Modeling Hantavirus Transmission with Immune Response Dynamics: Stability Analysis

Abstract

In this study, a deterministic mathematical model for the transmission dynamics of hantavirus incorporating immune response dynamics is formulated and analyzed. The model consists of susceptible and infected rodent populations together with susceptible, exposed, infectious, and recovered human compartments. An immune response compartment is introduced to describe immune activation and immune-mediated clearance of infection in humans. The proposed framework captures rodent-mediated transmission, disease progression, recovery, disease-induced mortality, and host immune interactions. The positivity and boundedness of solutions is proved, which yields that the system is biologically and mathematically well-posed in a feasible invariant region. The existence of the disease-free equilibrium is proved and the basic reproduction number is determined by the next-generation matrix method. We prove that the disease-free equilibrium is locally and globally asymptotically stable if $R_0 < 1$, while a unique endemic equilibrium point exists if $R_0 > 1$. It is found that the rodent ecology plays a primary role in hantavirus persistence, while the role of immune response is important for infection clearance and recovery in infected rodents. In particular, for strong immune stimulation and immune clearance rates, the infection burden is reduced, leading to disease suppression, while weak immune action can cause persistent infection and prolonged epidemic outbreaks. This work emphasizes the epidemiological role of incorporating the immune response in hantavirus transmission models, and gives insight into the integrated intervention strategies involving rodent population management, exposure reduction and immune protection enhancement for effective disease management.

Keywords: Mathematical Modeling, Disease Transmission Dynamics, Hantavirus, Immune Response Dynamics, Rodent Populations

1 Introduction

Hantavirus is an animal-borne virus that is mostly transmitted by rodents and contaminated environments. Characterized by single-stranded RNA viruses from the family *Hantaviridae*, the disease is associated with dangerous human diseases such as Hemorrhagic Fever with Renal Syndrome (HFRS) and Hantavirus Pulmonary Syndrome (HPS) [2]. Hantavirus outbreaks still present serious public health concerns in many parts of the world due to their high disease fatality, complexity of ecology, and growing relationship with environmental and climatic changes [3]. Rodents are the main hantavirus reservoirs, and they remain persistently infected for their entire lifetime. Hantavirus is transmitted by aerosolized virus shed in rodent urine, saliva, and feces [4]. Humans usually get infected by the respiratory route through contact with excretions, urine, and saliva from infected wild rodents [5]. Rarely, a bite from an infected rodent can transmit the virus directly. Occasionally, person-to-person transmission has been documented, but generally only for some South American strains, such as the Andes virus [6]. The rodent-to-human pathway dominates in nearly all other cases.

The means of disease maintenance is highly dependent on the ecology of its rodent reservoirs. Outbreaks and characteristic cyclical patterns of hantavirus are triggered and regulated by climate factors, such as rainfall, humidity, temperature, vegetation, and food sources that influence the population dynamics of its rodent reservoirs [7]. Periods of increased rainfall result in a population explosion in the disease's rodent hosts, increasing opportunities for spillover infections in humans [8]. The famous Four Corners

outbreak of 1993 in the United States demonstrates a strong relationship between ecology and hantavirus incidence [11]. Besides the powerful effects of ecology on population incidence, host immune response governs the fate of disease course within a human host. Upon infection, the immune system launches a response via several pathways: macrophagic, t-cell, cytokine, and antibody-related [12]. Klingstrm et al. reports that a cytotoxic T-cell response is important in controlling infection [14], and Raftery et al. demonstrates that immunopathology significantly contributes to the pathology of progression [15]. Thus, immune dynamics appear to have a strong influence on hantavirus progression, and so immune progression should be considered in mathematical models.

Mathematical modeling is an extremely important approach in the study of infectious diseases systems, especially for guiding intervention analysis. Ordinary differential equation compartmental epidemic models provide a powerful quantitative basis for analyzing dynamics, finding threshold conditions, studying behavior of the equilibria, and predicting the long-term future of spreading diseases [16, 17, 18]. In animal-borne diseases such as hantavirus, such constructive models are particularly useful due to their basis in ecological progression. Early quantitative work on hantavirus focused on mathematical modeling of ecological reservoir transmission. One of the first mathematical frameworks for hantavirus viral spread in nature was produced by Abramson and Kenkre [19]. They analyzed spatiotemporal transmission and found population movement to significantly affect epidemic persistence. Building on this work, Allen et al. developed deterministic models for infection spread in rodent populations [20, 21]. Their results demonstrate how ecology of the rodent hosts dominates disease dynamics, and they establish threshold conditions for persistence and extinction of the disease.

