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NUMERICAL AND BIFURCATION ANALYSIS WITH SENSITIVITY ASSESSMENT OF A DETERMINISTIC LEPTOSPIROSIS HANTAVIRUS CO-INFECTION MODEL

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Abstract In this paper, a deterministic compartmental model is developed in order to study the dynamics of the transmission of leptospirosis and hantavirus co-infection with particular emphasis on the human population. The model includes compartments of susceptible individuals, singly infected classes, co-infected individuals, and recovered individuals, as well as important pathogen exposure and disease progression mechanisms. Analytical features of the proposed model are addressed, such as positivity, boundedness, the existence of equilibrium points, and bifurcation analysis. The next-generation matrix approach is used to obtain the dominant transmission threshold, denoted by, $\mathcal{R}_0^c$ which determine the qualitative behavior of the system. The transmission-free equilibrium of the model is locally asymptotically stable when $\mathcal{R}_0^c < 1$ and unstable when $\mathcal{R}_0^c > 1$, indicating disease persistence in the population. In addition, bifurcation analysis is carried out to examine the system behavior near the critical threshold. The analysis confirm the occurrence of a backward transcritical bifurcation. The validity of the analytical results is confirmed through numerical simulations. In particular, the roles of transmission rates, recovery rates, and co-infection dynamics are studied in detail. Sensitivity analysis shows that parameters associated with infection transmission and recovery have the strongest influence on disease burden, with co-infection outcomes being especially responsive to changes in recovery rates. The results indicate that improving treatment efficiency and limiting transmission pathways can reduce both single infections and co-infection occurrences. Overall, this study highlights the importance of targeted control strategies for leptospirosis hantavirus co-infection and emphasizes the need for effective intervention measures to reduce public health risks.

Keywords Co-infection, leptospirosis disease, hantavirus infection, stability, numerical simulation, leptospirosis hantavirus co-infection.

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1. Introduction

Leptospirosis and hantavirus infection are zoonotic diseases that continuously affect public health across many parts of the world, particularly in the tropical and subtropical regions where climatic and environmental conditions facilitate their spread. Leptospirosis and hantavirus infection are both zoonotic diseases that continue to represent substantial public health challenges globally, particularly in tropical and subtropical regions. Leptospirosis is a bacterial disease caused by pathogenic *Leptospira* species and is transmitted to humans mainly through direct contact with soil or water contaminated with urine of infected animals. In contrast, hantavirus infection is a viral disease primarily transmitted to humans through inhalation of aerosols contaminated with excretions from infected rodents. Although the two diseases are induced by other pathogens, they have similar ecological and epidemiological characteristics since rodents are the key organisms that act as reservoirs of these diseases and the environmental factors have a significant impact on the level of transmission. Phosri et al. [21] explicated that the variability of rainfall is a powerful influence to the outbreak of leptospirosis. Engida et al. [6] established a coupled human rodent model and determined the importance of rodent reservoirs in sustaining endemic transmission. Costa et al. [19] demonstrated that even low-contact rate exposes humans to the presence of the bacteria Leptospira. All these studies indicate that rodent population and environmental factors are significant in the dynamics of transmission of zoonotic infections.

In 2023, mathematical modeling efforts increasingly incorporated nonlinear transmission mechanisms. Ahmad et al. [1] analyzed a nonlinear leptospirosis model and established threshold conditions for disease removal and persistence. Zhao et al. [24] introduced saturated incidence rates and demonstrated that linear transmission assumptions may overestimate infection levels. Kumar et al. [10] explained the effect of waning immunity and concluded that reinfection completely alters long-term dynamics. Similarly, Wang et al. [26] studied environmental contamination and showed that neglecting indirect transmission pathways underestimates outbreak severity. Recent studies have also advanced the modeling of hantavirus transmission. Ibarra-Cerdena, [4] investigated rodent population feedback mechanisms and showed that changes in environmental conditions affect hantavirus outbreaks. Xu and Watson [27] incorporated seasonal forcing and identified the coexistence of multiple equilibria. Kim et al. [18] analyzed optimal intervention strategies and found that reducing rodent-human contact is more effective than post-infection treatment alone. K. Park et al. [20] showed the persistence of hantavirus infection due to rodents even under control measures.

From 2024 onward, the attention of researchers shifted toward multi-pathogen and co-infection models. Ratti et al. [22] investigated malaria- cholera co-infection and proved that pathogen interaction modifies classical threshold dynamics. Singh et al. [11] studied tuberculosis-COVID-19 co-infection and demonstrated that co-infected individuals accelerate disease persistence. Zhang et al. [25] showed that co-infection can disturb disease-free equilibria and induce backward bifurcation. More recently, Malik et al. [12] emphasized that ignoring co-infection may lead to misleading policy recommendations. Despite these advances, only a limited number of studies have considered zoonotic co-infections. Asaduzzaman [2] studied leptospirosis- dengue co-infection and reported increased disease severity in co-infected individuals. Rahman et al. [9] studied and proved that human behavior plays a key role in transmission from rodents. Hassan et al. [23] studied the environmental effect on co-infection and showed that variation in climate factors is a source of infection persistence.

Mathematical modeling has played a fundamental role in the understanding of co-infection dynamics, where two or more pathogens interact within the same host population. Unlike single-

disease frameworks, co-infection models consider how one disease can either increase or decrease the impact of another, change the way infections spread, influence how quickly individuals recover, and alter host susceptibility. These models prove that interactions between pathogens can dramatically change epidemic thresholds, change the conditions of stability and even develop some complicated phenomena like multi-stability and backward bifurcation. The theoretical basis of the multi-compartment epidemic system analysis was laid down by the next-generation matrix methodology of Watmough et al. [5] that continues to play a pivotal role in computing the basic reproduction number of co-infection models.

Gumel et al. [7] showed that interactions between diseases can generate backward bifurcation analysis, it means that infection may persist even when the basic reproduction number is below one. Gao et al. [8] further studied that pathogen interaction can affect the overall disease burden and efficiently modify stability results. In a malaria–dengue co-infection framework, Sardar et al. [3] studied how disease interplay directly shapes control strategies and epidemic thresholds. The nonlinear transmission and interaction effects are important for understanding co-infection dynamics and interactions with feedback between pathogens which were studied by Zhang et al. [28], that linear models cannot easily capture. Such type of formulation accurately explains the co-infection disease spread within a population. Overall, these studies highlight the importance of co-infection modeling in understanding disease transmission patterns. With the help of non linear interaction we accurately describe how two diseases spread together, effect of one pathogen on another due to these reason, it is important to include nonlinear interaction effects in the model. Nadeem et al. [14, 15] have made a significant contribution to the study of nonlinear dynamical systems using different mathematical methods. Authors [17] studied (1+1)-dimensional Sawada–Kotera model using Lie symmetries analysis. They include their work on nanobeam-based N/MEMS models, invariant solutions, soliton dynamics, bifurcation, chaos analysis, and fuzzy methods to deal with uncertainty [15]. These works underline the significance of analytical and stability methods, which are directly connected with the current work in the study of complex systems behavior [16] and [13]. In short, co-infection modeling explains how two diseases spread, die out, and show responses to control strategies within a population. They also indicate that neglecting pathogen interactions may lead to an underestimation of outbreak severity and produce misleading conclusions regarding control measures. Therefore, studying interaction mechanisms in epidemic models is fundamental for reliable prediction and informed public health decision-making. This approach offers a more realistic representation of temporal dependence and pathogen interactions, thereby contributing to the growing literature on advanced co-infection modeling methodologies. However, none of these studies examined leptospirosis and hantavirus co-infection within a unified deterministic framework.

Motivated by these gaps, the present work develops a new mathematical model to investigate the co-infection dynamics of leptospirosis and hantavirus in humans. The disease-free equilibrium point, called transmission-free equilibrium, denoted by $\mathcal{E}^o_{TF}$, represents the absence of infection, while the endemic equilibrium point, called persistence infection equilibrium, is denoted by $\mathcal{E}^*_{PI}$. The basic reproduction number for the proposed model is called the dominant transmission threshold and is denoted by $\mathcal{R}^c_0$, governing the global dynamics of the system. The local stability is checked through Jacobian analysis, while the global stability of the model is checked through a Lyapunov function. Bifurcation and sensitivity analyses are performed to examine changes in equilibrium solutions with parameters and the influence of parameters on the disease, respectively. Numerical simulations are used to support the analytical section of the paper and to assess the effectiveness of potential control strategies.

