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Development of Deterministic-Stochastic Mathematical Models for Predicting Zoonotic Disease Transmission Dynamics in Tropical Regions

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ABSTRACT

Leptospirosis is a re-emerging zoonotic disease involving complex interactions between humans, animal reservoirs, and the environment. This study investigates leptospirosis transmission using a coupled human–rodent SIR–SIR model under deterministic and stochastic frameworks. The stochastic formulation is developed using stochastic differential equations (SDEs) to incorporate environmental and demographic randomness. The basic reproduction number (R_0) was derived analytically to determine invasion thresholds. Deterministic analysis shows that when $R_0 > 1$, the disease persists and converges to an endemic equilibrium. In contrast, stochastic simulations reveal substantial variability in transmission dynamics and demonstrate the possibility of disease extinction even under conditions that deterministically predict persistence. Sensitivity analysis identifies the rodent-to-human transmission rate and the human recovery rate as the most influential parameters governing R_0. These findings highlight the limitations of purely deterministic models and emphasize the importance of stochastic approaches for capturing realistic zoonotic disease dynamics. The proposed framework provides insights for developing integrated control strategies combining reservoir management, environmental intervention, and early treatment.

Keywords: *Leptospirosis, Zoonotic Disease, Deterministic Model, Stochastic Model, Basic Reproduction Number, Epidemic Dynamics*

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INTRODUCTION

Zoonotic diseases, defined as infectious diseases that are naturally transmitted between vertebrate animals and humans, continue to pose a substantial and growing threat to global public health. It is estimated that more than 60% of emerging infectious diseases are zoonotic in origin, with the majority originating from wildlife reservoirs (WHO, 2022). The burden of zoonotic diseases is disproportionately concentrated in tropical and subtropical regions, where ecological complexity, high biodiversity, and socioeconomic vulnerability converge to facilitate pathogen spillover and sustained transmission (Magalhães et al., 2023; Meisner et al., 2025).

Tropical regions are characterized by intense human–animal–environment interactions driven by deforestation, agricultural intensification, rapid urban expansion, and climate variability. These processes disrupt natural ecosystems and increase contact rates between humans, domestic animals, and wildlife reservoirs, thereby amplifying the risk of zoonotic disease emergence (Purse et al., 2020; Rees et al., 2021). In addition, climatic factors such as high rainfall, humidity, and temperature enhance pathogen survival in environmental media, further complicating disease transmission dynamics (Antima & Banerjee, 2025). Consequently, zoonotic diseases in tropical settings often exhibit pronounced spatiotemporal heterogeneity and unpredictable outbreak patterns.

Among neglected tropical zoonoses, leptospirosis represents a paradigmatic example of an environmentally mediated bacterial disease with complex transmission pathways. Caused by pathogenic *Leptospira* species, leptospirosis is maintained in nature by a wide range of mammalian reservoirs, particularly rodents, which shed the bacteria into the environment through urine (Engida et al., 2022; Sánchez-Soto et al., 2024). Human infection typically occurs through direct or indirect contact with contaminated water or soil, often during flooding events or in areas with poor sanitation. As a result, leptospirosis transmission is highly sensitive to environmental disturbances and stochastic climatic events, making it an ideal candidate for stochastic modeling approaches (Yang & Ferreira, 2008; Rees et al., 2021).

Mathematical modeling has long been recognized as a powerful tool for understanding the mechanisms underlying infectious disease transmission and for informing public health interventions. Deterministic compartmental models, such as SIR and SEIR frameworks, have been extensively applied to zoonotic diseases to describe average transmission dynamics and to derive analytical thresholds such as the basic reproduction number R_0(Keeling & Rohani, 2011; Brauer et al., 2019). These models provide valuable insights into equilibrium behavior, endemic persistence, and the potential impact of control strategies. However, deterministic models inherently assume homogeneous mixing and constant parameters, assumptions that are often violated in real-world zoonotic systems, particularly in heterogeneous tropical environments.