Sauvage et al. studied variables affecting rodent populations and found ecology to be a dominating factor in disease dynamics [22]. Many researchers continued to extend the classic hantavirus model, incorporating features such as stochasticity and spatial diffusion. Wesley and Allen discussed the effects of external, seasonal forcing on persistence and found that seasonality can drive the system to outbreak or oscillate [23]. Liu et al. incorporated time delays and found that they greatly affects stability of the system [24]. Recent studies have increasingly focused on integrating human infection dynamics into hantavirus models. Gutirrez-Jara et al. developed a mathematical model incorporating territorial structure and human mobility [25]. Their work demonstrated that spatial movement patterns significantly influence spillover transmission risk and outbreak propagation.

In recent years, growing attention has been directed toward the role of immune response in infectious disease modeling. Immune response dynamics have been extensively studied in viral infections such as HIV, hepatitis, influenza, Ebola, and COVID-19 [26, 27]. These models have shown that immune-mediated viral clearance and immune activation strongly affect disease progression and stability behavior. Several within-host viral models have incorporated immune effector cells and immune-mediated clearance mechanisms [28, 9, 10, 29]. Such approaches have improved understanding of viral persistence, immune exhaustion, cytokine storms, and therapeutic interventions. Despite these advances, relatively few mathematical studies have explicitly incorporated immune response dynamics into hantavirus transmission models. Since immune activation significantly influences disease severity, recovery, and mortality, neglecting immune interactions may limit the biological realism of epidemiological models.

The present study therefore develops a deterministic hantavirus transmission model incorporating immune response dynamics. The model integrates: rodent reservoir transmission, human infection progression, immune activation, immune-mediated clearance, and recovery and disease-induced mortality. The inclusion of immune dynamics provides a more realistic representation of host-pathogen interactions and allows investigation of how immune responses influence disease persistence and stability. The study focuses on rigorous qualitative analysis and stability theory. In particular, the objectives are to establish positivity and boundedness of solutions, derive the basic reproduction number, determine the disease-free equilibrium, analyze local and global stability properties, and investigate the role of immune response in infection control.

Understanding these mechanisms is essential for improving epidemiological prediction and designing effective disease control strategies. The results obtained in this work may contribute to future studies involving optimal control, immune therapy, stochastic effects, environmental transmission, and within-host hantavirus dynamics.

1.1 Model Assumptions with Immune Response Dynamics

The following assumptions are considered:

1. Rodents reproduce naturally.
2. Infected rodents remain infected throughout life.
3. Transmission among rodents occurs through contact.

4. Humans become infected through contact with infected rodents.
5. Human-to-human transmission is negligible.
6. Recovered humans acquire immunity.
7. The human immune system responds to hantavirus infection through activation of immune effector cells.
8. Immune cells help suppress infection by eliminating infectious human cells.
9. Immune response strength increases proportionally with the level of infection.
10. Immune cells decay naturally in the absence of infection.

1.2 Description of Variables

Table 1: Description of model variables

Variable	Description
$S_r(t)$	Susceptible rodents
$I_r(t)$	Infected rodents
$S_h(t)$	Susceptible humans
$E_h(t)$	Exposed humans
$I_h(t)$	Infectious humans
$R_h(t)$	Recovered humans
$M(t)$	Immune response level or immune effector cells

The total rodent population is

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

while the total human population is

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

1.3 Model Equations

The hantavirus transmission model with immune response dynamics is given by

$$\begin{aligned}
\frac{dS_r}{dt} &= \Pi_r - \lambda_r S_r I_r - \nu_r S_r, \\
\frac{dI_r}{dt} &= \lambda_r S_r I_r - \nu_r I_r, \\
\frac{dS_h}{dt} &= \Pi_h - \lambda_h S_h I_r - \nu_h S_h, \\
\frac{dE_h}{dt} &= \lambda_h S_h I_r - (\nu_h + \xi) E_h, \\
\frac{dI_h}{dt} &= \xi E_h - (\nu_h + \chi + \varepsilon) I_h - \psi M I_h, \\
\frac{dR_h}{dt} &= \chi I_h + \psi M I_h - \nu_h R_h, \\
\frac{dM}{dt} &= \omega I_h - \tau M.
\end{aligned} \tag{1}$$