2. Model formulation of leptospirosis and hantavirus co-infection

We formulate a deterministic compartmental model that describes the transmission dynamics of leptospirosis and hantavirus infection in the human-population only. The human population at time t, denoted by $N_p(t)$ is subdivided into five epidemiological classes: susceptible individuals ($S_p(t)$), infected individuals with leptospirosis only ($I_L(t)$), infected individuals with hantavirus only ($I_H(t)$), co-infected individuals ($I_C(t)$), and recovered individuals ($R_p(t)$). Thus, $N_p(t) = S_p(t) + I_L(t) + I_H(t) + I_C(t) + R_p(t)$.

Susceptible individuals acquire leptospirosis or hantavirus through effective contact with infectious individuals. The forces of infection for leptospirosis and hantavirus are given, respectively, by

$$\lambda_L = \beta_L \frac{I_L}{N_p}, \qquad \lambda_H = \beta_H \frac{I_H}{N_p},$$

where β_L and β_H denote the effective transmission rates of leptospirosis and hantavirus, respectively.

Individuals infected with one disease may acquire the second disease, leading to coinfection. Specifically, individuals in the leptospirosis class I_L acquire hantavirus at rate $\beta_{LH}\frac{I_H}{N_p}$, while individuals in the hantavirus class I_H acquire leptospirosis at rate $\beta_{HL}\frac{I_L}{N_p}$. Recovered individuals lose their immunity at the rate r and move back to the susceptible class. All individuals experience natural mortality at rate (μ_P). The proposed leptospirosis–hantavirus co-infection model is based on the following biological assumptions:

1. The human population is constant over the study period.
2. New individuals enter the population join the susceptible class with constant rate.
3. Susceptible individuals may acquire either leptospirosis or hantavirus infection.
4. Individuals infected with a single disease may progress to co-infection after exposure to the second pathogen.
5. The rodent population is not explicitly include in the model; instead, its epidemiological impact is captured indirectly through constant transmission rates acting on the human population.
6. Co-infected individuals recover without reverting to single-infection classes.
7. Recovery provides temporary immunity, which may wane over time.
8. Natural death occurs uniformly and disease-induced mortality is neglected in population.
9. Every newborn is considered susceptible and population is homogeneously mixed.

The model parameter and their biological description is listed in Table 1 and Figure 1 show its flowchart.

Based on these assumptions, the model is explain by the following system of ordinary differential equations:

$$\begin{aligned}
\frac{dS_p}{dt} &= \mu_p N_p - \beta_L \frac{I_L}{N_p} S_p - \beta_H \frac{I_H}{N_p} S_p + rR_p - \mu_p S_p,\\
\frac{dI_L}{dt} &= \beta_L \frac{I_L}{N_p} S_p - \beta_{LH} \frac{I_H}{N_p} I_L - (\gamma_L + \mu_p) I_L,
\end{aligned}$$

Table 1. Model parameters and their biological descriptions.

Parameter	Biological description
β_L	Transmission rate of leptospirosis infection
β_H	Transmission rate of hantavirus infection
β_{HL}	Co-infection rate from hantavirus to leptospirosis
β_{LH}	Co-infection rate from leptospirosis to hantavirus
γ_L	Recovery rate from leptospirosis infection
γ_H	Recovery rate from hantavirus infection
γ_C	Recovery rate from co-infection
μ_p	Natural mortality rate of the human population
r	Rate of loss of immunity

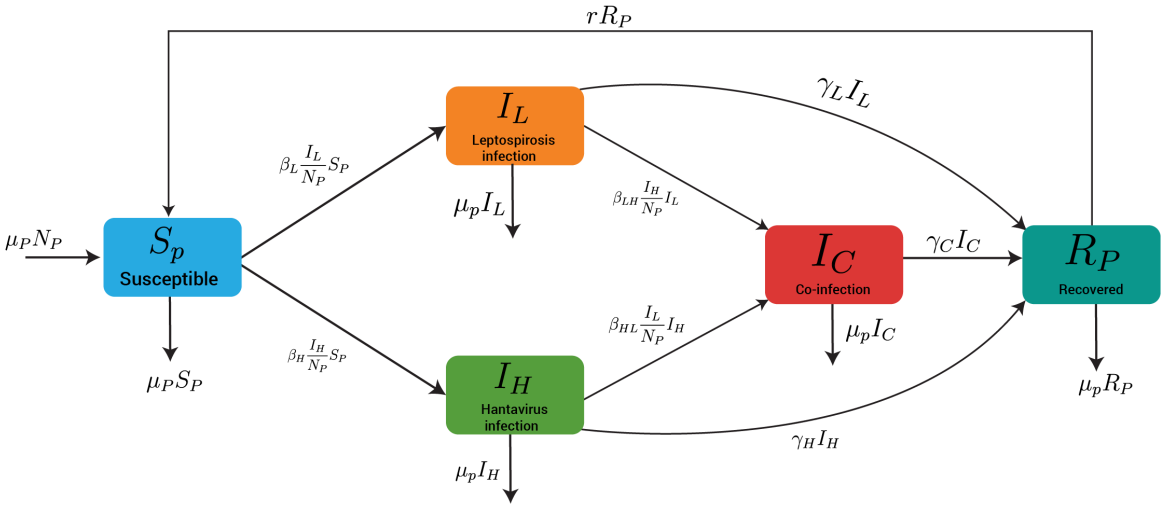


Figure 1. Flow chart of the leptospirosis-hantavirus co-infection transmission model.

$$\begin{aligned}
\frac{dI_H}{dt} &= \beta_H \frac{I_H}{N_p} S_p - \beta_{HL} \frac{I_L}{N_p} I_H - (\gamma_H + \mu_p) I_H, \\
\frac{dI_C}{dt} &= \beta_{LH} \frac{I_H}{N_p} I_L + \beta_{HL} \frac{I_L}{N_p} I_H - (\gamma_C + \mu_p) I_C, \\
\frac{dR_p}{dt} &= \gamma_L I_L + \gamma_H I_H + \gamma_C I_C - r R_p - \mu_p R_p.
\end{aligned} \tag{2.1}$$

3. Model properties

In this section, we establish the fundamental mathematical properties of proposed model, including non-negativity, boundedness of solutions, and the existence of biologically feasible invariant regions.

Theorem 3.1. *For any given non-negative initial conditions* $(S_p, I_L, I_H, I_C, R_p)(0) \in \mathbb{R}^5_+$, *the model his unique solution that exists for all* $t \geq 0$.

Proof. The right–hand sides of the system 2.1 consist of polynomial and rational functions of the state variables $(S_p, I_L, I_H, I_C, R_p)$ with denominators involving only the constant population size $N_p > 0$. Hence, the vector field is continuously differentiable on $\mathbb{R}^5_+$ and therefore locally Lipschitz continuous. By the Picard-Lindelöf (Cauchy–Lipschitz) theorem, the system admits a unique local solution for the given non–negative initial conditions.

