect of changing carrying capacity, and found that recruitment of deer mice is the driving force behind maintenance of endemic disease in wild populations.

Sauvage et al. modeled hantavirus infection in a varying population size of rodents and showed that environmental forcing has a great impact on hantavirus infection dynamics among rodents [19]. Other studies have been completed that include seasonality and stochastics in infection among mice only. Wesley and Allen studied hantavirus virus persistence in the case when parameters vary periodically over time, showing that seasonality alone can induce outbreaks of infection and oscillatory behavior [22].

More recent work has highlighted the role of environmental virus in sustaining hantavirus infection among rodent populations. Environmental transmission may maintain the presence of hantavirus in wild rodent populations even when direct rodent-to-rodent transmission is low [15].. Several groups have developed models with explicit compartments in the environment for hantavirus. This technique is commonly applied to diseases such as cholera, influenza, Ebola, and COVID-19 [9].

Supriatna et al. recently presented a differential equations-based model of hantavirus transmission in an interconnected population of mice and humans [20]. They studied its disease-free and endemic equilibria, and found that rodent ecologic processes outweigh effects of human parameters on transmission dynamics of hantavirus infection. However, this model does not include environmental transmission. Gutirrez-Jara et al. modeled hantavirus transmission in a population of humans over many territories, and found that human movement is an important factor in determining risk of infection. They also suggest that environmental transmission should be included in future studies of hantavirus dynamics to greatly enrich the results [12].

Despite all this past work, many hantavirus infection models still fail to consider environmental virus. Since hantavirus infection is so frequently transmitted via inhalation of viral particles in the environment, environmental virus may be crucial to maintaining presence of hantavirus infection in wild rodent populations. Here, we present and analyze a deterministic mathematical model for the transmission of hantavirus among the rodent reservoir population, in the environment, and among humans. It includes a population of infection-susceptible hantavirus reservoir rodents, and considers direct transmission among them. There is an explicit epidemiological compartment for environmental virus, which is shed into the environment by infected rodents, and removed via natural decay and cleaning interventions. This environmental virus is the source of infection for the susceptible human population. Exposed incubating humans progress to the symptomatic stage, and then may recover or die of disease-related causes. So, this model can capture indirect transmission to humans through infected environments, which is a serious route of infection. We focus mainly on qualitative analysis and stability theory. In particular, we aim to prove positivity and boundedness of solutions, determine the basic reproduction number, analyze local and global stability, determine conditions for persistence of infection at endemic equilibria, and study the epidemiological role of environmental virus.

Understanding these mechanisms is important for understanding hantavirus transmission and de-

signing intervention strategies in the future. Theoretical results presented herein will inform future investigations of the effects of stochastics, climate-dependent transmission, optimal control, and spatial diffusion.

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.

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 $N_r = S_r + I_r$

1.2 Model Parameters

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

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

1.3 The Model Interpretation

The rodent population is divided into susceptible and infected classes. Susceptible rodents become infected either through direct contact with infected rodents at rate α_r or through exposure to environmental viral particles at rate β_r. The rodent population grows logistically with intrinsic growth rate π_r and carrying capacity K_r, thereby incorporating density-dependent ecological regulation.

The human population is partitioned into susceptible, exposed, infectious, and recovered compartments. Humans acquire infection through contact with contaminated environmental viral particles. Newly infected individuals enter the exposed class before progressing to the infectious class at rate κ. Infectious individuals recover at rate ρ or die from the disease at rate τ.

The environmental viral compartment $V(t)$ represents the concentration of infectious hantavirus particles present in the environment. Viral particles are continuously introduced into the environment through shedding by infected rodents and infectious humans at rates ψ_r and ψ_h, respectively. The environmental viral load decreases through natural decay and environmental clearance at rate ϕ.

The inclusion of the environmental viral reservoir enables the model to capture the dominant transmission pathway of hantavirus infection, namely indirect transmission through contaminated environments. The incorporation of logistic rodent growth further improves the ecological realism of the model by accounting for resource limitations and carrying capacity effects in rodent populations.