Table 2: Description of model parameters

Parameter	Description
Π_r	Recruitment rate of rodents
ν_r	Natural death rate of rodents
λ_r	Transmission coefficient among rodents
Π_h	Recruitment rate of humans
ν_h	Natural death rate of humans
λ_h	Rodent-to-human transmission coefficient
ξ	Rate at which exposed humans become infectious
χ	Natural recovery rate of infectious humans
ε	Disease-induced mortality rate in humans
ψ	Immune-mediated clearance rate of infected humans
ω	Immune activation rate induced by infection
τ	Natural decay rate of immune response

1.4 Description of Parameters

1.5 Immune Dynamics Explained

The immune response variable

$$M(t)$$

represents immune effector activity generated against hantavirus infection.

The immune dynamics are governed by

$$\frac{dM}{dt} = \omega I_h - \tau M.$$

This implies that:

- immune activity increases in response to infectious humans,
- stronger infection induces stronger immune stimulation,
- immune cells decay naturally in the absence of infection.

The infectious human population evolves according to

$$\frac{dI_h}{dt} = \xi E_h - (\nu_h + \chi + \varepsilon) I_h - \psi M I_h.$$

The nonlinear term

$$\psi M I_h$$

describes immune-mediated elimination of infected individuals.

Thus:

- stronger immune response suppresses infection,
- immune activity enhances recovery,
- increased immune clearance lowers infectious burden.

Recovered humans satisfy

$$\frac{dR_h}{dt} = \chi I_h + \psi M I_h - \nu_h R_h,$$

showing that recovery occurs both naturally and through immune-mediated clearance.

2 Qualitative Analysis of the System

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

2.1 Positivity of Solutions

Consider the system (1).

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

$$t > 0.$$

Proof. Assume

$$S_r(0) > 0, \quad I_r(0) > 0, \quad S_h(0) > 0, \quad E_h(0) > 0, \quad I_h(0) > 0, \quad R_h(0) > 0, \quad M(0) > 0.$$

From the first equation,

$$\frac{dS_r}{dt} = \Pi_r - (\lambda_r I_r + \nu_r) S_r.$$

Hence,

$$\frac{dS_r}{dt} \geq -(\lambda_r I_r + \nu_r) S_r.$$

Integrating yields

$$S_r(t) \geq S_r(0) \exp\left(- \int_0^t (\lambda_r I_r + \nu_r) d\tau \right) > 0.$$

Similarly,

$$\frac{dI_r}{dt} = (\lambda_r S_r - \nu_r) I_r,$$

implies

$$I_r(t) = I_r(0) \exp\left(\int_0^t (\lambda_r S_r - \nu_r) d\tau \right) > 0.$$

Proceeding similarly for the remaining variables gives

$$S_h(t) > 0, \quad E_h(t) > 0, \quad I_h(t) > 0, \quad R_h(t) > 0, \quad M(t) > 0$$

for all

$$t > 0.$$

Therefore, the feasible region

$$\Omega \subset \mathbb{R}^7_+$$

is positively invariant.

□

2.2 Boundedness of Solutions

Theorem 2. *Solutions of system* (1) *remain uniformly bounded in a positively invariant region.*

Proof. Define the total rodent population

$$N_r = S_r + I_r.$$

Then

$$\frac{dN_r}{dt} = \Pi_r - \nu_r N_r.$$

Solving gives

$$N_r(t) = N_r(0) e^{-\nu_r t} + \frac{\Pi_r}{\nu_r} \left(1 - e^{-\nu_r t}\right).$$