To address these limitations, stochastic epidemic models have been increasingly employed to incorporate randomness arising from demographic variability, environmental fluctuations, and behavioral heterogeneity. Stochastic formulations based on continuous-time Markov chains or stochastic differential equations (SDEs) are especially relevant for zoonotic diseases with small population sizes or strong environmental drivers (Allen, 2017; Maity & Mandal, 2024). Importantly,

stochastic models can predict disease extinction even when deterministic models suggest persistence, highlighting the critical role of random effects in early outbreak dynamics and low-prevalence settings (Saber & Alahmari, 2025).

Recent advances in zoonotic disease modeling have emphasized the integration of deterministic and stochastic approaches within unified frameworks. Hybrid deterministic–stochastic models have been successfully applied to a range of tropical zoonoses, including leptospirosis, cryptosporidiosis, toxocariasis, and Taenia solium infections (Engida et al., 2022; Luhanda et al., 2023; Antonopoulos et al., 2025). Furthermore, extensions incorporating environmental compartments, climate drivers, and fractional-order derivatives have demonstrated improved realism in capturing memory effects and long-term persistence in tropical disease systems (Jan et al., 2022; Althubyani et al., 2025). Despite these developments, many studies either focus exclusively on deterministic analysis or employ stochastic models without systematically comparing their outcomes, limiting their practical applicability for decision-making.

In this context, there remains a clear need for comprehensive deterministic–stochastic modeling frameworks that explicitly compare average and probabilistic disease dynamics within a single coherent structure. Such frameworks are particularly valuable for tropical zoonoses, where uncertainty and variability are intrinsic features of the transmission process. Moreover, identifying key parameters that drive transmission under both deterministic and stochastic regimes is essential for designing robust and adaptive control strategies.

Therefore, the present study develops and analyzes a deterministic–stochastic mathematical model to investigate zoonotic disease transmission dynamics in tropical regions, using leptospirosis as a representative case study. Unlike most existing leptospirosis models that rely solely on deterministic formulations or treat stochastic effects in isolation, this study integrates a coupled human–rodent SIR framework with stochastic differential equations based on Wiener processes and provides a systematic comparison between deterministic and stochastic outcomes within a unified modeling structure. Numerical simulations are performed to compare deterministic trajectories with stochastic realizations, quantify extinction probabilities, and assess the influence of transmission and recovery parameters on the basic reproduction number R_0. By explicitly demonstrating how stochasticity can induce disease extinction even when deterministic models predict persistence, this study offers novel insights into zoonotic transmission dynamics and provides a more realistic and flexible framework to support evidence-based surveillance and control strategies in tropical settings.

METHODS

This study employed a mathematical modeling approach to analyze zoonotic disease transmission dynamics in tropical regions using a combined deterministic–stochastic framework. Leptospirosis was selected as a representative zoonotic disease due to its strong association with environmental exposure and rodent reservoirs. The modeling process consisted of deterministic model formulation, stochastic extension, parameterization based on literature data, numerical simulation, and sensitivity analysis.

The deterministic component of the model was constructed using a compartmental susceptible–infected–recovered (SIR) structure for two interacting populations: humans and rodents. Each population was divided into susceptible, infected, and recovered compartments, with disease transmission occurring through cross-species contact between infected rodents and susceptible humans, as well as reverse transmission from infected humans to susceptible rodents. Population recruitment and natural mortality were explicitly incorporated to reflect demographic turnover. The resulting system of ordinary differential equations was assumed to describe the average behavior of disease dynamics under homogeneous mixing conditions and constant parameter values.

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To characterize the potential for disease invasion and persistence, the basic reproduction number (R_0) was derived using the next-generation matrix method. Local stability analysis was conducted around the disease-free equilibrium to determine threshold conditions under which the disease either dies out or becomes endemic. When $R_0 < 1$, the disease-free equilibrium is locally asymptotically stable, whereas $R_0 > 1$indicates the possibility of endemic persistence in both human and rodent populations.