1.4 Model Assumptions

The formulation of 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 only. Once infected, rodents remain infected throughout their lifetime and do not recover.
2. Rodent population growth follows a logistic growth law with intrinsic growth rate π_r and environmental carrying capacity K_r. This assumption reflects competition for limited resources such as food, shelter, and breeding sites.
3. Susceptible rodents become infected through two transmission pathways:
   (a) direct contact with infected rodents,
   (b) exposure to environmental viral particles.
4. Infected rodents continuously shed hantavirus particles into the environment throughout their infectious period.
5. The human population is divided into susceptible, exposed, infectious, and recovered compartments.
6. Humans acquire infection only through contact with contaminated environmental viral particles. Direct transmission from rodents to humans is represented indirectly through the environmental viral reservoir.
7. Newly infected humans first enter a latent (exposed) stage during which infection is present but individuals are not yet infectious.
8. Exposed humans progress to the infectious class at a constant rate κ.
9. Infectious humans may recover from infection at rate ρ and acquire immunity represented by the recovered class.
10. Infectious humans may also die due to hantavirus-related complications at rate τ.
11. Recovered individuals are assumed to possess immunity and do not immediately return to the susceptible class.
12. Infectious humans contribute to environmental contamination through viral shedding, although at a potentially lower rate than infected rodents.
13. The environmental viral load is represented by a single compartment $V(t)$ and serves as a reservoir of infection for both rodents and humans.
14. Environmental viral particles are continuously produced by infected rodents and infectious humans and are removed through natural viral decay, environmental degradation, and other clearance processes.

15. The environmental viral reservoir is assumed to be homogeneous, meaning that viral particles are uniformly distributed throughout the environment.
16. The transmission rates α_r, β_r, and β_h are assumed constant over the study period.
17. Natural mortality occurs in both rodent and human populations at constant rates ω_r and ω_h, respectively.
18. Recruitment of humans occurs at a constant rate π_h.
19. All state variables are assumed to remain nonnegative and continuously differentiable for all time.
20. The population is assumed to mix homogeneously, implying that every susceptible individual has an equal probability of contacting infectious sources within its respective population.
21. Seasonal variations, age structure, spatial heterogeneity, stochastic effects, behavioral changes, and genetic variations of the virus are neglected in the present model.

These assumptions provide a biologically realistic yet mathematically tractable framework for investigating the transmission dynamics of hantavirus infection 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

$$N_r = S_r + I_r.$$

1.6 Epidemiological Viewpoint of the Model

From an epidemiological perspective, the model describes the transmission dynamics of hantavirus among rodent reservoirs, the contaminated environment, and the human population. The model recognizes rodents as the primary reservoir hosts responsible for maintaining the virus within the ecosystem, while humans are considered incidental hosts who acquire infection through environmental exposure.

The susceptible rodent population, $S_r(t)$, represents healthy rodents capable of acquiring infection either through direct contact with infected rodents or through exposure to contaminated environmental viral particles. The infected rodent population, $I_r(t)$, consists of rodents that carry and shed the virus throughout their lifetime. These infected rodents serve as the principal source of viral maintenance and environmental contamination.

The rodent subsystem therefore captures two important epidemiological mechanisms: direct rodent-to-rodent transmission and indirect transmission mediated by environmental viral contamination. The incorporation of logistic population growth reflects the ecological reality that rodent abundance is regulated by environmental carrying capacity and resource limitations. Since rodent density strongly influences disease prevalence, ecological regulation plays a significant role in the transmission dynamics.

The human population is divided into susceptible, exposed, infectious, and recovered compartments. Susceptible humans, $S_h(t)$, become infected through contact with contaminated environmental viral particles represented by $V(t)$. This transmission pathway reflects the dominant mechanism of hantavirus infection in humans, namely the inhalation of aerosolized viral particles originating from contaminated rodent excreta.

The exposed human class, $E_h(t)$, represents individuals who have acquired infection but are still within the incubation period and are not yet infectious. The transition from the susceptible to the exposed class, $E_h(t)$, signifies the acquisition of infection. This class represents individuals who are infected, but not yet infectious. Individuals leave this compartment and enter the infectious class, $I_h(t)$, where they are able to contribute to disease progression and environmental contamination, after the duration of the average latent period. Individuals who have survived infection and become immune enter the recovered class, $R_h(t)$.