Moreover, since all solutions are bounded in a positively invariant region (as established in Theorem 3.3), no finite–time blow–up can occur. Consequently, the local solution extends uniquely to a global solution defined for all $t \geq 0$. □

Theorem 3.2. *For any non-negative initial conditions* $(S_p, I_L, I_H, I_C, R_p)(0)$*, the model solutions remain non-negative for all time t.*

Proof. We prove positivity using the comparison principle. Consider the susceptible population S_p,

$$\frac{dS_p}{dt} = \mu_p N_p - \beta_L \frac{I_L}{N_p} S_p - \beta_H \frac{I_H}{N_p} S_p + r R_p - \mu_p S_p.$$

Since $\mu_p N_p \geq 0$ and $r R_p \geq 0$, we remove these positive terms to obtain

$$\frac{dS_p}{dt} \geq -\left(\beta_L \frac{I_L}{N_p} + \beta_H \frac{I_H}{N_p} + \mu_p\right) S_p.$$

Dividing both sides by S_p and integrating from 0 to t yields

$$\ln S_p(t) \; - \; \ln S_p(0) \; \geq \; - \int_0^t \left(\beta_L \frac{I_L(\tau)}{N_p} + \beta_H \frac{I_H(\tau)}{N_p} + \mu_p\right) d\tau \; \geq \; 0.$$

Exponentiating both sides, we obtain

$$S_p(t) \; \geq \; S_p(0) \; \exp\!\left(-\int_0^t \psi(\tau)\, d\tau\right) \; \geq 0 \quad \text{for all } t \geq 0,$$

where

$$\psi(\tau) = \left(\beta_L \frac{I_L(\tau)}{N_p} + \beta_H \frac{I_H(\tau)}{N_p} + \mu_p\right).$$

Hence, $S_p(t)$ remains non-negative for all $t \geq 0$. By applying the same argument to the remaining equations of the system we get

$$I_L(t) \; \geq \; I_L(0) \; \exp\!\left(-\int_0^t \left(\beta_{LH} \frac{I_H(\tau)}{N_p} + \gamma_L + \mu_p\right) d\tau\right) \geq 0,$$

$$I_H(t) \; \geq I_H(0) \; \exp\!\left(-\int_0^t \left(\beta_{HL} \frac{I_L(\tau)}{N_p} + \gamma_H + \mu_p\right) d\tau\right) \geq 0,$$

$$I_C(t) \; \geq I_C(0) \; \exp\!\left(-\int_0^t (\gamma_C + \mu_p)\, d\tau\right) \geq 0,$$

$$R_p(t) \; \geq \; R_p(0) \; \exp\!\left(-\int_0^t (r + \mu_p)\, d\tau\right) \geq 0.$$

Therefore, all state variables $S_p(t)$, $I_L(t)$, $I_H(t)$, $I_C(t)$, and $R_p(t)$ remain non-negative for time t. □

Theorem 3.3. *The model solutions are uniformly bounded in the positively invariant region* (Ω_{LH})

$$\Omega_{LH} = \left\{(S_p, I_L, I_H, I_C, R_p) \in \mathbb{R}^5_+ : S_p + I_L + I_H + I_C + R_p \leq N_p\right\}.$$

Proof. Let the total population be

$$N_p = S_p + I_L + I_H + I_C + R_p.$$

Summing all equations of the system gives

$$\frac{dN_p}{dt} = \mu_p N_p - \mu_p N_p = 0.$$

Hence

$$N_p(t) = N_p(0) \quad \text{for all } t \geq 0.$$

Therefore, human population remains constant and bounded. Since each compartment is non-negative and their sum is bounded by N_p all state variables are uniformly bounded.

Thus, the region

$$\Omega_{LH} = \left\{(S_p, I_L, I_H, I_C, R_p) \in \mathbb{R}^5_+ : S_p + I_L + I_H + I_C + R_p \leq N_p\right\}$$

is positively invariant. □

Remark 3.1. The invariance of Ω_{LH} ensures that the model is epistemologically well posed, as the total population is conserved and no compartment exceeds biologically meaningful bounds.

3.1. Equilibrium points of leptospirosis and hantavirus co-infection model

An equilibrium point of the system is defined as a state at which the population variables remain constant over time, that is,

$$\frac{dS_p}{dt} = \frac{dI_L}{dt} = \frac{dI_H}{dt} = \frac{dI_C}{dt} = \frac{dR_p}{dt} = 0.$$

Solving the resulting algebraic system leads to two biologically meaningful equilibria.

(i) Transmission–Free Equilibrium. The equilibrium corresponding to the complete absence of infection in the population is called the Transmission–Free Equilibrium and is denoted by $\mathcal{E}^o_{\mathrm{TF}}$. This notation is used throughout the paper to represent the disease–free state of the system. Setting $I_L = I_H = I_C = 0$, we obtain the unique Transmission–Free Equilibrium

$$\boxed{\mathcal{E}^o_{\mathrm{TF}} = (N_p,\ 0,\ 0,\ 0,\ 0)\,.}$$

This equilibrium always exists and represents a completely susceptible population.

(ii) Persistent Infection Equilibrium. The equilibrium associated with sustained transmission of infection is called the Persistent Infection Equilibrium and is denoted by $\mathcal{E}^*_{\mathrm{PI}}$. When at least one infection lies in the population, the system admits a Persistent Infection Equilibrium of the form

$$\boxed{\mathcal{E}^*_{\mathrm{PI}} = \left(S^*_p,\ I^*_L,\ I^*_H,\ I^*_C,\ R^*_p\right).}$$

The components of $\mathcal{E}^*_{\mathrm{PI}}$ is

$$S_p^* = \frac{N_p}{\beta_L}\left(\gamma_L + \mu_p + \beta_{LH}\frac{I_H^*}{N_p}\right) = \frac{N_p}{\beta_H}\left(\gamma_H + \mu_p + \beta_{HL}\frac{I_L^*}{N_p}\right),$$

$$I_C^* = \frac{(\beta_{LH}+\beta_{HL})\, I_L^* I_H^*}{N_p(\gamma_C+\mu_p)}, \quad R_p^* = \frac{\gamma_L I_L^* + \gamma_H I_H^* + \gamma_C I_C^*}{r+\mu_p}, \quad I_H^* = \frac{N_p(\gamma_C+\mu_p)I_C^*}{(\beta_{LH}+\beta_{HL})I_L^*}.$$

Theorem 3.4. *If the Persistent Infection Equilibrium of system 2.1 exists, then it is unique in the biologically feasible region*

$$\Omega_{LH} = \left\{(S_p, I_L, I_H, I_C, R_p) \in \mathbb{R}^5_+ :\ S_p + I_L + I_H + I_C + R_p = N_p\right\}.$$

Proof. Assume that an Persistent Infection Equilibrium $\mathcal{E}^*_{PI} = (S_p^*, I_L^*, I_H^*, I_C^*, R_p^*)$ exists with$I_L^* > 0$ and $I_H^* > 0$. At equilibrium, the infected subsystem satisfies

$$S_p^* = \frac{N_p}{\beta_L}\left(\gamma_L + \mu_p + \beta_{LH}\frac{I_H^*}{N_p}\right), \tag{3.1}$$

$$S_p^* = \frac{N_p}{\beta_H}\left(\gamma_H + \mu_p + \beta_{HL}\frac{I_L^*}{N_p}\right). \tag{3.2}$$

From equation (3.1) susceptible population S_p^* can be expressed a strictly increasing function of I_H^*, and from (3.2) the susceptible population S_p^* can be expressed a strictly increasing function of I_H^*. Equating the two expressions for S_p^* yields a system of two algebraic equations in the two unknowns (I_L^*, I_H^*). Each equation defines a linear, monotone increasing relationship between the infected classes.

Since the right-hand sides are continuous and strictly increasing functions, the corresponding curves intersect at exactly one point in the positive quadrant. Hence, the pair (I_L^*, I_H^*) is unique.

Given the uniqueness of I_L^* and I_H^*, the remaining equilibrium components

$$I_C^* = \frac{(\beta_{LH}+\beta_{HL})I_L^* I_H^*}{N_p(\gamma_C+\mu_p)}, \qquad R_p^* = \frac{\gamma_L I_L^* + \gamma_H I_H^* + \gamma_C I_C^*}{r+\mu_p}$$

are uniquely determined.

Therefore, the Persistent Infection Equilibrium $(\mathcal{E}^*_{PI})$ is unique in Ω_{LH}. □

Summary. The model admits a unique Transmission–Free Equilibrium $\mathcal{E}^o_{TF}$ and a unique Persistent Infection Equilibrium $\mathcal{E}^*_{PI}$ whenever infection invasion is possible.

3.2. Derivation of the dominant transmission threshold

The basic reproduction number, is measure the number of secondary infections generated by a typical infected individual in a fully susceptible population, in this study called the dominant transmission threshold and is denoted by $\mathcal{R}_0^c$. To derive this threshold quantity, we apply the next–generation matrix method, which compares the rate of appearance of new infections to the rate of removal from infected compartments. The infected subsystem of the model consists of the compartments (I_L, I_H, I_C). Among these, the single–infection classes I_L and I_H generate new infections, whereas the coinfected class I_C does not contribute directly to disease transmission.

