

**MATHEMATICAL MODELLING AND ANALYSIS OF EMERGING
AND RE-EMERGING OF RABIES TRANSMISSION DYNAMICS:
DETERMINISTIC AND STOCHASTIC APPROACHES WITH
CONTROL STRATEGIES**

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**A Dissertation Submitted in Partial Fulfilment of the Requirements for the Degree of
Doctor of Philosophy in Mathematical and Computer Sciences and Engineering of the
Nelson Mandela African Institution of Science and Technology**

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ABSTRACT

Rabies remains a critical global public health concern, particularly in regions with dense free roaming dog populations serving as viral reservoirs. This study develops a mathematical model using ordinary differential equations (ODEs) to analyze the transmission dynamics of rabies among humans, free-range, and domestic dogs. By leveraging next-generation matrices, the basic reproduction number $\mathcal{R}_0$ is derived to assess disease persistence. The study establishes that the disease-free equilibrium is globally asymptotically stable when $\mathcal{R}_0 < 1$, while the endemic equilibrium remains stable when $\mathcal{R}_0 \geq 1$. To identify the key drivers of rabies transmission, the Partial Rank Correlation Coefficient (PRCC) analysis was conducted. The results revealed that contact rates between infectious agents and hosts play a significant role in influencing the spread of rabies. A comparative analysis between Stochastic Continuous-Time Markov Chain (CTMC) and deterministic models provides insights into rabies persistence. The stochastic threshold was determined using a multitype branching process, demonstrating the influence of stochasticity on disease extinction probabilities. Simulations using 10 000 sample paths affirmed that stochastic models closely align with deterministic predictions while capturing variability in outbreak probabilities, particularly in low-infection scenarios. The integration of environmental reservoirs and multi-host interactions further strengthens the model's applicability while also recommending keeping dogs indoors. Numerical simulations employing mesh and contour plots illustrate an inverse relationship between the effective reproduction number $\mathcal{R}_e$ and control measures. Strategies such as domestic dog vaccination, health promotion, and post-exposure treatment effectively reduce rabies transmission, with vaccination and treatment emerging as the most impactful interventions for achieving disease elimination within five years. Optimal control models substantiate these findings, emphasizing the need for sustained community engagement and multifaceted intervention strategies. To align with the Global Rabies Control (GRC) initiative by 2030, this study advocates for comprehensive control measures prioritizing the regulation of free-range and domestic dogs, increased vaccination efforts, and targeted health interventions. By integrating deterministic and stochastic modeling approaches, this research provides a robust framework for understanding rabies dynamics and optimizing control strategies to mitigate its public health burden.

AUTHOR'S DECLARATION

I, Mfano Charles, do hereby declare to the Senate of Nelson Mandela African Institution of Science and Technology that this dissertation titled: Mathematical Modelling and Analysis of Emerging and Re-emerging of Rabies Transmission Dynamics: Deterministic and Stochastic Approaches with Control Strategies is my own original work and that it has neither been submitted nor presented for degree award in any other institution.

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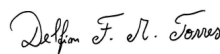
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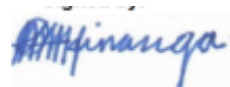
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CERTIFICATION

The undersigned certify that they have read and hereby recommend for acceptance by the Nelson Mandela African Institution of Science and Technology the dissertation entitled: Mathematical Modeling and Analysis of Emerging and Re-emerging of Rabies Transmission Dynamics: Deterministic and Stochastic Approaches with Control Strategies, in fulfillment of the requirements for the degree of Doctor of Philosophy in Mathematical and Computer Sciences and Engineering of the Nelson Mandela African Institution of Science and Technology.

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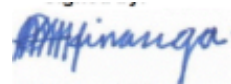
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DEDICATION

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LIST OF ABBREVIATIONS

CDC	: Centers for Disease Control and Prevention
CTMC	: Continuous Time Markov Chain
DALYs	: Disability adjusted life Years
FAO	: Food and Agriculture Organization of the United Nations
LGDs	: Livestock Guardian Dogs
LMICs	: Low-and middle income Countries
MDV	: Mass dog vaccination
MoLF	: Ministry of Livestock and Fisheries
NRCs	: The National Rabies Control Strategy
PEP	: Post-exposure Prophylaxis
PFU/mL	: Plaque Forming Units per milliliter
SEIV	: Susceptible-Exposed-Infectious-Vaccinated
WHO	: World Health Organization

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CHAPTER ONE

INTRODUCTION

1.1 Background of the Problem

Rabies is a zoonotic viral disease that affects mammals, including humans, and is caused by the *Rabies lyssavirus*. Following exposure, the virus travels from the infection site to the central nervous system, resulting in severe neuroinflammation and progressive neurological decline (De Benedictis *et al.*, 2022; Kumar *et al.*, 2023). This disease is present on every continent except Antarctica and remains a significant public health threat, particularly in low- and middle-income countries (LMICs).

Globally, approximately 95% of human rabies deaths occur in Asia and Africa, with an estimated 35 000 deaths annually in Asia and 25 000 in Africa (Mbilo *et al.*, 2021; Tian *et al.*, 2018). These numbers are widely regarded as underestimates due to substantial under-reporting, especially in rural and remote areas where limited healthcare infrastructure hampers timely case detection and diagnosis (Acharya *et al.*, 2021). Factors such as weak surveillance systems, a lack of understanding of dog population dynamics, inadequate control of animal reservoirs, and limited access to both pre-exposure and post-exposure prophylaxis (PEP) further obscure the true burden of the disease (Patane *et al.*, 2025).

In many endemic regions, free-roaming and unvaccinated dogs represent the primary reservoirs for transmission, complicating control efforts (Charles *et al.*, 2024; Zhu & Guo, 2016; Organization, 2018). Tanzania exemplifies these challenges and, similar to many LMICs, is particularly vulnerable to zoonotic diseases (Lushasi *et al.*, 2021). Although rabies has been documented in Tanzania since the 1930s, transmission persists, with approximately 1500 human cases reported each year (Rweyemamu *et al.*, 1973; Mpolya *et al.*, 2017).

An estimated 1.5 million Tanzanians live in proximity to wildlife livestock human interfaces, heightening the risk of disease spillover to humans, livestock, and domestic dogs (Mtema *et al.*, 2016). Despite the long-standing presence of rabies in the region, key epidemiological characteristics including spatial spread, reservoir interactions, and mechanisms of persistence remain insufficiently characterized. Addressing these gaps necessitates the strengthening of surveillance systems and the implementation of evidence-based, sustainable control strategies (Komol *et al.*, 2020).

1.1.1 Economic Burden of Rabies

Rabies represents a significant global economic burden, with annual losses estimated at approximately \$8.6 billion (Rather & Vasavi, 2024). According to FAO (2017), roughly 6% of these losses are attributable to livestock-related impacts. The economic burden associated with human rabies cases, measured in disability-adjusted life years (DALYs), is estimated to be nearly \$4 billion each year (Anothaisintawee *et al.*, 2019). Additionally, preventive and control measures, including mass vaccination campaigns and post-exposure prophylaxis (PEP), contribute another \$500 million to \$583 million annually across Africa and Asia. Acknowledging the urgency of this public health issue, the World Health Organization (WHO) initiated the “Zero Rabies by 2030” program, which aims to eradicate human deaths from dog-mediated rabies by 2030 (Akinsulie *et al.*, 2024). A crucial component of this strategy is mass dog vaccination (MDV), which is widely regarded as a highly cost-effective intervention (Kanda *et al.*, 2022).

1.1.2 Rabies Control Strategies and Costs in Tanzania

In Tanzania, vaccinating a single dog costs between \$2.50 and \$22.49, whereas treating an exposed individual can cost approximately 200 000 Tanzanian shillings (about \$75.64) (Stephen *et al.*, 2022). Economic analyses highlight the need for at least 40% vaccination coverage to achieve cost-effectiveness, particularly in high-risk areas such as Ngorongoro and Serengeti (Ramsey, 2017). Tanzania’s Rabies Control Strategy and projected budget for 2019–2028 (Table 1), submitted to the World Organisation for Animal Health (WOAH), emphasizes improving access to affordable vaccines and strengthening national eradication efforts. Consistent with this strategic direction, local governments have begun allocating dedicated funds for MDV, underscoring a growing commitment to sustainable and long-term rabies control (NCRs, 2019).

Table 1: Projected Budget for the Rabies Elimination Strategy (2019–2028) in millions TZS (NRCs, MoLE, 2019)

<i>Strategic Objective</i>	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028
Prevention of human rabies	559.00	559.45	590.67	570.10	480.85	364.01	377.21	497.81	522.70	548.84
Control and eliminate dog rabies	3 680.80	3864.84	4 058.09	4 260.99	4474.04	4687.22	4921.58	5167.66	5426.04	5697.34
Strengthen rabies surveillance using the one-health approach	90.00	82.50	86.63	90.96	80.78	84.81	89.05	94.36	99.07	104.03
Strengthening institutional capacity for control and eventual elimination of rabies	128.00	133.40	124.07	130.27	116.40	122.22	128.33	153.37	139.78	146.77
Conduct advocacy, communication and social mobilization on rabies	60.00	71.00	58.05	60.95	64.00	54.84	57.58	60.46	63.48	66.65
Conduct rabies operational research	15.00	30.00	31.50	10.00	5.00	30.00	31.50	7.00	7.35	7.72
Strengthen mechanism for monitoring and evaluation of rabies control programme	60.00	52.00	54.60	137.33	60.20	63.21	66.37	189.68	0.00	0.00

Continued on next page

Table 1 continued from previous page

<i>Strategic Objective</i>	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028
Enhance resource mobilization for rabies control and elimination	31.00	32.55	2.10	2.21	2.32	2.43	2.56	2.68	2.82	2.96
Enhance partnership and intersectoral collaboration	40.00	42.00	44.10	46.31	48.62	51.05	53.60	56.28	59.10	62.05

1.1.3 Role of Mathematical Modelling in Rabies Research

Mathematical modeling has played a central role in understanding infectious disease dynamics and has been particularly valuable in rabies research. These models help elucidate transmission pathways, assess the impact of interventions, and account for key determinants such as human–dog contact patterns and environmental differences between urban and rural settings. They support predictive analyses and guide the development of effective control strategies. A wide range of modeling frameworks has been applied to study rabies transmission in human and dog populations (Abdulmajid & Hassan, 2021; Amoako *et al.*, 2021; Ruan, 2017a; Ayoade *et al.*, 2019; Chapwanya & Dumani, 2022; Kadowaki *et al.*, 2018; Ayoade & Ibrahim, 2023; Tulu & Koya, 2017; Pantha *et al.*, 2021). Collectively, these studies have identified major drivers of transmission and evaluated diverse intervention strategies, contributing to evidence-based policy design for rabies control and elimination.

1.1.4 Transmission Dynamics of the Rabies Diseases

Mammals, including humans, are at risk of contracting rabies through bites from rabid animals, non-bite exposures such as scratches, open wounds exposed to infected saliva or other contagious material, and inhalation of aerosolized rabies virus (Peng *et al.*, 2022). Upon entering the body through a bite, the rabies virus travels to the brain via nerve cells, where it rapidly replicates. This results in pronounced inflammation of the central nervous system, particularly affecting the brain and spinal cord, leads to rapid neurological degeneration and death (Oertli, 2019; Grieve, 2022).

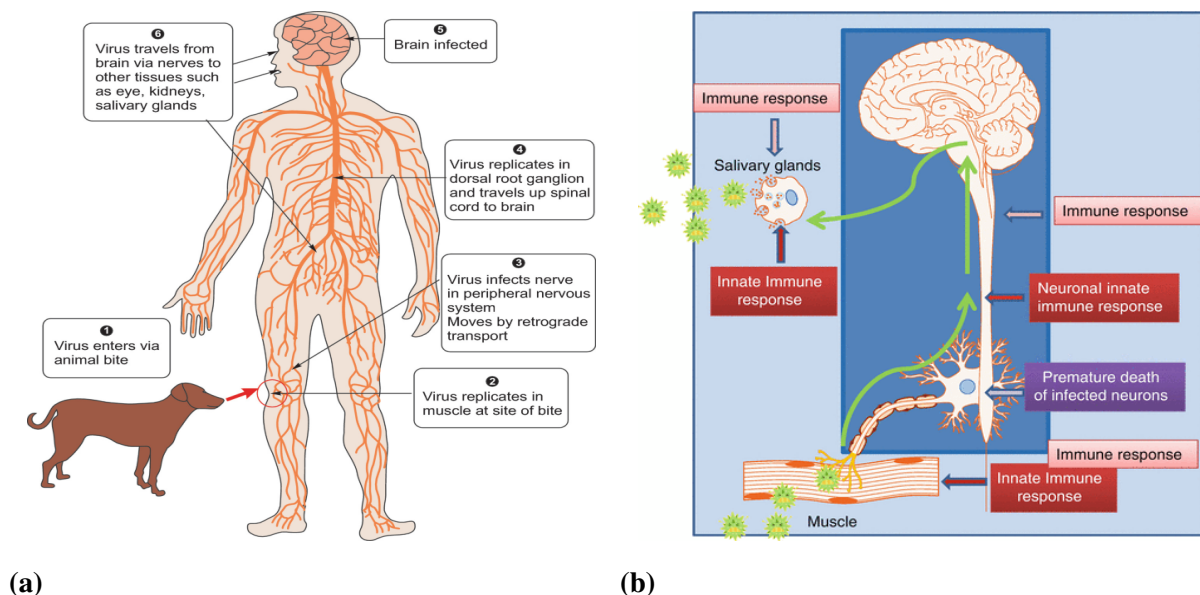


Figure 1: Transmission dynamics of rabies to humans (Paul, 2024)

According to the World Health Organization (WHO, 2017), dogs are the primary source of rabies transmission for over 99% in endemic regions, with an estimated three billion people living in rabies-affected areas worldwide (Organization, 2018; Pongsumpun & Puntani, 2022; Jawaz *et al.*, 2022). The CDC states that casual contact with infected individuals or exposure to non-infectious bodily fluids such as urine, blood, or feces does not pose a risk of rabies transmission. Furthermore, receiving a rabies vaccination does not constitute exposure, nor does it require PEP (World Health Organization, 2006; Laishram *et al.*, 2016).

1.1.5 Symptoms of Rabies Diseases

Symptoms of rabies typically appear between 20 days to 3 months after exposure, though they may surface as early as one week or as late as one year, depending on factors such as the location of the virus entry and the viral load. Common symptoms include fever, headaches, fatigue, restlessness, anxiety, hallucinations, hydrophobia, difficulty swallowing, and paralysis. In rare cases, the incubation period may extend up to 7 years. If not treated promptly with appropriate medical care, such as vaccination, the disease can progress to a comatose state and ultimately result in death (Nigg & Walker, 2009; Johnson *et al.*, 2010; Hailemichael *et al.*, 2022).

1.1.6 Environmental Determinants of Rabies Transmission

Although rabies is primarily transmitted through direct contact with virus-laden saliva via animal bites, the possibility of indirect environmental transmission cannot be entirely excluded (Rees *et al.*, 2021). The rabies virus loses infectivity upon drying or exposure to sunlight and is rapidly inactivated at temperatures above 50°C; however, it can survive for several hours to days at ambient temperatures (Lugelo *et al.*, 2020; Wilson, 2025). This environmental persistence raises concerns about indirect exposure through contaminated surfaces, animal carcasses, and shared resources, particularly in areas with poor sanitation, high densities of free-roaming dogs, and inadequate veterinary infrastructure (Simon *et al.*, 2019).

Urbanization and anthropogenic land-use changes further complicate rabies dynamics by increasing contact between wildlife reservoirs (e.g., bats, foxes, jackals), humans, and domestic animals (Marrana, 2022). Inadequate waste management in urban and peri-urban areas exacerbates the problem by attracting scavengers key rabies reservoirs such as stray dogs and wild carnivores (Wright *et al.*, 2021). These conditions sustain dense animal populations and promote rabies endemicity, especially where vaccination coverage is low.

Environmental factors also contribute to seasonal patterns in rabies transmission. Cooler temperatures enhance viral survival, while breeding seasons and migration cycles among reservoir

hosts increase the number of susceptible individuals and intensify interspecies contact (Moran, 2020; Gilbert, 2023). Recognizing these ecological patterns is vital for effective control strategies and predictive modeling.

1.2 Statement of the Problem

WHO estimates that rabies is responsible for approximately 60 000 deaths globally each year. The majority of these fatalities occur in Asia and Africa, with an estimated 35 000 and 25 000 deaths, respectively, collectively comprising 86% of global rabies-induced mortality (Zhu & Guo, 2016; Organization, 2018). In Tanzania, rabies accounts for approximately 1500 deaths annually, representing 6% of Africa's total rabies-related fatalities (Sambo *et al.*, 2022). Notably, nearly 40% of rabies cases occur in individuals under the age of 15, underscoring the disproportionate impact of the disease on pediatric populations in endemic regions (Ndii *et al.*, 2018). Despite the availability of rabies vaccination and post-exposure prophylaxis (PEP), access remains limited due to high costs, leaving many infected individuals without treatment and at risk of mortality.

The situation is further complicated by environmental factors, such as varying interaction rates between dogs and humans, climate change and seasonality, which can exacerbate the spread of rabies. Furthermore, conventional methods for treating and managing rabies remain challenging, exacerbated by the lack of diagnostic tests for pre-clinical rabies infection. Although substantial research has been conducted (Sambo *et al.*, 2013; Mtema *et al.*, 2016; Mpolya *et al.*, 2017; Asamoah *et al.*, 2017; Aydogan *et al.*, 2020; Hou *et al.*, 2012; Jawaz *et al.*, 2022), a deeper understanding of rabies persistence in dog populations and the associated human health risks requires integrating stochastic and deterministic modeling approaches (Rysava *et al.*, 2020). Therefore, it is imperative to investigate the incidence of dog bites, estimate the likelihood of rabies transmission, and assess the likelihood of clinical disease onset.

1.3 Research Objectives

1.3.1 Main Objective

The general objective of the study is to develop and analyze mathematical models of dog bites and estimate probability of developing rabies.

1.3.2 Specific Objectives

The specific objectives are:

- (i) To formulate and analyze a deterministic model that captures the transmission dynamics of rabies in human and dog populations.
- (ii) To conduct parameter estimation and sensitivity analysis for a deterministic model of rabies transmission in human and dog populations.
- (iii) To quantitatively assess the performance of the deterministic model in predicting rabies transmission .
- (iv) To formulate and analyze a CTMC stochastic model for predicting the probability of getting rabies disease.
- (v) To assess the impact of various interventions used to control rabies disease transmission using optimal control theory .

1.4 Research Questions

This study is influenced by the following research questions:

- (i) How can we quantify the interactions and contact rates between humans and dogs to incorporate them into the mathematical model?
- (ii) What are the potential intervention strategies for controlling rabies transmission, and how can they be represented in the mathematical model?
- (iii) What are the key parameters and transition rates that should be incorporated into the CTMC model to accurately predict the probability of contracting rabies?
- (iv) How does uncertainty and parameter estimation influence the prediction of dog bite incidents and the probability of rabies transmission?
- (v) What are the key indicators and metrics to measure the performance of the model in predicting the risk of rabies transmission?

1.5 Rationale of the Study

Dog bites represent a substantial global public health challenge, with millions of individuals affected annually. Among the most severe consequences of dog bites is the transmission of rabies, a fatal viral zoonosis spread primarily through the saliva of infected animals, particularly dogs. Understanding the probability of rabies transmission and the factors driving its emergence or re-emergence is therefore essential for effective prevention and control.

Mathematical models play a central role in predicting future patterns of disease transmission. These models incorporate key determinants such as dog population demographics, vaccination coverage, and environmental influences. By examining these variables, researchers can estimate the likelihood and dynamics of rabies spread, thereby supporting more targeted public health interventions and preventive strategies.

A comprehensive assessment of dog bites and rabies transmission requires a One Health perspective, which recognizes the interconnections of human, animal, and environmental health. Mathematical modeling provides an effective platform for integrating insights from veterinary science, epidemiology, ecology, and the social sciences. This interdisciplinary orientation deepens understanding of the complex interactions among dog behavior, human exposure, and rabies epidemiology, ultimately enhancing the design of robust and holistic control measures.

Therefore, conducting a study on the mathematical modeling of dog bites and the probability of rabies transmission is crucial for addressing the substantial public health burden posed by this disease. Through the combined use of epidemiological data, analytical modeling techniques, and interdisciplinary collaboration, researchers can generate evidence that strengthens policy development, informs intervention design, and supports long-term strategies to reduce dog-bite incidence and mitigate rabies transmission risk.

1.6 Significance of the Study

Rabies has remained a global zoonosis, posing public health risks and economic losses worldwide (Salyer *et al.*, 2017). Statistics from WHO revealed that rabies transmitted by dogs is responsible for the loss of over 1.8 million DALYs (disability adjusted life years) every year, with direct and indirect economic costs (PEP, animal tests, dog vaccination, and livestock losses) totaling \$5.5 billion per year (Shwiff *et al.*, 2018; Lembo *et al.*, 2010). In addition, the costs linked to the potential for human mortality contributed to the approximately \$120 billion global cost of canine rabies (Shwiff *et al.*, 2018).

Therefore, this study has comprehensively examined the dynamics of rabies transmission between humans and dogs by employing mathematical modeling integrated with stochastic approaches. These methodologies capture the intrinsic randomness and uncertainty associated with disease transmission, offering a more realistic and resilient analytical framework for assessing the probability of rabies outbreaks. Consequently, the study provides critical insights that can inform the design and implementation of strategic interventions. The outcomes of this research contribute to the effective management and control of rabies, with the ultimate goal of minimizing its public health burden and socioeconomic impacts through the following key

contributions:

- (i) Enhancing public awareness of the transmission dynamics of rabies among human and dog populations, thereby promoting proactive behavioral and health interventions.
- (ii) Supporting nomadic pastoralist communities residing near wildlife reserves by identifying practical strategies to protect their livestock from rabies infection, thereby safeguarding their livelihoods and improving economic resilience.
- (iii) Assisting educators, health professionals, and community leaders in disseminating knowledge on rabies prevention and control through training programs, awareness campaigns, workshops, media engagement, and educational seminars.
- (iv) Recommending cost-effective and evidence-based control measures aimed at reducing the risk of rabies transmission and mitigating its public health impact.
- (v) Contributing to the body of existing literature by offering a multidisciplinary perspective, thus serving as a foundation for future research and policy development in rabies control and prevention.

1.7 Delineation of the Study

This study focuses on modeling the transmission of rabies in humans and dogs. For the numerical simulations, most parameter values were sourced from existing literature, while others were estimated using least squares fitting based on data from the WHO. The optimal control model takes into account four intervention measures: vaccination, treatment, and public awareness campaigns. Strategies can involve any combination of one, two, or all four measures. The prediction models were developed based on the dynamics of rabies transmission and include human, domestic dog, and free-ranging dog populations. The epidemiological data were gathered from veterinary and public health reports.

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction

Rabies modeling has advanced considerably over the past three decades, alongside major developments in mathematical methods for studying infectious disease ecology. These advancements have enabled a deeper understanding of rabies virus dynamics and host–pathogen interactions (Asamoah *et al.*, 2017; Jeon *et al.*, 2019). Classic models examining the spread of fox rabies in Western Europe typically divide populations into four epidemiological compartments: susceptible (S), exposed (E), infectious (I), and recovered or removed (R), and employ both ordinary differential equations (ODEs) and stochastic agent-based simulations.

More recent work has strengthened these frameworks by integrating host demographic variability, spatial heterogeneity, and environmental stochasticity, leading to more realistic depictions of transmission dynamics. As a result, contemporary rabies models provide strong predictive capacity for tracking spatial spread and now serve as essential tools for guiding public health planning, especially when combined with optimal control strategies. In this chapter, we review key mathematical models addressing rabies transmission in humans and dogs, continuous-time Markov chain models for infectious diseases, optimal control and cost-effectiveness analyses for environmentally transmitted infections, and relevant non-mathematical studies on rabies.

2.2 Mathematical Models on the Transmission Dynamics of Rabies

Renald *et al.* (2020) developed a deterministic SEIV model to assess the impact of vaccination and culling on rabies transmission dynamics among domestic, stray, and Maasai dogs in Arusha, Tanzania. The model was validated using five years of incidence data from the region. The next-generation matrix method was employed to compute the basic reproduction number $\mathcal{R}_0$ and the effective reproduction number $\mathcal{R}_e$, which characterize disease persistence in the absence of interventions and disease extinction under implemented control strategies, respectively. Local and global stability of the disease-free equilibrium were established using eigenvalue analysis and the Metzler matrix approach. Analytical findings demonstrated that the disease-free equilibrium is locally and globally asymptotically stable whenever $\mathcal{R}_0 < 1$. Simulation results evaluating the relationship between $\mathcal{R}_0$, vaccination coverage, and culling rates further revealed that vaccination remains the most effective strategy for reducing rabies transmission.

Asamoah *et al.* (2021) developed a deterministic SEIR-type model to evaluate the impact of dog vaccination and public health interventions on rabies transmission. Stability and sensitivity analyses were employed to derive analytical expressions for the basic reproduction number $\mathcal{R}_0$, demonstrating that the stability of the endemic equilibrium is contingent upon maintaining vaccination coverage above a critical threshold. Through threshold theory and equilibrium analysis, the study provides strong analytical clarity, offering public health authorities a concrete target for vaccination efforts. Numerical simulations further illustrated that achieving vaccination levels above the calculated threshold substantially reduces transmission and can drive the system toward disease elimination.

Ayoade and Ibrahim (2023) developed a first-order nonlinear compartmental model (VDSD EDTD ID: Vaccinated-Susceptible-Exposed-Treatment-Infected Dogs) to analyze rabies transmission and control specifically within the dog population of Lagos State, Nigeria. Applying threshold theory and stability analysis, the study derived the effective reproduction number R_D and established conditions for disease-free and endemic equilibria. Using sensitivity analysis and the concept of herd immunity, the key finding was that while the government's campaign to vaccinate 60% of dogs is a positive step, it is mathematically insufficient for eradication. The model calculated that a herd immunity threshold of approximately 87% vaccination coverage is required to eliminate the disease. More critically, bifurcation analysis revealed that a vaccination-only strategy is inadequate due to the phenomenon of backward bifurcation, where the disease can persist even when the reproduction number is below one. For a successful outcome, the mass vaccination program must be integrated with two complementary interventions: enhanced surveillance for the early detection and treatment of exposed dogs, and the culling of infectious animals.

Hampson *et al.* (2020) used a metapopulation modelling framework based on interconnected deterministic compartmental systems to quantify the global rabies burden, with a focus on Africa and Asia. Their methodology integrated SEIR-type transmission dynamics with empirical data on spatial dog movement and connectivity between subpopulations, utilizing a data-driven parameterization approach that synthesized demographic, epidemiological, and mobility datasets. Applying spatial simulation and burden estimation techniques, this approach highlighted how high dog population turnover and unrestricted mobility are fundamental drivers of disease persistence across regions. The model's numerical simulations produced global burden estimates, indicating substantial under-reporting and predicting that targeted vaccination in high-mobility corridors could significantly reduce regional transmission. However, a significant limitation persists: the uncertainty in foundational data, particularly for dog population estimates and inter-regional contact rates, which can propagate through the model and affect the precision of burden estimates in numerical outputs.

Leung *et al.* (2021) constructed a stochastic branching-process model using probabilistic methods and Monte Carlo simulation to explore rabies spillover risks from domestic dog reservoirs into wildlife populations. Their methodology involved parameterizing the branching process with estimated reproduction numbers and cross-species transmission probabilities to quantify the distribution of potential outbreak sizes following a spillover event. Their approach excelled at capturing the inherent variability and chance in rare cross-species transmission events, dynamics often smoothed over or missed by deterministic mean-field models. The numerical results from extensive simulations demonstrated that the tail of the outbreak size distribution is heavy, leading to the key conclusion that even low-probability spillover events, when they occur, can seed and sustain independent transmission cycles in wildlife reservoirs, thereby complicating long-term elimination efforts by creating new maintenance reservoirs.

Samanta and Chattopadhyay (2022) introduced time-dependent control strategies using an SEIRS-type model, applying Pontryagin's Maximum Principle from optimal control theory to evaluate cost-effective vaccination and culling policies. Their methodology involved formulating an objective function that minimized the total cost of interventions and infections over a finite time horizon, then deriving the necessary optimality conditions to solve for the time-varying control profiles. The numerical results, obtained by solving the resulting two-point boundary value problem, revealed that a combined, dynamically adjusted strategy yields the most significant and economically efficient reduction in rabies prevalence over time, with optimal resource allocation shifting from high-intensity initial culling to sustained vaccination.

Zhao *et al.* (2023) extended classical deterministic frameworks by incorporating environmental stochasticity random fluctuations in transmission rates using stochastic differential equations (SDEs) based on Wiener processes. Their methodology involved deriving the probability density function of the infected population and performing stochastic stability analysis. Numerical simulations using Euler-Maruyama methods demonstrated that the intensity of this "noise" strongly influences the probability of disease extinction, with simulation results showing that even when $\mathcal{R}_0 > 1$, there remains a 15-30% probability of stochastic extinction within one year depending on the noise amplitude. This finding underscores that chance environmental factors can lead to unexpected outbreak fade-out or persistence.

Ali *et al.* (2024) developed a hybrid deterministic–stochastic rabies model where human infections followed deterministic ordinary differential equations (ODEs), while dog transmission was modelled with randomness via a continuous-time Markov chain (CTMC). Their methodology included Gillespie algorithm simulations for the stochastic component and analytical threshold derivation for the deterministic framework. Numerical results from 10 000 Monte Carlo simulations demonstrated "stochastic fade-out," showing that infections can die out by chance in 22%

of outbreaks in small, localized dog populations $N < 100$ even when the deterministic threshold suggests endemicity $\mathcal{R}_0 > 1$.

Mtema *et al.* (2025) constructed a spatially explicit deterministic model using reaction-diffusion partial differential equations (PDEs) to analyse the interplay between dog mobility and heterogeneous vaccination coverage. Their methodology involved finite difference numerical solutions on a two-dimensional grid with heterogeneous diffusion coefficients representing differential mobility. Numerical simulations demonstrated that patchy or spatially clustered vaccination creates transmission hotspots that act as sources for re-invasion, allowing rabies to persist regionally despite adequate average coverage with simulations showing that even with 80% overall coverage, local pockets of 40% coverage led to persistent hotspots that maintained regional transmission.