Hence,

$$0 \leq N_r(t) \leq \frac{\Pi_r}{\nu_r}.$$

Similarly, define the total human population

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

Adding the human equations yields

$$\frac{dN_h}{dt} = \Pi_h - \nu_h N_h - \varepsilon I_h.$$

Since

$$\varepsilon I_h \geq 0,$$

we obtain

$$\frac{dN_h}{dt} \leq \Pi_h - \nu_h N_h.$$

Thus,

$$0 \leq N_h(t) \leq \frac{\Pi_h}{\nu_h}.$$

Finally, consider the immune response equation:

$$\frac{dM}{dt} = \omega I_h - \tau M.$$

Since

$$I_h \leq N_h \leq \frac{\Pi_h}{\nu_h},$$

we have

$$\frac{dM}{dt} \leq \frac{\omega \Pi_h}{\nu_h} - \tau M.$$

Hence,

$$0 \leq M(t) \leq \frac{\omega \Pi_h}{\tau \nu_h}.$$

Therefore, all trajectories remain bounded in the feasible region

$$\Omega = \left\{ (S_r, I_r, S_h, E_h, I_h, R_h, M) \in \mathbb{R}_+^7 : N_r \leq \frac{\Pi_r}{\nu_r},\, N_h \leq \frac{\Pi_h}{\nu_h},\, M \leq \frac{\omega \Pi_h}{\tau \nu_h} \right\}.$$

□

2.3 Disease-Free Equilibrium

The disease-free equilibrium is obtained by setting

$$I_r = E_h = I_h = R_h = M = 0.$$

Thus,

$$E_0 = \left(\frac{\Pi_r}{\nu_r}, 0, \frac{\Pi_h}{\nu_h}, 0, 0, 0, 0 \right).$$

2.4 Basic Reproduction Number

Using the next-generation matrix method, we define the infected vector

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

The new infection matrix is

$$F = \begin{pmatrix} \lambda_r S_r I_r \\ \lambda_h S_h I_r \\ 0 \end{pmatrix},$$

while the transition matrix is

$$V = \begin{pmatrix} \nu_r I_r \\ (\nu_h + \xi) E_h \\ (\nu_h + \chi + \varepsilon + \psi M) I_h - \xi E_h \end{pmatrix}.$$

Evaluating at the disease-free equilibrium gives

$$F = \begin{pmatrix} \frac{\lambda_r \Pi_r}{\nu_r} & 0 & 0 \\ \frac{\lambda_h \Pi_h}{\nu_h} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

and

$$V = \begin{pmatrix} \nu_r & 0 & 0 \\ 0 & \nu_h + \xi & 0 \\ 0 & -\xi & \nu_h + \chi + \varepsilon \end{pmatrix}.$$

Hence,

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

where

$$\rho$$

denotes the spectral radius.

After simplification,

$$R_0 = \frac{\lambda_r \Pi_r}{\nu_r^2}.$$

Thus:

$$R_0 < 1$$

implies disease elimination, while

$$R_0 > 1$$

implies persistence.

3 Stability Analysis

Here, in this section, we shall consider the local and global stability of the equilibrium points.

3.1 Local Stability of the Disease-Free Equilibrium

Theorem 3. *The disease-free equilibrium*

$$E_0$$

is locally asymptotically stable whenever

$$R_0 < 1,$$

and unstable whenever

$$R_0 > 1.$$

Proof. The Jacobian matrix of system (1) evaluated at

$$E_0$$

is

$$J(E_0) = \begin{pmatrix} -\nu_r & -\frac{\lambda_r \Pi_r}{\nu_r} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\lambda_r \Pi_r}{\nu_r} - \nu_r & 0 & 0 & 0 & 0 & 0 \\ 0 & -\frac{\lambda_h \Pi_h}{\nu_h} & -\nu_h & 0 & 0 & 0 & 0 \\ 0 & \frac{\lambda_h \Pi_h}{\nu_h} & 0 & -(\nu_h + \xi) & 0 & 0 & 0 \\ 0 & 0 & 0 & \xi & -(\nu_h + \chi + \varepsilon) & 0 & 0 \\ 0 & 0 & 0 & 0 & \chi & -\nu_h & 0 \\ 0 & 0 & 0 & 0 & \omega & 0 & -\tau \end{pmatrix}.$$

The eigenvalues are:

$$\lambda_1 = -\nu_r,$$
$$\lambda_2 = \frac{\lambda_r \Pi_r}{\nu_r} - \nu_r,$$
$$\lambda_3 = -\nu_h,$$
$$\lambda_4 = -(\nu_h + \xi),$$
$$\lambda_5 = -(\nu_h + \chi + \varepsilon),$$
$$\lambda_6 = -\nu_h,$$
$$\lambda_7 = -\tau.$$

Observe that

$$\lambda_2 < 0 \iff \frac{\lambda_r \Pi_r}{\nu_r^2} < 1.$$

Hence,

$$R_0 < 1$$

implies all eigenvalues have negative real parts.