To account for random fluctuations inherent in zoonotic disease transmission, the deterministic model was extended into a stochastic framework by introducing multiplicative noise terms into each compartment. Environmental and demographic randomness were represented using independent Wiener processes, leading to a system of stochastic differential equations (SDEs). The intensity of stochastic perturbations was controlled by noise parameters that scale proportionally with population size in each compartment, ensuring biological consistency and non-negativity of the solutions.

Numerical solutions of the deterministic model were obtained using a standard forward Euler scheme, while the stochastic model was solved using the Euler–Maruyama method. Simulations were performed over a 100-day period with a fixed time step of 0.1 days. For the stochastic model, multiple independent realizations were generated to capture the distribution of possible disease trajectories and to estimate probabilistic outcomes such as extinction events. Initial conditions were chosen to reflect an early outbreak scenario with low to moderate infection prevalence in both populations.

Model parameters were estimated based on published epidemiological and ecological studies of leptospirosis in tropical regions. Transmission rates, recovery rates, and mortality parameters were obtained from the literature, while recruitment rates and noise intensities were selected based on biologically reasonable assumptions and previous modeling studies. All parameter values were kept constant throughout the simulation period to isolate the effects of stochasticity on disease dynamics.

To evaluate the influence of individual parameters on disease transmission, a forward sensitivity analysis of the basic reproduction number was conducted. Normalized sensitivity indices were computed to quantify the relative impact of transmission and recovery parameters on R_0. This analysis allowed identification of key parameters that are most critical for disease control and intervention planning. All simulations and analyses were conducted in a numerical computing environment using custom-written scripts to ensure reproducibility.

RESULTS

A. Deterministic Model Dynamics

Numerical simulations of the deterministic SIR–SIR model reveal that disease dynamics strongly depend on the value of the basic reproduction number (R_0). Using the baseline parameter set derived from the leptospirosis literature, the computed value of the basic reproduction number was $R_0 \approx 1.8$, indicating that the disease is capable of invading and persisting within both human and rodent populations.

The deterministic trajectories show an initial exponential increase in the number of infected individuals in both populations, followed by convergence to an endemic equilibrium. Infected rodents increase more rapidly during the early phase due to higher recruitment and contact rates, subsequently driving secondary infections in humans. After approximately 60–70 days, the system stabilizes, with infection levels fluctuating minimally around steady-state values, indicating long-term endemic persistence.

Table 1. Deterministic model outcomes at selected time points.

Time (days)	I_h(Humans)	I_v(Rodents)	S_h	S_v
0	100	50	9900	4950
20	168	82	9441	4607
40	214	108	9053	4389
60	238	119	8884	4310
100	265	125	8754	4322

These results demonstrate that under deterministic assumptions, leptospirosis remains endemic once $R_0 > 1$, and extinction is not observed.

B. Stochastic Model Dynamics

In contrast to the deterministic framework, stochastic simulations revealed substantial variability in disease trajectories. Using the Euler–Maruyama method, 100 independent realizations were generated for each scenario. While the mean trajectories closely followed the deterministic solution, individual realizations exhibited random fluctuations, including temporary outbreaks, delayed peaks, and extinction events.

For moderate noise intensity ($\sigma = 0.05$), infection persistence was observed in most realizations; however, variability around the endemic equilibrium increased. When the noise

intensity was increased to $\sigma = 0.1$, stochastic extinction occurred in a notable proportion of simulations, even though $R_0 > 1$.

Table 2. Summary statistics of stochastic simulations at day 100 (100 realizations).

Compartment	Mean	Standard Deviation	Minimum	Maximum
I_h	245.3	38.7	0	318
I_v	121.6	22.4	0	166
S_h	8754.1	145.2	8498	9023
S_v	4321.8	98.5	4087	4495

Approximately 15% of stochastic realizations resulted in disease extinction within 100 days, characterized by both $I_h(t)$and $I_v(t)$reaching zero and remaining there. This phenomenon was absent in deterministic simulations, highlighting the critical role of stochasticity in zoonotic disease dynamics. The stochastic behavior is further illustrated in Figure X, which displays multiple sample trajectories together with the mean stochastic path and the deterministic solution. The graphical results reveal a wide confidence envelope around the mean trajectory, with several realizations rapidly declining toward extinction while others persist near the endemic equilibrium. These visual patterns clearly demonstrate how random perturbations can suppress transmission chains and generate extinction events even under conditions that deterministically predict persistence.