Mbu *et al.* (2025) introduced a network-based mathematical model that captures heterogeneity in dog-to-dog contact patterns by representing individual dogs as nodes and their interactions as edges. Their approach employed both synthetic scale-free networks and empirically derived contact structures informed by observational studies. The numerical results demonstrated that high-degree nodes typically dominant or widely roaming dogs act as critical super-spreaders that sustain rabies transmission. Notably, targeted vaccination of the top 20% highest-degree individuals reduced the final outbreak size by approximately 73%, compared with only 52% under random vaccination at equivalent coverage.

2.3 Non-Mathematical Modelling on the Transmission Dynamics of Rabies

Sambo *et al.* (2014) provides one of the most influential non-mathematical investigations into rabies by examining community knowledge, attitudes, and practices in Tanzania. Their cross-sectional survey revealed that although most respondents recognised rabies as a fatal disease, preventive behaviour such as timely post-exposure prophylaxis (PEP) seeking and routine dog vaccination remained remarkably low. This gap between awareness and practice highlighted how socio cultural beliefs, distance to health facilities, and reliance on traditional healers continue to hinder effective rabies prevention.

Qin *et al.* (2025) analyzed the global burden of rabies from 1990 to 2019 using Age-Standardized Incidence Rates (ASIRs) and Estimated Annual Percentage Changes (EAPCs) from Global Burden of Disease data. The study found a significant global decline of 43.1% in rabies cases, with an EAPC of -3.35% , driven by major reductions in low- and middle-income regions. However, progress was unequal, with the highest burden persisting in low Socio-demographic Index areas, while some high-income regions saw increasing trends. The

analysis concludes that rabies remains a major endemic threat in socio-economically disadvantaged regions, underscoring the critical need for targeted, equity-focused control strategies.

Acharya *et al.* (2020) explored the application of the One Health framework for rabies control. Through qualitative policy analysis, they found that weak intersectoral coordination and limited diagnostic capacity frequently undermine integrated rabies management despite strong theoretical endorsement of One Health approaches.

Bunuma *et al.* (2025), whose scoping review showed that rabies remains heavily underreported in Mainland Tanzania due to fragmented data systems, poor laboratory infrastructure, and inconsistent reporting between veterinary and human health departments.

Selleck *et al.* (2025) demonstrated that the financial burden of rabies especially indirect costs such as transportation to clinics and lost productivity is substantially underestimated. Their review further indicated that while free or subsidised dog vaccination is widely considered cost-effective, many low-income countries continue to lack sustainable financing structures for long-term campaign implementation.

2.4 Key Observation in Rabies Research Agenda

Despite a substantial body of reviewed research on mathematical models (Asamoah *et al.*, 2021; Ayoade & Ibrahim, 2023; Hampson *et al.*, 2020; Mtema *et al.*, 2025; Samanta & Chattopadhyay, 2022), a significant gap persists. Many existing studies overlook important interconnected factors, particularly the interactions between various host populations, such as domestic and free-range dogs, as well as environmental reservoirs. This deficiency underscores the urgent need for integrated, system-level approaches that capture the full ecological complexity of rabies dynamics. Such frameworks should extend beyond single-host or purely deterministic models to encompass stochastic events, host behavioural. Furthermore, although global trends suggest some progress, the burden of the disease remains heavily concentrated in high-risk, low-resource regions, where the challenges surrounding surveillance and intervention logistics are particularly pronounced. To address these shortcomings, our study develops an advanced modeling framework designed to formulate effective, multi-faceted control strategies for emerging and re-emerging outbreaks. Importantly, we include critical environmental and socio-behavioural deterrent factors, such as habitat connectivity and community compliance dynamics, which are frequently overlooked in previous models. By integrating these elements, our work aims to provide a more comprehensive and realistic understanding of systems, thereby informing the design of cost-effective, sustainable, and locally adaptable interventions.

CHAPTER THREE

MATERIALS AND METHODS

3.1 Introduction

In this chapter, we developed a comprehensive mathematical framework using ordinary differential Equations (ODEs) to capture the dynamic interactions among susceptible individuals, infectious dog populations (both domestic and free-roaming), and the environmental reservoir of the rabies virus. The modelling approach is systematically structured to transition from a deterministic formulation to a more nuanced stochastic representation, reflecting the evolution and increasing sophistication of epidemiological modelling techniques.

Initially, we formulated a deterministic Model (3.5) to describe the average behavior of the system over time, which provides foundational insights into the transmission dynamics of rabies under the assumption of homogeneous mixing and continuous population changes. This model clarifies key thresholds such as the basic reproduction number, equilibrium states, and the conditions under which the disease may persist or be eradicated. Next, to account for the inherent randomness and discrete nature of real-world disease transmission, we formulated a stochastic Model (3.57) based on a continuous-time Markov chain (CTMC) framework. This stochastic formulation introduces variability in the transmission process, capturing fluctuations due to demographic stochasticity, which are particularly important in low-population settings or during the early phases of an outbreak. Both models are presented alongside a detailed methodological explanation, including the formulation of transition rates, model assumptions, parameter estimation strategies, and numerical implementation procedures. This rigorous approach ensures methodological transparency and facilitates a more robust interpretation of the simulation results. Furthermore, the chapter discusses the broader epidemiological implications of the modeling outcomes (3.83), offering insights into effective intervention strategies and public health policies aimed at controlling and ultimately eliminating rabies transmission in both human and dog populations.

3.2 Formulation of the Model without Intervention

The model for the transmission dynamics of rabies is structured into three interacting components: the human population, the dog population, and rabies-causing viruses in the environment. The dog population is further subdivided into two distinct subgroups, namely free-range dogs and domestic dogs. Free-range dogs are defined as dogs that roam in public areas without direct human control. This category includes stray dogs, street dogs, feral dogs, village dogs, and African wild dogs, whose global population is estimated to be approximately 900 million.

In contrast, domestic dogs are community-owned animals that remain under continuous direct human control (Gompper, 2013; Lescureux & Linnell, 2014).

The free-range dogs population defined by N_F is divided into three compartments; first compartment is susceptible denoted as S_F , these are group of free-range dogs who are not infected but may get infection after adequately contact with either infected free-range dogs symbolized as I_F , infected domestic dogs symbolized as I_D or with environment that contains rabies virus symbolized as M . The second compartment is exposed free-range dogs symbolized as E_F which include all the free-range dogs who have contracted the disease but can not transmit the disease and do not have symptoms of rabies infections. The third compartment is I_F made up of dogs who have contracted rabies virus with fully developed rabies symptoms and are infectious.

The domestic dogs population defined by N_D is divided into four compartments; first compartment is susceptible denoted by S_D , these are group of domestic dogs who are not infected but may get infection after they adequately contact with either infected free-range dogs, infected domestic dogs or environment that contains rabies virus. The second compartment is exposed symbolized as E_D which include all the domestic dogs who have contracted the disease but can not transmit the disease and do not have rabies disease symptoms. The third compartment is infectious domestic dogs denoted as I_D made up of dogs who have contracted rabies virus and are infectious. The fourth compartment is recovered denoted as R_D these are domestic dogs who get post-exposure prophylaxis after they contact with rabies infectious agent and develop temporary immunity, but can become susceptible due to temporary immunity loss.

Environment represents the virus causing rabies disease that are in the physical object or any other materials in the environment. These virus containing material are considered as virus transmitting media or the infectious agent and denoted as $M(t)$. Once the virus is shed into the environment, it can survive on surfaces for some time, making it possible for humans and animals to contract the virus if they come into adequate contact with the contaminated areas or objects. Therefore, proper hygiene, handling of animals, and vaccination are important measures to prevent the spread of rabies.

Infected dogs can shed the rabies causing virus to the environment through several ways: Saliva: Infected dogs can shed the virus in their saliva and spread it through coughing, sneezing, or breathing. Feces: Dogs infected with some viruses like parvovirus or distemper virus can shed the virus in their feces for a long time. This can contaminate the environment, including soil, water, and surfaces. Infected dogs with some viruses like Lyme disease or Rocky Mountain spotted fever can shed the virus in their blood, which can transmit to other dogs or humans through contact with infected blood or ticks. It's important to note that proper hygiene and

disinfection practices can help reduce the spread of viruses in the environment.

The Human population denoted by N_H , is divided into four compartments; first compartment are those human who have not acquired the rabies infection but may get if they adequately get into contact with infectious I_F , I_D or contaminated environment $\lambda (M)$ referred to susceptible human and denoted by S_H . The second compartment is exposed Human symbolized as E_H which are individual who have contracted the disease but can not transmit the rabies virus and do not have symptoms of rabies infections. The third compartment is the infected Human symbolized as I_H which consist of individual who have contracted the rabies virus showing all the symptoms of rabies and are infectious. The fourth compartment are recovered human beings denoted by R_H which consist of individuals who get post-exposure prophylaxis after they contact and develop temporary immunity.

3.2.1 Model Assumptions

The mathematical model describing the transmission dynamics of rabies among human populations, free-range dogs, domestic dogs, and environmental reservoirs is grounded in a set of biologically and epidemiologically plausible assumptions. These assumptions are critical in defining the structure, compartments, and interactions within the model, ensuring both mathematical tractability and relevance to real-world rabies transmission patterns.

(i) All non-infectious humans and dogs are subject to natural death:

This assumption captures the background mortality rate affecting all susceptible, exposed (non-infectious) and recovered individuals. It is biologically reasonable to consider that individuals who are not currently infected still face risks of death due to aging, accidents, or other unrelated diseases. The inclusion of natural mortality in non-infectious compartments allows for accurate modelling of population turnover.

(ii) There is reduced chance for domestic dogs to get the disease due to protection from human beings:

Domestic dogs often live under close human supervision and may receive vaccinations or veterinary care, reducing their exposure to infectious free-ranging animals and contaminated environments. This assumption introduces a differential risk between domestic and free-range dogs, acknowledging the role of human intervention in disease prevention among companion animals.

(iii) The recruitment rate for each population is greater than natural mortality:

This assumption implies a net population growth or sustainability, ensuring that the populations of humans, domestic dogs, and free-range dogs are not declining due to natural

causes alone. A recruitment rate exceeding natural mortality maintains a dynamic population structure and supports the long-term persistence of the system being modeled. It also facilitates sustained disease transmission dynamics.

- (iv) **Each population is homogeneously mixed, that is, each individual (human or dog) has an equal chance of getting the disease:**

The homogeneous mixing assumption simplifies the model by treating individuals within the same population as having equal probabilities of interacting with infectious agents. While real-world interactions may be heterogeneous due to spatial or social factors, this assumption is necessary for analytical tractability and is commonly employed in compartmental models to represent average transmission dynamics.

- (v) **No Post-exposure Prophylaxis (PEP) or Pre-exposure Prophylaxis (PrEP) is applied for free-range dogs:**

This assumption reflects the practical reality that free-ranging or stray dogs typically do not have access to medical intervention or vaccination, especially in resource-limited settings. It also highlights a critical gap in rabies control, making free-range dogs significant contributors to the maintenance and spread of rabies within the ecosystem.

- (vi) **The environment is any material or object that is contaminated by the viruses that cause rabies disease:**

This assumption expands the transmission pathways to include indirect routes via environmental contamination. Saliva from infected animals can contaminate surfaces or fomites, serving as a potential reservoir for the virus. While environmental transmission is less common than direct bites, this inclusion enhances the comprehensiveness of the model, particularly in areas where exposure to contaminated materials may occur.

- (vii) **All humans and dogs are recruited at a constant rate:**

This assumption implies a continuous and uniform inflow of new individuals (births or migration) into the respective compartments, such as susceptible humans or dog populations. Constant recruitment ensures that the model reflects sustained population renewal over time, which is essential for capturing long-term disease dynamics.

- (viii) **Upon exposure, human beings and domestic dogs receive Post-exposure Prophylaxis (PEP):**

This assumption acknowledges existing public health interventions aimed at preventing disease progression following exposure. Post-exposure Prophylaxis is effective in humans and domestic dogs when administered promptly and correctly.

3.2.2 Description of Model Interaction

Susceptible humans S_H are constantly recruited at a rate of θ_1 . When they come into adequate contact with I_F , I_D , or the virus in the environment $\lambda(M)$, individuals in the S_H category become exposed at rates τ_1 , τ_2 , and τ_3 respectively. Thus the force of infection for human is given by Equation (3.1).

$$\chi_1 = (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H, \quad (3.1)$$

Where;

$$\lambda(M) = \frac{M}{M+C}.$$

After contracting rabies infections, susceptible humans become exposed individuals, denoted by E_H , typically for 1 to 3 months. Those in the E_H category who receive PEP recover at the rate β_2 . Since PEP does not confer permanent immunity, individuals in the R_H category can lose immunity and become susceptible again at the rate β_3 . The remaining proportion of the exposed class progresses to the infectious state I_H at a rate β_1 . Infected humans can die due to the disease at a rate σ_1 . All human compartments experience natural death at a rate of μ_1 .

Free-range dogs that are susceptible to rabies are continuously introduced into the population at a rate denoted by θ_2 . These dogs become infected when they get into contact with infected free-range dogs (I_F), infected domestic dogs (I_D), or the virus in the environment, at rates κ_1 , κ_2 , and κ_3 , respectively. The force of infection for free-range dogs is calculated using Equation (3.2).

$$\chi_2 = (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F. \quad (3.2)$$

After contracting the rabies infection, free-range dogs that are susceptible progress to the exposed state, E_F , which lasts for 1 to 3 months. The exposed free-range dogs then progress to the infectious state, I_F , at a rate of γ . Infected free-range dogs may die due to the disease at a rate of σ_2 . Additionally, all free-range dog compartments experience natural death at a rate of μ_2 .

Susceptible domestic dogs are constantly recruited at a rate of θ_3 . These dogs become exposed when they come into adequate contact with I_F , I_D , or the rabies virus in the environment, at the rates ψ_1 , ψ_2 , and ψ_3 , respectively. Since domestic dogs are under human control, they have a lower risk of contracting the disease. Hence, the force of infection for free-range dogs is given by Equation (3.3).

$$\chi_3 = \left(\frac{\psi_1 I_F}{1+\rho_1} + \frac{\psi_2 I_D}{1+\rho_2} + \frac{\psi_3}{1+\rho_3} \lambda(M) \right) S_D. \quad (3.3)$$

After contracting a rabies infection, domestic dogs enter a state E_D , where they stay for several

months at a rate of β_1 . If dogs in the E_D category receive post-exposure prophylaxis, they move to the R_D category at a rate of γ_2 . However, PEP does not provide permanent immunity, and dogs in the R_D category can lose their immunity and become susceptible again at a rate of γ_3 . Meanwhile, the remaining proportion of the exposed class progresses to the infectious state I_D at a rate of γ_1 . Infected domestic dogs can die from the disease at a rate of σ_3 . All compartments of domestic dogs experience natural death at a rate of μ_3 .

Rabies virus is spread in the environment through shedding by infected free-range and domestic dogs, as well as humans, at rates denoted by v_2 , v_3 , and v_1 , respectively. Equation (3.4) can be used to estimate the virus recruitment in the environment.

$$\theta_4 = (v_1 I_H + v_2 I_F + v_3 I_D) M. \quad (3.4)$$

The viruses are removed from the environment at a rate μ_4 .

3.3 Model Parameters and Variables

The state variables and model parameters utilized in the formulation of the rabies transmission dynamics are comprehensively defined in Tables (2) and (3). These tables provide precise descriptions and relevant notations that underpin the structure of the mathematical model, thereby facilitating clarity in the interpretation and analysis of the system's behavior, respectively.

3.3.1 Description of State Variables

Table 2: Model state variables and their descriptions

Variable	Description
$S_H(t)$	susceptible Human at time t
$E_H(t)$	Exposed Human at time t
$I_H(t)$	Infected Human at time t
$R_H(t)$	Recovered Human at time t
$S_F(t)$	Susceptible free- range dog at time t
$E_F(t)$	Exposed free-range dog at time t
$I_F(t)$	Infected free-range dog at time t
$S_D(t)$	Susceptible domestic dog at time t
$E_D(t)$	Exposed domestic dog at time t
$I_D(t)$	Infected domestic dog at time t
$R_D(t)$	Recovered domestic dog at time t
$M(t)$	Number of rabies virus per unit volume in the environment at time t

3.3.2 Description of Model Parameters

Table 3: Model parameters and their descriptions

Parameters	Descriptions
θ_1	Recruitment rate of susceptible human
τ_1	The rate that S_H gets infection from I_F .
τ_2	The rate that S_H gets infection from I_D .
τ_3	The rate that S_H gets infection from M .
β_1	Progression rate out of E_H to I_H .
β_2	Recovery rate of E_H .
β_3	Rate of immunity loss of human being.
μ_1	Natural death rate of human being.
σ_1	Disease induced death rate for I_H .
θ_2	Recruitment rate of free-range dogs.
κ_1	The rate that S_F gets infection from I_F .
κ_2	The rate that S_F gets infection from I_D .
κ_3	The rate that S_F gets infection from M .
σ_2	Disease induced death rate of I_F
μ_2	Natural mortality rate of free-range dogs
θ_3	Recruitment rate of domestic dog population
ψ_1	The rate that S_D gets infection from I_D .
ψ_2	The rate that S_D gets infection from I_F .
ψ_3	The rate that S_D gets infection from M .
μ_3	Natural death rate for domestic dog population.
σ_3	Disease induced death rate for I_D .
γ	The rate at which E_F becomes I_F .
γ_1	The rate at which E_D becomes I_D .
γ_2	Recovery rate of E_D
γ_3	Rate of loss of temporary immunity for R_D .
v_1, v_2, v_3	Environmental virus shedding rate from I_H , I_F and I_D respectively
μ_4	Natural removal rate of rabies from the environment.
ρ_1	The deterrent coefficient of domestic dog from I_F .
ρ_2	The deterrent coefficient of domestic dog from I_D .
ρ_3	The deterrent coefficient of domestic dog from M .
C	Concentration of rabies in the environment.

3.3.3 Flow Diagram

Based on the model description, underlying assumptions, and the compartmental flow diagram, a system of twelve differential equations was systematically formulated, as presented in Equation (3.5). The corresponding dynamics of rabies transmission among free-range dogs, domestic dogs, humans, and the environment are comprehensively depicted in Fig. 2.

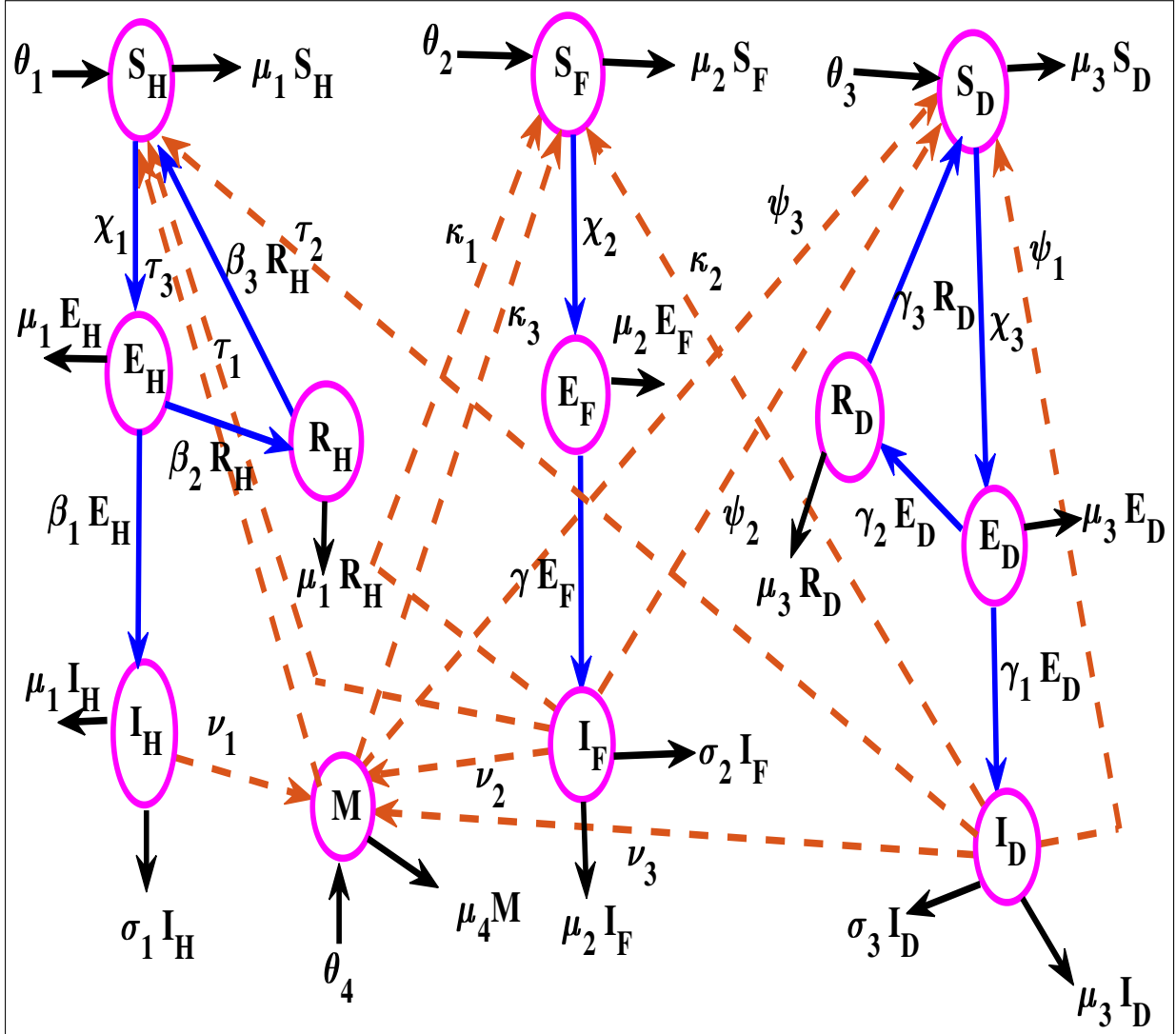


Figure 2: Basic Compartmental Model Diagram

3.3.4 Model Equations

Based on an analysis of the model parameters and their relationships to the state variables, we construct a model in the form of a system of ODEs as delineated in Equation (3.5).

$$\left\{ \begin{array}{l} \dot{S}_H = \theta_1 + \beta_3 R_H - \mu_1 S_H - \chi_1, \\ \dot{E}_H = \chi_1 - (\mu_1 + \beta_1 + \beta_2) E_H, \\ \dot{I}_H = \beta_1 E_H - (\sigma_1 + \mu_1) I_H, \\ \dot{R}_H = \beta_2 E_H - (\beta_3 + \mu_1) R_H, \\ \dot{S}_F = \theta_2 - \chi_2 - \mu_2 S_F, \\ \dot{E}_F = \chi_2 - (\mu_2 + \gamma) E_F, \\ \dot{I}_F = \gamma E_F - (\mu_2 + \sigma_2) I_F, \\ \dot{S}_D = \theta_3 - \mu_3 S_D - \chi_3 + \gamma_3 R_D, \\ \dot{E}_D = \chi_3 - (\mu_3 + \gamma_1 + \gamma_2) E_D, \\ \dot{I}_D = \gamma_1 E_D - (\mu_3 + \sigma_3) I_D, \\ \dot{R}_D = \gamma_2 E_D - (\mu_3 + \gamma_3) R_D, \\ \dot{M} = (v_1 I_H + v_2 I_F + v_3 I_D) - \mu_4 M. \end{array} \right. \quad (3.5)$$

Subject to the following non-negative conditions;

$$\begin{aligned} S_H(0) > 0, \quad E_H(0) \geq 0, \quad I_H(0) \geq 0, \quad R_H(0) \geq 0, \quad S_F(0) > 0, \quad E_F(0) \geq 0, \quad I_F(0) \geq 0, \\ S_D(0) > 0, \quad E_D(0) \geq 0, \quad I_D(0) \geq 0, \quad R_D(0) \geq 0. \end{aligned}$$

3.4 Properties of the Rabies Model

3.4.1 Model's Invariant Region

In this subsection, we consider the Model (3.5) under the assumption that all state variables and parameters are non-negative for all $t \geq 0$. Under this framework, Theorem (3.1) is employed to analyze the system within a biologically meaningful and mathematically feasible region. This analysis leads to the determination of the invariant region of the rabies model, as described below:

Theorem 3.1

Solution of the rabies Model (3.5) is uniformly bounded if $\Omega \in \mathbb{R}_+^{12}$ and $\Omega = \Omega_H \cup \Omega_D \cup \Omega_F \cup$

$\Omega_M \in \mathbb{R}_+^4 \times \mathbb{R}_+^3 \times \mathbb{R}_+^4 \times \mathbb{R}_+^1$ where,

$$\begin{aligned}\Omega_H &= \left\{ (S_H, E_H, I_H, R_H) \in \mathbb{R}_+^4 : 0 \leq N_H \leq \frac{\theta_1}{\mu_1} \right\}, \\ \Omega_F &= \left\{ (S_F, E_F, I_F) \in \mathbb{R}_+^3 : 0 \leq N_F \leq \frac{\theta_2}{\mu_2} \right\}, \\ \Omega_D &= \left\{ (S_D, E_D, I_D, R_D) \in \mathbb{R}_+^4 : 0 \leq N_D \leq \frac{\theta_3}{\mu_3} \right\}.\end{aligned}$$

and Ω is the positive invariant region.

Proof. By utilizing Theorem 3.1, we derive the invariant region of the rabies model as follows. Consider the population of the human from Equation (3.5) as:

$$\frac{dN_H}{dt} = \frac{dS_H}{dt} + \frac{dE_H}{dt} + \frac{dI_H}{dt} + \frac{dR_H}{dt}. \quad (3.6)$$

Then, Equation (3.6) implies to Equation (3.7) as follows:

$$\frac{dN_H}{dt} = \theta_1 - (S_H + E_H + I_H + R_H)\mu_1 - \sigma_1 I_H. \quad (3.7)$$

But if $N_H = S_H + E_H + I_H + R_H$, then Equation (3.7) can be written as:

$$\frac{dN_H}{dt} = \theta_1 - N_H\mu_1, \quad (3.8)$$

and by utilizing the integrating factor in Equation (3.8) and $t \rightarrow 0$, we have:

$$N_H(0) \leq \frac{\theta_1}{\mu_1} + Ce^0 \implies N_H(0) - \frac{\theta_1}{\mu_1} \leq C. \quad (3.9)$$

By simplifying Equation (3.9) and after simple manipulations, it follows that,

$$\Omega_H = \left\{ (S_H, E_H, I_H, R_H) \in \mathbb{R}_+^4 : 0 \leq N_H \leq \frac{\theta_1}{\mu_1} \right\}.$$

Employing the same procedures for free range and domestic dog populations, we obtain:

$$\Omega_F = \left\{ (S_F, E_F, I_F) \in \mathbb{R}_+^3 : 0 \leq N_F \leq \frac{\theta_2}{\mu_2} \right\}, \quad \Omega_D = \left\{ (S_D, E_D, I_D, R_D) \in \mathbb{R}_+^4 : 0 \leq N_D \leq \frac{\theta_3}{\mu_3} \right\}. \quad (3.10)$$

Again, from the environment that contains rabies virus,

$$\dot{M} = (v_1 I_H + v_2 I_F + v_3 I_D) - \mu_4 M. \quad (3.11)$$

Since $N_H \leq \frac{\theta_1}{\mu_1}$, $N_F \leq \frac{\theta_2}{\mu_3}$, and $N_D \leq \frac{\theta_3}{\mu_3}$, it follows that $I_H \leq \frac{\theta_1}{\mu_1}$, $I_F \leq \frac{\theta_2}{\mu_2}$, and $I_D \leq \frac{\theta_3}{\mu_3}$. Therefore, Equation (3.11) can be expressed as:

$$\dot{M} \leq \left(\frac{v_1 \theta_1}{\mu_1} + \frac{v_2 \theta_2}{\mu_2} + \frac{v_3 \theta_3}{\mu_3} \right) - \mu_4 M. \quad (3.12)$$

Now, let Y be the solution which is unique to the initial value problem such that:

$$\left. \begin{aligned} \dot{Y} &\leq \left(\frac{v_1 \theta_1}{\mu_1} + \frac{v_2 \theta_2}{\mu_2} + \frac{v_3 \theta_3}{\mu_3} \right) - \mu_4 M, \text{ for } t > 0 \\ Y(0) &= M(0). \end{aligned} \right\} \quad (3.13)$$

By using integration factor Equation (3.13) as $t \rightarrow \infty$, we get the expression:

$$\left(M(0) - \left(\frac{v_1 \theta_1}{\mu_1} + \frac{v_2 \theta_2}{\mu_2} + \frac{v_3 \theta_3}{\mu_3} \right) \frac{1}{\mu_4} e^{\mu_4 t} \right) \quad (3.14)$$

and as Equation (3.14) goes to zero, we have:

$$M(t) \leq \Omega_M. \quad (3.15)$$

Thus, the Model (3.5) is biologically and mathematically meaningfully such that their solution relies in the region Ω . $\square$

3.4.2 Positivity of the Solution

For the Model (3.5) to be epidemiologically meaningfully and well-posed, we need to prove that the state variables are non-negative such that $\forall t \geq 0$.

Lemma 3.1

All solutions of the system that start in the region $\Omega \subset \mathbb{R}_+^{12}$ remain positive all the time.