Therefore,

$$E_0$$

is locally asymptotically stable.

Conversely, if

$$R_0 > 1,$$

then

$$\lambda_2 > 0,$$

implying instability.

□

3.2 Existence of Endemic Equilibrium

Theorem 4. *System* (1) *admits a unique endemic equilibrium whenever*

$$R_0 > 1.$$

Proof. At equilibrium,

$$\frac{dI_r}{dt} = 0.$$

Thus,

$$\lambda_r S_r^* I_r^* - \nu_r I_r^* = 0.$$

Since

$$I_r^* > 0,$$

we obtain

$$S_r^* = \frac{\nu_r}{\lambda_r}.$$

Substituting into the susceptible rodent equation gives

$$I_r^* = \frac{\Pi_r}{\nu_r} - \frac{\nu_r}{\lambda_r}.$$

Therefore,

$$I_r^* > 0 \iff \frac{\lambda_r \Pi_r}{\nu_r^2} > 1.$$

Hence,

$$R_0 > 1$$

guarantees existence of a biologically feasible endemic equilibrium.

□

3.3 Global Stability of the Disease-Free Equilibrium

Theorem 5. *The disease-free equilibrium is globally asymptotically stable whenever*

$$R_0 < 1.$$

Proof. Consider the Lyapunov function

$$\mathcal{L} = I_r.$$

Differentiating along solutions of system (1) gives

$$\dot{\mathcal{L}} = \lambda_r S_r I_r - \nu_r I_r.$$

Since

$$S_r \leq \frac{\Pi_r}{\nu_r},$$

we obtain

$$\dot{\mathcal{L}} \leq \left(\frac{\lambda_r \Pi_r}{\nu_r} - \nu_r \right) I_r.$$

Thus,

$$\dot{\mathcal{L}} = \nu_r (R_0 - 1) I_r.$$

Hence,

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

whenever

$$R_0 < 1.$$

Furthermore,

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

only if

$$I_r = 0.$$

By LaSalle's Invariance Principle, all trajectories converge to the disease-free equilibrium. Therefore, the disease-free equilibrium is globally asymptotically stable whenever

$$R_0 < 1.$$

□

4 Discussion and Conclusion

In this study, a deterministic mathematical model incorporating immune response dynamics was formulated and analyzed to investigate the transmission dynamics of hantavirus between rodent reservoirs and human populations. The model developed here is an extension of existing classical hantavirus transmission models to include a mechanistic immune response compartment and immune-mediated clearance within infeted humans. Its inclusion helps to monitor the progression of hantavirus to within-host immune responses, since activating host immunity strongly contributes to severity of infection and subsequent recovery or progression to worse disease. The qualitative analysis revealed that the system maintains several important mathematical properties. The positivity analysis showed that the solutions to the system are positive, with positive solutions for all time guaranteed by starting the trajectories with positive initial conditions. This is an important property to check in epidemiological models whose variables represent populations, since negative population numbers in simulation settings can lead to unreliable behavior and biologically irrelevant results.

The analysis of boundedness of solutions further revealed that all solutions to the system are finite, contained within a positively invariant feasible region in $\mathbb{R}^7_+$. In particular, it was proven that the rodent and human populations are contained by birth and death processes, but also that immune activity remains finite. This continues to be a biologically sound result, since it does not make sense for population sizes nor immune reaction strengths to grow to infinity in finite times. In particular, this result is mathematically significant because it prevents singularities and blow-up in finite time, so solutions are guaranteed to exist. The disease-free equilibrium represents the infection-free human and rodent population, with no infected rodents, no exposed humans, no exposed infectious humans, and no activity in the immune compartment. Practically, this is the equilibrium corresponding to eradication of spread of hantavirus in

both rodents and humans. The immune response variable, M, is zero at this equilibrium because there is no infectious virus present to stimulate activation of immune cells.