A key epidemiological aspect of the model is the inclusion of the environmental viral reservoir, $V(t)$. This compartment forms the intermediate transmission step between infected rodents and infectious humans and susceptible hosts. Expressed as a concentration of viral particles per square meter, new "particles" are contributed by shedding from infected rodents and infectious humans, and particles disappear through natural viral decay and environmental removal.

In this way, the model captures the feedback of the epidemiological system on the ecological system: infected rodents increase environmental contamination, which in turn leads to new rodent and human infections, and infectious humans may additionally contribute to viral persistence in the environment.

Epidemiologically, disease persistence in this model can occur by three mechanisms: prevelance of infection in the rodent reservoir, accumulation of viral particles in the environment, and exposure of susceptible humans to the environment. Thus, in this system, efficacious disease interventions would include attmepts to reduce prevalence of rodent infection, reduce environmental contamination, increase environmental cleanup, and reduce human exposure.

In summary, the model provides an epidemiological basis for understanding hantavirus infection in rodents and spillover to humans given ecological forces and intrahost and environment-host transmission dynamics.

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. *All solutions of system* (1) *with nonnegative initial conditions remain nonnegative for all future time.*

Proof. Consider the boundary

$$S_r = 0.$$

Then

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

Similarly,

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

Also,

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

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

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

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

and

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

Thus every vector field points inward on the boundary of

$$\mathbb{R}^7_+.$$

Hence trajectories cannot leave the positive orthant.

Therefore,

$$S_r, I_r, S_h, E_h, I_h, R_h, V \geq 0$$

for all

$$t > 0.$$

□

2.3 Invariant Region and Boundedness

Theorem 3. *System* (1) *is uniformly bounded in a positively invariant region.*

Proof. Define

$$N_r = S_r + I_r.$$

Adding the rodent equations gives

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

Thus

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

Hence

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

Similarly, define

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

Adding the human equations gives

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

Hence

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

Therefore

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

For the environmental virus,

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

Using

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

we obtain

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

Thus

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

Hence all solutions remain 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

Using the next-generation matrix approach, let

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

The new infection matrix is

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

where

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

The transition matrix is

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

Hence

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

which simplifies to

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

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. *The disease-free equilibrium is globally asymptotically stable whenever*

$$R_0 < 1.$$

Proof. Define the Lyapunov function

$$\mathcal{L} = a_1 I_r + a_2 E_h + a_3 I_h + a_4 V,$$

where

$$a_i > 0.$$

Differentiating gives

$$\dot{\mathcal{L}} = a_1 \dot{I}_r + a_2 \dot{E}_h + a_3 \dot{I}_h + a_4 \dot{V}.$$

Choosing

$$a_2 = \frac{a_3 \kappa}{\omega_h + \kappa},$$

and

$$a_4 = \frac{a_1\beta_r S_r^0 + a_2\beta_h S_h^0}{\phi},$$

yields

$$\dot{\mathcal{L}} \leq -\Theta(1 - R_0)(I_r + E_h + I_h + V),$$

for some

$$\Theta > 0.$$

Hence

$$\dot{\mathcal{L}} \leq 0$$

whenever

$$R_0 < 1.$$

Furthermore,

$$\dot{\mathcal{L}} = 0$$

only if

$$I_r = E_h = I_h = V = 0.$$

By LaSalle's Invariance Principle,

$$\lim_{t\to\infty}(S_r, I_r, S_h, E_h, I_h, R_h, V) = E_0.$$

Therefore the disease-free equilibrium is globally asymptotically stable whenever

$$R_0 < 1.$$

□

2.8 Existence of the Endemic Equilibrium

Suppose that

$$R_0 > 1.$$

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 yields

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

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

$$\Pi_h - \eta_h S_h^* V^* - \omega_h S_h^* = 0, \tag{4}$$

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

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

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

and

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

2.9 Human Equilibrium Components

From (4),

$$S_h^* = \frac{\Pi_h}{\omega_h + \eta_h V^*}.$$

From (5),

$$E_h^* = \frac{\eta_h S_h^* V^*}{\omega_h + \kappa}.$$

Substituting S_h^*,

$$E_h^* = \frac{\eta_h \Pi_h V^*}{(\omega_h + \kappa)(\omega_h + \eta_h V^*)}.$$

From (6),

$$I_h^* = \frac{\kappa E_h^*}{\omega_h + \rho + \tau}.$$

Therefore

$$I_h^* = \frac{\kappa \eta_h \Pi_h V^*}{(\omega_h + \eta_h V^*)(\omega_h + \kappa)(\omega_h + \rho + \tau)}.$$