Proof. To prove the existence of a solution to Model (3.5), we consider initial conditions and apply the integral operator $\int_0^t (\cdot) ds$ on each compartment of the Model (3.5) as:

$$\dot{S}_H = \theta_1 + \beta_3 R_H - \mu_1 S_H - (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H. \quad (3.16)$$

Integrating (3.16) both sides over $[0, t]$, we get that:

$$\int_0^t \dot{S}_H dt = \int_0^t (\theta_1 + \beta_3 R_H - \mu_1 S_H - (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H) dt. \quad (3.17)$$

Then, the left-hand side of (3.17) leads to $S_H(t) - S_H(0)$ and the right-hand side of the same Equation leads to:

$$\int_0^t \theta_1 dt + \int_0^t \beta_3 R_H dt - \int_0^t \mu_1 S_H dt - \int_0^t (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H dt. \quad (3.18)$$

By simplifying each integral in (3.18), we have:

$$\int_0^t f(s) ds = \begin{cases} \theta_1 t, & \text{if } f(s) = \theta_1, \\ \beta_3 \int_0^t R_H(s) ds, & \text{if } f(s) = \beta_3 R_H(s), \\ \mu_1 \int_0^t S_H(s) ds, & \text{if } f(s) = \mu_1 S_H(s), \\ \int_0^t (\tau_1 I_F(s) + \tau_2 I_D(s) + \tau_3 \lambda(M)) S_H(s) ds, & \text{for disease dynamics.} \end{cases} \quad (3.19)$$

By combining and rearranging the results in (3.19), it follows that:

$$S_H(t) = S_H(0) + \theta_1 t + \beta_3 \int_0^t R_H(s) ds - \mu_1 \int_0^t S_H(s) ds - \int_0^t (\tau_1 I_F(s) + \tau_2 I_D(s) + \tau_3 \lambda(M)) S_H(s) ds. \quad (3.20)$$

Thus, we conclude with the non-negativity of the integral terms:

$$S_H(t) \geq S_H(0) + \theta_1 t + \beta_3 \int_0^t R_H(s) ds - \mu_1 (\tau_1 I_F(s) + \tau_2 I_D(s) + \tau_3 \lambda(M)) \int_0^t S_H(s) ds, \quad (3.21)$$

since $S_H(0) \geq 0$, $\theta_1 \geq 0$, $\beta_3 R_H(s) \geq 0$, $s \in [0, t]$. It follows that $S_H(t) \geq 0$ for all $t \geq 0$. Similarly, we prove the positivity of $I_H(t)$ by considering:

$$\dot{I}_H = \beta_1 E_H - (\sigma_1 + \mu_1) I_H. \quad (3.22)$$

Rearranging Equation (3.22) and applying the integrating factor $\mu(t)$, it results in Equation (3.23):

$$e^{(\sigma_1 + \mu_1)t} I_H = I_H(0) + \int_0^t \beta_1 e^{(\sigma_1 + \mu_1)s} E_H(s) ds. \quad (3.23)$$

By solving for $I_H(t)$ in Equation (3.23), we obtain:

$$I_H(t) = e^{-(\sigma_1 + \mu_1)t} \left(I_H(0) + \int_0^t \beta_1 e^{(\sigma_1 + \mu_1)s} E_H(s) ds \right).$$

Since $I_H(0) \geq 0$ and $E_H(s) \geq 0$ for $s \geq 0$, it follows that:

$$I_H(t) \geq 0 \quad \forall t \geq 0. \quad (3.24)$$

Using the same procedure, we conclude that:

$$\begin{aligned} S_H(t) &> 0, E_H(t) \geq 0, I_H(t) \geq 0, R_H(t) \geq 0, S_F(t) > 0, E_F(t) \geq 0, I_F(t) \geq 0, \\ S_D(t) &\geq 0, E_D(t) \geq 0, I_D(t) \geq 0, R_D(t) \geq 0, \forall t \geq 0. \end{aligned}$$

and the proof is complete. $\square$

3.5 Model Analysis

3.5.1 Rabies Disease Free Equilibrium Point ($\mathcal{E}_0$)

The term $\mathcal{E}_0$ represents the state of no rabies disease within a population. To achieve $\mathcal{E}_0$ in both human and dog populations, all infectious compartments in Equation (3.5) must be set to zero, resulting in:

$$\mathcal{E}_0 = \left(\frac{\theta_1}{\mu_1}, 0, 0, 0, \frac{\theta_2}{\mu_2}, 0, 0, \frac{\theta_3}{\mu_3}, 0, 0, 0, 0 \right).$$

3.5.2 The Basic Reproduction Number, $\mathcal{R}_0$

The $\mathcal{R}_0$ predicts whether rabies will spread across the community or die out. If $\mathcal{R}_0 < 1$, it means that every infectious individual will cause less than one secondary infection, hence the disease will die out in the community. If $\mathcal{R}_0 > 1$, it means that every infectious individual will cause more than one secondary infection, leading to the persistence of rabies in the entire population. To compute the $\mathcal{R}_0$, we utilize the next-generation matrix approach, following the methods of LaSalle (1976) and Yang (2014), in conjunction with the Jacobian matrix, as follows:

$$\frac{dx_i}{dt} = \mathcal{F}_i(x) - (\mathcal{V}_i^+(x) - \mathcal{V}_i^-(x)) \quad (3.25)$$

Where $\mathcal{F}_i$ is the new infections in the compartment i while $\mathcal{V}_i^+$ and $\mathcal{V}_i^-$ are the transfer terms in and out of the compartment i respectively. From Equation (3.5), $\mathcal{F}_i$ and $\mathcal{V}_i$ can be defined as;

$$\mathcal{F}_i = \begin{pmatrix} (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H \\ 0 \\ (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F \\ 0 \\ \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right) S_D \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V}_i = \begin{pmatrix} (\mu_1 + \beta_1 + \beta_2) E_H \\ (\sigma_1 + \mu_1) I_H - \beta_1 E_H \\ (\mu_2 + \gamma) E_F \\ (\mu_2 + \sigma_2) I_F - \gamma E_F \\ (\mu_3 + \gamma_1 + \gamma_2) E_D \\ (\mu_3 + \delta_3) I_D - \gamma_1 E_D \\ Q \end{pmatrix} \quad (3.26)$$

For $Q = \mu_4 M - (v_1 I_H + v_2 I_F + v_3 I_D)$. The Jacobian Matrices F and V at $\mathcal{E}_0$ is given by Equation (3.27) ;

$$F = \frac{\partial \mathcal{F}_i(\mathcal{E}_0)}{\partial x_j}, V = \frac{\partial \mathcal{V}_i(\mathcal{E}_0)}{\partial x_j} \quad (3.27)$$

From Equation (3.26) , F and V at $\mathcal{E}_0$ is given in Equation (3.28) and (3.29) respectively;

$$\left. \frac{\partial \mathcal{F}_i}{\partial x_j} \right|_{\mathcal{E}_0} = F = \begin{pmatrix} 0 & 0 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\psi_1 \theta_3}{(1 + \rho_1) \mu_3} & 0 & \frac{\psi_2 \theta_3}{(1 + \rho_2) \mu_3} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}. \quad (3.28)$$

In the linearized system (3.28), the entry F_{ij} represents the rate at which individuals in the infected state j give rise to or develop new infections in individuals in the infected state i , with reference to the infected states indexed by i and j for $i, j \in [1, 2, 3, 4, 5, 6, 7]$. As a result, when a person in an infected condition j does not instantly produce any new instances in an infected

state i , we have $F_{ij} = 0$, and,

$$\left. \frac{\partial \mathcal{V}_i}{\partial x_j} \right|_{\mathcal{E}_0} = V = \begin{pmatrix} \mu_1 + \beta_1 + \beta_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\beta_1 & \sigma_1 + \mu_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \mu_2 + \gamma & 0 & 0 & 0 & 0 \\ 0 & 0 & -\gamma & \mu_2 + \sigma_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \mu_3 + \gamma_1 + \gamma_2 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\gamma_1 & \mu_3 + \sigma_3 & 0 \\ 0 & -v_1 & 0 & -v_2 & 0 & -v_3 & \mu_4 \end{pmatrix}. \quad (3.29)$$

Then, the inverse of matrix V is obtained using Maple software, and its result is given as:

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu_1 + \beta_1 + \beta_2} & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_1}{(\sigma_1 + \mu_1)(\mu_1 + \beta_1 + \beta_2)} & \frac{1}{\sigma_1 + \mu_1} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{\mu_2 + \gamma} & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\gamma}{(\mu_2 + \gamma)(\mu_2 + \sigma_2)} & \frac{1}{\mu_2 + \sigma_2} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{\mu_3 + \gamma_1 + \gamma_2} & 0 & 0 \\ 0 & 0 & 0 & 0 & A & \frac{1}{\mu_3 + \sigma_3} & 0 \\ B & C & \frac{v_2 \gamma}{(\mu_2 + \gamma)(\mu_2 + \sigma_2)\mu_4} & \frac{v_2}{(\mu_2 + \sigma_2)\mu_4} & D & \frac{v_3}{(\mu_3 + \sigma_3)\mu_4} & \frac{1}{\mu_4} \end{pmatrix} \quad (3.30)$$

Where;

$$\begin{cases} A = \frac{\gamma_1}{(\mu_3 + \sigma_3)(\mu_3 + \gamma_1 + \gamma_2)}, & B = \frac{v_1 \beta_1}{(\sigma_1 + \mu_1)(\mu_1 + \beta_1 + \beta_2)\mu_4}, \\ C = \frac{v_1}{(\sigma_1 + \mu_1)\mu_4}, & D = \frac{v_3 \gamma_1}{(\mu_3 + \sigma_3)(\mu_3 + \gamma_1 + \gamma_2)\mu_4}. \end{cases}$$

In the context of computing the $\mathcal{R}_0$ in epidemiology, the $(V^{-1})_{ij}$ obtained in Equation (3.30) represents the generation matrix. The generation matrix describes the expected number of newly infected individuals that a single infectious individual in each of the different susceptible classes generates. Meanwhile, the diagonal elements represent the rate of leaving the corresponding susceptible class due to other causes such as recoveries caused by administration of PEP. In particular, $\frac{1}{\mu_1 + \beta_1 + \beta_2}$, $\frac{1}{\mu_2 + \gamma}$, $\frac{1}{\mu_3 + \gamma_1 + \gamma_2}$ respectively, represent the average incubation pe-

riod for rabies in humans, free-range dogs, and domestic dogs. Meanwhile, $\frac{1}{\mu_1 + \sigma_1}$, $\frac{1}{\mu_2 + \sigma_2}$, $\frac{1}{\mu_3 + \sigma_3}$ respectively, represent the average time spent by an infective human, free-range dog, and domestic dog in the infectious state, and $\frac{1}{\mu_4}$ is the average time the rabies virus spends in M. The next generation Matrix FV^{-1} is then calculated as in Equation (3.31):

$$FV^{-1} = \begin{pmatrix} 0 & 0 & R_{13} & R_{14} & R_{15} & R_{16} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & R_{33} & R_{34} & R_{35} & R_{36} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & R_{53} & R_{54} & R_{55} & R_{56} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}. \quad (3.31)$$

Where,

$$\left\{ \begin{array}{l} R_{13} = \frac{\tau_1 \theta_1 \gamma}{\mu_1 (\mu_2 + \gamma) (\mu_2 + \sigma_2)}, \quad R_{14} = \frac{\tau_1 \theta_1}{\mu_1 (\mu_2 + \sigma_2)}, \quad R_{15} = \frac{\tau_2 \theta_1 \gamma}{\mu_1 (\mu_3 + \gamma_1 + \gamma_2) (\mu_3 + \sigma_3)}, \\ R_{33} = \frac{\kappa_1 \theta_2 \gamma}{\mu_2 (\mu_2 + \gamma) (\mu_2 + \sigma_2)}, \quad R_{34} = \frac{\kappa_1 \theta_2}{\mu_2 (\mu_2 + \sigma_2)}, \quad R_{35} = \frac{\kappa_1 \theta_2 \gamma}{\mu_2 (\mu_3 + \gamma_1 + \gamma_2) (\mu_3 + \sigma_3)}, \\ R_{53} = \frac{\psi_1 \theta_3 \gamma}{(1 + \rho_1) (\mu_2 + \gamma) (\mu_2 + \sigma_2) \mu_3}, \quad R_{54} = \frac{\psi_1 \theta_3}{(1 + \rho_1) \mu_3 (\mu_2 + \sigma_2)}, \\ R_{56} = \frac{\psi_2 \theta_3}{(1 + \rho_2) \mu_3 (\mu_3 + \sigma_3)}, \quad R_{16} = \frac{\tau_2 \theta_1}{\mu_1 (\mu_3 + \sigma_3)}, \quad R_{36} = \frac{\kappa_1 \theta_2}{\mu_2 (\mu_3 + \sigma_3)}, \\ R_{55} = \frac{\psi_2 \theta_3 \gamma}{(1 + \rho_2) (\mu_3 + \gamma_1 + \gamma_2) (\mu_3 + \sigma_3) \mu_3}. \end{array} \right. \quad (3.32)$$

Where R_{13} denotes the average number of humans who become exposed following adequate contact with infected free-range dogs, and R_{14} represents the average number of humans who progress to infection after being exposed through contact with free-range dogs. Similarly, R_{15} is the average number of humans exposed after adequate contact with infected domestic dogs, while R_{16} captures the average number of humans who progress to infection after exposure resulting from contact with domestic dogs.

Furthermore, R_{33} denotes the average number of free-range dogs exposed through adequate contact with infected free-range dogs, whereas R_{34} represents the average number of free-range dogs that become infected following progression from the exposed class due to contact with infected free-range dogs. Likewise, R_{53} is the average number of domestic dogs exposed through adequate contact with infected domestic dogs, and R_{54} represents the average number of domestic dogs that become infected after progressing from exposure due to contact with infected

free-range dogs.

Finally, R_{55} denotes the average number of domestic dogs exposed through adequate contact with infected domestic dogs, while R_{56} represents the average number of domestic dogs that become infected after progressing from exposure resulting from contact with infected domestic dogs.

From Equation (3.31), we obtained the eigenvalues as:

$$\left(0, 0, 0, 0, 0, \frac{1}{2}R_{55} + \frac{1}{2}R_{33} + \frac{1}{2}\sqrt{R_{33}^2 - 2R_{33}R_{55} + 4R_{35}R_{53} + R_{55}^2}, \right. \\ \left. \frac{1}{2}R_{55} + \frac{1}{2}R_{33} - \frac{1}{2}\sqrt{R_{33}^2 - 2R_{33}R_{55} + 4R_{35}R_{53} + R_{55}^2} \right). \quad (3.33)$$

The (i, k) element of the FV^{-1} of the next generation matrix represents the expected number of secondary infections in the compartment i caused by individuals in compartment k , assuming that the individual's environments remain consistent throughout the infection. It is worth noting that FV^{-1} Matrix is non-negative, meaning has a non-negative eigenvalue. This non-negative eigenvalue corresponds to a non-negative eigenvector that represents the distribution of infected individuals who generate the highest number of secondary infections per generation, also known as $\mathcal{R}_0$. $\mathcal{R}_0$ according to Dharmaratne *et al.* (2020) is the largest eigenvalue of FV^{-1} given by;

$$\mathcal{R}_0 = \rho(FV^{-1}). \quad (3.34)$$

The basic reproduction number, $\mathcal{R}_0$, is given by the spectral radius of the next-generation matrix:

$$\mathcal{R}_0 = \rho(FV^{-1}) = \frac{(R_{55} + R_{33}) + \sqrt{R_{33}(R_{33} - 2R_{55}) + 4R_{35}R_{53} + R_{55}^2}}{2}. \quad (3.35)$$

Where,

$$\begin{cases} R_{33} = \frac{\kappa_1 \theta_2 \gamma}{\mu_2(\mu_2 + \gamma)(\mu_2 + \sigma_2)}, & R_{35} = \frac{\kappa_1 \theta_2 \gamma}{\mu_2(\mu_3 + \gamma_1 + \gamma_2)(\mu_3 + \sigma_3)} \\ R_{53} = \frac{\psi_1 \theta_3 \gamma}{(1 + \rho_1)(\mu_2 + \gamma)(\mu_2 + \sigma_2)\mu_3}, \\ R_{55} = \frac{\psi_2 \theta_3 \gamma}{(1 + \rho_2)(\mu_3 + \gamma_1 + \gamma_2)(\mu_3 + \sigma_3)\mu_3}. \end{cases} \quad (3.36)$$

Where R_{33} denote the average number of free-range dogs exposed through interactions between susceptible and infected free-range dogs, while R_{35} represents exposure of free-range dogs resulting from contact with infected domestic dogs. Likewise, R_{53} and R_{55} correspond to the

average number of domestic dogs exposed through interactions with infected free-range and infected domestic dogs, respectively.

3.5.3 Existence of the Steady State Solution $\mathcal{E}^*$

The endemic equilibrium point is the steady state where rabies is present in humans, free-range dogs, and domestic dogs. To find this point, we set the Equations of the Model (3.5) to zero and solve the resulting system simultaneously. The state variables for each compartment are represented by;

$$\mathcal{E}^* (S_H^*, E_H^*, I_H^*, R_H^*, S_F^*, E_F^*, I_F^*, S_D^*, E_D^*, I_D^*, R_D^*, M^*).$$

$$\left\{ \begin{array}{l} R_H^* = \frac{\beta_2 (\sigma_1 + \mu_1) I_H^*}{\beta_1 (\beta_3 + \mu_1)}, \quad E_H^* = \frac{(\sigma_1 + \mu_1) I_H^*}{\beta_1}, \\ I_H^* = \frac{\beta_1 (\beta_3 + \mu_3) (\sigma_1 + \mu_1)^2 (\beta_1 + \beta_2 + \beta_3) \mu_1 + \beta_1 \beta_3 (\sigma_1 + \mu_1)^2}{(\sigma_1 + \mu_1)^2 ((\beta_1 + \beta_2 + \beta_3) \mu_1 + \beta_1 \beta_3)} \\ \quad - \frac{\beta_1 (\beta_3 + \mu_3) (\sigma_1 + \mu_1)^2 \beta_3 - \theta_1 (\beta_3 + \mu_3) (\sigma_1 + \mu_1)^2}{(\sigma_1 + \mu_1)^2 ((\beta_1 + \beta_2 + \beta_3) \mu_1 + \beta_1 \beta_3)}, \\ S_H^* = \frac{\beta_3 \beta_2 (\sigma_1 + \mu_1) I_H^*}{\beta_1 (\beta_3 + \mu_1) \mu_1} - \frac{(\mu_1 + \beta_1 + \beta_2) (\sigma_1 + \mu_1) I_H^*}{\beta_1 \mu_1} + \frac{\theta_1}{\mu_1} \end{array} \right.$$

Re-writing Eq. (3.35) as

$$(R_0 - 1) (R_0 - R_{33} - R_{55} + 1) + (1 - R_{33}) (1 - R_{55}) - R_{35} R_{53} = 0, \quad (3.37)$$

where R_{33} , R_{55} , R_{35} , and R_{53} are defined in Equation (3.32), we get:

$$\left\{ \begin{array}{l} I_D^* = \frac{\gamma_1 \psi_1 I_F^* (1 + \rho_2) (1 + \rho_3) M^* + \gamma_1 \psi_3 M^* (1 + \rho_1) (1 + \rho_2)}{(\mu_3 + \gamma_1 + \gamma_2)^2 - \gamma_1 \psi_2 (1 + \rho_1) (1 + \rho_3) M^* (\mu_3 + \gamma_1 + \gamma_2)}, \\ E_D^* = \frac{(\mu_3 + \sigma_3) I_D^*}{\gamma_1}, \quad R_D^* = \frac{\gamma_2 (\mu_3 + \sigma_3) I_D^*}{\gamma_1 (\mu_3 + \gamma_3)}, \quad E_F^* = \frac{(\mu_2 + \sigma_2) I_F^*}{\gamma}, \\ S_D^* = \frac{\gamma_3 (\mu_3 + \sigma_3) I_D^*}{\mu_3 \gamma_1} - \frac{(\mu_3 + \gamma_1 + \gamma_2) \gamma_2 (\mu_3 + \sigma_3) I_D^*}{\gamma_1 (\mu_3 + \gamma_3) \mu_3} + \frac{\theta_3}{\mu_3}, \\ S_F^* = \frac{\theta_2}{\mu_2} - \frac{(\mu_2 + \gamma) (\mu_2 + \sigma_2) I_F^*}{\gamma \mu}, \quad M^* = \frac{v_3 I_D^* + v_2 I_F^* + v_1 I_H^*}{\mu_4}, \end{array} \right.$$

where;

$$\left\{ \begin{array}{l} \theta_2 = \frac{(\mu_2 + \gamma) \mu_2 (1 + (R_0 - 1)) (1 + \rho_1) \mu_3 (\mu_2 + \sigma_2)}{[\mu_3 (1 + \rho_1) (1 + \rho_2) (\mu_3 + \sigma_3) (\mu_3 + \gamma_1 + \gamma_2) R_0 - \theta_3 \gamma_1 (\psi_2 (1 + \rho_1) \mu_3 + \psi_1 (1 + \rho_2))] \gamma \kappa_1} \\ \quad \times [(1 + \rho_2) (\mu_3 + \sigma_3) (\mu_3 + \gamma_1 + \gamma_2) (1 + (R_0 - 1)) - \theta_3 \psi_2 \gamma_1], \\ \theta_3 = \frac{[-\mu_2 (\mu_2 + \sigma_2) (\mu_2 + \gamma) R_0 + \gamma \kappa_1 \theta_2] (1 + (R_0 - 1)) (1 + \rho_1) \mu_3 (1 + \rho_2) (\mu_3 + \sigma_3) (\mu_3 + \gamma_1 + \gamma_2)}{[-\mu_2 (\mu_2 + \sigma_2) (\mu_2 + \gamma) (1 + (R_0 - 1)) + \gamma \kappa_1 \theta_2] (1 + \rho_1) \psi_2 \mu_3 + \gamma \kappa_1 \theta_2 \psi_1 (1 + \rho_2)} \gamma_1. \end{array} \right.$$

The endemic equilibrium point $\mathcal{E}^*$ of the rabies disease persists when $I_H, I_F, I_D, M > 0$ and $\mathcal{R}_0 \geq 1$, as summarized in Theorem 3.2.

Theorem 3.2

The system (3.5) has a unique $\mathcal{E}^*$ if $\mathcal{R}_0 \geq 1$ and $I_H, I_F, I_D, M > 0$.

3.6 Stability Analysis

3.6.1 Local Stability of the Disease Free Equilibrium Point $\mathcal{E}_0$

We prove local stability of the disease free equilibrium point with the help of the Routh–Hurwitz criterion.

Theorem 3.3

$\mathcal{E}_0$ is locally asymptotically stable if $\mathcal{R}_0 < 1$ and all eigenvalues of the Jacobian matrix ($J(\mathcal{E}_0)$) evaluated at $\mathcal{E}_0$ have negative real parts.

Proof. To show that all the eigenvalues of the matrix $J(\mathcal{E}_0)$ in Equation (3.38) of the Model (3.5) at the $\mathcal{E}_0$ have negative real parts. Subsequently, since the endemic equilibrium exists if, and only if, $\mathcal{R}_0 < 1$, we utilize the Jacobian matrix at the disease-free state $J(\mathcal{E}_0)$, which is expressed as:

$$J(\mathcal{E}_0) = \begin{pmatrix} -\mu_1 & 0 & 0 & 0 & 0 & 0 & -\frac{\tau_1 \theta_1}{\mu_1} & 0 & 0 & -\frac{\tau_2 \theta_1}{\mu_1} & 0 & 0 \\ 0 & -a_1 & 0 & 0 & 0 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 & 0 \\ 0 & \beta_1 & -a_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_2 & 0 & -a_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_2 & 0 & -\frac{\kappa_1 \theta_2}{\mu_2} & 0 & 0 & -\frac{\kappa_2 \theta_2}{\mu_2} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -a_4 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & 0 & \frac{\kappa_2 \theta_2}{\mu_2} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma & -a_5 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\psi_1 \theta_3}{\mu_3(1+\rho_1)} & -\mu_3 & 0 & -\frac{\psi_2 \theta_3}{\mu_3(1+\rho_2)} & \gamma_3 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\psi_1 \theta_3}{\mu_3(1+\rho_1)} & 0 & -a_6 & \frac{\psi_2 \theta_3}{\mu_3(1+\rho_1)} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma & -a_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_2 & 0 & -a_8 & 0 \\ 0 & 0 & \nu_1 & 0 & 0 & 0 & \nu_2 & 0 & 0 & \nu_3 & 0 & -\mu_4 \end{pmatrix}, \quad (3.38)$$

where;

$$\begin{aligned} a_1 &= \mu_1 + \beta_1 + \beta_2, \quad a_2 = \mu_1 + \sigma_1, \quad a_3 = \mu_3 + \beta_3, \quad a_4 = \mu_2 + \gamma, \\ a_5 &= \mu_2 + \sigma_2, \quad a_6 = \mu_3 + \gamma_1 + \gamma_2, \quad a_7 = \mu_3 + \sigma_3, \quad a_8 = \mu_3 + \gamma_3. \end{aligned}$$

The first, fourth, fifth, eighth, and twelfth columns of $J(\mathcal{E}_0)$ in Equation (3.38) contain the diagonal terms. It is obvious from the eigenvalues $\lambda_1 = -\mu_1$, $\lambda_2 = -a_3$, $\lambda_3 = -\mu_2$, $\lambda_4 = -\mu_3$,

and $\lambda_5 = -\mu_4$, respectively. Thus, $J(\mathcal{E}_0)$ reduces to:

$$J_1(\mathcal{E}_0) = \begin{pmatrix} -a_1 & 0 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ \beta_1 & -a_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -a_4 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} & 0 \\ 0 & 0 & \gamma & -a_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\psi_1 \theta_3}{\mu_3(1+\rho_1)} & -a_6 & \frac{\psi_2 \theta_3}{\mu_3(1+\rho_1)} & 0 \\ 0 & 0 & 0 & 0 & \gamma & -a_7 & 0 \\ 0 & 0 & 0 & 0 & \gamma_2 & 0 & -a_8 \end{pmatrix}. \quad (3.39)$$

Again, the second and seventh columns of the $J_1(\mathcal{E}_0)$ in (3.39) contain the diagonal terms. It is obvious from the eigenvalues $\lambda_6 = -a_2$ and $\lambda_7 = -a_8$. Thus $(J_1(\mathcal{E}_0))$ reduces to;

$$J_2(\mathcal{E}_0) = \begin{pmatrix} -a_1 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} \\ 0 & -a_4 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} \\ 0 & \gamma & -a_5 & 0 & 0 \\ 0 & 0 & \frac{\psi_1 \theta_3}{\mu_3(1+\rho_1)} & -a_6 & \frac{\psi_2 \theta_3}{\mu_3(1+\rho_1)} \\ 0 & 0 & 0 & \gamma & -a_7 \end{pmatrix}. \quad (3.40)$$

Again, the first column of $J_2(\mathcal{E}_0)$ in Equation (3.40) contains the diagonal term, and it is obvious from eigenvalues $\lambda_8 = -a_1$. Thus $J_2(\mathcal{E}_0)$ reduces to;

$$J_3(\mathcal{E}_0) = \begin{pmatrix} -a_4 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} \\ \gamma & -a_5 & 0 & 0 \\ 0 & \frac{\psi_1 \theta_3}{\mu_3(1+\rho_1)} & -a_6 & \frac{\psi_2 \theta_3}{\mu_3(1+\rho_1)} \\ 0 & 0 & \gamma & -a_7 \end{pmatrix}. \quad (3.41)$$

Computing the eigenvalues of the given matrix $J_3(\mathcal{E}_0)$ in Equation (3.41) involves solving the

characteristic polynomial equation:

$$P(\lambda) = \det(J_3(\mathcal{E}_0) - \lambda I) = 0,$$

where I is the identity matrix and λ represents the eigenvalues. Thus,

$$\lambda^4 + C_1\lambda^3 + C_2\lambda^2 + C_3\lambda + C_4 = 0. \quad (3.42)$$

Equations (3.38), (3.39), and (3.40) evidence that $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7$, and λ_8 exhibit negative real parts. By applying the Routh–Hurwitz criterion, the other four eigenvalues of the matrix Equation (3.41) will also have negative real parts if all coefficients in Equation (3.42) are greater than zero.

$$\begin{cases} C_1 > 0, \\ C_1C_2 - C_3 > 0, \\ C_1C_4 - (C_1C_2 - C_3) > 0, \\ C_4 > 0. \end{cases} \quad (3.43)$$

If these conditions hold, all eigenvalues have negative real parts, ensuring local asymptotic stability of $\mathcal{E}_0$. Then, the other four eigenvalues of the matrix Equation (3.41) will also have negative real parts if all coefficients in Equation (3.42) are greater than zero. Then,

$$\begin{cases} C_1 = a_7 + a_6 + a_5 + a_4 > 0, \\ C_2 = -\gamma b_1 - \gamma b_4 + a_5a_4 + a_6a_4 + a_7a_4 + a_6a_5 + a_7a_5 + a_7a_6 > 0, \\ C_3 = ((a_6 + a_7)a_5 - \gamma b_4 + a_7a_6)a_4 + (-\gamma b_4 + a_7a_6)a_5 - \gamma b_1(a_6 + a_7) > 0, \\ C_4 = \gamma^2 b_1 b_4 - \gamma^2 b_2 b_3 - \gamma a_4 a_5 b_4 - \gamma a_6 a_7 b_1 + a_4 a_5 a_6 a_7 > 0, \end{cases}$$

where;

$$\begin{cases} b_1 = \frac{\kappa_1 \theta_2}{\mu_2}, \quad b_2 = \frac{\psi_1 \theta_3}{\mu_3(1 + \rho_1)}, \\ b_3 = \frac{\kappa_2 \theta_2}{\mu_2}, \quad b_4 = \frac{\psi_2 \theta_3}{\mu_3(1 + \rho_1)}. \end{cases}$$

Since all eigenvalues of $J(\mathcal{E}_0)$ evaluated at $\mathcal{E}_0$ have negative real parts, the Model (3.5) at the $\mathcal{E}_0$ is locally asymptotically stable if $\mathcal{R}_0 < 1$. $\square$

3.7 Global Stability of the Disease Free Equilibrium Point

The model behavior $\mathcal{E}_0$, is investigated using a Metzler matrix as applied by Castillo-Chavez *et al.* (2002). Let X_s denote non-transmitting class, X_m be transmitting class and $X_{\mathcal{E}_0}$ be the Disease Free equilibrium, whereby:

$$\begin{cases} \frac{dX_s}{dt} = B(X_s - X_{\mathcal{E}_0}) + B_1 X_m, \\ \frac{dX_m}{dt} = B_2 X_m. \end{cases} \quad (3.44)$$

Then, from the Model (3.5), it can be deduced that:

$$X_s = (S_H, R_H, S_F, S_D, R_D)^T, \quad X_m = (E_H, I_H, E_F, I_F, E_D, I_D, M)^T,$$

$$X_s - X_{\mathcal{E}_0} = \begin{pmatrix} S_H - \frac{\theta_1}{\mu_1} \\ R_H \\ S_F - \frac{\theta_2}{\mu_2} \\ S_D - \frac{\theta_3}{\mu_3} \\ R_D \end{pmatrix}, \quad B = \begin{pmatrix} -\mu & \beta_3 & 0 & 0 & 0 \\ 0 & -(\beta_3 + \mu_1) & 0 & 0 & 0 \\ 0 & 0 & -\mu_2 & 0 & 0 \\ 0 & 0 & 0 & -\mu_3 & \gamma_3 \\ 0 & 0 & 0 & 0 & -(\mu_3 + \gamma_3) \end{pmatrix}.$$

$$B_1 = \begin{pmatrix} 0 & 0 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ \beta_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} & 0 \\ 0 & 0 & 0 & \frac{\psi_1 \theta_3}{\mu_3(1+\rho_1)} & 0 & \frac{\psi_2 \theta_3}{\mu_3(1+\rho_2)} & 0 \\ 0 & 0 & 0 & 0 & \gamma_2 & 0 & 0 \end{pmatrix}, \text{ and}$$

$$B_2 = \begin{pmatrix} -\mu_1 - \beta_1 - \beta_2 & 0 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ \beta_1 & -\sigma_1 - \mu_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\mu_2 - \gamma & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} & 0 \\ 0 & 0 & \gamma & -\mu_2 - \sigma_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\psi_1 \theta_3}{\mu_3(1+\rho_1)} & -\mu_3 - \gamma_1 - \gamma_2 & \frac{\psi_2 \theta_3}{\mu_3(1+\rho_1)} & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\mu_3 - \sigma_3 & 0 \\ 0 & v_1 & 0 & v_2 & 0 & v_3 & -\mu_4 \end{pmatrix}.$$

Matrix B_2 is a Metzler matrix due to its non-negative off-diagonal entries. To establish its stability, we must show it meets the criteria for a Metzler matrix and that its eigenvalues have negative real parts. We will use the concept of a stable Metzler matrix and apply the following Lemma 3.2.