Let $\mathbf{x} = (I_L, I_H, I_C)^T$. Following the standard formulation, the dynamics of the infected classes can be written as

$$\frac{d\mathbf{x}}{dt} = \mathcal{F}_c(\mathbf{x}) - \mathcal{V}_c(\mathbf{x}),$$

where $\mathcal{F}_c$ represents the rate of new infection and $\mathcal{V}_c$ represents the rate of transfer and removal.

$$\mathcal{F}_c(\mathbf{x}) = \begin{bmatrix} \beta_L \dfrac{I_L}{N_p} S_p \\ \beta_H \dfrac{I_H}{N_p} S_p \\ \beta_{HL} \dfrac{I_H}{N_p} I_L + \beta_{HL} \dfrac{I_L}{N_p} I_H \end{bmatrix}, \qquad \mathcal{V}_c(\mathbf{x}) = \begin{bmatrix} \beta_{HL} \dfrac{I_H}{N_p} I_L + (\gamma_L + \mu) I_L \\ \beta_{HL} \dfrac{I_L}{N_p} I_H + (\gamma_H + \mu) I_H \\ (\gamma_C + \mu) I_C \end{bmatrix}.$$

Evaluating the system at the transmission–free equilibrium, the Jacobian matrix of the new infection terms is denoted by $\mathcal{F}_{k1}$, while the Jacobian matrix of the removal terms is denoted by $\mathcal{V}_{k2}$. These matrices are given by

$$\mathcal{F}_{k1} = \frac{\partial \mathcal{F}_c}{\partial (I_L, I_H, I_C)} = \begin{bmatrix} \beta_L & 0 & 0 \\ 0 & \beta_H & 0 \\ 0 & 0 & 0 \end{bmatrix}, \qquad \mathcal{V}_{k2} = \frac{\partial \mathcal{V}_c}{\partial (I_L, I_H, I_C)} = \begin{bmatrix} \gamma_L + \mu_p & 0 & 0 \\ 0 & \gamma_H + \mu_p & 0 \\ 0 & 0 & \gamma_C + \mu_p \end{bmatrix}.$$

The next–generation matrix is defined as FV^{-1}, which yields

$$\mathcal{F}_{k1} \mathcal{V}_{k2}^{-1} = \begin{bmatrix} \dfrac{\beta_L}{\gamma_L + \mu_p} & 0 & 0 \\ 0 & \dfrac{\beta_H}{\gamma_H + \mu_p} & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

The threshold quantity is obtained as the spectral radius of $\mathcal{F}_{k1} \mathcal{V}_{k2}^{-1}$. Hence

$$\mathcal{R}_0^c = \max\left\{ \frac{\beta_L}{\gamma_L + \mu_p}, \frac{\beta_H}{\gamma_H + \mu_p} \right\}, \qquad \text{where} \quad \mathcal{R}_0^L = \frac{\beta_L}{\gamma_L + \mu_p}, \quad \mathcal{R}_0^H = \frac{\beta_H}{\gamma_H + \mu_p}.$$

The dominant transmission threshold ($\mathcal{R}_0^c$) reflects the most transmissible single–infection pathway and determines whether infection can invade the population. Co-infection contributes to disease burden but does not modify the invasion threshold.

4. Stability analysis of co-infection model

Theorem 4.1. *If $\mathcal{R}_0^c < 1$, then the transmission–free equilibrium ($\mathcal{E}_{TF}^o$) is locally–asymptotically stable in Ω_{LH} and and unstable if $\mathcal{R}_0^c > 1$.*

Proof. To investigate the local stability of transmission–free equilibrium, We compute the Jacobian matrix of the infected subsystem with respect to (I_L, I_H, I_C) and evaluate it at $\mathcal{E}_{TF}^o$.

The Jacobian matrix is

$$J_I(\mathcal{E}_{TF}^o) = \begin{bmatrix} \beta_L - (\gamma_L + \mu_p) & 0 & 0 \\ 0 & \beta_H - (\gamma_H + \mu_p) & 0 \\ 0 & 0 & -(\gamma_C + \mu_p) \end{bmatrix}.$$

The eigenvalues of $J_I(\mathcal{E}_0)$ are

$$\lambda_{k1} = \beta_L - (\gamma_L + \mu_p), \quad \lambda_{k2} = \beta_H - (\gamma_H + \mu_p), \quad \lambda_{k3} = -(\gamma_C + \mu_p).$$

Since $\gamma_C + \mu_p > 0$, we have $\lambda_3 < 0$. Thus, all eigenvalues have negative real parts if and only if

$$\beta_L - (\gamma_L + \mu_p) < 0 \quad \text{and} \quad \beta_H - (\gamma_H + \mu_p) < 0$$

which is equivalent to

$$\frac{\beta_L}{\gamma_L + \mu_p} < 1 \quad \text{and} \quad \frac{\beta_H}{\gamma_H + \mu_p} < 1.$$

Hence if $\mathcal{R}_0^c < 1$, the transmission–free equilibrium is locally–asymptotically stable. □

Theorem 4.2. *The persistence infection equilibrium $\mathcal{E}_{PI}^*$ of system (1) is globally asymptotically-stable in the feasible region (Ω_{LH}) whenever $\mathcal{R}_0^c < 1$.*

Proof. Define a Lyapunov function on the infected classes such that

$$V_{LH}(I_L, I_H, I_C) = I_L + I_H + I_C.$$

Clearly, $V_{LH} \geq 0$ for all $I_L, I_H, I_C \geq 0$, and $V = 0$ if and only if $I_L = I_H = I_C = 0$. Differentiate V_{LH} along solutions of the modem with respect to t

$$\frac{dV_{LH}}{dt} = \frac{dI_L}{dt} + \frac{dI_H}{dt} + \frac{dI_C}{dt}.$$

Substituting the model equations yields

$$\frac{dV_{LH}}{dt} = \beta_L \frac{S_p}{N_p} I_L - (\gamma_L + \mu_p) I_L + \beta_H \frac{S_p}{N_p} I_H - (\gamma_H + \mu_p) I_H - (\gamma_C + \mu_p) I_C.$$

Since $0 < S_p \leq N_p$ for all t, we have $\frac{S_p}{N_p} \leq 1$. Hence

$$\frac{dV_{LH}}{dt} \leq \big(\beta_L - (\gamma_L + \mu_p)\big) I_L + \big(\beta_H - (\gamma_H + \mu_p)\big) I_H - (\gamma_C + \mu_p) I_C.$$

If

$$\frac{\beta_L}{\gamma_L + \mu_p} < 1 \quad \text{and} \quad \frac{\beta_H}{\gamma_H + \mu_p} < 1, \quad \text{implies that} \quad \beta_L - (\gamma_L + \mu_p) < 0, \quad \beta_H - (\gamma_H + \mu_p) < 0$$

and clearly $\gamma_C + \mu_p > 0$. Therefore, $\frac{dV_{LH}}{dt} \leq 0$ with equality if and only if $I_L = I_H = I_C = 0$.

By Lyapunov's direct method and Lassalle invariance principle, all solutions starting in the feasible region converge to $\mathcal{E}_{TF}^o$ as $t \to \infty$. Thus, the persistence infection equilibrium $\mathcal{E}_{TF}^o$ is globally asymptotically stable. □

Theorem 4.3. *Assume that the persistent infection equilibrium $(\mathcal{E}_{PI}^*)$ exists and If $\mathcal{R}_0^c > 1$ then the persistent infection equilibrium $(\mathcal{E}_{PI}^*)$ of model is locally–asymptotically stable.*

Proof. The Jacobian matrix of the infected subsystem evaluated at $\mathcal{E}_{PI}^*$ is

$$J(\mathcal{E}_{PI}^*) = \begin{bmatrix} \beta_L \dfrac{S_p^*}{N_p} - \beta_{LH} \dfrac{I_H^*}{N_p} - (\gamma_L + \mu_p) & -\beta_{LH} \dfrac{I_L^*}{N_p} & 0 \\ -\beta_{HL} \dfrac{I_H^*}{N_p} & \beta_H \dfrac{S_p^*}{N_p} - \beta_{HL} \dfrac{I_L^*}{N_p} - (\gamma_H + \mu_p) & 0 \\ \beta_{LH} \dfrac{I_H^*}{N_P} + \beta_{HL} \dfrac{I_H^*}{N_p} & \beta_{LH} \dfrac{I_L^*}{N_p} + \beta_{HL} \dfrac{I_L^*}{N_p} & -(\gamma_C + \mu_p) \end{bmatrix}.$$

The eigenvalue corresponding to the third equation is, $\lambda_{k3} = -(\gamma_C + \mu_p) < 0$. Consider the upper-left 2×2 principal sub matrix J_2. At the Persistent Infection Equilibrium, the equilibrium conditions imply

$$\beta_L \frac{S_p^*}{N_p} > (\gamma_L + \mu_p), \qquad \beta_H \frac{S_p^*}{N_p} > (\gamma_H + \mu_p),$$

which is equivalent to $\mathcal{R}_0^c > 1$.