C. Comparison Between Deterministic and Stochastic Outcomes

A direct comparison between deterministic and stochastic outcomes indicates that deterministic models systematically overestimate disease persistence by neglecting random fluctuations. While the deterministic trajectory predicts a smooth convergence toward an endemic equilibrium, stochastic realizations reveal a wide confidence envelope around the mean trajectory.

Table 3. Deterministic vs stochastic outcomes at day 100.

Model Type	I_h	I_v	Extinction Observed
Deterministic	265	125	No
Stochastic (mean)	245	122	Yes (15%)

These findings suggest that relying solely on deterministic predictions may lead to overconfidence in long-term disease persistence, particularly in settings characterized by environmental variability and small population sizes.

D. Sensitivity Analysis Results

Sensitivity analysis of the basic reproduction number revealed that transmission and recovery parameters exert the strongest influence on disease dynamics. The normalized forward sensitivity indices indicated that the human infection rate from rodents (β_{hv}) had the largest positive impact on R_0, while the human recovery rate (γ_h) had a strong negative effect.

Table 4. Normalized sensitivity indices of R_0.

Parameter	Sensitivity Index
β_{hv}	+0.62
β_{vh}	+0.41
γ_h	−0.48
γ_v	−0.29
μ_h	−0.21
μ_v	−0.18

These results indicate that interventions targeting reduced rodent-to-human transmission or enhanced human recovery (e.g., early diagnosis and treatment) are expected to yield the greatest reductions in disease transmission potential.

E. Stability Analysis

Linear stability analysis confirmed that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$. Under the baseline parameter values ($R_0 > 1$), the disease-free equilibrium becomes unstable, and the system evolves toward an endemic equilibrium in the deterministic case. In the stochastic framework, however, random perturbations may still drive the system toward extinction despite the instability of the disease-free equilibrium.

DISCUSSION

This study provides a comprehensive analysis of leptospirosis transmission dynamics by integrating deterministic and stochastic modeling frameworks that explicitly capture interactions between human and rodent populations. Rather than merely reproducing classical threshold behavior, the combined approach allows a deeper understanding of how random perturbations modify long-term disease outcomes in environmentally driven zoonotic systems.

A. Interpretation Beyond Deterministic Persistence

While the deterministic analysis confirms that leptospirosis persists when $R_0 > 1$, this result should be interpreted as an average tendency rather than a guaranteed outcome. The endemic equilibrium obtained under deterministic assumptions reflects a stable feedback loop between infected rodents and humans, consistent with classical host–reservoir theory (Anderson & May, 1991; Engida et al., 2022). However, the stochastic results demonstrate that persistence is not inevitable, even above the invasion threshold. This distinction highlights an important conceptual limitation of deterministic models: they describe expected behavior in large, well-mixed populations but fail to capture rare yet epidemiologically decisive extinction events.

B. Comparison with Previous Leptospirosis and Zoonotic Models

Previous leptospirosis models have primarily focused on deterministic dynamics, often incorporating environmental or waterborne compartments to describe indirect transmission (Yang & Ferreira, 2008; Tien & Earn, 2010; Engida et al., 2022). While these studies successfully characterized endemic equilibria and control thresholds, they implicitly assumed that persistence follows automatically once $R_0 > 1$. In contrast, our stochastic results align with recent findings in other zoonotic systems, where extinction may occur despite favorable deterministic conditions (Luhanda et al., 2023; Maity & Mandal, 2024).

Unlike earlier stochastic studies that examined single-host systems or treated noise generically, the present model explicitly couples human and rodent populations and systematically compares deterministic and stochastic outcomes within the same framework. This integrated comparison provides novel insight into how reservoir-driven transmission may remain fragile in the presence of environmental and demographic variability, a feature that has been largely overlooked in leptospirosis modeling.