Lemma 3.2 ((Kamgang & Sallet, 2008))

Let N be a square Metzler matrix written in block form

$$N = \begin{pmatrix} A & B \\ C & D \end{pmatrix}. \quad (3.45)$$

where A , B and D are rectangular matrices while C is a square matrix. Then N is Metzler stable if, and only if, matrices A and $D - CA^{-1}B$ are Metzler stable.

In our case one has

$$A = \begin{pmatrix} -\mu_1 - \beta_1 - \beta_2 & 0 & 0 \\ \beta_1 & -\sigma_1 - \mu_1 & 0 \\ 0 & 0 & -\mu_2 - \gamma \end{pmatrix},$$

$$B = \begin{pmatrix} \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ 0 & 0 & 0 & 0 \\ \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 \\ 0 & 0 & 0 & 0 \\ \frac{\psi_1 \theta_3}{\mu_3(1+\rho_1)} & 0 & \frac{\psi_2 \theta_3}{\mu_3(1+\rho_1)} & 0 \\ 0 & 0 & 0 & 0 \\ v_1 & 0 & v_2 & 0 \end{pmatrix}, \quad C = \begin{pmatrix} 0 & 0 & 0 \\ \gamma & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix},$$

and

$$D = \begin{pmatrix} -\mu_2 - \sigma_2 & 0 & 0 \\ 0 & -\mu_3 - \gamma_1 - \gamma_2 & 0 \\ 0 & \gamma & -\mu_3 - \sigma_3 \end{pmatrix}.$$

Since the off-diagonal elements of A are β_1 and 0, both of which are non-negative, we conclude that matrix A is Metzler stable.

Next, matrix $D - CA^{-1}B$ can be calculated as follows:

$$CA^{-1}B = \begin{pmatrix} \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} \\ 0 & 0 & 0 \\ 0 & \gamma & -\mu_2 - \sigma_2 \end{pmatrix}.$$

Subtracting $CA^{-1}B$ from D gives:

$$D - CA^{-1}B = \begin{pmatrix} 0 & 0 & 0 \\ 0 & -\mu_1 - \sigma_1 & 0 \\ 0 & 0 & -\mu_2 - \gamma - \sigma_2 \end{pmatrix}.$$

Since the eigenvalues of the Matrix B are $\lambda_1 = -\mu_3$, $\lambda_2 = -\mu_2$, $\lambda_3 = -\mu_1$, $\lambda_4 = -(\mu_3 + \gamma_3)$, and $\lambda_5 = -(\beta_3 + \mu_1)$ and the off-diagonal elements of $D - CA^{-1}B$ are all 0, which are non-negative, thus matrix $D - CA^{-1}B$ is also Metzler stable. Since both matrices A and $D - CA^{-1}B$ have non-negative off-diagonal elements, they are Metzler stable. Thus, B_2 is also Metzler stable. Therefore, $\mathcal{E}_0$ of the Model (3.5) is globally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable otherwise.

3.8 Global Stability of Endemic Equilibrium Point $\mathcal{E}^*$

Theorem 3.4

$\mathcal{E}^*$ of the rabies Model (3.5) is globally asymptotically stable whenever $\mathcal{R}_0 \geq 1$.

Proof. To prove Theorem (3.4), we adopted the approach of Chapwanya & Dumani (2022) and Charles *et al.* (2024) by constructing a Lyapunov function of the form

$$\mathcal{H} = \sum_{i=1}^n \mathcal{D}_i \left(U_i - U_i^* + U_i^* \ln \left(\frac{U_i}{U_i^*} \right) \right), \mathcal{D}_i > 0 \text{ for } i = 1, 2, 3, \dots, n,$$

where $\mathcal{D}_i$ represents a positive constant that needs to be determined, U_i stands for the population variable at compartment i , and U_i^* denotes the equilibrium point of the rabies model at compartment i for $i \in \{1, 2, 3, \dots, 12\}$. Therefore, we define the Lyapunov $\mathcal{H}$ for Model (3.5) as follows:

$$\mathcal{H} = \begin{pmatrix} \mathcal{D}_1 \left(S_H - S_H^* + S_H \ln \left(\frac{S_H}{S_H^*} \right) \right) + \mathcal{D}_2 \left(E_H - E_H^* + E_H \ln \left(\frac{E_H}{E_H^*} \right) \right) \\ + \mathcal{D}_3 \left(I_H - I_H^* + I_H \ln \left(\frac{I_H}{I_H^*} \right) \right) + \mathcal{D}_4 \left(R_H - R_H^* + R_H \ln \left(\frac{R_H}{R_H^*} \right) \right) \\ + \mathcal{D}_5 \left(S_F - S_F^* + S_F \ln \left(\frac{S_F}{S_F^*} \right) \right) + \mathcal{D}_6 \left(E_F - E_F^* + E_F \ln \left(\frac{E_F}{E_F^*} \right) \right) \\ + \mathcal{D}_7 \left(I_F - I_F^* + I_F \ln \left(\frac{I_F}{I_F^*} \right) \right) + \mathcal{D}_8 \left(S_D - S_D^* + S_D \ln \left(\frac{S_D}{S_D^*} \right) \right) \\ + \mathcal{D}_9 \left(E_D - E_D^* + E_D \ln \left(\frac{E_D}{E_D^*} \right) \right) + \mathcal{D}_{10} \left(I_D - I_D^* + I_D \ln \left(\frac{I_D}{I_D^*} \right) \right) \\ + \mathcal{D}_{11} \left(R_D - R_D^* + R_D \ln \left(\frac{R_D}{R_D^*} \right) \right) + \mathcal{D}_{12} \left(M - M^* + M \ln \left(\frac{M}{M^*} \right) \right) \end{pmatrix}. \quad (3.46)$$

Evaluating Equation (3.46) at $\mathcal{E}^*$ gives

$$\mathcal{H} = \mathcal{E}^*(S_H^*, E_H^*, I_H^*, R_H^*, S_F^*, E_F^*, I_F^*, S_D^*, E_D^*, I_D^*, R_D^*, M^*) = 0.$$

Then, using the time derivative of $\mathcal{H}$ in Equation (3.46) gives

$$\frac{d\mathcal{H}}{dt} \left\{ \begin{array}{l} = \mathcal{D}_1 \left(1 - \frac{S_H^*}{S_H} \right) \frac{dS_H}{dt} + \mathcal{D}_2 \left(1 - \frac{E_H^*}{E_H} \right) \frac{dE_H}{dt} + \mathcal{D}_3 \left(1 - \frac{I_H^*}{I_H} \right) \frac{dI_H}{dt} \\ + \mathcal{D}_4 \left(1 - \frac{R_H^*}{R_H} \right) \frac{dR_H}{dt} + \mathcal{D}_5 \left(1 - \frac{S_F^*}{S_F} \right) \frac{dS_F}{dt} + \mathcal{D}_6 \left(1 - \frac{E_F^*}{E_F} \right) \frac{dE_F}{dt} \\ + \mathcal{D}_7 \left(1 - \frac{I_F^*}{I_F} \right) \frac{dI_F}{dt} + \mathcal{D}_8 \left(1 - \frac{S_D^*}{S_D} \right) \frac{dS_D}{dt} + \mathcal{D}_9 \left(1 - \frac{E_D^*}{E_D} \right) \frac{dE_D}{dt} \\ + \mathcal{D}_{10} \left(1 - \frac{I_D^*}{I_D} \right) \frac{dI_D}{dt} + \mathcal{D}_{11} \left(1 - \frac{R_D^*}{R_D} \right) \frac{dR_D}{dt} + \mathcal{D}_{12} \left(1 - \frac{M^*}{M} \right) \frac{dM}{dt}. \end{array} \right. \quad (3.47)$$

Consider $\mathcal{E}^*$ of Equation (3.5) such that

$$\left\{ \begin{array}{l} \theta_1 = (\tau_1 I_F^* + \tau_2 I_D^* + \tau_3 \lambda(M^*)) S_H^* + \mu_1 S_H^* - \beta_3 R_H^*, \\ \mu_1 + \beta_1 + \beta_2 = \frac{(\tau_1 I_F^* + \tau_2 I_D^* + \tau_3 \lambda(M^*)) S_H^*}{E_H^*}, \\ \sigma_1 + \mu_1 = \frac{\beta_1 E_H^*}{I_H^*}, \quad \beta_3 + \mu_1 = \frac{\beta_2 E_H^*}{R_H^*}, \\ \theta_2 = (\kappa_1 I_F^* + \kappa_2 I_D^* + \kappa_3 \lambda(M^*)) S_F^* + \mu_2 S_F^*, \\ \mu_2 + \gamma = \frac{(\kappa_1 I_F^* + \kappa_2 I_D^* + \kappa_3 \lambda(M^*)) S_F^*}{E_F^*}, \quad \sigma_2 + \mu_2 = \frac{\gamma E_F^*}{I_F^*}, \\ \theta_3 = \left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} + \frac{\psi_3 \lambda(M^*)}{1 + \rho_3} \right) S_D^* + \mu_3 S_D^* - \gamma_3 R_D^*, \\ \mu_3 + \gamma_1 + \gamma_2 = \frac{\left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} + \frac{\psi_3 \lambda(M^*)}{1 + \rho_3} \right) S_D^*}{E_D^*}, \\ \sigma_3 + \mu_3 = \frac{\gamma_1 E_D^*}{I_D^*}, \quad \gamma_3 + \mu_3 = \frac{\gamma_2 E_D^*}{R_D^*}, \quad \mu_4 = \frac{(\nu_1 I_H^* + \nu_2 I_F^* + \nu_3 I_D^*)}{M^*}. \end{array} \right. \quad (3.48)$$

Then, by substituting (3.48) into (3.5) , we have

$$\frac{d\mathcal{H}}{dt} = \left\{ \begin{array}{l} \mathcal{D}_1 \left(1 - \frac{S_H^*}{S_H} \right) (\theta_1 + \beta_3 R_H - \mu_1 S_H - \chi_1) \\ + \mathcal{D}_2 \left(1 - \frac{E_H^*}{E_H} \right) (\chi_1 - (\mu_1 + \beta_1 + \beta_2) E_H) \\ + \mathcal{D}_3 \left(1 - \frac{I_H^*}{I_H} \right) (\beta_1 E_H - (\sigma_1 + \mu_1) I_H) \\ + \mathcal{D}_4 \left(1 - \frac{R_H^*}{R_H} \right) (\beta_2 E_H - (\beta_3 + \mu_1) R_H) \\ + \mathcal{D}_5 \left(1 - \frac{S_F^*}{S_F} \right) (\theta_2 - \chi_2 - \mu_2 S_F) \\ + \mathcal{D}_6 \left(1 - \frac{E_F^*}{E_F} \right) (\chi_2 - (\mu_2 + \gamma) E_F) \\ + \mathcal{D}_7 \left(1 - \frac{I_F^*}{I_F} \right) (\gamma E_F - (\mu_2 + \sigma_2) I_F) \\ + \mathcal{D}_8 \left(1 - \frac{S_D^*}{S_D} \right) (\theta_3 - \mu_3 S_D - \chi_3 + \gamma_3 R_D) \\ + \mathcal{D}_9 \left(1 - \frac{E_D^*}{E_D} \right) (\chi_3 - (\mu_3 + \gamma_1 + \gamma_2) E_D) \\ + \mathcal{D}_{10} \left(1 - \frac{I_D^*}{I_D} \right) (\gamma_1 E_D - (\mu_3 + \delta_3) I_D) \\ + \mathcal{D}_{11} \left(1 - \frac{R_D^*}{R_D} \right) (\gamma_2 E_D - (\mu_3 + \gamma_3) R_D) \\ + \mathcal{D}_{12} \left(1 - \frac{M^*}{M} \right) ((v_1 I_H + v_2 I_F + v_3 I_D) - \mu_4 M) . \end{array} \right. \quad (3.49)$$

Using $\mathcal{E}^*$ described in Equation (3.48), we simplify the Equation (3.49) as:

$$\frac{d\mathcal{H}}{dt} = \left\{ \begin{aligned} &= \mathcal{D}_1 \left(1 - \frac{S_H^*}{S_H} \right) \left((\tau_1 I_F^* + \tau_2 I_D^* + \tau_3 \lambda(M^*)) S_H^* + \mu_1 S_H^* - \beta_3 R_H^* - \mu_1 S_H \right. \\ &\quad \left. - (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H + \beta_3 R_H \right) \\ &+ \mathcal{D}_2 \left(1 - \frac{E_H^*}{E_H} \right) \left((\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H - \frac{(\tau_1 I_F^* + \tau_2 I_D^* + \tau_3 \lambda(M^*)) S_H^* E_H}{E_H^*} \right) \\ &+ \mathcal{D}_3 \left(1 - \frac{I_H^*}{I_H} \right) \left(\beta_1 E_H - \frac{\beta_1 E_H^* I_H}{I_H^*} \right) + \mathcal{D}_4 \left(1 - \frac{R_H^*}{R_H} \right) \left(\beta_2 E_H - \frac{\beta_2 E_H^* R_H}{R_H^*} \right) \\ &+ \mathcal{D}_5 \left(1 - \frac{S_F^*}{S_F} \right) \left((\kappa_1 I_F^* + \kappa_2 I_D^* + \kappa_3 \lambda(M^*)) S_F^* + \mu_2 S_F^* - \mu_2 S_F \right. \\ &\quad \left. - (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F \right) \\ &+ \mathcal{D}_6 \left(1 - \frac{E_F^*}{E_F} \right) \left((\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F - \frac{(\tau_1 I_F^* + \kappa_2 I_D^* + \kappa_3 \lambda(M^*)) S_F^* E_F}{E_F^*} \right) \\ &+ \mathcal{D}_7 \left(1 - \frac{I_F^*}{I_F} \right) \left(\gamma E_F - \frac{\gamma E_F^* I_F}{I_F^*} \right) \\ &+ \mathcal{D}_8 \left(1 - \frac{S_D^*}{S_D} \right) \left(\left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} \frac{\psi_3 \lambda(M^*)}{1 + \rho_3} \right) S_D^* + \mu_3 S_D^* - \gamma_3 R_D^* \right. \\ &\quad \left. - \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} \frac{\psi_3 \lambda(M)}{1 + \rho_3} \right) S_D - \mu_3 S_D + \gamma_3 R_D \right) \\ &+ \mathcal{D}_9 \left(1 - \frac{E_D^*}{E_D} \right) \left(\left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} \frac{\psi_3 \lambda(M)}{1 + \rho_3} \right) S_D \right. \\ &\quad \left. - \frac{\left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} \frac{\psi_3 \lambda(M^*)}{1 + \rho_3} \right) S_D^* E_D}{E_D^*} \right) \\ &+ \mathcal{D}_{10} \left(1 - \frac{I_D^*}{I_D} \right) \left(\gamma_1 E_D - \frac{\gamma_1 E_D^* I_D}{I_D^*} \right) + \mathcal{D}_{11} \left(1 - \frac{R_D^*}{R_D} \right) \left(\gamma_2 E_D - \frac{\gamma_2 E_D^* R_D}{R_D^*} \right) \\ &+ \mathcal{D}_{12} \left(1 - \frac{M^*}{M} \right) \left(v_1 I_H + v_2 I_F + v_3 I_D - \frac{(v_1 I_H^* + v_2 I_F^* + v_3 I_D^*) M}{M^*} \right). \end{aligned} \right. \quad (3.50)$$

Then, Equation (3.50) can be expressed as follows:

$$\begin{aligned}
\frac{d\mathcal{H}}{dt} = & \left[\begin{aligned}
& -\mathcal{D}_1 \mu_1 S_H \left(1 - \frac{S_H^*}{S_H}\right)^2 + \mathcal{D}_1 \tau_1 S_H I_F \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{I_F^* S_H^*}{I_F S_H} - 1\right) \\
& + \mathcal{D}_1 \tau_2 S_H I_D \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{I_D^* S_H^*}{I_D S_H} - 1\right) \\
& + \mathcal{D}_1 \tau_3 S_H \lambda(M) \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{\lambda(M^*) S_H^*}{\lambda(M) S_H} - 1\right) + \mathcal{D}_1 \beta_3 R_H \left(1 - \frac{S_H^*}{S_H}\right) \left(1 - \frac{R_H^*}{R_H}\right) \\
& + \mathcal{D}_2 \tau_1 S_H I_F \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{I_F^* S_H^* E_H}{I_F S_H E_H^*}\right) + \mathcal{D}_2 \tau_2 S_H I_D \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{I_D^* S_H^* E_H}{I_D S_H E_H^*}\right) \\
& + \mathcal{D}_2 \tau_3 S_H \lambda(M) \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{\lambda(M^*) S_H^* E_H}{\lambda(M) S_H E_H^*}\right) \\
& + \mathcal{D}_3 \beta_1 E_H \left(1 - \frac{I_H^*}{I_H}\right) \left(1 - \frac{E_H^* I_H}{E_H I_H^*}\right) + \mathcal{D}_4 \beta_2 E_H \left(1 - \frac{R_H^*}{R_H}\right) \left(-\frac{E_H^* R_H}{E_H R_H^*}\right) \\
& - \mathcal{D}_5 \mu_2 S_F \left(1 - \frac{S_F^*}{S_F}\right)^2 + \mathcal{D}_5 \kappa_1 S_F I_F \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{I_F^* S_F^*}{I_F S_F} - 1\right) \\
& + \mathcal{D}_5 \kappa_2 S_F I_D \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{I_D^* S_F^*}{I_D S_F} - 1\right) \\
& + \mathcal{D}_5 \kappa_3 S_F \lambda(M) \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{\lambda(M^*) S_F^*}{\lambda(M) S_F} - 1\right) + \mathcal{D}_6 \kappa_1 S_F I_F \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{I_F^* S_F^* E_F}{I_F S_F E_F^*}\right) \\
& + \mathcal{D}_6 \kappa_2 S_F I_D \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{I_D^* S_F^* E_F}{I_D S_F E_F^*}\right) \\
& + \mathcal{D}_6 \kappa_3 S_F \lambda(M) \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{\lambda(M^*) S_F^* E_F}{\lambda(M) S_F E_F^*}\right) + \mathcal{D}_7 \gamma E_F \left(1 - \frac{I_F^*}{I_F}\right) \left(1 - \frac{E_F^* I_F}{E_F I_F^*}\right) \\
& - \mathcal{D}_8 \mu_3 S_D \left(1 - \frac{S_D^*}{S_D}\right)^2 + \frac{\psi_1 S_D I_F \mathcal{D}_8}{(1 + \rho_1)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{I_F^* S_D^*}{I_F S_D} - 1\right) \\
& + \frac{\psi_2 S_D I_F \mathcal{D}_8}{(1 + \rho_2)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{I_D^* S_D^*}{I_D S_D} - 1\right) \\
& + \frac{\psi_3 S_D \lambda(M) \mathcal{D}_8}{(1 + \rho_3)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{\lambda(M^*) S_D^*}{\lambda(M) S_D} - 1\right) + \mathcal{D}_8 \gamma_3 R_D \left(1 - \frac{S_D^*}{S_D}\right) \left(1 - \frac{R_D^*}{R_D}\right) \\
& + \frac{\psi_1 S_D I_F \mathcal{D}_9}{(1 + \rho_1)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{I_F^* S_D^* E_D}{I_F S_D E_D^*}\right) + \frac{\psi_2 S_D I_F \mathcal{D}_9}{(1 + \rho_2)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{I_D^* S_D^* E_D}{I_D S_D E_D^*}\right) \\
& + \frac{\psi_3 S_D I_F \mathcal{D}_9}{(1 + \rho_3)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{\lambda(M^*) S_D^* E_D}{\lambda(M) S_D E_D^*}\right) \\
& + \mathcal{D}_{10} \gamma_1 E_D \left(1 - \frac{I_D^*}{I_D}\right) \left(1 - \frac{E_D^* I_D}{E_D I_D^*}\right) + \mathcal{D}_{11} \gamma_2 E_D \left(1 - \frac{R_D^*}{R_D}\right) \left(-\frac{E_D^* R_D}{E_D R_D^*}\right) \\
& + \mathcal{D}_{12} v_1 I_H \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_H^* M}{I_H M^*}\right) + \mathcal{D}_{12} v_2 I_F \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_F^* M}{I_F M^*}\right) \\
& + \mathcal{D}_{12} v_3 I_D \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_D^* M}{I_D M^*}\right).
\end{aligned} \right]
\end{aligned}
\tag{3.51}$$

Equation (3.51) can be written as:

$$\frac{d\mathcal{H}}{dt} = \mathcal{G} + \mathcal{P},$$

where;

$$\mathcal{P} = -\mathcal{D}_1\mu_1S_H \left(1 - \frac{S_H^*}{S_H}\right)^2 - \mathcal{D}_5\mu_2S_F \left(1 - \frac{S_F^*}{S_F}\right)^2 - \mathcal{D}_8\mu_3S_D \left(1 - \frac{S_D^*}{S_D}\right)^2$$

and

$$\mathcal{G} = \left\{ \begin{aligned} & \mathcal{D}_1\tau_1S_HI_F \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{I_F^*S_H^*}{I_FS_H} - 1\right) + \mathcal{D}_1\tau_2S_HI_D \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{I_D^*S_H^*}{I_DS_H} - 1\right) \\ & + \mathcal{D}_1\tau_3S_H\lambda(M) \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{\lambda(M^*)S_H^*}{\lambda(M)S_H} - 1\right) + \mathcal{D}_1\beta_3R_H \left(1 - \frac{S_H^*}{S_H}\right) \left(1 - \frac{R_H^*}{R_H}\right) \\ & + \mathcal{D}_2\tau_1S_HI_F \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{I_F^*S_H^*E_H}{I_FS_HE_H^*}\right) + \mathcal{D}_2\tau_2S_HI_D \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{I_D^*S_H^*E_H}{I_DS_HE_H^*}\right) \\ & + \mathcal{D}_2\tau_3S_H\lambda(M) \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{\lambda(M^*)S_H^*E_H}{\lambda(M)S_HE_H^*}\right) \\ & + \mathcal{D}_3\beta_1E_H \left(1 - \frac{I_H^*}{I_H}\right) \left(1 - \frac{E_H^*I_H}{E_HI_H^*}\right) + \mathcal{D}_4\beta_2E_H \left(1 - \frac{R_H^*}{R_H}\right) \left(-\frac{E_H^*R_H}{E_HR_H^*}\right) \\ & + \mathcal{D}_5\kappa_1S_FI_F \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{I_F^*S_F^*}{I_FS_F} - 1\right) + \mathcal{D}_5\kappa_2S_FI_D \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{I_D^*S_F^*}{I_DS_F} - 1\right) \\ & + \mathcal{D}_5\kappa_3S_F\lambda(M) \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{\lambda(M^*)S_F^*}{\lambda(M)S_F} - 1\right) + \mathcal{D}_6\kappa_1S_FI_F \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{I_F^*S_F^*E_F}{I_FS_FE_F^*}\right) \\ & + \mathcal{D}_6\kappa_2S_FI_D \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{I_D^*S_F^*E_F}{I_DS_FE_F^*}\right) \\ & + \mathcal{D}_6\kappa_3S_F\lambda(M) \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{\lambda(M^*)S_F^*E_F}{\lambda(M)S_FE_F^*}\right) + \mathcal{D}_7\gamma E_F \left(1 - \frac{I_F^*}{I_F}\right) \left(1 - \frac{E_F^*I_F}{E_FI_F^*}\right) \\ & + \frac{\psi_1S_DI_F\mathcal{D}_8}{(1+\rho_1)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{I_F^*S_D^*}{I_FS_D} - 1\right) + \frac{\psi_2S_DI_F\mathcal{D}_8}{(1+\rho_2)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{I_D^*S_D^*}{I_DS_D} - 1\right) \\ & + \frac{\psi_3S_D\lambda(M)\mathcal{D}_8}{(1+\rho_3)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{\lambda(M^*)S_D^*}{\lambda(M)S_D} - 1\right) + \mathcal{D}_8\gamma_3R_D \left(1 - \frac{S_D^*}{S_D}\right) \left(1 - \frac{R_D^*}{R_D}\right) \\ & + \frac{\psi_1S_DI_F\mathcal{D}_9}{(1+\rho_1)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{I_F^*S_D^*E_D}{I_FS_DE_D^*}\right) + \frac{\psi_2S_DI_F\mathcal{D}_9}{(1+\rho_2)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{I_D^*S_D^*E_D}{I_DS_DE_D^*}\right) \\ & + \frac{\psi_3S_DI_F\mathcal{D}_9}{(1+\rho_3)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{\lambda(M^*)S_D^*E_D}{\lambda(M)S_DE_D^*}\right) \\ & + \mathcal{D}_{10}\gamma_1E_D \left(1 - \frac{I_D^*}{I_D}\right) \left(1 - \frac{E_D^*I_D}{E_DI_D^*}\right) + \mathcal{D}_{11}\gamma_2E_D \left(1 - \frac{R_D^*}{R_D}\right) \left(-\frac{E_D^*R_D}{E_DR_D^*}\right) \\ & + \mathcal{D}_{12}v_1I_H \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_H^*M}{I_HM^*}\right) + \mathcal{D}_{12}v_2I_F \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_F^*M}{I_FM^*}\right) \\ & + \mathcal{D}_{12}v_3I_D \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_D^*M}{I_DM^*}\right). \end{aligned} \right. \quad (3.52)$$

To simplify (3.52), let

$$a = \frac{S_H}{S_H^*}, b = \frac{E_H}{E_H^*}, c = \frac{I_H}{I_H^*}, d = \frac{R_H}{R_H^*}, e = \frac{S_F}{S_F^*}, f = \frac{E_F}{E_F^*}, g = \frac{I_F}{I_F^*},$$

$$h = \frac{S_D}{S_D^*}, r = \frac{E_D}{E_D^*}, n = \frac{I_D}{I_D^*}, m = \frac{\lambda(M)}{\lambda(M^*)}, l = \frac{R_D}{R_D^*}, \text{ and } k = \frac{M}{M^*}.$$

We express the Equation (3.52) as:

$$\mathcal{Q} = \left\{ \begin{aligned} & \tau_1 S_H I_F \left(1 - \frac{b}{ac} + \frac{1}{ac} - \frac{1}{b} \right) + \tau_2 S_H I_D \left(\frac{1}{an} - 1 + \frac{1}{a^2 n} + \frac{1}{a} \right) \\ & + \tau_3 S_H \lambda(M) \left(\frac{1}{am} - 1 + \frac{1}{a^2 m} + \frac{1}{a} \right) + \beta_3 R_H \left(1 - \frac{1}{d} - \frac{1}{a} + \frac{1}{ad} \right) \\ & + \tau_1 S_H I_F \left(1 - \frac{b}{af} - \frac{1}{b} + \frac{1}{af} \right) + \tau_2 S_H I_D \left(1 - \frac{b}{an} - \frac{1}{b} + \frac{1}{an} \right) \\ & + \tau_3 S_H \lambda(M) \left(1 - \frac{b}{af} - \frac{1}{b} + \frac{1}{af} \right) + \tau_2 S_H I_D \left(1 - \frac{b}{am} - \frac{1}{b} + \frac{1}{am} \right) \\ & + \beta_1 E_H \left(1 - \frac{b}{c} - \frac{1}{c} + \frac{b}{c^2} \right) + \beta_2 E_H \left(1 - \frac{b}{d} - \frac{1}{d} + \frac{b}{d^2} \right) \\ & + \kappa_1 S_F I_F \left(\frac{1}{ef} - 1 - \frac{1}{e^2 f} + \frac{1}{e} \right) + \kappa_2 S_F I_D \left(\frac{1}{en} - 1 - \frac{1}{e^2 n} + \frac{1}{e} \right) \\ & + \kappa_3 S_F \lambda(M) \left(\frac{1}{em} - 1 - \frac{1}{e^2 m} + \frac{1}{e} \right) + \kappa_1 S_F I_F \left(1 - \frac{f}{en} - \frac{1}{f} + \frac{1}{en} \right) \\ & + \kappa_2 S_F I_D \left(1 - \frac{f}{en} - \frac{1}{f} + \frac{1}{en} \right) + \kappa_3 S_F \lambda(M) \left(1 - \frac{f}{me} - \frac{1}{f} + \frac{1}{me} \right) \\ & + \gamma E_F \left(1 - \frac{g}{f} - \frac{1}{f} + \frac{g}{f^2} \right) + \frac{\psi_1 S_D I_F}{(1+\rho_1)} \left(1 - \frac{1}{h} - \frac{1}{h^2 g} + \frac{1}{h} \right) \\ & + \frac{\psi_2 S_D I_F}{(1+\rho_2)} \left(1 - \frac{1}{h} - \frac{1}{h^2 g} + \frac{1}{h} \right) + \frac{\psi_3 S_D \lambda(M)}{(1+\rho_3)} \left(\frac{1}{mh} - 1 - \frac{1}{h^2 m} + \frac{1}{h} \right) \\ & + \gamma_3 R_D \left(1 - \frac{1}{l} - \frac{1}{h} + \frac{1}{hl} \right) + \frac{\psi_1 S_D I_F}{(1+\rho_1)} \left(1 - \frac{r}{hg} - \frac{1}{r} + \frac{1}{hg} \right) \\ & + \frac{\psi_2 S_D I_F}{(1+\rho_2)} \left(1 - \frac{r}{hn} - \frac{1}{r} + \frac{1}{hn} \right) + \frac{\psi_3 S_D I_F}{(1+\rho_3)} \left(1 - \frac{r}{hm} - \frac{1}{r} + \frac{1}{hm} \right) \\ & + \gamma_1 E_D \left(1 - \frac{l}{r} - \frac{1}{l} + \frac{1}{r} \right) + \gamma_2 E_D \left(1 - \frac{r}{hg} - \frac{1}{r} + \frac{1}{hg} \right) \\ & + v_1 I_H \left(1 - \frac{k}{c} - \frac{1}{k} + \frac{1}{c} \right) + v_2 I_F \left(1 - \frac{k}{g} - \frac{1}{k} + \frac{1}{g} \right) + v_3 I_D \left(1 - \frac{k}{n} - \frac{1}{k} + \frac{1}{n} \right). \end{aligned} \right. \quad (3.53)$$

Now we make use of the following basic inequality.