Hence,

$$\mathrm{tr}(J_2) < 0, \qquad \det(J_2) > 0.$$

Therefore, each eigenvalues of $J(\mathcal{E}_{PI}^*)$ have negative real parts, and the persistent infection equilibrium $\mathcal{E}_{PI}^*$ is locally–asymptotically stable. □

Theorem 4.4. *Assume that the dominant transmission threshold satisfies $\mathcal{R}_0^c > 1$ so that the system admits a unique persistent infection equilibrium $\mathcal{E}_{PI}^* = (S_p^*, I_L^*, I_H^*, I_C^*, R_p^*)$ with all components positive. Then the Persistent Infection Equilibrium $(\mathcal{E}_{PI}^*)$ is globally–asymptotically stable in feasible region Ω_{LH}*

Proof. Define a Lyapunov function,

$$V_{LH} = \left(I_L - I_L^* - I_L^* \ln \frac{I_L}{I_L^*}\right) + \left(I_H - I_H^* - I_H^* \ln \frac{I_H}{I_H^*}\right) + \left(I_C - I_C^* - I_C^* \ln \frac{I_C}{I_C^*}\right).$$

Since the function $h - h^* - h^* \ln \frac{h}{h^*}$ is nonnegative for all $h > 0$ and equals zero if and only if $h = h^*$, it follows that $V_{LH} \geq 0$ in Ω_{LH} and $V_{LH} = 0$ if and only if $(S_p, I_L, I_H, I_C, R_p) = \mathcal{E}_{PI}^*$.

Taking the derivative of V_{LH} along solutions of model, we obtain

$$\frac{dV_{LH}}{dt} = \left(1 - \frac{I_L^*}{I_L}\right)\frac{dI_L}{dt} + \left(1 - \frac{I_H^*}{I_H}\right)\frac{dI_H}{dt} + \left(1 - \frac{I_C^*}{I_C}\right)\frac{dI_C}{dt}.$$

Substituting the system equations into the derivative of the Lyapunov function and using the equilibrium identities satisfied by $\mathcal{E}_{PI}^*$, we obtain

$$\frac{dV_{LH}}{dt} = I_{h1} - I_{h2},$$

where

$$I_{h1} = \beta_L \frac{S_p}{N_p}(I_L - I_L^*) + \beta_H \frac{S_p}{N_p}(I_H - I_H^*) + \frac{\beta_{LH} + \beta_{HL}}{N_p} I_L I_H \left(1 - \frac{I_C^*}{I_C}\right),$$

and

$$\begin{aligned} I_{h2} = {} & \beta_{LH} \frac{I_H}{N_p}(I_L - I_L^*) + \beta_{HL} \frac{I_L}{N_p}(I_H - I_H^*) + (\gamma_L + \mu_p)(I_L - I_L^*) \\ & + (\gamma_H + \mu_p)(I_H - I_H^*) + (\gamma_C + \mu_p)(I_C - I_C^*). \end{aligned}$$

Thus, if $I_{h2} \geq I_{h1}$ then,

$$\begin{aligned} & \frac{dV_{LH}}{dt} \leq 0 \text{ in } \ \Omega_{LH}, \text{ and } \frac{dV_{LH}}{dt} = 0, \\ & \text{if and only if } \ S_p = S_p^*, \ I_L = I_L^*, \ I_H = I_H^*, \ I_C = I_C^*, \ R_p = R_p^*. \end{aligned}$$

Therefore, largest invariant set contained in $\{x \in \Omega_{LH} : \dot{V}_{LH} = 0\}$ is $\{\mathcal{E}_{PI}^*\}$. By Lassalle invariance principle, every solution with initial condition in Ω_{LH} converges to $\mathcal{E}_{PI}^*$ as $t \to \infty$.

Consequently, the Persistent Infection Equilibrium point is globally-asymptotically stable in Ω_{LH}. □

5. Bifurcation analysis

Bifurcation analysis is fundamental qualitative tool use for the study of nonlinear dynamical systems and play essential role in understanding the behavior of models. It describes how the qualitative nature of equilibrium solutions changes as system parameters change. In particular, bifurcation theory explains how small changes in certain parameters, referred to as *bifurcation parameters*, may lead to significant changes in the stability or number of equilibrium points of the system.

For the proposed model, bifurcation analysis is used to show how variations in transmission-related parameters which influence stability of equilibrium points. The value $\mathcal{R}_0^c = 1$ serves as a critical bifurcation point separating disease-free and endemic states.

When $\mathcal{R}_0^c < 1$, the transmission-free equilibrium is locally asymptotically stable, indicating that the infection will die out from population. When $\mathcal{R}_0$ becomes greater than one, the disease-free state is no longer stable, and the infection starts to persist in the population. This transition is commonly associated with a *forward (transcritical) bifurcation*, where the persistent infection equilibrium ($\mathcal{E}_{PI}^*$) becomes locally asymptotically stable for $\mathcal{R}_0 > 1$. To understand the influence of each parameters in disease transmission, bifurcation analysis is complemented by sensitivity analysis.

The bifurcation and sensitivity analyses together provide a complete understanding of nonlinear behavior of the proposed model and investigate important insights for designing effective disease control and prevention strategies.

Confirmation of bifurcation. To confirm the bifurcation analysis in proposed model, we check jacobian matrix of the system at the transmission-free equilibrium his zero eigenvalue when $\mathcal{R}_0^c = 1$.

$$\mathcal{E}_{TF}^o = (I_L^0, I_H^0, I_C^0) = (N_p, 0, 0).$$

Let $J_k(\mathcal{E}_{TF}^o)$ be the Jacobian matrix of the model evaluated $\mathcal{E}_{TF}^o$ then,

$$J_k(\mathcal{E}_{TF}^o) = \begin{bmatrix} \beta_L - (\gamma_L + \mu_p) & 0 & 0 \\ 0 & \beta_H - (\gamma_H + \mu_p) & 0 \\ 0 & 0 & -(\gamma_C + \mu_p) \end{bmatrix}.$$

The eigenvalues of $J_k(\mathcal{E}_{TF}^o)$ are

$$\lambda_1 = \beta_L - (\gamma_L + \mu_p), \quad \lambda_2 = \beta_H - (\gamma_H + \mu_p), \quad \lambda_3 = -(\gamma_C + \mu_p).$$

Define the threshold parameters

$$\mathcal{R}_0 = \max\{\mathcal{R}_0^L, \mathcal{R}_0^H\}, \qquad \mathcal{R}_0^L = \frac{\beta_L}{\gamma_L + \mu_p}, \qquad \mathcal{R}_0^H = \frac{\beta_H}{\gamma_H + \mu_p}.$$

When $\mathcal{R}_0^c < 1$, all eigenvalues of $J(\mathcal{E}_{TF}^o)$ have negative real parts, and hence the transmission-free equilibrium is locally asymptotically stable. As $\mathcal{R}_0^c$ increases and crosses the critical value such that $\mathcal{R}_0^c = 1$, at least one eigenvalue becomes zero while the remaining eigenvalues remain negative. This change in stability confirms the occurrence of bifurcation at $\mathcal{R}_0^c = 1$. Therefore, the transmission free equilibrium undergoes a transcritical bifurcation when $\mathcal{R}_0^c = 1$, leading to the emergence of an Persistent Infection Equilibrium (E_{PI}^*) when $\mathcal{R}_0 > 1$.