The basic reproduction number, R_0, was calculated as the critical parameter that determines persistence or extinction of the disease in the rodent population. It can be interpreted as the average number of secondary infections in susceptible rodents caused by a new infected rodent introduced into a population from a rodent external to the population. From its definition, several interesting biological observations arise. The rodent transmission coefficient increases the reproductive number by its appearance in the numerator, showing how more rodent population increases the probability of disease persistence. Similarly, increased rodent recruitment enhances disease persistence because favorable ecological conditions often support rapid rodent population growth. In contrast, increased rodent mortality strongly suppresses transmission, suggesting that rodent control measures may substantially reduce outbreak intensity and disease persistence.

The local stability analysis established that the disease-free equilibrium is locally asymptotically stable whenever $R_0 < 1$, and unstable whenever $R_0 > 1$. Biologically, this means that when the reproduction number is below unity, small introductions of infected rodents cannot sustain transmission and the disease gradually disappears from the population. However, when the reproduction number exceeds unity, infection persists and may spread throughout the rodent and human populations. The reproduction number therefore acts as the fundamental threshold separating disease eradication from endemic persistence.

The existence of an endemic equilibrium whenever $R_0 > 1$ demonstrates that hantavirus can persist permanently within the rodent-human system under sufficiently strong transmission conditions. At endemicity, infected rodents, infectious humans, and immune activity remain positive. The model has a very important immune feature. The immune equation indicates that the immune response increases when there are more infectious humans, and decays in the absence of infection. In practical sense, this represents the stimulation of immune effector cells under the influence of a viral infection, and their slow decline after the pathogen is cleared. The immune-mediated clearance term in the infectious human equation reveals that high levels of immune activity suppress infection and promote recovery. As such, increased immune activity leads to decreased infectious burden. Conversely, very fast immune decay may lead to more persistent infection.

The global stability analysis proved that the disease-fee equilibrium is globally asymptotically stable whenever $R_0 < 1$. Here, global stability is a much stronger result than local stability, which only guarantees convergence to equilibrium of small outbreaks. The analysis uses Lyapunov functions and LaSalle's Invariance Principle, and shows that infected rodents are guaranteed to decrease as long as the reproduction number is constrained to be less than one. This means the disease will be extinguished from the system and immune activation will also disappear. This result is important because it means that bringing the reproduction number below one will ultimately ensure the eradication of hantavirus from the population.

The results of this work also have several relevant epidemiological and public health implications. Since rodent transmission dynamics are overwhelmingly important for sustaining infection, reducing rodent density and rodent contact should remain a top priority for disease control. Environmental sanitation and habitat management may substantially lower transmission intensity by reducing opportunities for rodent-human interaction. In addition, strengthening immune response mechanisms through therapeutic or immunological interventions may improve disease outcomes and accelerate recovery. Public health strategies should therefore combine rodent population management, environmental sanitation, reduction of human exposure, and improved healthcare interventions.

Despite its usefulness, the model possesses several limitations. The system assumes homogeneous mixing among populations and does not account for spatial heterogeneity, stochastic environmental effects, seasonal transmission, environmental viral persistence, cytokine storm mechanisms, or immune exhaustion effects. These simplifications may limit the ability of the model to fully capture the complexity of real hantavirus outbreaks. Future extensions could therefore incorporate environmental viral dynamics, nonlinear immune activation, delayed immune responses, stochastic perturbations, fractional-order effects, and spatial diffusion processes. Such improvements would provide a more comprehensive understanding of hantavirus epidemiology and enhance the predictive capability of the model in complex ecological environments.

Generally, the study provides a rigorous theoretical framework for understanding hantavirus transmission dynamics and the role of immune response in disease progression and control. The integration of epidemiological and immunological processes offers valuable insights into the mechanisms governing disease persistence, stability behavior, and outbreak control. The model may therefore serve as a foundation for future investigations involving optimal control strategies, environmental transmission, stochastic effects, and advanced immune-mediated dynamics.

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