Theorem 3.5

If $\varepsilon(y) = 1 - y + \ln y$, then $\varepsilon(y) \leq 0$ such that $1 - y \leq -\ln y$ if and only if $y > 0$.

From Equation (3.53), we have:

$$1 - \frac{1}{d} - \frac{1}{a} + \frac{1}{ad} = \left(1 - \frac{1}{d}\right) + \left(1 - \frac{1}{a}\right) - \left(1 - \frac{1}{ad}\right). \quad (3.54)$$

Using Theorem (3.5) and the concept of geometric mean, Equation (3.54) can be written as:

$$\begin{aligned} \left(1 - \frac{1}{d}\right) + \left(1 - \frac{1}{a}\right) - \left(1 - \frac{1}{ad}\right) &\leq -\ln\left(\frac{1}{d}\right) - \ln\left(\frac{1}{a}\right) + \ln\left(\frac{1}{ad}\right) \\ &\leq \ln\left(a \times d \times \frac{1}{ad}\right) = \ln(1) = 0. \end{aligned} \quad (3.55)$$

Following similar procedures in (3.55), we get:

$$1 - \frac{c}{b} - \frac{1}{c} + \frac{1}{b} \leq 0, \quad 1 - \frac{p}{m} - \frac{1}{p} + \frac{1}{m} \leq 0, \quad 1 - \frac{d}{b} - \frac{1}{d} + \frac{1}{b} \leq 0.$$

From Equation (3.51), the global stability holds only if $\frac{d\mathcal{H}}{dt} \leq 0$. Now, if $\mathcal{P} < \mathcal{G}$, then $\frac{d\mathcal{H}}{dt}$ will be negative definite, which implies that $\frac{d\mathcal{H}}{dt} < 0$ and $\frac{d\mathcal{H}}{dt} = 0$ only at $\mathcal{E}^*$. Hence, by LaSalle's invariance principle (LaSalle, 1976), any solution to the rabies Model (3.5) which intersect the interior $\mathbb{R}_+^{12}$ limits to $\mathcal{E}^*$ is globally asymptotically stable whatever $\mathcal{R}_0 \geq 1$. $\square$

3.9 Rabies Stochastic Model

In this section, we formulate a stochastic framework in form of (3.56):

$$dX_t = f(X_t)dt + g(X_t)dW_t, \quad (3.56)$$

Where dW_t is a Wiener process, integrating a deterrent factor, Gaussian noise as (Brownian motion) $dL_i(t)$, the integral of stochastic jumps state-dependent scaling $\int_{R^{12} \setminus \{0\}}$ with intensity $z_i(\xi)$. By adopting a stochastic approach, the Model (3.5) captures the inherent randomness and variability in the transmission process, recognizing that real-world outcomes often deviate from average trends due to chance events. This extension is particularly useful for understanding the unpredictable nature of transmission, especially in smaller populations, where random fluctuations can result in unexpected outbreaks or even the elimination of the disease. The dynamics

of the rabies stochastic process are described by system (3.57) as:

$$\left\{ \begin{array}{l} \dot{S}_H = \theta_1 + \beta_3 R_H - \mu_1 S_H - \chi_1 + S_H dL_1(t) + \int_{R^{12} \setminus \{0\}} z_1(\xi) S_H(t^-) Z_1(dt, d\xi), \\ \dot{E}_H = \chi_1 - (\mu_1 + \beta_1 + \beta_2) E_H + E_H dL_2(t) + \int_{R^{12} \setminus \{0\}} z_2(\xi) E_H(t^-) Z_2(dt, d\xi), \\ \dot{I}_H = \beta_1 E_H - (\sigma_1 + \mu_1) I_H + I_H dL_3(t) + \int_{R^{12} \setminus \{0\}} z_3(\xi) I_H(t^-) Z_3(dt, d\xi), \\ \dot{R}_H = (\beta_2 + u_4) E_H - (\beta_3 + \mu_1) R_H + R_H dL_4(t) + \int_{R^{12} \setminus \{0\}} z_4(\xi) R_H(t^-) Z_4(dt, d\xi), \\ \dot{S}_F = \theta_2 - \chi_2 - \mu_2 S_F + S_F dL_5(t) + \int_{R^{12} \setminus \{0\}} z_5(\xi) S_F(t^-) Z_5(dt, d\xi), \\ \dot{E}_F = \chi_2 - (\mu_2 + \gamma) E_F + E_F dL_6(t) + \int_{R^{12} \setminus \{0\}} z_6(\xi) E_F(t^-) Z_6(dt, d\xi), \\ \dot{I}_F = \gamma E_F - (\mu_2 + \sigma_2) I_F + I_F dL_7(t) + \int_{R^{12} \setminus \{0\}} z_7(\xi) I_F(t^-) Z_7(dt, d\xi), \\ \dot{S}_D = \theta_3 - \mu_3 S_D - \chi_3 + \gamma_3 R_D + S_D dL_8(t) + \int_{R^{12} \setminus \{0\}} z_8(\xi) S_D(t^-) Z_8(dt, d\xi), \\ \dot{E}_D = \chi_3 - (\mu_3 + \gamma_1 + \gamma_2 + u_4) E_D + E_D dL_9(t) + \int_{R^{12} \setminus \{0\}} z_9(\xi) E_D(t^-) Z_9(dt, d\xi), \\ \dot{I}_D = \gamma_1 E_D - (\mu_3 + \sigma_3) I_D + I_D dL_{10}(t) + \int_{R^{12} \setminus \{0\}} z_{10}(\xi) I_D(t^-) Z_{10}(dt, d\xi), \\ \dot{R}_D = (\gamma_2 + u_4) E_D - (\mu_3 + \gamma_3) R_D + R_D dL_{11}(t) + \int_{R^{12} \setminus \{0\}} z_{11}(\xi) R_D(t^-) Z_{11}(dt, d\xi), \\ \dot{M} = (v_1 I_H + v_2 I_F + v_3 I_D) - \mu_4 M + M dL_{12}(t) + \int_{R^{12+} \setminus \{0\}} z_{12}(\xi) M(t^-) Z_{12}(dt, d\xi), \end{array} \right. \quad (3.57)$$

subject to initial non-negative conditions:

$$\begin{aligned} S_H(0) &> 0, E_H(0) \geq 0, I_H(0) \geq 0, R_H(0) \geq 0, S_F(0) > 0, E_F(0) \geq 0, I_F(0) \geq 0, \\ S_D(0) &\geq 0, E_D(0) \geq 0, I_D(0) \geq 0, R_D(0) \geq 0. \end{aligned}$$

3.9.1 Positivity of the Model (3.57)

Proof. Consider the stochastic differential equation for infected free range dogs:

$$\begin{aligned} dI_F(t) &= [\gamma E_F(t) - (\mu_2 + \sigma_2) I_F(t)] dt + I_F(t) dL_7(t) \\ &\quad + \int_{R^{12} \setminus \{0\}} z_7(\xi) I_F(t^-) Z_7(dt, d\xi), \end{aligned} \quad (3.58)$$

with $\gamma, \mu_2, \sigma_2 > 0$, $I_F(0) > 0$, and $z_7(\xi) > -1$.

Theorem 3.6 (Itô's Formula for Jump-Diffusion Processes)

Let X_t be a jump-diffusion process defined by:

$$dX_t = \mu(t, X_t) dt + \sigma(t, X_t) dB_t + \int_{\mathbb{R}^n \setminus \{0\}} h(t, \xi) Z(dt, d\xi),$$

where B_t is a standard Brownian motion, $Z(dt, d\xi)$ is a Poisson random measure, and $h(t, \xi)$ is the jump size function. Let $f(t, x) \in C^{1,2}$. Then Itô's formula gives:

$$\begin{aligned} df(t, X_t) = & \left(\frac{\partial f}{\partial t} + \mu(t, X_t) \frac{\partial f}{\partial x} + \frac{1}{2} \sigma^2(t, X_t) \frac{\partial^2 f}{\partial x^2} \right) dt \\ & + \sigma(t, X_t) \frac{\partial f}{\partial x} dB_t + \int_{\mathbb{R}^n \setminus \{0\}} [f(X_{t-} + h(t, \xi)) - f(X_{t-})] Z(dt, d\xi). \end{aligned} \quad (3.59)$$

Equation (3.58) rewrites as:

$$\frac{dI_F(t)}{I_F(t^-)} = \left(\frac{\gamma E_F(t)}{I_F(t^-)} - (\mu_2 + \sigma_2) \right) dt + dL_7(t) + \int_{\mathbb{R}^{12} \setminus \{0\}} z_7(\xi) Z_7(dt, d\xi). \quad (3.60)$$

From (3.59), Define $Y(t) = \ln I_F(t)$. Applying (3.59) with jumps yields:

$$\begin{aligned} dY(t) = & \left(\frac{\gamma E_F(t)}{I_F(t)} - (\mu_2 + \sigma_2) \right) dt + dL_7(t) \\ & + \int_{\mathbb{R}^{12} \setminus \{0\}} \ln(1 + z_7(\xi)) Z_7(dt, d\xi). \end{aligned} \quad (3.61)$$

Hence, the explicit solution of (3.58) is:

$$I_F(t) = I_F(0) \exp \left(\int_0^t \left[\frac{\gamma E_F(s)}{I_F(s)} - (\mu_2 + \sigma_2) \right] ds + L_7(t) + \sum_{0 < s \leq t} \ln(1 + \Delta J_s) \right). \quad (3.62)$$

Given $I_F(0) > 0$ and $z_7(\xi) > -1$, the exponential term in (3.62) is finite a.s., ensuring $I_F(t) > 0$ for all $t > 0$. Thus, the positivity of $I_F(t)$ is preserved. $\square$

Again, Consider the stochastic differential equation for $S_H(t)$ given by:

$$\begin{aligned} dS_H(t) = & [\theta_1 + \beta_3 R_H(t) - \mu_1 S_H(t) - \chi_1] dt + S_H(t) dL_1(t) \\ & + \int_{\mathbb{R}^{12} \setminus \{0\}} z_1(\xi) S_H(t^-) Z_1(dt, d\xi) \end{aligned} \quad (3.63)$$

To ensure $S_H(t) > 0$, we apply (3.59) to $V(t) = \ln S_H(t)$. Let $b(t) = \theta_1 + \beta_3 R_H(t) - \mu_1 S_H(t) - \chi_1$, $\sigma(t) = \sigma_1 S_H(t)$, and $\gamma(t, \xi) = z_1(\xi)$. The result from (3.59) is:

$$\begin{aligned} d\ln S_H(t) = & \frac{1}{S_H(t)} [\theta_1 + \beta_3 R_H(t) - \mu_1 S_H(t) - \chi_1] dt + dL_1(t) \\ & - \frac{1}{2} \sigma_1^2 dt + \int_{\mathbb{R}^{12} \setminus \{0\}} [\ln(1 + z_1(\xi)) - z_1(\xi)] v(d\xi) dt \\ & + \int_{\mathbb{R}^{12} \setminus \{0\}} \ln(1 + z_1(\xi)) \tilde{N}(dt, d\xi) \end{aligned} \quad (3.64)$$

For $\ln S_H(t)$ to remain well-defined, we require $1 + z_1(\xi) > 0$, such that, $z_1(\xi) > -1$. The process update due to jumps is:

$$S_H(t^+) = S_H(t^-)(1 + z_1(\xi)) \quad (3.65)$$

Thus, for $S_H(0) > 0$ and $z_1(\xi) > -1$, $S_H(t)$ remains positive for all t . The drift term does not push $S_H(t)$ to zero when $S_H \rightarrow 0^+$ if $\theta_1 + \beta_3 R_H > \chi_1$. Combining Equations (3.63), (3.64), and (3.65), we conclude that $S_H(t) > 0$ for all $t \geq 0$

under the conditions $S_H(0) > 0$, $z_1(\xi) > -1$, and bounded coefficients. Thus using the same steps as in (3.58) to (3.65) the stochastic Model (3.57) is positive for all $t \geq 0$, $z_i(\xi) > -1$.

3.9.2 Rabies Continuous Time Markov Chain (CTMC) Stochastic Model

Stochastic CTMC models utilize Galton–Watson Branching Processes to delineate the probabilities of various events, offering valuable insights into dynamics, control strategies, prediction of expected case numbers, extinction time, and the assessment of surveillance and response system effectiveness. While deterministic models rely on the basic reproduction number ($\mathcal{R}_0$) to determine whether a disease persists or diminishes in a population, stochastic models view $\mathcal{R}_0$ as a stochastic threshold, recognizing that the disease can still cease to exist even if the threshold exceeds one, contingent upon the initial number of infectives introduced into a susceptible population.

A Stochastic CTMC model for rabies transmission dynamics has been developed based on the assumptions employed in the stochastic Model (3.57). For the sake of simplicity, the same notations and parameters as those used in the deterministic Model (3.5) have been adopted. Let $S_H, E_H, I_H, R_H, S_F, E_F, I_F, S_D, E_D, I_D, R_D, M$, denote the discrete random variable for suscepti-

ble humans, exposed humans, infectious humans, recovered humans, susceptible free-range dogs, exposed free-range dogs, infectious free-range dogs, susceptible domestic dogs, exposed domestic dogs, infectious domestic animals, recovered domestic dogs, and environment respectively. Let

$$\mathbf{X}(t) = (S_H, E_H, I_H, R_H, S_F, E_F, I_F, S_D, E_D, I_D, R_D, M)^T$$

be the associated random vector for all discrete random variables $S_H, E_H, I_H, R_H, S_F, E_F, I_F, S_D, E_D, I_D, R_D$, and M . Given the time-homogeneous nature of CTMC model and its adherence to the Markov property, it is established that the future state of the process at $(t + \Delta t)$ hinges entirely upon the current state at time t . As a result, the interval between events follows an exponential distribution characterized by a specific parameter:

$$\left\{ \begin{aligned} \Psi(\mathbf{X}) &= \theta_1 + \beta_3 R_H + \mu_1 N_H + \tau_1 I_F S_H + \tau_2 I_D S_H + \left(\frac{\tau_3 M}{M+C} \right) S_H + \beta_1 E_H \\ &\quad + \kappa_1 I_F S_F + \kappa_2 I_D S_F + \left(\frac{\kappa_3 M}{M+C} \right) S_F + \mu_2 N_F + \gamma E_F + \sigma_2 I_F + \theta_3 \\ &\quad + \left(\frac{\psi_1}{1+\rho_1} \right) I_F S_D + \left(\frac{\psi_2}{1+\rho_2} \right) I_D S_D + \left(\frac{\psi_3 M}{(1+\rho_3)(M+C)} \right) S_D \\ &\quad + \mu_3 N_D + \gamma_3 R_D + \gamma_1 E_D + \gamma_2 E_D + \sigma_3 I_D + \nu_1 I_H + \nu_2 I_F + \nu_3 I_D \\ &\quad + \mu_4 M + \beta_2 E_H + \sigma_1 I_H + \theta_2, \end{aligned} \right. \quad (3.66)$$

where;

$$N_H = S_H + E_H + I_H + R_H, N_F = S_F + E_F + I_F \text{ and } N_D = S_D + E_D + I_D + R_D.$$

For modeling the transmission dynamics of rabies between humans and dogs using a CTMC model, event transitions and their corresponding rates are typically derived from the deterministic Model (3.5) using Equation (3.67) and (3.68).

$$(S, I, M) \rightarrow (S', I', M') \quad \text{with rate:} \quad (3.67)$$

$$q(S, I, M) = \begin{cases} \psi_{DD} S_D I_D, & (S_D - 1, E_D + 1) \text{ (Dog-to-dog transmission)} \\ \tau_{DH} S_H I_D, & (S_H - 1, E_H + 1, I_D) \text{ (Dog-to-human transmission)} \\ \nu_{DM} S_D M, & (S_D - 1, E_D + 1, M) \text{ (Environmental transmission to dogs)} \\ \nu_{HM} S_H M, & (S_H - 1, E_H + 1, M) \text{ (Environmental transmission to humans)} \\ \lambda_D I_D, & (I_D, M + 1) \text{ (Virus shedding by infected dogs)} \\ \mu M, & (M - 1) \text{ (Environmental virus decay)} \\ \mu_1 I_H, & (I_H - 1) \text{ (Removal of infected humans)} \\ \mu_3 I_D, & (I_D - 1) \text{ (Removal of infected dogs)} \end{cases} \quad (3.68)$$

These transitions occur as individuals move between compartments due to recruitment or movement, assuming an initial presence of only one individual while other sub-populations are not yet established. Table 4 summarizes the events and their associated transition rates, where the values 1, -1, and 0 represent an increase by 1, a decrease by 1, and no change in state, respectively, for the variable from time t to $t + \Delta t$.

Table 4: State transitions and rates of occurrence for the CTMC

Event	Rate, r	Transition $\Delta \tilde{Z}(t)$
Recruitment of S_H	θ_1	$(1, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)$
Natural death of S_H	$\mu_1 S_H$	$(-1, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)$
Contact of S_H and I_F	$\tau_1 I_F S_H$	$(-1, 1, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)$
Contact of S_H and I_D	$\tau_2 I_D S_H$	$(-1, 1, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)$
Contact of S_H and M	$\frac{M \tau_3}{M + C} S_H$	$(-1, 1, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)$
Progression from E_H to I_H	$\beta_1 E_H$	$(0, -1, 1, 0, 0, 0, 0, 0, 0, 0, 0, 0)$
Recovery of E_H	$\beta_2 E_H$	$(0, -1, 0, 1, 0, 0, 0, 0, 0, 0, 0, 0)$
Natural death of E_H	$\mu_1 E_H$	$(0, -1, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)$
Disease induced death of I_H	$\sigma_1 I_H$	$(0, 0, -1, 0, 0, 0, 0, 0, 0, 0, 0, 0)$
natural death of I_H	$\mu_1 I_H$	$(0, 0, -1, 0, 0, 0, 0, 0, 0, 0, 0, 0)$
Natural death of R_H	$\mu_1 R_H$	$(0, 0, 0, -1, 0, 0, 0, 0, 0, 0, 0, 0)$
Rate of immunity loss of R_H	$\beta_3 R_H$	$(1, 0, 0, -1, 0, 0, 0, 0, 0, 0, 0, 0)$
Recruitment of S_F	θ_2	$(0, 0, 0, 0, 1, 0, 0, 0, 0, 0, 0, 0)$
Natural death of S_F	$\mu_2 S_F$	$(0, 0, 0, 0, -1, 0, 0, 0, 0, 0, 0, 0)$
Contact of S_F and I_F	$\kappa_1 I_F S_F$	$(0, 0, 0, 0, -1, 1, 0, 0, 0, 0, 0, 0)$
Contact of S_F and I_D	$\kappa_2 I_D S_F$	$(0, 0, 0, 0, -1, 1, 0, 0, 0, 0, 0, 0)$

Continued on next page

Table4 – Continued from previous page

Event	Rate, r	Transition $\Delta\tilde{Z}(t)$
Contact of S_F and M	$\frac{M\kappa_3}{M+C}S_F$	$(0, 0, 0, 0, -1, 1, 0, 0, 0, 0, 0, 0)$
Progression of E_F to I_F	γE_F	$(0, 0, 0, 0, 0, -1, 1, 0, 0, 0, 0, 0)$
Natural death of E_F	$\mu_2 E_F$	$(0, 0, 0, 0, 0, -1, 0, 0, 0, 0, 0, 0)$
Natural death of I_F	$\mu_2 I_F$	$(0, 0, 0, 0, 0, 0, -1, 0, 0, 0, 0, 0)$
Disease induced death of I_F	$\sigma_2 I_F$	$(0, 0, 0, 0, 0, 0, -1, 0, 0, 0, 0, 0)$
Recruitment of S_D	θ_3	$(0, 0, 0, 0, 0, 0, 0, 1, 0, 0, 0, 0)$
Natural death of S_D	$\mu_3 S_D$	$(0, 0, 0, 0, 0, 0, 0, -1, 0, 0, 0, 0)$
Contact of S_D and I_F	$\frac{\psi_1 I_F}{1 + \rho_1} S_D$	$(0, 0, 0, 0, 0, 0, 0, -1, 1, 0, 0, 0)$
Contact of S_D and I_D	$\frac{\psi_2 I_D}{1 + \rho_2} S_D$	$(0, 0, 0, 0, 0, 0, 0, -1, 1, 0, 0, 0)$
Contact of S_D and M	$\frac{\psi_3 M}{(1 + \rho_3)(C + M)} S_D$	$(0, 0, 0, 0, 0, 0, 0, -1, 1, 0, 0, 0)$
Progression of E_D to I_D	$\gamma_1 E_D$	$(0, 0, 0, 0, 0, 0, 0, 0, -1, 1, 0, 0)$
Recovery of E_D	$\gamma_2 E_D$	$(0, 0, 0, 0, 0, 0, 0, 0, -1, 0, 1, 0)$
Disease induced death of I_D	$\sigma_3 I_D$	$(0, 0, 0, 0, 0, 0, 0, 0, 0, -1, 0, 0)$
Natural death of E_D	$\mu_3 E_D$	$(0, 0, 0, 0, 0, 0, 0, 0, -1, 0, 0, 0)$
Natural death of I_D	$\gamma_3 R_D$	$(0, 0, 0, 0, 0, 0, 0, 0, 0, -1, 0, 0)$
Natural death of R_D	$\mu_3 R_D$	$(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, -1, 0)$
Remove of rabies in the environment	$\mu_4 M$	$(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, -1)$
shading of I_H to M	$v_1 I_H$	$(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 1)$
shading of I_F to M	$v_2 I_F$	$(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 1)$
shading of I_D to M	$v_3 I_D$	$(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 1)$

3.9.3 Multitype Branching Process

The multitype branching process framework is utilized to examine the dynamics of the nonlinear Continuous-Time Markov Chain (CTMC) in the vicinity of the rabies-free equilibrium point $\mathcal{E}_0$. This analytical approach is critical for estimating the probabilities of disease extinction or outbreak under varying initial conditions. In CTMC-based models, the branching process either exhibits exponential growth or converges to extinction, particularly when the initial number of infective individuals is small, as typically observed at the early stages of an outbreak. In order for the multitype branching process to be applicable, it necessitates a sufficiently large initial susceptible population. As per the parameters of this study, we have established the initial susceptible populations as follows: $S_H(0) = \frac{\theta_1}{\mu_1}$, $S_F(0) = \frac{\theta_2}{\mu_2}$, and $S_D(0) = \frac{\theta_3}{\mu_3}$. We assume that

infectives of type i , I_i , produce infectives of type j , I_j , and the number of offspring produced by an individual of type I_i is independent of the number of offspring produced by either type I_i or type I_j , where $j \neq i$. The term 'birth' describes the infection transmission between susceptible humans, infected humans, susceptible domestic dogs, infected domestic dogs, susceptible free range dogs, infected free range dogs, and the rabies in the environment. Given that the multitype branching process exhibits linear behavior near the disease-free equilibrium, birth and death events occur independently. Probability generating functions (PGFs) are defined for rabies-related births and deaths within the environment, infected humans, and both domestic and free-ranging dog populations. These functions are instrumental in quantifying the probability of rabies extinction or outbreak among human and canine hosts.

Let $\{Y_{ji}\}_{j=1}^n$ be the offspring random variable for type i , where $i = 1, 2, \dots, n$ infectious hosts. Here, Y_{ji} represents the number of offspring of type j produced by an infective of type i . The offspring probability generating function (pgf) for the infectious population I_i is defined under the condition that there is initially one infectious host at the beginning of the disease outbreak, i.e., $I_i(0) = 1$, and all other types are zero: $I_j = 0$. The offspring pgf $f_i : [0, 1]^n \rightarrow [0, 1]$ for type i individuals, given $I_i(0) = 1$ and $I_j(0) = 0$ for $j \neq i$, is expressed as:

$$f_i(u_1, u_2, \dots, u_n) = \sum_{\ell_1=0}^{\infty} \sum_{\ell_2=0}^{\infty} \cdots \sum_{\ell_n=0}^{\infty} P_i(\ell_1, \ell_2, \dots, \ell_n) u_1^{\ell_1} u_2^{\ell_2} \cdots u_n^{\ell_n}, \quad (3.69)$$

where;

$$P_i(\ell_1, \ell_2, \dots, \ell_n) = \text{Prob}\{Y_{1j} = \ell_1, Y_{2j} = \ell_2, \dots, Y_{nj} = \ell_n\} \quad (3.70)$$

is the likelihood that a single infectious individual of type i will produce k offspring of type j . Equation (3.69) is utilized to establish an $n \times n$ non-negative and irreducible expectation matrix $\mathcal{M}_1 = [m_{ji}]$, where m_{ji} denotes the expected number of offspring of type j generated by an infected individual of type i . The elements of matrix $\mathcal{M}_1$ are calculated by finding the derivative f_i with respect to u_j and then evaluating all u variables at 1 (Lahodny *et al.*, 2015; Maity & Mandal, 2022), meaning that

$$m_{ji} = \left. \frac{\partial f_i}{\partial u_j} \right|_{u=1} < \infty. \quad (3.71)$$

The likelihood of disease extinction or outbreak is evaluated by the size of the spectral radius of the expectation matrix $\mathcal{M}_1$, $\rho(\mathcal{M}_1)$. If $\rho(\mathcal{M}_1) \leq 1$, then the likelihood of disease extinction is one, that is,

$$\mathbb{P}_0 = \lim_{t \rightarrow \infty} \text{Prob}\{\tilde{I}(t) = \tilde{0}\} = 1, \quad (3.72)$$

and if $\rho(\mathcal{M}_1) > 1$, then there exists a positive chance such that the possibility of disease extinc-

tion is given by

$$\mathbb{P}_0 = \lim_{t \rightarrow \infty} \text{Prob}\{\tilde{I}(t) = \tilde{0}\} = q_1^{i_1} q_2^{i_2} \dots q_k^{i_k} < 1, \quad (3.73)$$

where $(q_1, q_2, \dots, q_k)$ is the unique fixed point of the k offspring pgf, $f_i(q_1, q_2, \dots, q_k) = q_i$, and $0 < q_i < 1, i = 1, 2, \dots, k$ (Lahodny *et al.*, 2015; Lahodny & Allen, 2013; Maliyoni *et al.*, 2023). The likelihood of disease outbreak is

$$1 - \mathbb{P}_0 = 1 - q_1^{i_1} q_2^{i_2} \dots q_k^{i_k}, \quad (3.74)$$

where $\mathbb{P}_0$ is the possibility of extinction or outbreak (Lahodny *et al.*, 2015; Maity & Mandal, 2022). Predictions concerning disease extinction and the occurrence of outbreaks can be made using stochastic epidemic theory, which focuses on the number of infectious individuals within each group. If a disease originates from an infectious group with a reproduction number ($\mathcal{R}_0 > 1$), and i infective individuals are introduced into an entirely susceptible population, the probability of a significant outbreak is approximately $1 - \left(\frac{1}{\mathcal{R}_0}\right)^i$. Conversely, the probability of the disease becoming extinct is approximately $\left(\frac{1}{\mathcal{R}_0}\right)^i$. In the early stages of a rabies outbreak, with only a few infected dogs, there is a limited potential for generating infectious humans and dogs. Exposed humans and both free-range and domestic dogs can progress to infectious classes. The offspring probability generating function for the exposed class E_H , given that $E_H(0) = 1, I_H(0) = 0, E_F(0) = 0, I_F(0) = 0, E_D(0) = 0, I_D(0) = 0$, and $M(0) = 0$, is given by

$$f_1(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\beta_1 u_2 + \beta_2 + \mu_1}{\beta_1 + \beta_2 + \mu_1}. \quad (3.75)$$

The expression $\beta_1/\beta_1 + \beta_2 + \mu_1$ represents the likelihood of exposed individual move to the infectious class I_H . The term $\beta_2/\beta_1 + \beta_2 + \mu_1$ represents the probability of exposed individuals recovering as a result of Rabies PEP, while $\mu_1/\beta_1 + \beta_2 + \mu_1$ indicates the likelihood of exposed individuals naturally dying before transitioning to the infected class.

If $E_H(0) = 0, I_H(0) = 1, E_F(0) = 0, I_F(0) = 0, E_D(0) = 0, I_D(0) = 0$, and $M(0) = 0$, then the offspring likelihood generating function for I_H is given by

$$f_2(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{v_1 u_2 u_7 + \sigma_1 + \mu_1}{v_1 + \sigma_1 + \mu_1}. \quad (3.76)$$

In pgf (3.76), the term $v_1/\mu_1 + v_1 + \sigma_1$ represents the probability of infected humans shedding the rabies virus in the environment; $\mu_1/\mu_1 + v_1 + \sigma_1$ signifies the probability of infected humans dying naturally; and the term $\sigma_1/\mu_1 + v_1 + \sigma_1$ refers to the likelihood of infected humans dying due to the disease.

The offspring probability generating function for E_F , such that $E_H(0) = 0$, $I_H(0) = 0$, $E_F(0) = 1$, $I_F(0) = 0$, $E_D(0) = 0$, $I_D(0) = 0$, and $M(0) = 0$, is given by

$$f_3(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\gamma u_4 + \mu_2}{\gamma + \mu_2}, \quad (3.77)$$

where $\gamma/\gamma + \mu_2$ denotes the chance of the exposed free range dogs class progressing to the infected class, and $\mu_2/\gamma + \mu_2$ represents the possibility of exposed free dogs dying naturally before progressing to the infected class.

The offspring probability for I_F , given that $E_H(0) = 0$, $I_H(0) = 0$, $E_F(0) = 0$, $I_F(0) = 1$, $E_D(0) = 0$, $I_D(0) = 0$, and $M(0) = 0$, is given by

$$f_4(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\hat{\lambda}_1 u_1 u_4 + \hat{\lambda}_2 u_4 u_5 + \hat{\lambda}_3 u_3 u_4 + \mu_2 + \sigma_2 + v_2 u_4 u_7}{\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + v_2}, \quad (3.78)$$

for $\hat{\lambda}_1 = \tau_1 S_H^0$, $\hat{\lambda}_2 = \frac{\psi_1}{1 + \rho_1} S_D^0$, $\hat{\lambda}_3 = \kappa_1 S_F^0$.