From (7),

$$R_h^* = \frac{\rho I_h^*}{\omega_h}.$$

Hence

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

2.10 Rodent Equilibrium Components

Adding (2) and (3) gives

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

Since

$$N_r^* > 0,$$

we obtain

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

Therefore

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

Using

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

equation (3) becomes

$$S_r^* \left(\alpha_r I_r^* + \eta_r V^*\right) = \omega_r I_r^*.$$

Hence

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

Substituting into

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

yields

$$\frac{\omega_r I_r^*}{\alpha_r I_r^* + \eta_r V^*} + I_r^* = K_r \left(1 - \frac{\omega_r}{\Pi_r}\right).$$

After simplification,

$$\alpha_r (I_r^*)^2 + \left[\eta_r V^* + \omega_r - \alpha_r N_r^*\right] I_r^* - \eta_r N_r^* V^* = 0.$$

Thus I_r^* satisfies the quadratic equation

$$\alpha_r (I_r^*)^2 + B(V^*) I_r^* + C(V^*) = 0,$$

where

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

and

$$C(V^*) = -\eta_r N_r^* V^*.$$

Therefore

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

The positive root is selected since $I_r^* > 0$.
Once I_r^* is known,

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

2.11 Reduction to a Single Equation

From (8),

$$V^* = \frac{\psi_r I_r^* + \psi_h I_h^*}{\phi}.$$

Substituting the expressions for

$$I_r^*$$

and

$$I_h^*$$

into the above equation gives

$$F(V^*) = 0.$$

Specifically,

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

Hence the endemic equilibrium is completely determined by the positive root of

$$F(V^*) = 0.$$

Theorem 6. *If*

$$R_0 > 1,$$

then the algebraic equation

$$F(V^*) = 0$$

admits at least one positive solution. Consequently, system (1) *possesses at least one biologically meaningful endemic equilibrium*

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

2.12 Local Stability of the Endemic Equilibrium

Theorem 7. *Let*

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

be the endemic equilibrium of system (1)*, which exists whenever*

$$R_0 > 1.$$

Then E^* is locally asymptotically stable if all Hurwitz determinants associated with the characteristic polynomial of the Jacobian matrix evaluated at E^* are positive.

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

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

The Jacobian matrix evaluated at E^* is

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

where

$$J_{11} = \Pi_r - \frac{2\Pi_r N_r^*}{K_r} - \alpha_r I_r^* - \eta_r V^* - \omega_r,$$

$$J_{12} = \Pi_r - \frac{2\Pi_r N_r^*}{K_r} - \alpha_r S_r^*,$$

$$J_{21} = \alpha_r I_r^* + \eta_r V^*,$$

$$J_{22} = \alpha_r S_r^* - \omega_r,$$

and

$$J_{33} = -\eta_h V^* - \omega_h.$$

Observe that the recovered class does not feed back into any other compartment. Consequently,

$$\lambda_1 = -\omega_h$$

is immediately an eigenvalue of the Jacobian matrix and satisfies

$$\lambda_1 < 0.$$

Therefore,

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

where

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

Hence the remaining six eigenvalues are obtained from the roots of

$$Q(\lambda) = 0.$$

The coefficient b_1 is obtained from the trace of the reduced 6×6 infected subsystem matrix M:

$$b_1 = -\operatorname{tr}(M).$$

Thus

$$b_1 = -(J_{11} + J_{22} + J_{33}) + (\omega_h + \kappa) + (\omega_h + \rho + \tau) + \phi.$$

Similarly,

$$b_2 = \sum \text{all principal minors of order 2},$$

$$b_3 = \sum \text{all principal minors of order 3},$$

$$b_4 = \sum \text{all principal minors of order 4},$$

$$b_5 = \sum \text{all principal minors of order 5},$$

and

$$b_6 = \det(M).$$

Consequently,

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

The Hurwitz matrix associated with $Q(\lambda)$ is

$$H = \begin{pmatrix} b_1 & b_3 & b_5 & 0 & 0 & 0 \\ 1 & b_2 & b_4 & b_6 & 0 & 0 \\ 0 & b_1 & b_3 & b_5 & 0 & 0 \\ 0 & 1 & b_2 & b_4 & b_6 & 0 \\ 0 & 0 & b_1 & b_3 & b_5 & 0 \\ 0 & 0 & 1 & b_2 & b_4 & b_6 \end{pmatrix}.$$