Direction of the bifurcation with respect to β_L. We analyze the direction of the bifurcation occurring at thetransmission free equilibrium ($\mathcal{E}^o_{TF}$) of the infected subsystem by applying center manifold theory of Castillo–Chavez and Song. The transmission rate β_L is chosen as the bifurcation parameter.

Let $x = (x_1, x_2, x_3)^T = (I_L, I_H, I_C)^T$.

The Jacobian matrix of the infected sub system evaluated at E_0 is

$$J_I(E_0) = \begin{bmatrix} \beta_L - (\gamma_L + \mu_p) & 0 & 0 \\ 0 & \beta_H - (\gamma_H + \mu_p) & 0 \\ 0 & 0 & -(\gamma_C + \mu_p) \end{bmatrix}.$$

Setting

$$\beta_L = \beta_L^* = \gamma_L + \mu_p, \quad \beta_H < \gamma_H + \mu_p,$$

the Jacobian has a single zero eigenvalue, while the remaining eigenvalues are strictly negative.

Right and left eigenvectors. Let $U_k = (u_{k1}, u_{k2}, u_{k3})^T$ is right eigenvector associated with the zero eigenvalue then,

$$J_I(E_0)U_k = 0, \qquad \begin{bmatrix} \beta_L - (\gamma_L + \mu_p) & 0 & 0 \\ 0 & \beta_H - (\gamma_H + \mu_p) & 0 \\ 0 & 0 & -(\gamma_C + \mu_p) \end{bmatrix} \cdot \begin{bmatrix} u_{k1} \\ u_{k2} \\ u_{k3} \end{bmatrix} = \begin{bmatrix} 0 \\ 0 \\ 0 \end{bmatrix},$$

yields

$$u_{k2} = u_{k3} = 0, \quad u_{k1} \neq 0.$$

Choosing $u_{k1} = 1$, we obtain

$$U_k = (1, 0, 0)^T.$$

Similarly, the left eigenvector $V_k = (v_{k1}, v_{k2}, v_{k3})$ satisfies

$$V_k J_I(E_0) = 0, \qquad \text{where} \quad V_k = (1, 0, 0)$$

which gives

$$V_k J_I(E_0) = 0, \quad \text{implies that} \quad \begin{cases} \beta_L^* - (\gamma_L + \mu_P)v_{k1} & = 0 \\ \beta_H - (\gamma_H + \mu_P)v_{k2} & = 0 \\ -(\gamma_C + \mu_P)v_{k3} & = 0 \end{cases} \quad \text{implies that} \quad V_k = (1, 0, 0).$$

Computation of bifurcation coefficients. Let f_{k1} denote the $k1$-th component of the reduced system. Since $V = (1, 0, 0)$, only the first equation contributes to the bifurcation coefficients.

The coefficient a is defined by

$$a_k = \sum_{i,j,k} v_{k1} u_{i1} u_{j1} \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\mathcal{E}^o_{TF}), \quad a_k = \frac{\partial f_1^2}{\partial x_1^2} \quad \text{implies that} \quad a_k = 0.$$

The coefficient b_k is given by

$$b_k = \sum_{i,k} v_{k1} u_{i1} \frac{\partial^2 f_k}{\partial x_i \partial \beta_L}(\mathcal{E}^o_{TF}) = \frac{\partial^2 f_1}{\partial I_L \partial \beta_L} = 1 \quad \text{implies that} \quad b_k > 0.$$

Since $a_k = 0$ and $b_k > 0$ the system his backward transcritical bifurcation at the critical parameter value. A similar result is obtained when β_H is taken as the bifurcation parameter.

6. Sensitivity analysis of leptospirosis, hantavirus co-infection

The relative importance of model parameters on the transmission dynamics of leptospirosis and hantavirus co-infection is investigated using normalized forward sensitivity analysis. Sensitivity indices provide quantitative information on how variations in parameters influence the dominant transmission threshold. This analysis is particularly useful for identifying key parameters that should be targeted by control strategies. The normalized forward sensitivity index of the basic reproduction number $\mathcal{R}_0^c$ with respect to a parameter p_i is defined as

$$\Upsilon_{p_i}^{\mathcal{R}_0^c} = \frac{\partial \mathcal{R}_0^c}{\partial p_i} \cdot \frac{p_i}{\mathcal{R}_0^c}.$$

From this index we measures the proportional change in $\mathcal{R}_0^c$ due to proportional change in the parameter p_i. A positive sensitivity index indicates that increase in parameter leads to increase in $\mathcal{R}_0^c$, whereas a negative index implies a reducing effect on disease transmission. Using the above formulation, the sensitivity indices of $\mathcal{R}_0^c$ with respect to the epidemiological parameters are computed and summarized in Table 2.

Table 2. Normalized sensitivity indices of the $\mathcal{R}_0^c$.

Parameter	Sensitivity index	Direction of effect	Qualitative impact
β_L	$+1$	Positive	Maximum effect
γ_L	-0.9994	Negative	Maximum reducing effect
β_H	$+1$	Positive	Maximum effect
γ_H	-0.9977	Negative	Strong reducing effect
μ_p	-0.0023	Negative	Minimal effect

The results indicate that the transmission rates of leptospirosis and hantavirus (β_L and β_H) have the strongest positive influence on the $\mathcal{R}_0^c$, with sensitivity indices equal to unity. This implies that a 20% increase in any parameter related transmission leads to corresponding 20% increase in $\mathcal{R}_0^c$. On the other hand recovery rates γ_L and γ_H his negative influence, on $\mathcal{R}_0^c$ which show that improvements in treatment and recovery largely reduce disease spread. The natural death rate μ_p has a negligible effect on $\mathcal{R}_0^c$, suggesting that demographic effects play a minor role as compared to transmission and recovery rates. These findings highlight the importance of reducing environmental exposure and improving clinical management to treat leptospirosis and hantavirus infections.

7. Numerical simulation of co-infection model

This section provides, numerical simulations of leptospirosis and hantavirus co-infection model in order to illustrate and support the qualitative results developed in the above analytical section of the model. Two parameter regimes are considered: One corresponding to $\mathcal{R}_0^c < 1$, where the disease is expected to die out, and another corresponding to $\mathcal{R}_0^c > 1$, where the disease persists and approaches an persistence infection equilibrium. These numerical experiments confirm the theoretical predictions regarding stability and long-term behavior of the model. This final part validates the theoretical results through numerical simulations performed in Goggle Colab by using Rk4 method. The parameter sets corresponding to the cases $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 > 1$ are taken from already published papers and some are calculated and fitted with model that summarized in Table 3.

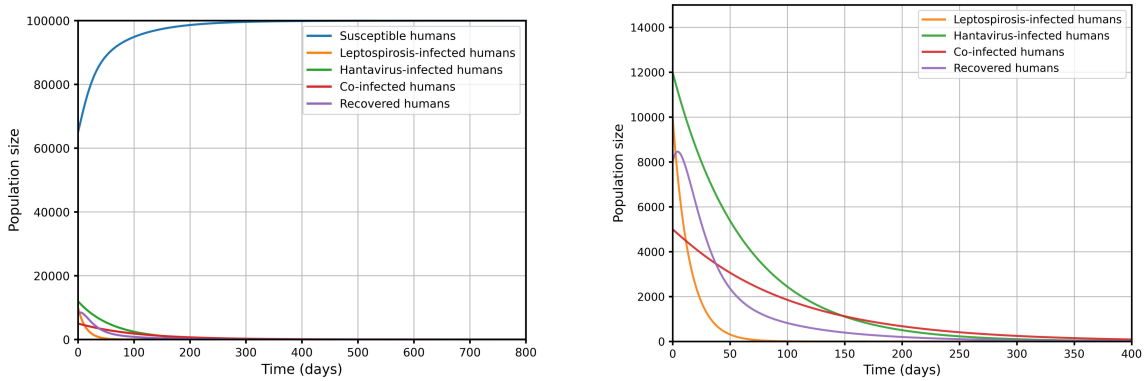
Table 3. Parameter values for $\mathcal{R}_0^c < 1$ and $\mathcal{R}_0^c > 1$.