In pgf (3.78), the term $\hat{\lambda}_1/\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + v_2$ represents the probability of infected free range dogs to cause rabies infection to susceptible humans, $\hat{\lambda}_2/\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + v_2$ signifies the probability of an infected free range dogs causing infection to susceptible domestic dogs, $\hat{\lambda}_3/\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + v_2$ denotes an infected free range dogs causing infection to free range dogs, $v_2/\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + v_2$ represents the probability of free range dogs to shade rabies virus in the environment, $\mu_2/\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + v_2$ denotes the probability of infected free range dogs dying naturally, and the term $\sigma_2/\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + v_2$ is the probability of infected free range dogs dying from rabies disease.

If $E_H(0) = 0$, $I_H(0) = 0$, $E_F(0) = 0$, $I_F(0) = 0$, $E_D(0) = 1$, $I_D(0) = 0$, and $M(0) = 0$, then the offspring probability of generating function for E_D is given by

$$f_5(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\gamma_1 u_6 + \gamma_2 + \mu_3}{\gamma_1 + \gamma_2 + \mu_3}, \quad (3.79)$$

where $\gamma_1/\gamma_1 + \gamma_2 + \mu_3$ represents the probability of exposed domestic dog progressing to infected class, $\gamma_2/\gamma_1 + \gamma_2 + \mu_3$ denotes the probability of exposed domestic dogs recovering from exposed class as a result of Rabies PEP before progressing to infected class, and $\mu_3/\gamma_1 + \gamma_2 + \mu_3$ signifies the probability of infected domestic dogs dying naturally before progressing to infected class.

The offspring probability generating function for I_D , such that $E_H(0) = 0$, $I_H(0) = 0$, $E_F(0) =$

0, $I_F(0) = 0$, $E_D(0) = 0$, $I_D(0) = 1$, and $M(0) = 0$, is given by

$$f_6(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\hat{\lambda}_4 u_5 u_6 + \hat{\lambda}_5 u_1 u_6 + \hat{\lambda}_6 u_3 u_6 + \mu_3 + \sigma_3 + v_3 u_6 u_7}{\hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + v_3}, \quad (3.80)$$

for $\hat{\lambda}_4 = \frac{\psi_2}{1 + \rho_2} S_D^0$, $\hat{\lambda}_5 = \tau_2 S_H^0$, $\hat{\lambda}_6 = \kappa_2 S_F^0$.

In pgf (3.80), the term $\hat{\lambda}_4 / \hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + v_3$ represents the probability of infected domestic dogs causing rabies infection to susceptible domestic dogs, $\hat{\lambda}_5 / \hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + v_3$ signifies the probability of a domestic dogs causing infection to susceptible humans, $\hat{\lambda}_6 / \hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + v_3$ denotes domestic dogs causing infection to susceptible free range dogs, $v_3 / \hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + v_3$ represents the probability of domestic dogs to shed rabies virus in the environment, $\mu_3 / \hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + v_3$ denotes the probability of infected domestic dogs dying naturally, and the term $\sigma_3 / \hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + v_3$ is the probability of infected domestic dogs dying from rabies disease.

If $E_H(0) = 0$, $I_H(0) = 0$, $E_F(0) = 0$, $I_F(0) = 0$, $E_D(0) = 0$, $I_D(0) = 0$, and $M(0) = 1$, then the offspring probability of generating function for M is given by

$$f_7(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\hat{\lambda}_7 u_5 u_7 + \hat{\lambda}_8 u_1 u_7 + \hat{\lambda}_9 u_3 u_7 + \mu_4}{\hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4}, \quad (3.81)$$

for $\hat{\lambda}_7 = \frac{\psi_3}{1 + \rho_3} S_D^0$, $\hat{\lambda}_8 = \tau_3 S_H^0$, $\hat{\lambda}_9 = \kappa_3 S_F^0$,

where $\hat{\lambda}_7 / \hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4$ represents the probability of rabies in environment causing infection to susceptible domestic dogs, $\hat{\lambda}_8 / \hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4$ signifies the probability of rabies in environment causing infection to susceptible humans, $\hat{\lambda}_9 / \hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4$ denotes the probability of rabies in environment causing infection to susceptible free range dogs, and $\mu_4 / \hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4$ denotes the probability of removal of rabies in environment.

The expectation matrix $\mathcal{M}$ of the branching process is a 7×7 matrix, which is defined by Equation (3.82). It is derived from the offspring probability generating functions (pgfs) given

in Equations (3.75) to (3.81), with all variables $(u_1, u_2, u_3, u_4, u_5, u_6, u_7) = (1, 1, 1, 1, 1, 1, 1)$:

$$\mathcal{M} = \begin{pmatrix} 0 & 0 & 0 & \frac{\hat{\lambda}_1}{J_1} & 0 & \frac{\hat{\lambda}_5}{J_2} & \frac{\hat{\lambda}_8}{J_3} \\ \frac{\beta_1}{J_4} & \frac{v_1}{J_5} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\hat{\lambda}_3}{J_6} & 0 & \frac{\hat{\lambda}_6}{J_7} & \frac{\hat{\lambda}_9}{J_8} \\ 0 & 0 & \frac{\gamma}{(\gamma + \mu_2)} & \frac{\mathbb{G}_1}{J_1} & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\hat{\lambda}_4}{J_1} & 0 & \frac{\hat{\lambda}_4}{J_2} & \frac{\hat{\lambda}_7}{J_3} \\ 0 & 0 & 0 & 0 & \frac{\gamma_1}{(\gamma_1 + \gamma_2 + \mu_3)} & \frac{\mathbb{G}_2}{J_2} & 0 \\ 0 & \frac{v_1}{(v_1 + \mu_1 + \sigma_1)} & 0 & \frac{v_2}{J_1} & 0 & \frac{v_3}{J_2} & \frac{\hat{\lambda}_9}{J_3} \end{pmatrix}, \quad (3.82)$$

where;

$$\begin{aligned} J_1 &= \hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + v_2, \quad J_2 = \hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + v_3, \quad J_3 = \hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4, \\ J_4 &= \beta_1 + \beta_2 + \mu_1, \quad J_5 = v_1 + \sigma_1 + \mu_1, \quad J_6 = \hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \mu_2 + \sigma_2 + v_2, \quad \mathbb{G}_2 = \hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + v_3, \\ J_7 &= \hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \mu_3 + \sigma_3 + v_3, \quad J_8 = \hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4, \quad \mathbb{G}_1 = \hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + v_2. \end{aligned}$$

The Continuous-Time Markov Chain (CTMC) model identifies a stochastic threshold that determines whether rabies will die out or lead to an outbreak in human and dog populations. This threshold is represented by the spectral radius of the expectation matrix, $\rho(\mathcal{M})$. There is a relationship between $\rho(\mathcal{M})$ in the stochastic model and the basic reproduction number $\mathcal{R}_0$ in the deterministic model. For rabies to be eliminated from both human and dog populations, it is required that $\rho(\mathcal{M}) \leq 1$ or $\mathcal{R}_0 < 1$. Conversely, in deterministic models, rabies persists in humans and dogs if $\mathcal{R}_0 > 1$. The relationship between the deterministic and stochastic thresholds for rabies extinction can be expressed as $\mathcal{R}_0 < 1 \iff \rho(\mathcal{M}) < 1$. In stochastic models, when $\rho(\mathcal{M}) < 1$, there is a possibility of either an outbreak or extinction of the *Rabies lyssavirus*, depending on the initial number of infectives at the onset of the disease outbreak. Conversely,

if $\rho(\mathcal{M}) > 1$, a fixed point

$$(f_1, f_2, f_3, f_4, f_5, f_6, f_7) \in (0, 1)^7$$

can be determined using offspring generating functions, which are then used to assess the probability of disease extinction. These generating functions are nonlinear, making analytical computation challenging, and thus numerical methods are typically employed for their calculation.

3.10 Mathematical Model with Control Strategies

Time-dependent control variables to Model (3.5) were administered to assess their efficacy in mitigating the transmission of rabies disease in both humans and dogs. The selection of these variables is guided by strategic interventions aimed at decreasing the likelihood of rabies infections in host population, as well as minimizing the shedding rate of rabies virus by dogs within the environment, including both domestic and free-range populations. To achieve optimal control over rabies transmission in humans, domesticated, and free-roaming dogs, we integrated four distinct time-dependent control variables that are meticulously tailored to address the intricacies of this complex epidemiological scenario: $u_1(t)$ measures the effect of promoting good health practices and management (surveillance and monitoring) in reducing the possibility of human and dog infections with rabies; $u_2(t)$ represents the control effort due to vaccination of domestic dogs; $u_3(t)$ measures the impact of educating communities about rabies related risks and preventive measures against exposure (public awareness and education); $u_4(t)$ represents treatment efforts for individuals exposed to suspected rabid animals through bites or scratches (Post-Exposure Prophylaxis-PEP). Figure 3 illustrates the transmission of rabies and incorporates model assumptions, variable definitions, and parameter specifications.

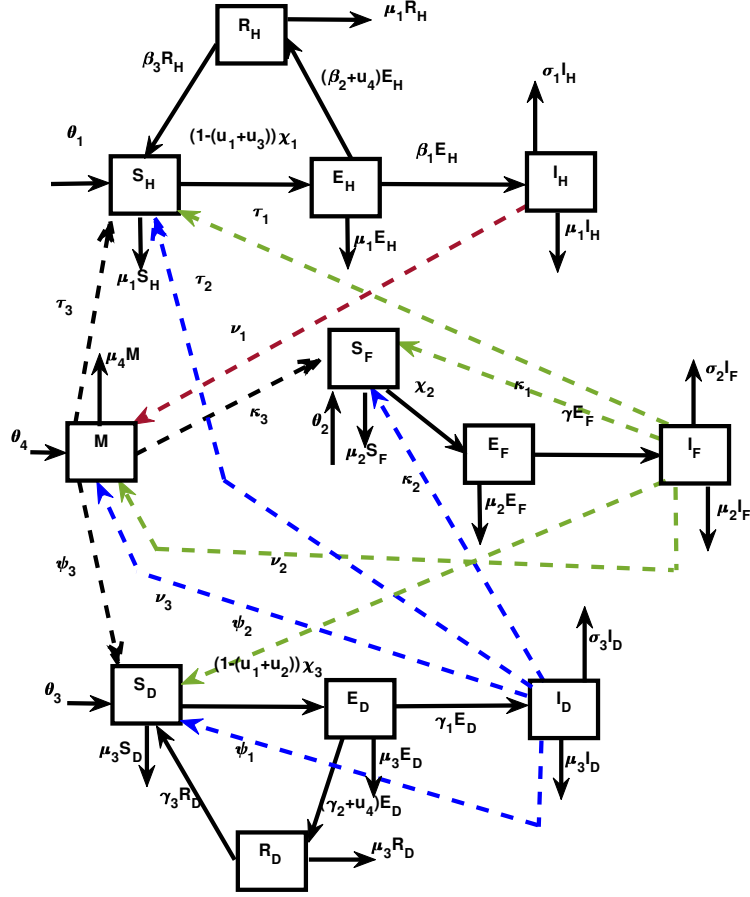


Figure 3: Compartmental diagram for rabies transmission with control

Ordinary differential Equations (ODEs) depict the dynamics of rabies as follows:

$$\left\{ \begin{array}{l}
 \dot{S}_H = \theta_1 + \beta_3 R_H - \mu_1 S_H - (1 - (u_1 + u_3)) (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H, \\
 \dot{E}_H = (1 - (u_1 + u_3)) (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H - (\mu_1 + \beta_1 + \beta_2 + u_4) E_H, \\
 \dot{I}_H = \beta_1 E_H - (\sigma_1 + \mu_1) I_H, \\
 \dot{R}_H = (\beta_2 + u_4) E_H - (\beta_3 + \mu_1) R_H, \\
 \dot{S}_F = \theta_2 - (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F - \mu_2 S_F, \\
 \dot{E}_F = (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F - (\mu_2 + \gamma) E_F, \\
 \dot{I}_F = \gamma E_F - (\mu_2 + \sigma_2) I_F, \\
 \dot{S}_D = \theta_3 - \mu_3 S_D - (1 - (u_1 + u_2)) \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right) S_D + \gamma_3 R_D, \\
 \dot{E}_D = (1 - (u_1 + u_2)) \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right) S_D - (\mu_3 + \gamma_1 + \gamma_2 + u_4) E_D, \\
 \dot{I}_D = \gamma_1 E_D - (\mu_3 + \sigma_3) I_D, \\
 \dot{R}_D = (\gamma_2 + u_4) E_D - (\mu_3 + \gamma_3) R_D, \\
 \dot{M} = (\nu_1 I_H + \nu_2 I_F + \nu_3 I_D) - \mu_4 M,
 \end{array} \right. \quad (3.83)$$

where

$$\lambda(M) = \frac{M}{M+C},$$

subject to initial non-negative conditions

$$\begin{aligned} S_H(0) > 0, E_H(0) \geq 0, I_H(0) \geq 0, R_H(0) \geq 0, S_D(0) > 0, E_D(0) \geq 0, I_D(0) \geq 0, \\ S_D(0) > 0, E_D(0) \geq 0, I_D(0) \geq 0, R_D(0) \geq 0. \end{aligned}$$

3.11 Formulation of the Optimal Control Problem

We aim to reduce the infected human and dog populations while also optimizing the costs linked to the implementation of control strategies. The objective function that reduces the M , E_H , I_H , E_D and I_D is given by:

$$J(u_j)_{1 \leq j \leq 4} = \min_{(u_1, u_2, u_3, u_4)} \int_{t_0}^{t_f} \left(K_1 M + K_2 E_H + K_3 I_H + K_4 E_D + K_5 I_D - K_6 S_D + \frac{1}{2} \sum_{j=1}^4 A_j u_j^2 \right) dt \quad (3.84)$$

subject to the control system (3.83), where K_1 represents the constant weight for the environment while K_2 and K_4 are the constant weights for the exposed classes of humans and dogs, respectively. Likewise, K_3 and K_5 are the constant weights for the infectious classes of humans and dogs, respectively, and K_6 is the constant weight for vaccinated domestic dogs. The coefficients A_1 , A_2 , A_3 , and A_4 are relative cost weights allocated to each specific control measure to transform the integral into the cost occurred over a time frame of t_f years. This duration represents the time period for implementing the controls. In essence, the cost associated with each control scenario is considered to follow a non-linear quadratic pattern, denoted as $\frac{A_1 u_1^2}{2}$. This cost reflects the expenses incurred in implementing control strategies aimed at promoting environmentally sustainable health practices and management, specifically surveillance and monitoring. Similarly, $\frac{A_2 u_2^2}{2}$ represents the cost of vaccinating domestic dogs, while $\frac{A_3 u_3^2}{2}$ is linked to community education about the risks of rabies exposure and prevention (public awareness and education). Meanwhile, $\frac{A_4 u_4^2}{2}$ pertains to the gradual treatment of infected individuals by potentially rabid animals (PEP). Each of these costs contributes to the overall expenditure required to address and mitigate the risks associated with rabies.

Considering $J(u_1, u_2, u_3, u_4)$, the primary goal is to minimize the number of exposed and infected for both human and domestic dogs while simultaneously maximizing the number of recovered humans and domestic dogs. Consequently, our aim is to determine u_1^* ,

u_2^*, u_3^*, u_4^* that satisfy the condition $J(u_1^*, u_2^*, u_3^*, u_4^*) = \min J(u_1, u_2, u_3, u_4)$, where $U = \{u : u \text{ is measurable and } 0 \leq u_j(t) \leq 1, j = 1, \dots, 4, \text{ for } t \in [t_0, t_f]\}$ is the control set, with the state variables $S_H(t), E_H(t), I_H(t), R_H(t), S_F(t), E_F(t), I_F(t), S_D(t), E_D(t), I_D(t), R_D(t)$, and $M(t)$ satisfying the control system (3.83) and given initial conditions (3.10).

3.12 Analysis of the Optimal Control Problem

This can be proved by characterizing the optimal control via Pontryagin's maximum principle, elucidating its essential features and providing a comprehensive understanding of its structure and dynamics.

3.12.1 Existence of a Unique Optimal Control

The solution to our optimal control problem is ensured by applying the results found in Stoddart (1967) and Sagamiko *et al.* (2015).

Theorem 3.7

The optimal control problem formulated in Section 3.11 admits a solution.

Proof. We want to show that there exists a control $\bar{u} = (\bar{u}_1, \bar{u}_2, \bar{u}_3, \bar{u}_4) \in U$ such that $J(\bar{u}) \leq J(u)$ for all $u = (u_1, u_2, u_3, u_4) \in U$ subject to (3.5)–(3.10). This follows from Stoddart (1967); Sagamiko *et al.* (2015) noting that: (a) the control system (3.83) is linear with respect to the control variables; (b) the control set U is bounded, convex and closed, and (c) the integrand of the objective functional (3.84) is convex in U . $\square$

Theorem 3.8

The solution to the optimal control problem formulated in Section 3.11 is unique.

Proof. Assume that there exists optimal controls $\bar{u}_1, \bar{u}_2, \bar{u}_3, \bar{u}_4$ and $\tilde{u}_1, \tilde{u}_2, \tilde{u}_3, \tilde{u}_4$ for the problem of Section 3.11, both achieving the minimum value of the objective functional:

$$J(\bar{u}_1, \bar{u}_2, \bar{u}_3, \bar{u}_4) = J(\tilde{u}_1, \tilde{u}_2, \tilde{u}_3, \tilde{u}_4) = \min_{u_1, u_2, u_3, u_4} J(u_1, u_2, u_3, u_4). \quad (3.85)$$

Consider the difference between the two optimal controls such that:

$$\delta u_j = \bar{u}_j - \tilde{u}_j \quad \text{for } j = 1, 2, 3, 4.$$

Since both controls are optimal, we have:

$$J(\tilde{u}_1, \tilde{u}_2, \tilde{u}_3, \tilde{u}_4) \leq J(\tilde{u}_1 + \delta u_1, \tilde{u}_2 + \delta u_2, \tilde{u}_3 + \delta u_3, \tilde{u}_4 + \delta u_4). \quad (3.86)$$

Expanding the expression (3.86), and subtracting $J(\tilde{u}_1, \tilde{u}_2, \tilde{u}_3, \tilde{u}_4)$ from both sides, we get:

$$\begin{cases} J(\tilde{u}_1 + \delta u_1, \tilde{u}_2 + \delta u_2, \tilde{u}_3 + \delta u_3, \tilde{u}_4 + \delta u_4) - J(\tilde{u}_1, \tilde{u}_2, \tilde{u}_3, \tilde{u}_4) \\ = \int_{t_0}^{t_f} \left(\sum_{j=1}^4 2A_j \tilde{u}_j \delta u_j + \sum_{j=1}^4 A_j \delta u_j^2 \right) dt. \end{cases} \quad (3.87)$$

Since all terms in expression (3.87) have non-negative integrand,

$$0 \leq \int_{t_0}^{t_f} \left(\sum_{j=1}^4 2A_j \tilde{u}_j \delta u_j + \sum_{j=1}^4 A_j \delta u_j^2 \right) dt. \quad (3.88)$$

Now, since $0 \leq \tilde{u}_j(t) \leq 1$ and $0 \leq \delta u_j(t) \leq 1$ for all $t \in [t_0, t_f]$ and $j = 1, 2, 3, 4$, we have:

$$0 \leq 2A_j \tilde{u}_j \delta u_j \leq 2A_j |\delta u_j|$$

and

$$0 \leq A_j \delta u_j^2 \leq A_j |\delta u_j|,$$

where $|\delta u_j|$ represents the absolute value of δu_j . Therefore,

$$0 \leq \int_{t_0}^{t_f} \left(\sum_{j=1}^4 2A_j \tilde{u}_j \delta u_j + \sum_{j=1}^4 A_j \delta u_j^2 \right) dt \leq \int_{t_0}^{t_f} \left(\sum_{j=1}^4 2A_j |\delta u_j| + \sum_{j=1}^4 A_j |\delta u_j| \right) dt = 0. \quad (3.89)$$

The inequality (3.89) holds if $\delta u_j(t) = 0$ for all $t \in [t_0, t_f]$ and $j = 1, 2, 3, 4$. This implies that $\bar{u}_j(t) = \tilde{u}_j(t)$ for all $t \in [t_0, t_f]$ and $j = 1, 2, 3, 4$, which means the two optimal controls are the same. $\square$

3.12.2 Characterization of the Optimal Control

Pontryagin's Maximum Principle (PMP), as applied in Silva *et al.* (2021) and Yamada *et al.* (2019), is now employed to characterize the solution in Section 3.11. The conditions outlined by the PMP transform the optimal control problem into the task of minimizing a pointwise function.

We introduce the Hamiltonian H given by:

$$\begin{aligned}
 H = & K_1 M + K_2 E_H + K_3 I_H + K_4 E_D + K_5 I_D - K_6 S_D + \frac{1}{2} u_1^2 A_1 + \frac{1}{2} u_2^2 A_2 \\
 & + \frac{1}{2} u_3^2 A_3 + \frac{1}{2} u_4^2 A_4 + \lambda_1 (\theta_1 + \beta_3 R_H - \mu_1 S_H - (1 - (u_1 + u_3)) \chi_1) \\
 & + \lambda_2 ((1 - (u_1 + u_3)) \chi_1 - (\mu_1 + \beta_1 + \beta_2 + u_4) E_H) + \lambda_3 (\beta_1 E_H - (\sigma_1 + \mu_1) I_H) \\
 & + \lambda_4 ((\beta_2 + u_4) E_H - (\beta_3 + \mu_1) R_H) + \lambda_5 (\theta_2 - \chi_2 - \mu_2 S_F) \\
 & + \lambda_6 (\chi_2 - (\mu_2 + \gamma) E_F) + \lambda_7 (\gamma E_F - (\mu_2 + \sigma_2) I_F) \\
 & + \lambda_8 (\theta_3 - \mu_3 S_D - (1 - (u_1 + u_2)) \chi_3 + \gamma_3 R_D) \\
 & + \lambda_9 ((1 - (u_1 + u_2)) \chi_3 - (\mu_3 + \gamma_1 + \gamma_2 + u_4) E_D) \\
 & + \lambda_{10} (\gamma_1 E_D - (\mu_3 + \sigma_3) I_D) + \lambda_{11} ((\gamma_2 + u_4) E_D - (\mu_3 + \gamma_3) R_D) \\
 & + \lambda_{12} ((v_1 I_H + v_2 I_F + v_3 I_D) - \mu_4 M),
 \end{aligned}
 \tag{3.90}$$

where;

$$\begin{aligned}
 \chi_1 &= (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H, \quad \chi_2 = (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F, \\
 \chi_3 &= \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right) S_D, \quad \lambda(M) = \frac{M}{M + C},
 \end{aligned}$$

and $\lambda_1, \dots, \lambda_{12}$ represent the adjoint variables. The result stated below is a direct implication of Pontryagin's maximum principle and the results of Section 3.12.1.

Theorem 3.9

If the optimal control to the problem formulated in Section 3.11 is $u^* = (u_1^*, u_2^*, u_3^*, u_4^*)$, then there exist corresponding adjoint variables $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7, \lambda_8, \lambda_9, \lambda_{10}, \lambda_{11}$, and λ_{12} that satisfy the adjoint condition:

$$\lambda'_j(t) = -\frac{\partial H}{\partial y_j}$$

subject to $\lambda_j(t_f) = 0$, for $1 \leq j \leq 12$. Furthermore, the optimal controls u_i^* , $i = 1, \dots, 4$, are characterized by the maximality condition: $\lambda_j(t_f) = 0$ for $j = 1, 2, 3, \dots, 12$ for control set u_i . Hence, we have:

$$\frac{\partial H}{\partial u_i} = 0 \quad \text{where } i = 1, 2, 3, 4,$$

The solutions for the adjoint and optimality conditions are presented in Equations (3.91) and

(3.92), respectively.

$$\frac{d\lambda_i}{dt} = \begin{cases} -\lambda_1 \left(-\mu_1 - (1 - u_1 - u_3) \left(\tau_2 I_D^* + \tau_1 I_F^* + \frac{\tau_3 M^*}{M^* + C} \right) \right) \\ \quad - \lambda_2 (1 - u_1 - u_3) \left(\tau_2 I_D^* + \tau_1 I_F^* + \frac{\tau_3 M^*}{M^* + C} \right), & i = 1, \\ -K_2 + \lambda_2 (\mu_1 + \beta_1 + \beta_2 + u_4) - \lambda_3 \beta_1 - \lambda_4 (\beta_2 + u_4), & i = 2, \\ -K_3 + \lambda_3 (\mu_1 + \sigma_1) - \lambda_{12} v_1, & i = 3, \\ -\lambda_1 \beta_3 + \lambda_4 (\beta_3 + \mu_1), & i = 4, \\ \lambda_5 (\kappa_1 I_F^* + \kappa_2 I_D^* + \frac{\kappa_3 M^*}{M^* + C} + \mu_2) - \lambda_6 (\kappa_1 I_F^* + \kappa_2 I_D^* + \frac{\kappa_3 M^*}{M^* + C}), & i = 5, \\ \lambda_6 (\mu_2 + \gamma) - \lambda_8 \gamma, & i = 6, \\ (\lambda_1 - \lambda_2) (1 - u_1 - u_3) \tau_1 S_H^* + (\lambda_5 - \lambda_6) \kappa_1 S_F^* \\ \quad + \lambda_7 (\mu_2 + \sigma_2) + \frac{S_D^* (1 - u_1 - u_2) (\lambda_8 - \lambda_9) \psi_1}{1 + \rho_1} - \lambda_{12} v_2, & i = 7, \\ K_6 - \lambda_8 \left(-\mu_3 - (1 - u_1 - u_2) \left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} + \frac{\psi_3 M^*}{(M^* + C)(1 + \rho_3)} \right) \right) \\ \quad - \lambda_9 (1 - u_1 - u_2) \left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} + \frac{\psi_3 M^*}{(M^* + C)(1 + \rho_3)} \right), & i = 8, \\ -K_4 + \lambda_9 (\mu_3 + \gamma_1 + \gamma_2 + u_4) - \lambda_{10} \gamma_1 - \lambda_{11} (\gamma_1 + u_4), & i = 9, \\ -K_5 + S_H^* (\lambda_1 - \lambda_2) (1 - u_1 - u_3) \tau_2 \\ \quad + S_F^* (\lambda_5 - \lambda_6) + \frac{(1 - u_1 - u_2) \psi_2 S_D^* (\lambda_8 - \lambda_9)}{1 + \rho_2} \kappa_2 \\ \quad + \lambda_{10} (\mu_3 + \sigma_3) - \lambda_{12} v_3, & i = 10, \\ -\lambda_8 \gamma_3 + \lambda_{11} (\mu_3 + \gamma_3), & i = 11, \\ -K_1 + (\lambda_1 - \lambda_2) (1 - u_1 - u_3) \left(\frac{\tau_3}{M^* + C} + \frac{\tau_3 M^*}{(M^* + C)^2} \right) S_H^* \\ \quad + (\lambda_5 - \lambda_6) \left(\frac{\kappa_1}{M^* + C} + \frac{\kappa_1 M^*}{(M^* + C)^2} \right) S_F^* \\ \quad + (\lambda_8 - \lambda_9) (1 - u_1 - u_2) \left(\frac{\psi_3}{(M^* + C)(1 + \rho_3)} + \frac{\psi_3 M^*}{(M^* + C)^2 (1 + \rho_3)} \right) S_D^* \\ \quad + \lambda_{12} \mu_4, & i = 12. \end{cases} \quad (3.91)$$

$$\mathbf{u}^* = \begin{bmatrix} u_1^* \\ u_2^* \\ u_3^* \\ u_4^* \end{bmatrix} = \max \left\{ \mathbf{0}, \min \left(\mathbf{1}, \left[\begin{array}{c} \frac{(\lambda_2 - \lambda_1) \left(\tau_2 I_D + \tau_1 I_F + \frac{\tau_3 M}{M+C} \right) S_H + (\lambda_9 - \lambda_8) G S_D}{A_1} \\ \frac{(\lambda_9 - \lambda_8) \left(\frac{\psi_1 I_F}{1+\rho_1} + \frac{\psi_2 I_D}{1+\rho_2} + \frac{\psi_3 M}{(M+C)(1+\rho_3)} \right) S_D}{A_2} \\ \frac{(\lambda_2 - \lambda_1) \left(\tau_1 I_F + \tau_2 I_D + \frac{\tau_3 M}{M+C} \right) S_H}{A_3} \\ \frac{E_H (\lambda_2 - \lambda_4) + E_D (\lambda_9 - \lambda_{11})}{A_4} \end{array} \right] \right\} \right\}. \quad (3.92)$$

Where:

$$G = \frac{\psi_1 I_F}{1+\rho_1} + \frac{\psi_2 I_D}{1+\rho_2} + \frac{\psi_3 M}{(M+C)(1+\rho_3)}.$$

In Section 4.4 we follow Maliyoni *et al.* (2023) to implement Theorem 3.9 in MATLAB in an algorithm way.

CHAPTER FOUR

RESULTS AND DISCUSSION

4.1 Introduction

In this chapter, mathematical models of rabies interactions presented in the chapter three were simulated in Matrix Laboratory (MATLAB) programming language to support analytical findings and demonstrate the strength and impact of mitigation strategies.