The associated Hurwitz determinants are

$$\Delta_1 = b_1,$$

$$\Delta_2 = \begin{vmatrix} b_1 & b_3 \\ 1 & b_2 \end{vmatrix} = b_1 b_2 - b_3$$

,

$$\Delta_3 = \begin{vmatrix} b_1 & b_3 & b_5 \\ 1 & b_2 & b_4 \\ 0 & b_1 & b_3 \end{vmatrix},$$

$$\Delta_4 = \begin{vmatrix} b_1 & b_3 & b_5 & 0 \\ 1 & b_2 & b_4 & b_6 \\ 0 & b_1 & b_3 & b_5 \\ 0 & 1 & b_2 & b_4 \end{vmatrix},$$

$$\Delta_5 = \begin{vmatrix} b_1 & b_3 & b_5 & 0 & 0 \\ 1 & b_2 & b_4 & b_6 & 0 \\ 0 & b_1 & b_3 & b_5 & 0 \\ 0 & 1 & b_2 & b_4 & b_6 \\ 0 & 0 & b_1 & b_3 & b_5 \end{vmatrix},$$

and

$$\Delta_6 = \det(H).$$

According to the Routh–Hurwitz criterion, every root of

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

has negative real part if and only if

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

and

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

Since

$$\lambda_1 = -\omega_h < 0,$$

and all roots of $Q(\lambda)$ possess negative real parts under the above conditions, it follows that every eigenvalue of

$$J(E^*)$$

has strictly negative real part.
Therefore,

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

is locally asymptotically stable.

□

Corollary 2.1. If

$$R_0 > 1,$$

and

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

together with

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

then small perturbations around the endemic equilibrium decay exponentially with time and the system returns to the endemic state. Consequently, hantavirus persists permanently in the population at the endemic level represented by E^*.

3 Discussion

The present study developed and rigorously analyzed a deterministic mathematical model describing the transmission dynamics of hantavirus among rodent reservoirs, humans, and the environmental viral reservoir. The model incorporates several important epidemiological mechanisms including direct rodent-to-rodent transmission, environmental transmission, logistic rodent population growth, human disease progression, environmental viral persistence, and viral shedding by both infected rodents and infectious humans. The inclusion of these mechanisms provides a comprehensive framework for investigating the ecological and epidemiological processes responsible for the emergence, persistence, and control of hantavirus infection.

The existence and uniqueness of solutions is a basic result here. Existence of unique solutions for for all nonnegative initial data is asserted by the fact that the equations in the model are continuously differentiable and locally Lipschitz in the biologically feasible region. Thus the model is well posed and solutions are unique given intial conditions. Biologically, this is desirable since the model will generate unambiguous disease courses.

It was shown that all state variables are nonnegative for all time if initial conditions are nonnegative.

This is an important property because state variables represent populations and viral load in the environment, in this case, both of which must remain nonnegative. Negative populations or infectious condition would destroy the epidemiological consistency of the model formulation.

Boundedness was shown by proving that solutions always remain outcome is that the three host populations are bounded above. The rodent population is never allowed to exceed a certain threshold depending on its carrying capacity, while the human population is bounded above by the ratio of recruitment to natural death rate. The infectious viral agent in the environment is also never allowed to become infinite, due to its own rate of death and the necessarily finite rate at which new virus can be shed into the environment. In other words, host populations are regulated by natural demography and contamination of the environment is eventually mitigated by the death of the virus.

A key result is the derivation of the disease free equilibrium E_0. It describes a disease-free environment in which rodents are at their ecological equilibrium population, humans are healthy, and the environment is uncontaminated. Epidemiologically, it is the state in which the hantavirus has been eradicated in rodents and humans.

The basic reproduction number R_0 was computed using the next-generation matrix approach. This quantity determines whether or not the disease can invade the population. The formula shows that there are three main epidemiological contributions to the reproduction number. The first term describes direct transmission from infectious rodent to infectious rodent, the second term involves rodents and infection transmission from the environment, and the third term involves only the environment and describes transmission from the contaminated environment to humans.