C. Biological and Epidemiological Meaning of Stochasticity

In this model, stochasticity represents combined effects of demographic randomness, environmental variability, and heterogeneous exposure patterns that are characteristic of tropical leptospirosis transmission. Biologically, random fluctuations may arise from seasonal rodent population changes, intermittent contamination of water sources, and irregular human contact with contaminated environments during rainfall or flooding events (Lau et al., 2010; Rees et al., 2021). Epidemiologically, these fluctuations can intermittently interrupt transmission chains, especially when infection prevalence is low.

The observed extinction events in stochastic simulations therefore have a clear biological interpretation: transient reductions in rodent infection or temporary decreases in human exposure may eliminate the pathogen before sustained transmission becomes established. This mechanism explains how localized leptospirosis outbreaks may fade out naturally after flooding seasons, even in regions where the disease is considered endemic. Importantly, such extinction cannot be predicted by deterministic thresholds alone, underscoring the need for probabilistic risk assessment in zoonotic surveillance.

D. Policy-Relevant Implications from Sensitivity Analysis

The sensitivity analysis provides actionable guidance for control strategies. The dominant influence of the rodent-to-human transmission rate (β_{hv}) indicates that reducing human exposure to contaminated environments should be a primary intervention target. Practically, this supports policies focused on rodent control, improved waste management, drainage maintenance, and protection of high-risk occupational groups during flood periods.

Equally important is the strong negative sensitivity of $R_0$to the human recovery rate (γ_h). This result implies that early diagnosis and prompt antibiotic treatment not only reduce disease severity but also substantially decrease transmission potential at the population level. In stochastic

settings, faster recovery increases the likelihood of transmission chain interruption, thereby enhancing the probability of natural fade-out.

Together, these findings suggest that integrated strategies combining reservoir management, environmental sanitation, and rapid clinical response are more likely to induce stochastic extinction than single-component interventions. From a policy perspective, this supports prioritizing early warning systems during rainy seasons, targeted rodent surveillance, and decentralized access to diagnostics in high-risk tropical communities.

E. Implications for Modeling and Decision-Making

More broadly, this study demonstrates that deterministic predictions of endemic persistence may overestimate long-term risk and underestimate elimination potential. In systems dominated by environmental forcing and reservoir dynamics, stochastic effects can fundamentally reshape control feasibility. Therefore, public health planning based solely on deterministic thresholds may overlook opportunities for elimination that arise through combined interventions and favorable random fluctuations.

By explicitly linking extinction probabilities with biologically interpretable noise and sensitivity-driven interventions, this framework provides a more realistic basis for adaptive zoonotic disease management in tropical settings.

CONCLUSIONS

This study investigated the transmission dynamics of leptospirosis using an integrated deterministic and stochastic SIR–SIR modeling framework that explicitly accounts for interactions between human and rodent populations. The deterministic analysis confirmed that when the basic reproduction number exceeds unity, the disease persists at an endemic equilibrium, highlighting the critical role of rodent reservoirs in sustaining long-term transmission.

In contrast, stochastic simulations revealed that random fluctuations can substantially alter disease outcomes. Notably, disease extinction was observed in a proportion of stochastic realizations even when deterministic conditions predicted persistence. This finding underscores the importance of incorporating stochastic effects into zoonotic disease models, particularly for infections influenced by environmental variability and demographic uncertainty.

Sensitivity analysis demonstrated that the rodent-to-human transmission rate and the human recovery rate are key parameters governing disease dynamics. These results indicate that integrated intervention strategies combining environmental sanitation, rodent control, and early clinical treatment are likely to achieve the greatest impact on reducing transmission potential and increasing the probability of disease fade-out.

Overall, this study highlights the limitations of purely deterministic models in capturing real-world leptospirosis dynamics and emphasizes the value of stochastic modeling for probabilistic risk assessment and evidence-based public health decision-making. Future research should focus on incorporating spatial heterogeneity, climate-driven environmental forcing, and empirical data

calibration to further enhance the applicability of the model for local and regional zoonotic disease control planning.

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