4.1.1 Model Fitting

The model is calibrated using real-world data from Tanzania spanning the period 1990 to 2018 (Fig.4) through optimization techniques. Specifically, the Nelder–Mead algorithm was employed to minimize the discrepancy between the model's predictions data $\hat{Y}(\Theta_i)$ and the observed data Y through discretization $I_H(t_i)$ of the system (3.5) without controls, as given below

$$I_H(t_i + \Delta t) = I_H(t_i) + (\beta_1 \cdot E_H(t_i) - (\sigma_1 + \mu_1) \cdot I_H(t_i)) \cdot \Delta t. \quad (4.1)$$

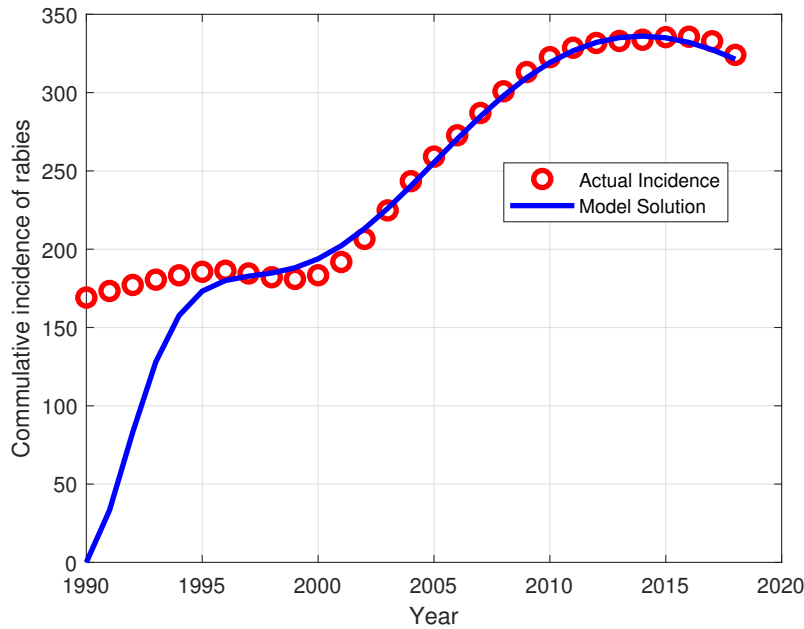


Figure 4: Model (3.5) fitting against reported incidence data of infected humans rabies $I_H(t)$ from 1990 to 2018

4.1.2 Parameter Value and Estimation

To generate the rabies model-predicted dataset of infected individuals, denoted as $RD(t_i; \Theta_i)$, at time t_i for a given parameter vector Θ_i , random Gaussian noise $\eta_i(t_i; \Theta_i)$ was incorporated into the measurements. This adjustment simulates real-world dynamics, where measurement errors are inherently present. Consequently, the observed dependent data were expressed as:

$$I(t_i; \Theta_i) = RD(t_i; \Theta_i) + \eta_i(t_i; \Theta_i) \text{ for each time } t_i \in [1, n], \quad (4.2)$$

where n is the number of data points. The objective function quantifies the difference between the model's predictions $I(t_i; \Theta_i)$ and the estimated data $I_{\text{estimated},i}$. We define the objective function $J(\Theta_i)$ as the sum of squared differences between model predictions and estimated dataset as

$$J(\Theta_i) = \sum_{i=1}^{33} a_i (I(t_i; \Theta_i) - I_{\text{estimated},i})^2, \quad (4.3)$$

where $I_{\text{estimated},i}$ is the estimated number of infected rabies individuals at time t_i . The term $(I(t_i; \Theta_i) - I_{\text{estimated},i})^2$ represents the squared difference between the model-predicted value $I(t_i; \Theta_i)$ and the estimated value $I_{\text{estimated},i}$ at time t_i ; a_i typically represents the weighting or importance assigned to each term in the summation. The goal is to find the parameter vector $\hat{\Theta}_i$ that minimizes the objective function $J(\Theta_i)$ given by

$$\hat{\Theta}_i = \arg \min J(\Theta_i). \quad (4.4)$$

The expression indicates that $\hat{\Theta}_i$ is the parameter vector that optimally fits the model to the estimated data by minimizing the objective function $J(\Theta_i)$. The symbol $\arg \min$ is used to find the argument (in this case, Θ_i) that minimizes the function $J(\Theta_i)$. To find the parameter value Θ_i , we apply partial derivatives on Eq. (3.5) with respect to each of the 33 parameters represented by Θ_i such that

$$\frac{\partial J(\Theta_i)}{\partial \Theta_i} = \sum_{i=1}^{33} 2 (I(t_i; \Theta_i) - I_{\text{estimated},i}) \frac{\partial (I(t_i; \Theta_i))}{\partial \Theta_i} = 0 \text{ for } i = 1, 2, \dots, 33, \quad (4.5)$$

where

$$\Theta_i = \{\rho_j, \theta_k, \tau_k, \beta_k, \mu_m, \sigma_m, \gamma_n, \kappa_k, \psi_k, \nu_p, C\}, \quad j, k \in \{1, 2, 3\}, m \in \{1, 2, 3, 4\}, n, p \in \{1, 2, 3\}.$$

The results of the simulation are shown in Table 5 and Fig. 8 through 10.

Table 5: Estimated model parameters (Year⁻¹), initial guess for parameters (Year⁻¹) and their respective source

Parameters	Baseline Value	Source	Estimated Value	Mean (μ) and Std (σ)
θ_1	2000	(Estimated)	1993.382113	$\mathcal{N}(1996.691056 \ 4.4679553)$
τ_1	0.0004	Tian <i>et al.</i> (2018)	0.000405	$\mathcal{N}(0.000402 \ 4 \times 10^{-6})$.
τ_2	0.0004	Tian <i>et al.</i> (2018)	0.000604	$\mathcal{N}(0.000502 \ 1.44 \times 10^{-4})$.
τ_3	[0.0003 0.0100]	(Estimated)	0.000303	$\mathcal{N}(0.000302 \ 2 \times 10^{-6})$.
β_1	$\frac{1}{6}$	Zhang <i>et al.</i> (2011) and Tian <i>et al.</i> (2018)	0.165581	$\mathcal{N}(0.166124 \ 7.68 \times 10^{-4})$.
β_2	[0.54 1]	Zhang <i>et al.</i> (2011)	0.540487	$\mathcal{N}(0.5402435 \ 3.7815 \times 10^{-4})$.
β_3	1	(Estimated)	0.999301	$\mathcal{N}(0.9996505 \ 1.6521 \times 10^{-4})$.
μ_1	0.0142	Organization (2010) and Organization (2013)	0.014417	$\mathcal{N}(0.014309 \ 1.53 \times 10^{-4})$.
σ_1	1	Zhang <i>et al.</i> (2011) and Abdulmajid & Hassan (2021)	1.006332	$\mathcal{N}(1.03166 \ 4.47 \times 10^{-3})$.
θ_2	1000	(Estimated)	1004.12044	$\mathcal{N}(1002.060222 \ 2.913594)$.
κ_1	0.00006	(Estimated)	0.000020	$\mathcal{N}(0.000040 \ 2.8 \times 10^{-5})$.
κ_2	0.00005	(Estimated)	0.000081	$\mathcal{N}(0.000066 \ 2.2 \times 10^{-5})$.
κ_3	[0.00001 0.00003]	(Estimated)	0.000040	$\mathcal{N}(0.000025 \ 2.1 \times 10^{-5})$.
γ	$\frac{1}{6}$	Zhang <i>et al.</i> (2011) and Tian <i>et al.</i> (2018)	0.166374	$\mathcal{N}(0.166520 \ 2.07 \times 10^{-4})$.
σ_2	0.09	Zhang <i>et al.</i> (2011)	0.089556	$\mathcal{N}(0.089778 \ 3.14 \times 10^{-4})$.
μ_2	0.067	Addo (2012)	0.066268	$\mathcal{N}(0.066634 \ 1.58 \times 10^{-4})$.
θ_3	1200	(Estimated)	1203.844461	$\mathcal{N}(1201.922230 \ 2.718444)$.
ψ_1	0.0004	Hampson <i>et al.</i> (2019)	0.000077	$\mathcal{N}(0.000238 \ 2.28 \times 10^{-4})$.
ψ_2	0.0004	Hailemichael <i>et al.</i> (2022)	0.000066	$\mathcal{N}(0.000233 \ 2.36 \times 10^{-4})$.
ψ_3	0.0003	(Estimated)	0.000030	$\mathcal{N}(0.0003 \ 1.91 \times 10^{-4})$.
μ_3	0.067	(Estimated)	0.080129	$\mathcal{N}(0.073565 \ 8.056 \times 10^{-3})$.
σ_3	0.08	Zhang <i>et al.</i> (2011)	0.091393	$\mathcal{N}(0.085697 \ 8.056 \times 10^{-3})$.
γ_1	$\frac{1}{6}$	Tian <i>et al.</i> (2018)	0.172489	$\mathcal{N}(0.169578 \ 4.117 \times 10^{-3})$.
γ_2	0.09	Zhang <i>et al.</i> (2011)	0.090308	$\mathcal{N}(0.090154 \ 2.18 \times 10^{-4})$.

Continued on next page

Table 5 – Continued from previous page

Parameters	Baseline Value	Source	Estimated Value	Mean (μ) and Std (σ)
γ_3	0.05	(Estimated)	0.050128	$\mathcal{N}(0.050128 \ 9.1 \times 10^{-5})$.
ν_1	0.001	(Estimated)	0.001958	$\mathcal{N}(0.001479 \ 6.77 \times 10^{-4})$.
ν_2	0.006	(Estimated)	0.008971	$\mathcal{N}(0.007485 \ 2.101 \times 10^{-3})$.
ν_3	0.001	(Estimated)	0.005735	$\mathcal{N}(0.003367 \ 3.3348 \times 10^{-3})$.
μ_4	0.08	(Estimated)	0.080625	$\mathcal{N}(0.080313 \ 4.42 \times 10^{-4})$.
ρ_1	10	Ruan (2017b)	9.920733	$\mathcal{N}(9.960366 \ 5.605 \times 10^{-2})$.
ρ_2	8	(Estimated)	8.116421	$\mathcal{N}(8.058211 \ 8.2322 \times 10^{-2})$.
ρ_3	15	(Estimated)	14.917005	$\mathcal{N}(14.958502 \ 5.8686 \times 10^{-2})$.
C	0.003 (PFU)/mL	(Estimated)	0.003011	$\mathcal{N}(0.003005 \ 8.0000 \times 10^{-6})$.

Figures 5 through 7 present the residual histograms, which validate the consistency of the model with the estimated parameters. The autocorrelation analysis illustrated in Figures 12 through 15 remains within the range of ± 0.2 , indicating a moderate to weak correlation among consecutive residuals. This finding suggests minimal error dependence and confirms that the model is unbiased, devoid of systematic trends. Furthermore, Figures 8 through 10 depict the dynamics of rabies, wherein an increase in infections $I(t)$ leads to a decrease in the susceptible $S(t)$ population, ultimately resulting in a non-negative equilibrium due to increase in contact rates.

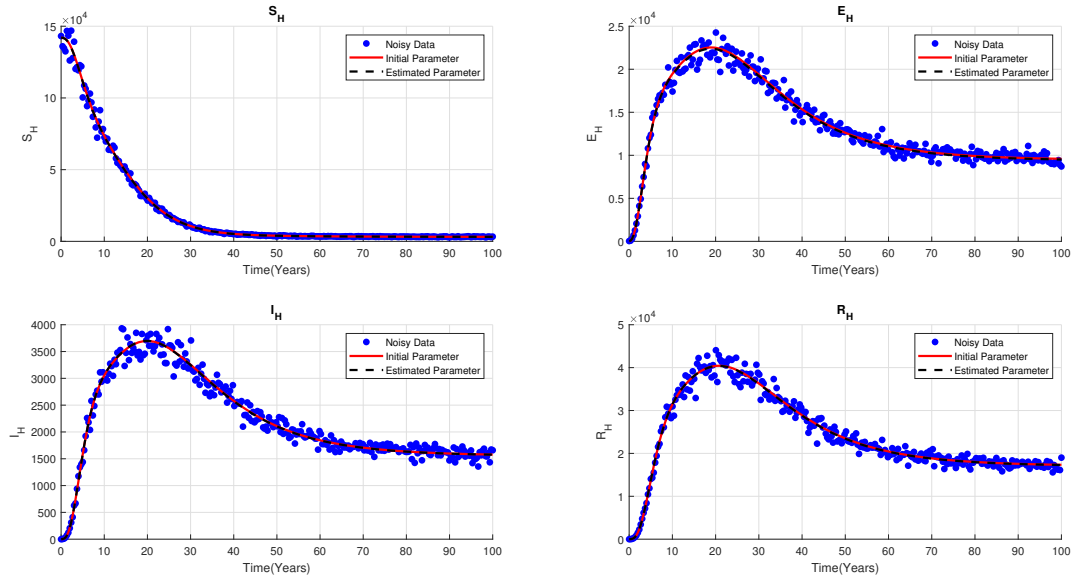


Figure 5: Model fitting and scatter plot with parameter estimate for human population with standard deviation $\sigma = 0.05$ and confidence interval (C.I.) = 95 %

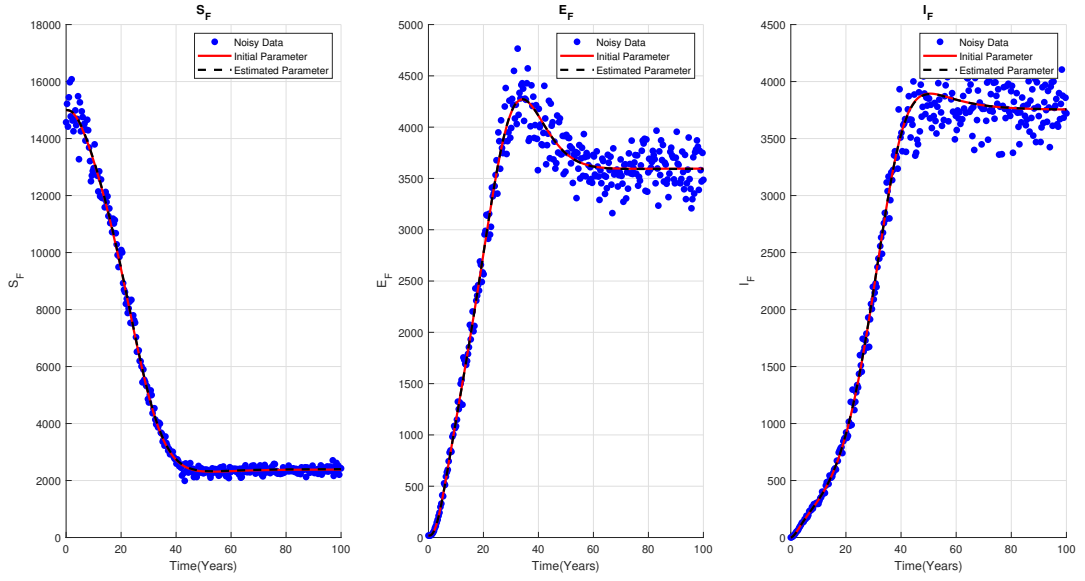


Figure 6: Model fitting and scatter plot with parameter estimate for free range dogs with standard deviation $\sigma = 0.05$ and confidence interval (C.I.) = 95 %

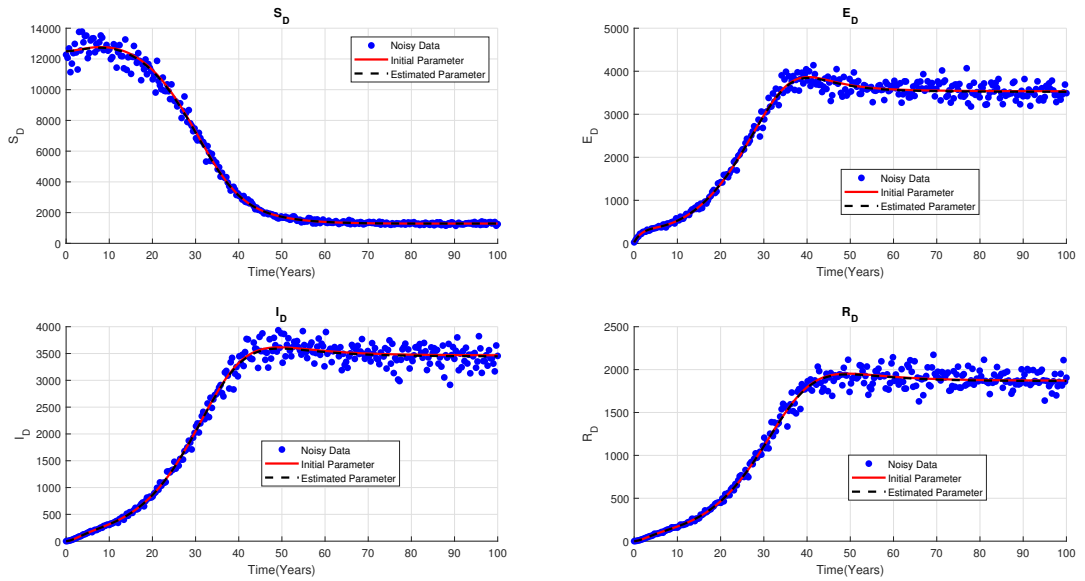


Figure 7: Model fitting and scatter plot with parameter estimate for domestic dogs with standard deviation $\sigma = 0.05$ and confidence interval (C.I.) = 95 %

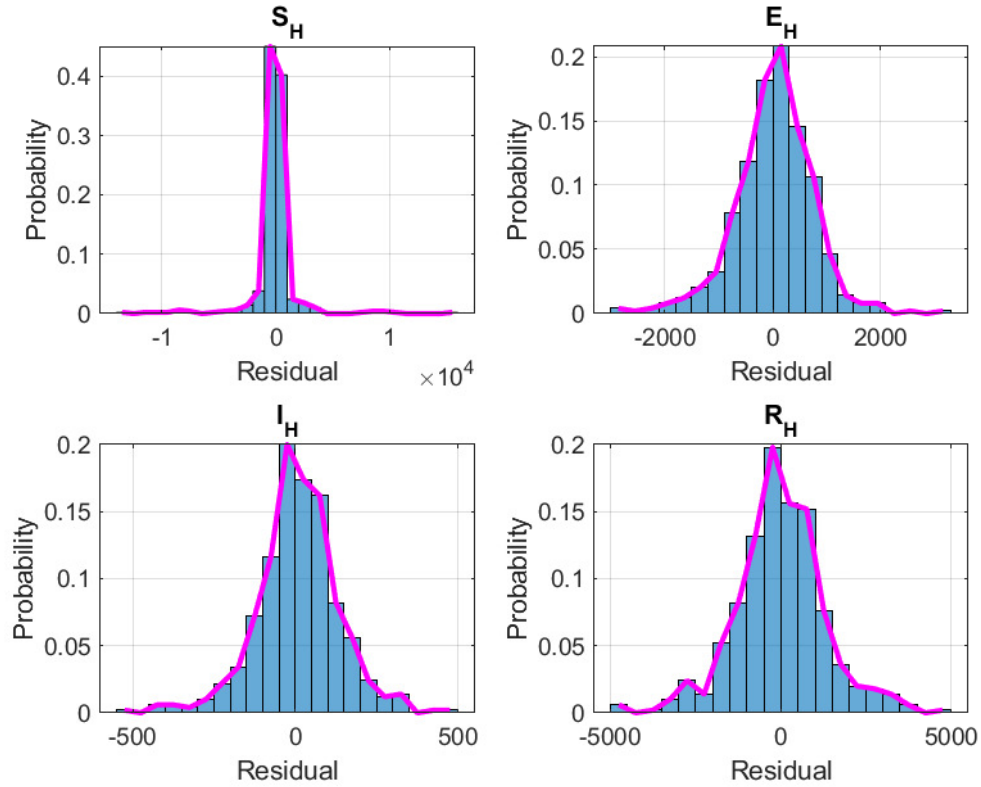


Figure 8: Normal distribution of the rabies model variable with standard deviation $\sigma=0.05$ and Confidence interval (C.I.)=95% for human population

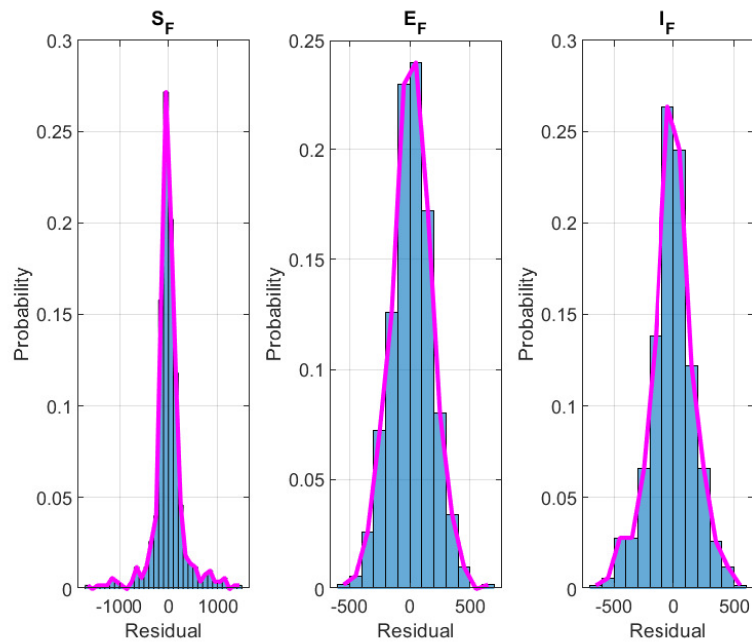


Figure 9: Normal distribution of the rabies model variables with standard deviation $\sigma=0.05$ and Confidence interval (C.I.)=95% for free-range dogs population

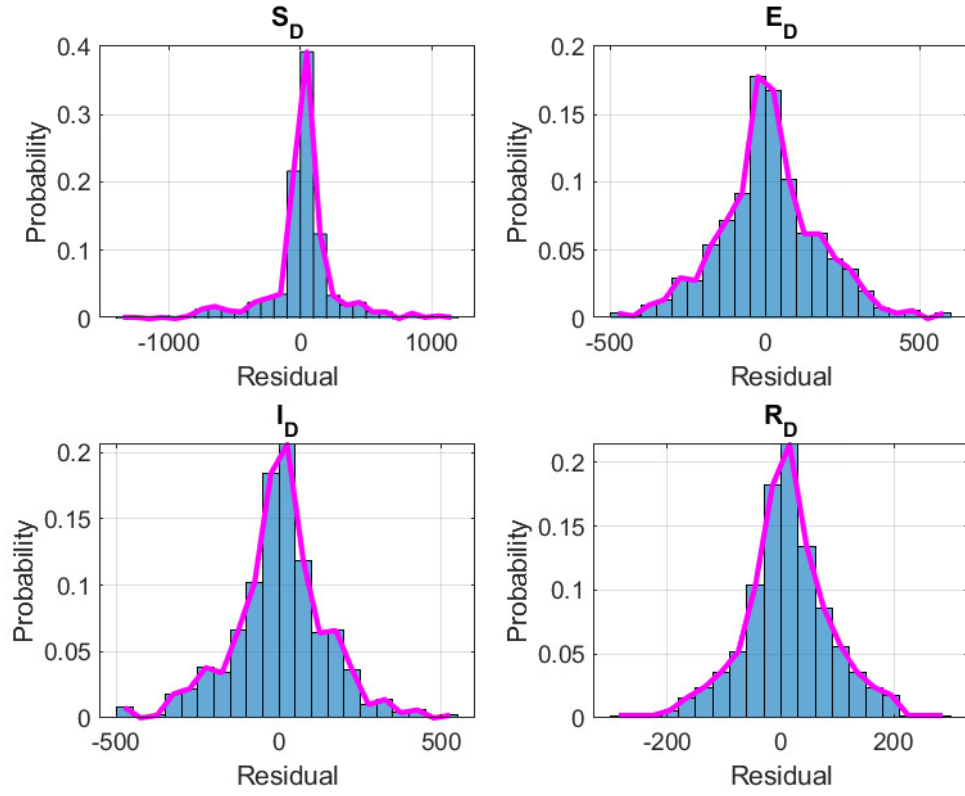


Figure 10: Normal distribution of the rabies model variable with standard deviation $\sigma=0.05$ and Confidence interval (C.I.)=95% for domestic dogs population

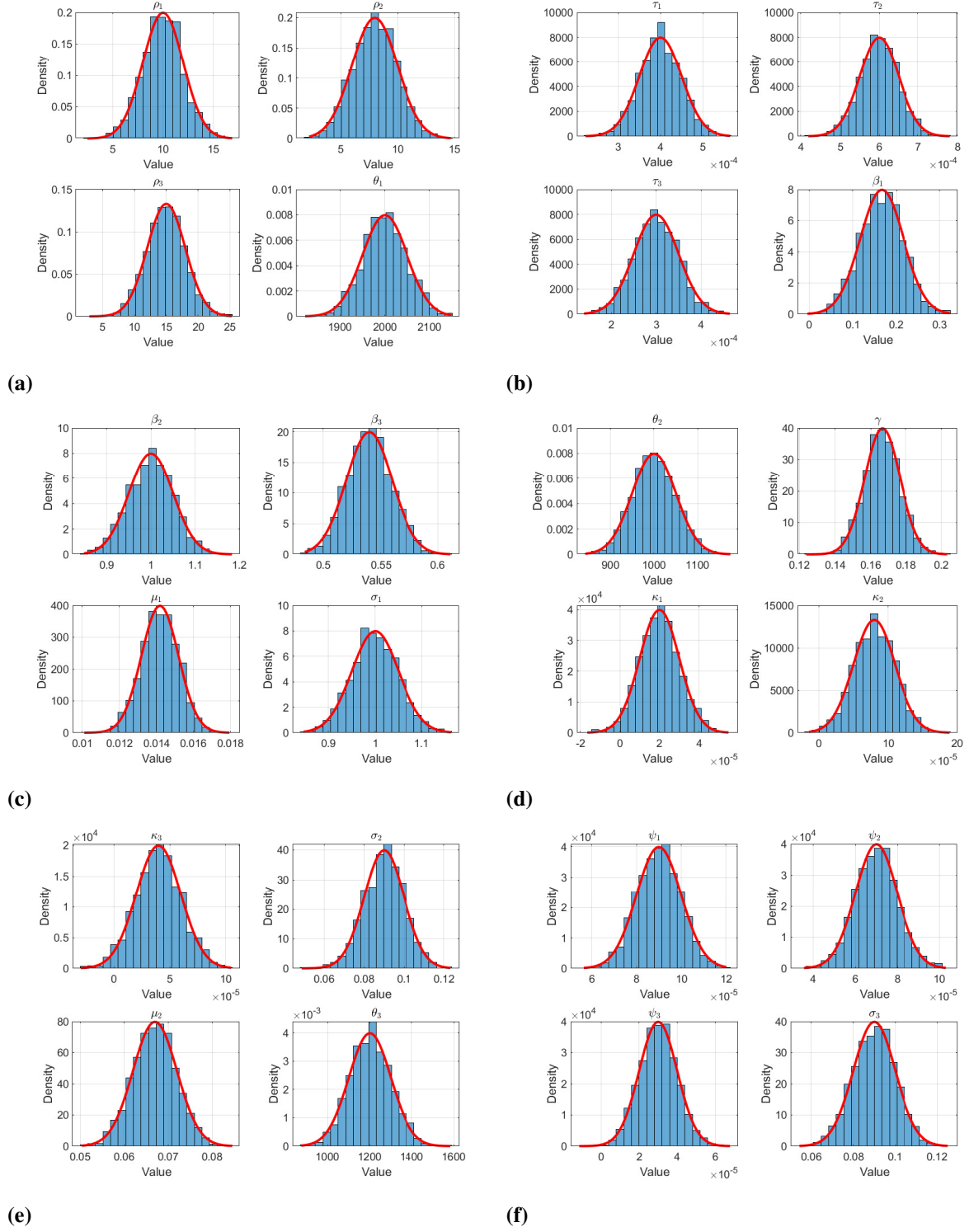


Figure 11: Normal distribution plots (a-f) of parameter values

All parameter values followed a normal distribution, as illustrated in Fig. 11 (a-f).