This separation is useful for biological interpretation. Rodent-to-rodent direct transmission, controlled by the parameter α_r, increases the ability of the disease to persist. The more efficient this transmission is, the more infections it will generate. Just as for the first term, the environmental transmission parameter β_r and the rate of shedding virus in the environment ψ_r in the numerator. The environmental decay rate of virus ϕ, appears in the denominator and acts to decrease the amount of time that virus particles are able to hang around in the environment and be transmitted to hosts.

The human component of the basic reproduction number shows that human infections are due to transmission from the environment, but that disease progression and recovery rates play a central role in whether or not transmission can occur to humans. Increased progression to lethal disease and less time spent in the infectious stage, as well as faster recovery, decrease the number of infections.

It was proven that the disease free equilibrium is locally stable whenever $R_0 < 1$. When this condition is satisfied, each of the three main epidemiological transmission mechanisms cannot generate enough secondary infections in hosts to sustain the circulation of the virus and the disease will die out. When the opposite case $R_0 > 1$ occurs, the disease can invade the population and the disease free equilibrium is unstable. This classical result emphasizes the importance of the basic reproduction number as a threshold determining disease dynamics.

The existance of an endemic equilibrium whenever $R_0 > 1$ shows that it is possible for the hantavirus to circulate endemically. Every infected class is positive, so rodents, the environment, and humans are all infected at endemicity.

The endemic equilibrium can be locally stable, according to the Routh, Hurwitz criteria, when all the eigenvalues of the Jacobian evaluated at the endemic equilibrium have negative real part. This means that when the virus is persisting endemically, small perturbations will always return to endemicity; it is very difficult to dislodge the virus from the system.

The environmental reservoir of virus is one of the most influential parts of this model. In the disease free equilibrium E_0, only the healthy hosts, human and rodent, exist at nonzero populations. The environment acts as a bridge between the two infected groups of humans and rodents. This is consistent with the epidemiology of hantavirus transmission, which is believed to be more via the environment than directly from host to host.

The logistic wild rodent population growth is another improvement to many classical models for hantavirus. Rodent population is determined by ecological factors as well as by infection. This suggests that control of rodent abundance in nature would be an effective mechanism for lowering the force of infection.

Biologically, these results suggest that decreasing rodent population density, decreasing level of contamination in the environment, increasing rate of death of the virus in environment, and decreasing human exposure to contaminated environments are feasible means of reducing the basic reproduction

number. This is consistent with the public health advice for how to avoid HPS disease in the United States.

This model has certain limitations. It is not spatially-explicit or age structured, and it does not include seasonality or stochasticity. It neglects human-to-human transmission, which is valid for most strains of hantavirus but not all. It does not address issues of immune response, or include time delays.

Possible extensions for future study include seasonal or spatially implicit forcing, diffusion or dispersal, stochasticity, inclusion of immune response and antiviral treatments, fractional-order derivatives, optimal control, and parameterization to data.

4 Conclusion

In this paper, we have formulated and analyzed a deterministic transmission model for hantavirus, which involves rodent, human, and environmental components. One can see that the system is fairly realistic, in that it includes both direct and indirect transmission, logistic growth of host rodent population and environmental concentration, as well as human disease progression. Mathematically, we have proved the existence, uniqueness, positivity, and boundedness of the solutions, so that the model is very well-posed, The disease-free equilibrium and reproduction number have been computed, and we showed that the DFE is locally asymptotically stable when $R_0 < 1$, unstable when $R_0 > 1$. The existence and local stability of the endemic equilibrium is also established when the reproduction number is larger than one.

Our results for this model show that a combination of infection in the rodent population, concentration in the environmental component, and transmission to the human population, are responsible for persistence of the disease. In particular, the environmental component is laced with infection from the rodents and then infects both rodent and human populations, forming a critical link. Control measures should reduce the level of hantavirus in the environment and reduce the contacts with susceptible rodents and humans to decrease the risk of infections. Overall, we have provided a rigorous framework (25) in which to understand the ecological principles behind persistence of virus in the environment. Our theoretical results could form the basis for a more in-depth mathematical analysis involving seasonal, spatial or immunological effects, or stochasticity and optimal control.

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