Parameter	$\mathcal{R}_0^c < 1$	$\mathcal{R}_0^c > 1$	Reference
μ_p	0.000913	0.000913	[6]
γ_H	0.0167	0.0167	[Calculated]
γ_L	0.0714285714	0.0714285714	[6]
γ_C	0.01	0.01	[fitted]
r	0.089	0.089	[6]
β_L	0.001	0.15	[Calculated]
β_{LH}	0.001	0.001	[fitted]
β_H	0.001	0.0456	[Calculated]
β_{HL}	0.0030	0.028	[fitted]

Figures 2–3 illustrate the dynamical behavior of the model under two epidemiological regimes. When the dominant transmission threshold satisfies $\mathcal{R}_0^c = 0.0597 < 1$, the solutions goes toward the $\mathcal{E}_{TF}^o$ shown in Figure 2(a), where all infected and co-infected together with recovered compartments, become zero shown in Figures 2(b). Conversely, when the dominant transmission threshold exceeds unity, specifically $\mathcal{R}_0^c = 2.7242 > 1$, the system settles at a stable Persistent Infection Equilibrium and the solution tend to $\mathcal{E}_{PI}^* = (46869.13, 17449.04, 12530.78, 6380.01, 16771.03)$ shown in Figure 3(a) and Figure 3(b).

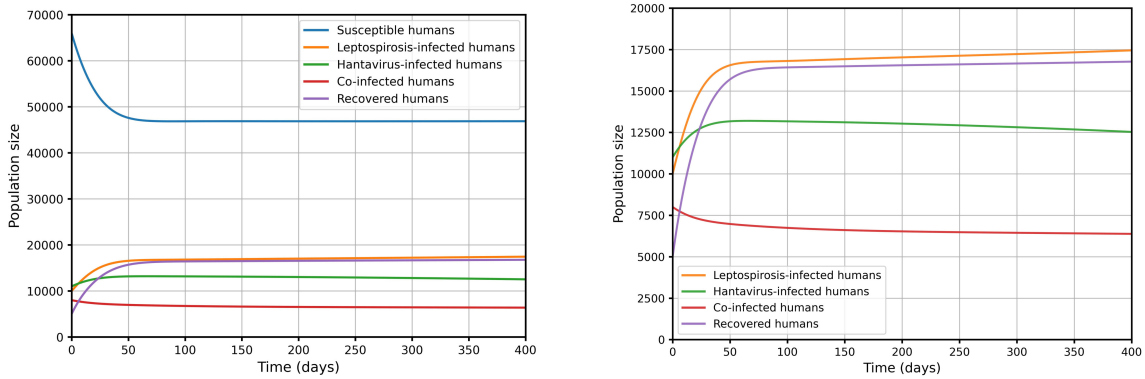
From the analytical expression of $\mathcal{R}_0^c$, it is clear that the parameters β_L, β_H, γ_H, and γ_L have a significant influence on the leptospirosis hantavirus co-infection transmission dynamics. In particular, the transmission rates of leptospirosis (β_L) and hantavirus (β_H) appear explicitly in $\mathcal{R}_0^c$, indicating their direct contribution to disease spread. An increase in either β_L in Figure 4(a) or β_H Figure 5(a) leads to a proportional increase in $\mathcal{R}_0$, there by enhancing the likelihood of sustained transmission and endemic persistence of the diseases. While Figure 4(b) and Figure 5(b) show the effect of β_{LH} and β_{HL} on leptospirosis infected humans and hantavirus infected humans respectively.

Figure 6 illustrates the dynamics of the co-infected human population $I_C(t)$ under different values of the transmission parameters β_{LH} and β_{HL}. These parameters represent the transmission rates contributing to the progression of individuals into the co-infected class. The Figure 6(b) shows increasing values of β_{LH} lead to a noticeable change in the trajectory of the



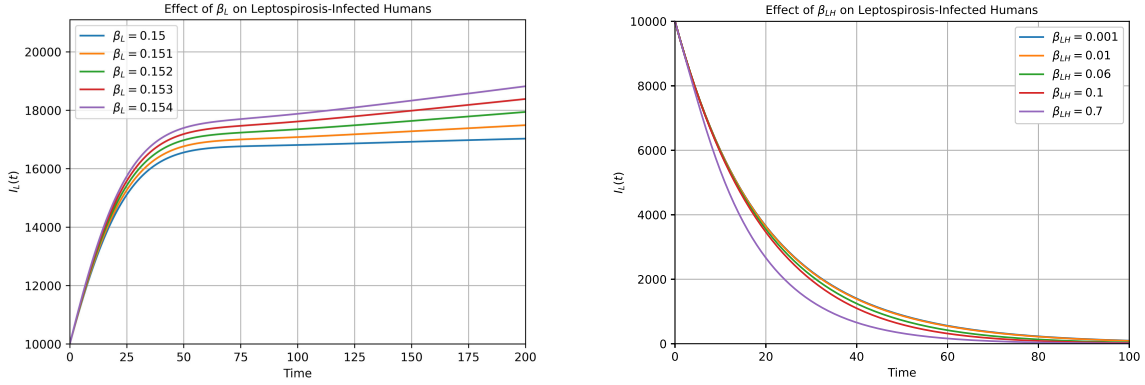
(a) (b)

Figure 2. Human population under $\mathcal{R}_0^c < 1$.



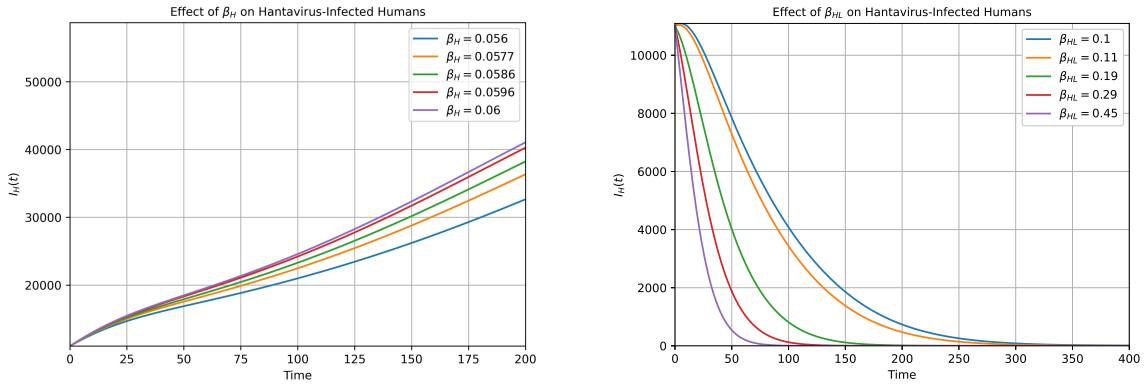
(a) (b)

Figure 3. Human population under $\mathcal{R}_0^c > 1$.



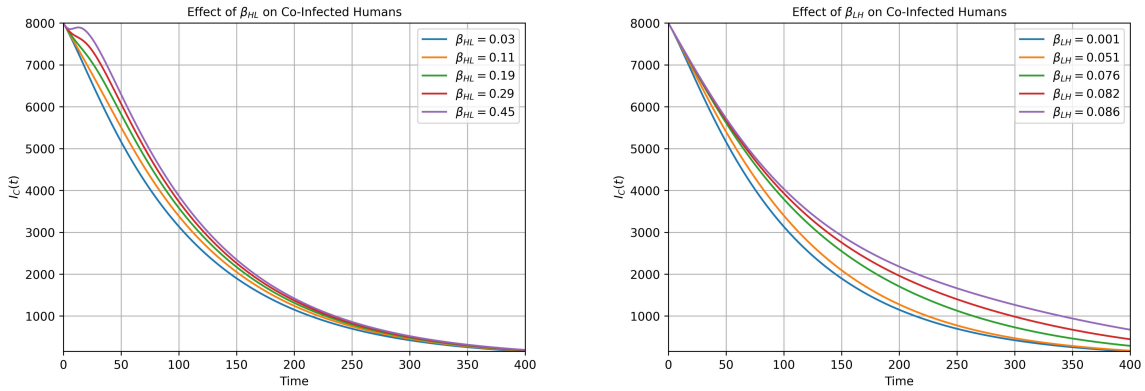
(a) (b)

Figure 4. Effect of β_L and β_{LH} on leptospirosis infected humans.



(a) (b)

Figure 5. Effect of β_H and β_{HL} on hantavirus infected humans.

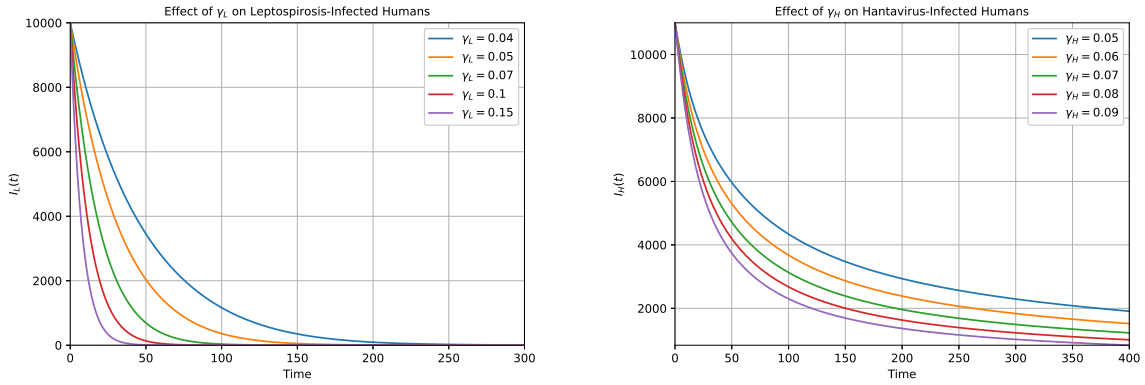


(a) (b)

Figure 6. Effect of β_{HL} and β_{LH} on co-infected humans.

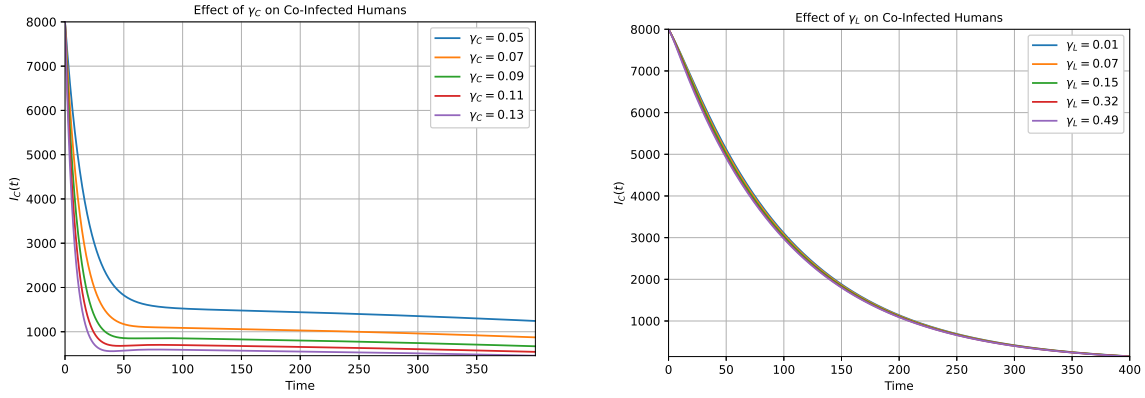
co-infected population. Higher transmission intensity accelerates the accumulation of co-infected individuals and sustains a larger burden over time. Although $I_C(t)$ eventually declines due to recovery and disease-induced mortality, larger values of β_{LH} significantly delay this decline, indicating prolonged persistence of co-infection within the population. Similarly, in Figure 6(a) show the influence of β_{HL} on the co-infected class. An increase in β_{HL} results in consistently higher levels of co-infected individuals throughout the simulation period. This behavior confirms that stronger cross-transmission between infected compartments enhances the risk of co-infection and increases the overall disease burden.

Figure 7–8 illustrates the impact of recovery rates on the dynamics of leptospirosis-infected, hantavirus-infected, and co-infected human populations. Figure 7(a) shows the effect of increasing the γ_L on the class $I_L(t)$. As γ_L increases, the number of individuals infected with leptospirosis declines more rapidly, which show that higher recovery rates reduce the infectious period and reduce disease persistence. Similarly, Figure 7(b) show the influence of the hantavirus recovery rate γ_H on the hantavirus-infected population $I_H(t)$. An increase in γ_H show fast decrease in infected individual over time. This confirms that enhancing recovery mechanisms,



(a) (b)

Figure 7. Effect of γ_L and γ_H on I_L and I_H respectively.



(a) (b)

Figure 8. Effect of γ_C and γ_L on I_C and I_L respectively.

such as effective treatment or immune response, plays a crucial role in controlling hantavirus transmission.

Figure 8 show the effect of recovery on the co-infected compartment I_C. Figure 8(a) show that increasing the co-infection recovery rate γ_C effectively reduces the size and duration of the co-infected class. Which show the importance of targeted treatment strategies for individuals suffering from both infections simultaneously. While Figure 8(a) and Figure 8(b) show the effect of γ_C and γ_L on co-infected humans.

Overall, the results show that the recovery rates γ_L, γ_H, and γ_C play important role in controlling the disease. When recovery increases, the number of single infections and co-infections decreases. In particular, faster recovery greatly reduces co-infection cases, helping in the long-term control and possible elimination of leptospirosis–hantavirus co-infection.

8. Conclusion

In this paper, we develop a deterministic mathematical model to investigate the transmission dynamics of leptospirosis and hantavirus co-infection in human populations. The qualitative

analysis of the model established several fundamental mathematical properties such as Positivity and boundedness of solutions, transmission-free equilibrium ($\mathcal{E}^o_{TF}$), and the persistence infection equilibrium ($\mathcal{E}^*_{PI}$) to ensure the biological feasibility of the model. The transmission-free equilibrium represents a disease-free scenario in which neither leptospirosis nor hantavirus persists in the population, while the persistence infection equilibrium corresponds to sustained transmission of one or both infections, including the possibility of co-infection. A key contribution of this study is the derivation and analysis of the dominant transmission threshold ($\mathcal{R}^c_0$) by using the next-generation matrix-approach. Specifically, when $\mathcal{R}^c_0 < 1$, the transmission-free equilibrium ($\mathcal{E}^o_{TF}$) is locally asymptotically stable, indicating that both infections will eventually die out from the population. On the other hand, when $\mathcal{R}^c_0 > 1$, the transmission-free equilibrium loses stability and the persistence infection equilibrium $\mathcal{E}^*_{PI}$ arising which show persistence of infection. This threshold condition show the critical role of $\mathcal{R}^c_0$ as a dominant indicator of epidemic potential in co-infection model. The direction of bifurcation at the critical threshold $\mathcal{R}^c_0 = 1$ was also investigated using center manifold theory. The analysis confirmed that the model undergoes a backward bifurcation, implying that the emergence of endemic infection occurs smoothly as the reproduction number crosses unity. This result is biologically important, as it suggests that small increases in transmission rates beyond the threshold can gradually lead to persistent infections. In particular, the numerical simulation confirmed that the trajectories of compartments converge to transmission-free equilibrium when $\mathcal{R}^c_0 < 1$ and to the persistence infection equilibrium when $\mathcal{R}^c_0 > 1$. The relative importance of model parameters on the transmission dynamics of leptospirosis and hantavirus co-infection is investigated using normalized forward sensitivity analysis. Sensitivity indices provide quantitative information on how variations in parameters influence the dominant transmission threshold. This analysis is particularly useful for identifying key parameters that should be targeted for control strategies. From an epidemiological point of view, the results highlight the importance of all control strategies targeting both pathogens simultaneously. Control measures such as reducing human contact with contaminated environments, improving sanitation, controlling rodent populations, and early diagnosis and treatment can effectively reduce the reproduction number and limit co-infection prevalence. Work with only one infection in regions where both pathogens co-circulate may be insufficient to eliminate disease transmission. As a future extension of the present co-infection model, we plan to incorporate rodent dynamics and environmental bacterial transmission in order to investigate their impact on disease spread, perform sensitivity analysis to identify key influential parameters.

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Conflict of interest. We declare no conflict of interest.

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