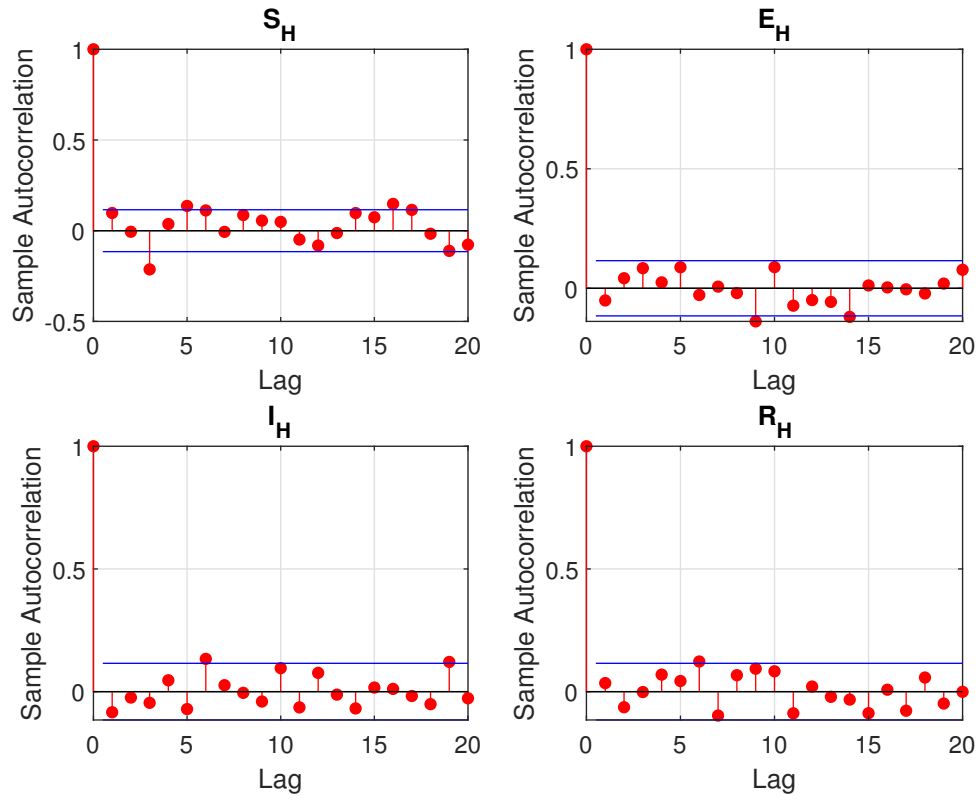


Figure 12: The sample autocorrelation of the residuals in relation to human population indicating the lack of significance at the 5% level

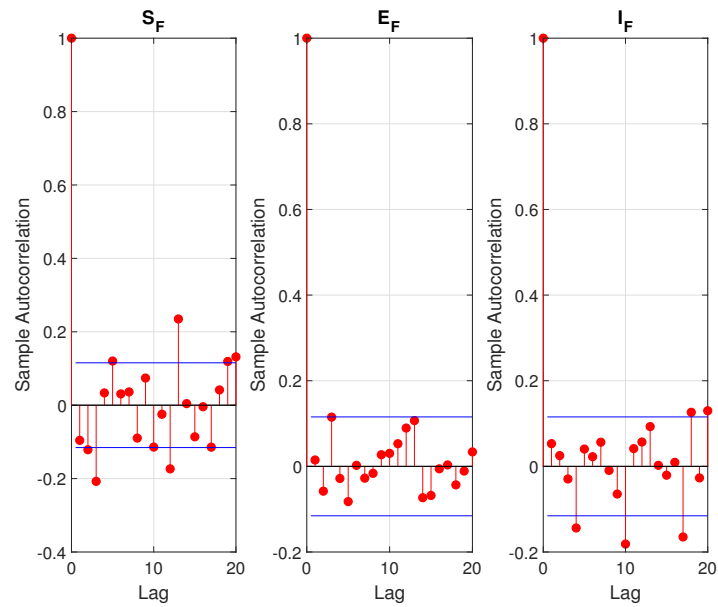


Figure 13: The sample autocorrelation of the residuals in relation to domestic dogs indicating the lack of significance at the 5% level

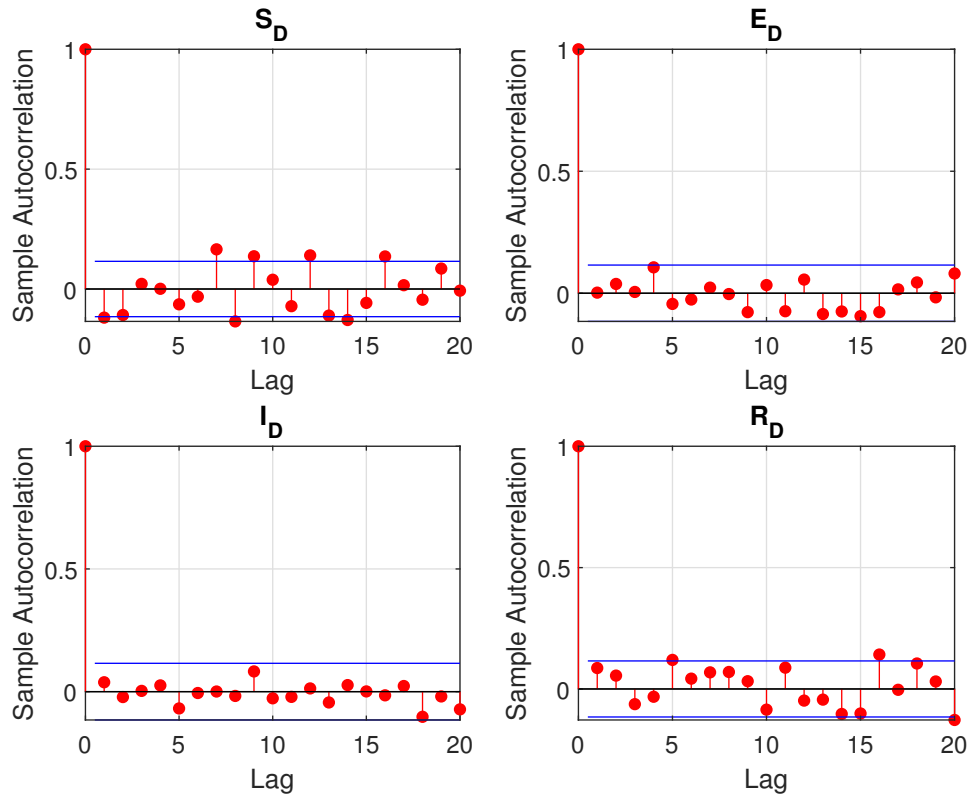


Figure 14: The sample autocorrelation of the residuals in relation to free range indicating the lack of significance at the 5% level

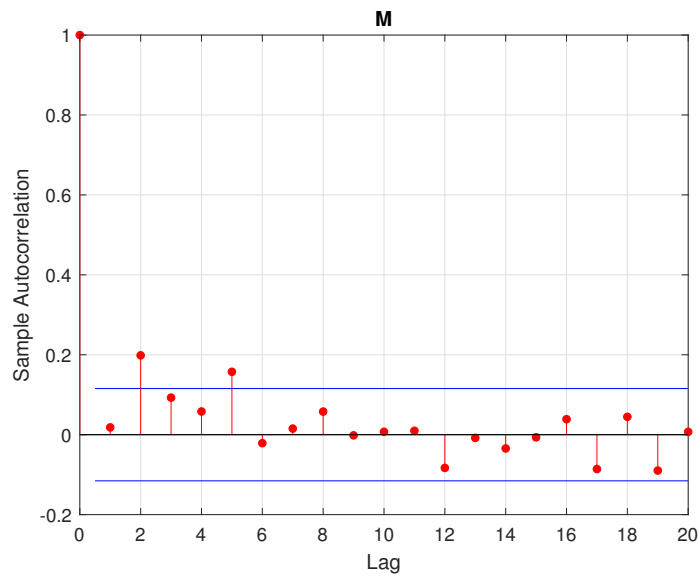


Figure 15: The sample autocorrelation of the residuals in relation to environment indicating the lack of significance at the 5% level

4.1.3 Evaluation of Error Metrics and Model Performance

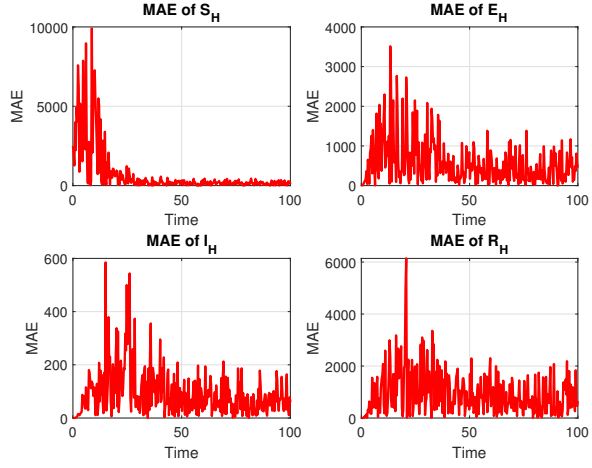
To evaluate the performance of the model given in equation (3.5), coefficient of determination (R^2), MAE and MRE were employed as metrics to quantify the deviation between the predicted and observed values. Let $X_{\text{obs},i}$ and $X_{\text{pred},i}$ represent the observed and predicted values of X for $i = 1, 2, \dots, n$. The MAE and MRE were computed using algorithm. Where $X \in \{S_H, E_H, I_H, R_H, S_F, E_F, I_F, S_D, E_D, I_D, R_D, M\}$, and $X \in \mathbb{R}^{12}$ represents the relevant variables. The results of these evaluations are provided in Table 6 and visualized in Fig. 16 through 22.

Table 6: Quantitative Assessment of Model Performance

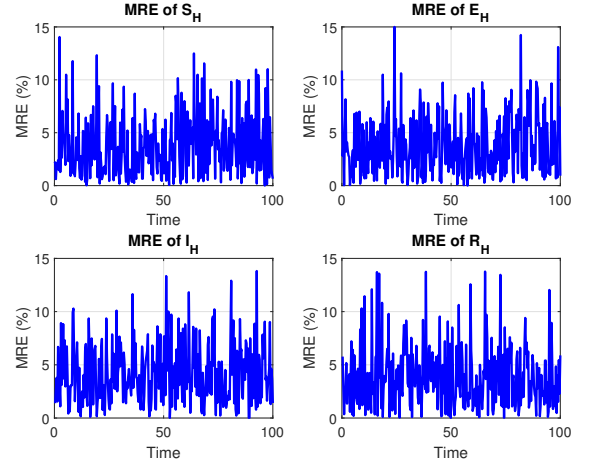
Variable	MAE	MRE (%)	R^2	Variable	MAE	MRE (%)	R^2
S_H	753.83	4.25	0.99656	I_F	126.11	4.00	0.98384
E_H	533.204	3.95	0.98500	S_D	168.23	4.20	0.99544
I_H	89.54	4.00	0.98543	E_D	123.00	4.20	0.97963
R_H	959.60	4.00	0.98407	I_D	104.33	4.00	0.98516
S_F	181.78	3.91	0.99381	R_D	62.74	4.10	0.98638
E_F	138.90	4.17	0.97505	M	20.37	3.85	0.98573

Table (6) presents a quantitative evaluation of model performance using Mean Absolute Error (MAE), Mean Relative Error (MRE%), and the coefficient of determination (R^2). The high R^2 values across all variables ranging from 0.97505 to 0.99656 indicate that the model captures the observed dynamics with strong accuracy and reliability. Among the human compartments, the susceptible humans (S_H) exhibit a high MAE of 753.83 and an R^2 of 0.99656, suggesting large absolute prediction errors but very good overall fit. The exposed (E_H) and infected (I_H) humans maintain moderate MAE values (533.204 and 89.54, respectively) with high R^2 values (> 0.985), reflecting the model's ability to closely approximate both latent and active stages of infection in humans. The recovered human compartment (R_H) also shows strong fit ($R^2 = 0.98407$) despite a high MAE. In free-ranging dogs, susceptible (S_F), exposed (E_F), and infected (I_F) exhibit relatively low MAEs (ranging from 126.11 to 181.78) and strong R^2 values (above 0.975), reinforcing their critical role in rabies transmission and confirming the model's capability in capturing their disease dynamics accurately. Similar patterns are observed in domestic dog compartments, with all categories (from susceptible to recovered) showing low errors and consistently high R^2 values above 0.979. This highlights the effectiveness of the model in representing rabies transmission within this important reservoir host group.

Finally, the environmental viral concentration (M) demonstrates the lowest MAE (20.37) and maintains a high R^2 of 0.98573, indicating excellent model performance in tracking the environmental component of rabies transmission. Overall, the model displays strong predictive power across all compartments, particularly in host categories that play central roles in disease spread, underscoring its suitability for simulating and evaluating rabies control strategies (Fig. 16a through 22).

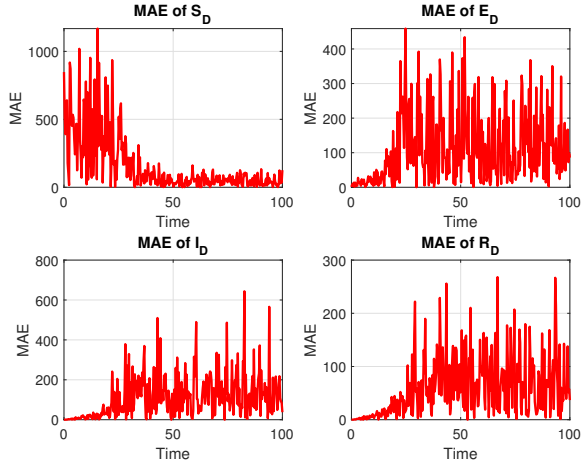


(a) Mean Absolute Error (MAE) for N_H

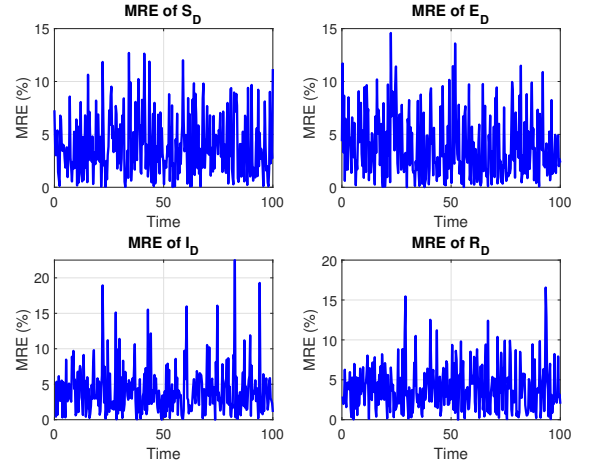


(b) Mean Relative Error (MRE) for N_H

Figure 16: Comparison of Mean Absolute and Relative Errors for N_H

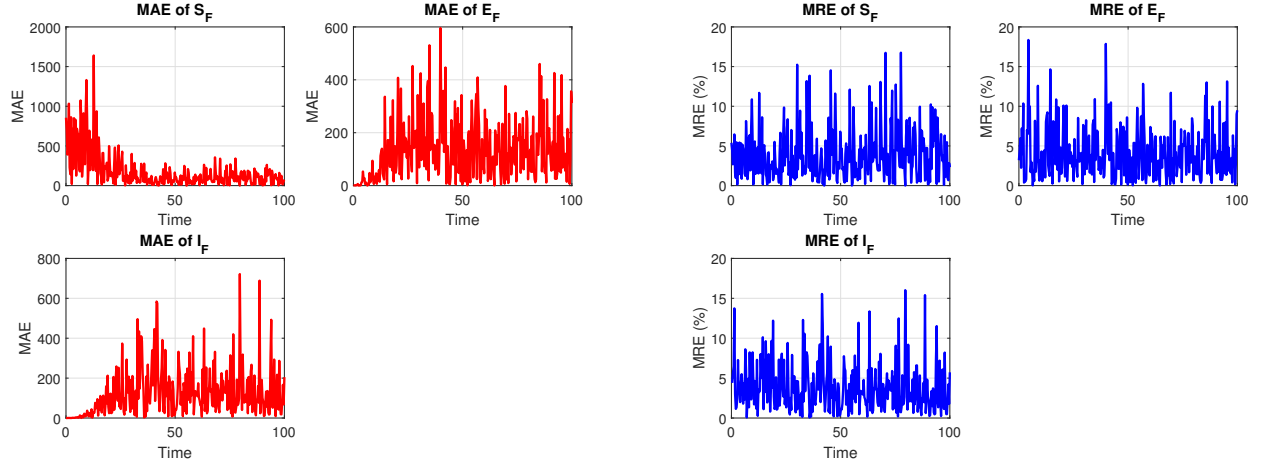


(a) Mean Absolute Error (MAE) for N_D



(b) Mean Relative Error (MRE) for N_D

Figure 17: Comparison of Mean Absolute and Relative Errors for N_D



(a) Mean Absolute Error (MAE) for N_F

(b) Mean Relative Error (MRE) for N_F

Figure 18: Comparison of Mean Absolute and Relative Errors for N_F

For comparative analysis, Fig. 16 through 22 presents the solutions from the literature alongside the estimated parameter values. It is evident that the solutions exhibit a strong alignment with each other.

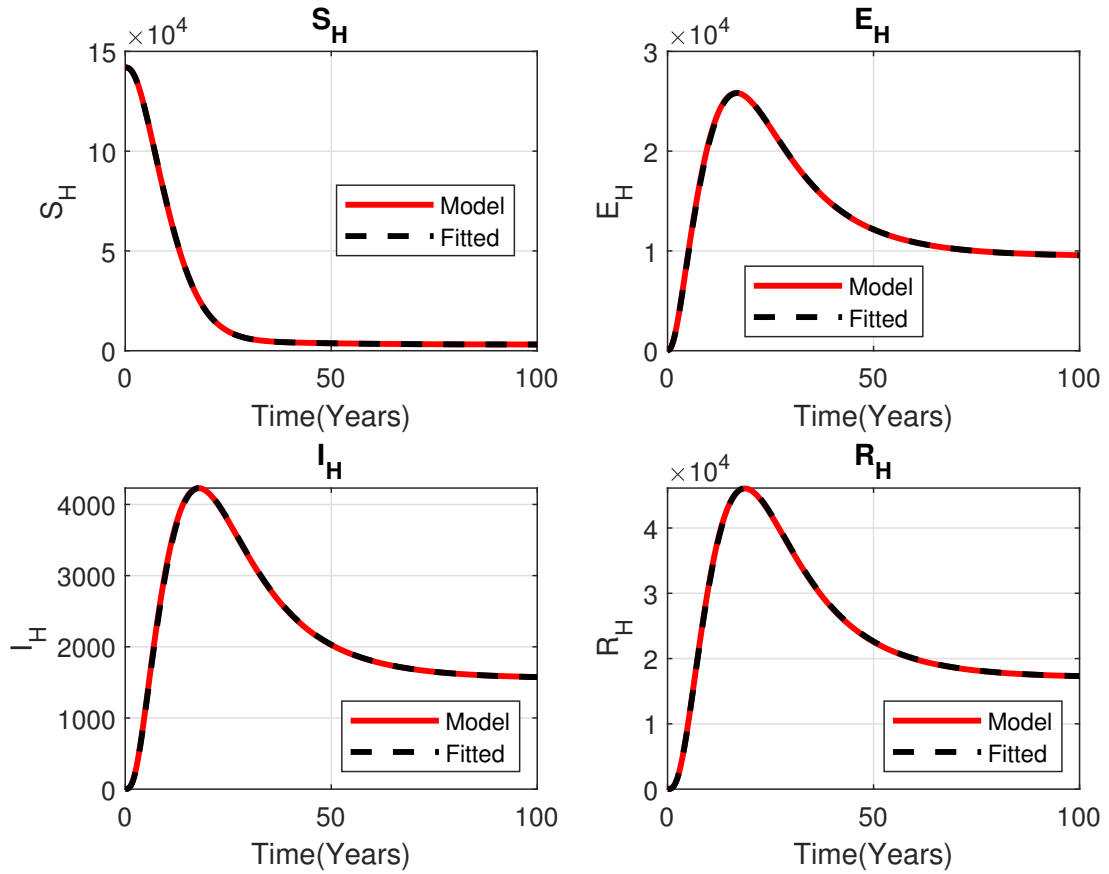


Figure 19: Comparison of the Model and Fitted Solution of model (3.5) for N_H

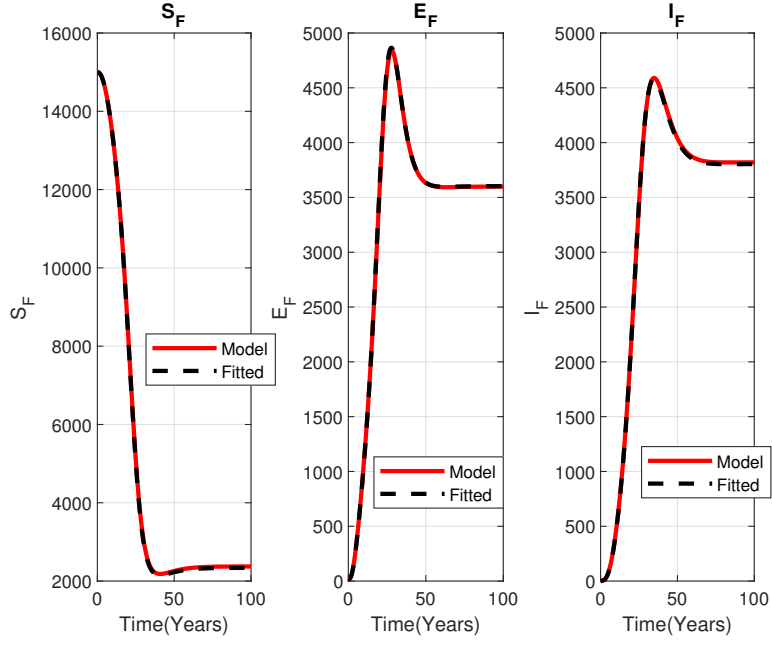


Figure 20: Comparison of the Model and Fitted Solution of model (3.5) for N_F

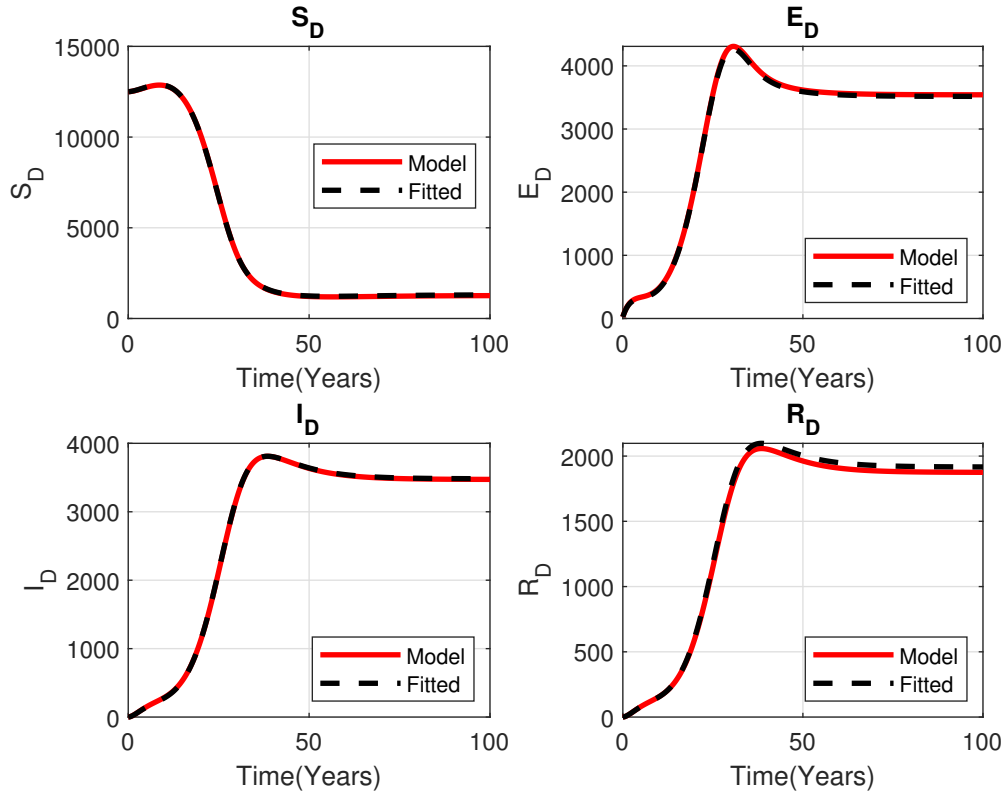


Figure 21: Comparison of the Model and Fitted Solution of model (3.5) for N_D .

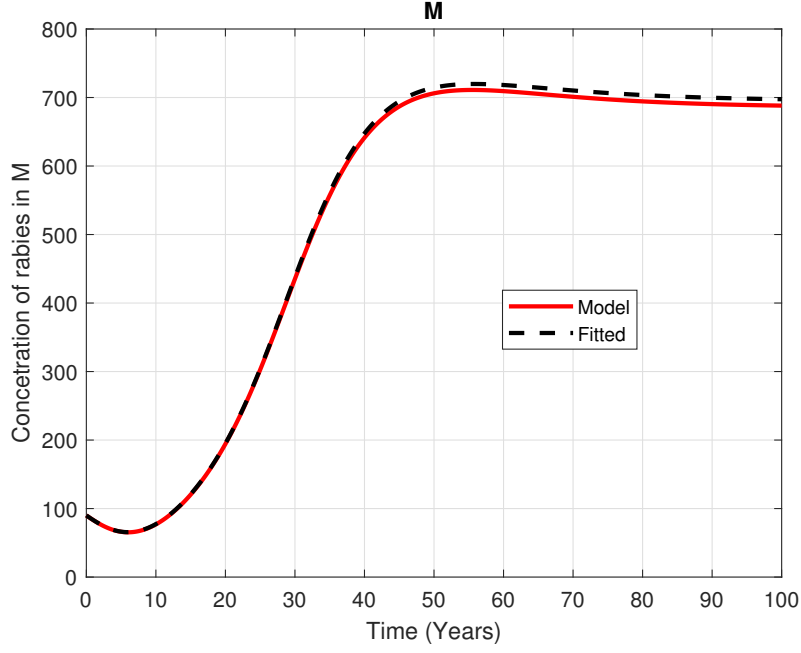


Figure 22: Comparison of the Model and Fitted Solution of model (3.5) for M

4.1.4 Local Sensitivity Analysis

In order to assess the sensitivity of the parameters within the rabies model in relation to the disease, a normalized sensitivity index was employed, as proposed by Zhang *et al.* (2011), Asamoah *et al.* (2017) and Bornaa *et al.* (2020) given by:

$$\gamma_{\mathcal{P}_i}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \mathcal{P}_i} \times \frac{\mathcal{P}_i}{\mathcal{R}_0} \quad (4.6)$$

Where $\mathcal{R}_0$ denotes the basic reproduction number for rabies, $\mathcal{P}_i$ signifies the parameters applied within the rabies model were derived using Equation (4.6) as follows:

$$\left\{ \begin{array}{ll} \gamma_{\gamma_1}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \gamma_1} \times \frac{\gamma_1}{\mathcal{R}_0} & = -0.105552 \\ \gamma_{\gamma_2}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \gamma_2} \times \frac{\gamma_2}{\mathcal{R}_0} & = -0.056998 \\ \gamma_{\kappa_1}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \kappa_1} \times \frac{\kappa_1}{\mathcal{R}_0} & = +0.891720 \\ \gamma_{\kappa_2}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \kappa_2} \times \frac{\kappa_2}{\mathcal{R}_0} & = +0.051422 \\ \gamma_{\mu_2}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \mu_2} \times \frac{\mu_2}{\mathcal{R}_0} & = -1.616021 \\ \gamma_{\mu_3}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \mu_3} \times \frac{\mu_3}{\mathcal{R}_0} & = -0.105358 \end{array} \right. \quad (4.7)$$

Thus, using Equation (4.6) and parameter values in Table 5, we obtain sensitivity indexes for each parameter as shown in Table 7.

Table 7: Sensitivity indexes for $\mathcal{R}_0$ parameters

Parameter	sensitivity index	Parameter	sensitivity index
γ_1	-0.105552	ψ_1	+0.051422
γ_2	-0.056998	ψ_2	+0.005436
κ_1	+0.897120	κ_2	+0.051422
μ_2	-1.616021	ρ_1	-0.046747
μ_3	-0.105358	ρ_2	-0.004832
σ_2	-0.540654	σ_3	-0.05144
θ_2	+0.941420	θ_3	+0.056858

The results presented in Table 7 clearly indicate that parameters ψ_1 , ψ_2 , κ_1 , and κ_2 , which are associated with the rabies model (3.5), positively influence the value of $\mathcal{R}_0$ when increased. In contrast, an increase in parameters γ_1 , γ_2 , μ_2 , μ_3 , ρ_1 , ρ_2 , σ_2 , and σ_3 is linked to a reduction in the value of $\mathcal{R}_0$. Specifically, a 20% increase in any of ψ_1 , ψ_2 , κ_1 , or κ_2 results in a proportional 20% increase in the value of $\mathcal{R}_0$. Conversely, increasing any of γ_1 , γ_2 , μ_2 , μ_3 , ρ_1 , ρ_2 , σ_2 , or σ_3 results in a comparatively smaller increase in $\mathcal{R}_0$.

Furthermore, the analysis suggests that ψ_1 , ψ_2 , κ_1 , and κ_2 have a positive influence on $\mathcal{R}_0$. This finding implies that intervention strategies significantly impact the prevalence of rabies within the population examined.

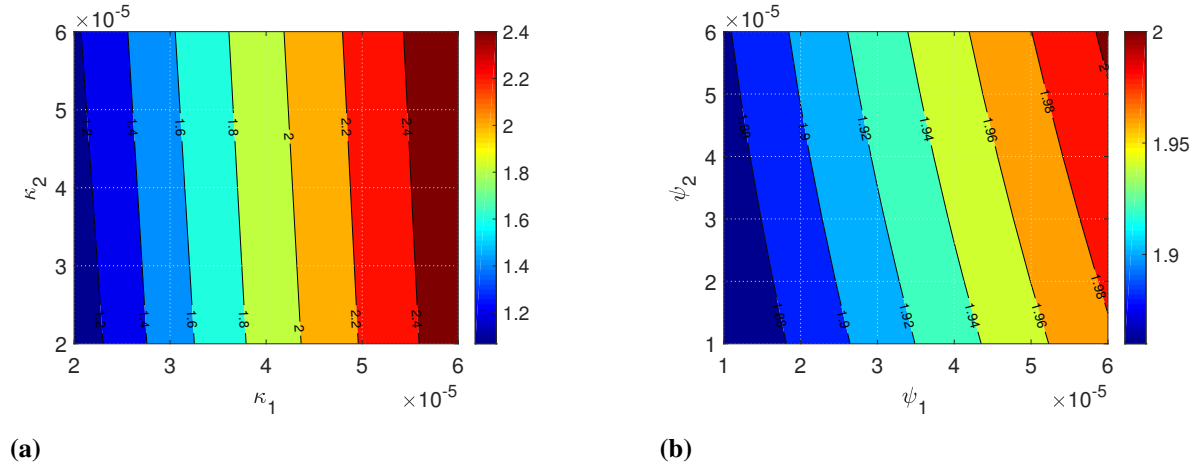


Figure 23: Effect of (a) κ_1 , κ_2 and (b) ψ_1 , ψ_2 with respect to $\mathcal{R}_0$

4.1.5 Global Sensitivity Analysis (GSA)

The Global Sensitivity Analysis (GSA) for modeling uncertainty and parameter estimation of rabies transmission for model (3.5) was conducted using Latin Hypercube Sampling (LHS) and the Partial Rank Correlation Coefficient (PRCC). The goal was to identify the key factors influencing the transmission of a new infection under a specific intervention, following the approach outlined in Organization (2013). This method involved using LHS to sample parameters that contribute to the variable $\mathcal{R}_0$ and calculating the PRCC. If X_i is the sampled input parameter, and Z_i is the model output corresponding to X_i , with means $\bar{X}$ and $\bar{Z}$ respectively, then the PRCC value r of X_i and Z_i was given by:

$$r = \frac{\text{Cov}(X_i, Z_i)}{\sqrt{\text{Var}(X_i)\text{Var}(Z_i)}} = \frac{\sum_{i=1}^N (X_i - \bar{X})(Z_i - \bar{Z})}{\sqrt{\sum_{i=1}^N (X_i - \bar{X})^2 \sum_{i=1}^N (Z_i - \bar{Z})^2}}. \quad (4.8)$$

for these parameters, as defined in equations (4.8) through (4.12). A total of 1,000 simulations were performed for each LHS run. Parameter sampling was based on a uniform distribution, treating the model parameters as input variables and $\mathcal{R}_0$ as the output variable. The PRCC values range from -1 to $+1$, where values closer to these extremes indicate stronger correlations on $\mathcal{R}_0$. If $p > 0.05$, it is assumed that a parameter is not statistically significant for $\mathcal{R}_0$. Positive PRCC values denote positive correlations, while negative values signify negative correlations. Values within the range of -0.2 to $+0.2$ represent weak or negligible correlations. The PRCC values greater than $+0.3$ or less than -0.3 indicate strong positive or strong negative correlations, respectively. Furthermore, values in the range of $0.2 < \text{PRCC} < 0.3$ or $-0.3 < \text{PRCC} < -0.2$ indicate moderate positive or moderate negative correlations.

The PRCC was calculated between a parameter θ_i and the output Y , while controlling for the effects of other variables Z . The mathematical formulation was as follows:

$$Y = f(\theta_1, \theta_2, \dots, \theta_n), \quad (4.9)$$

where Y is the model output, and θ_i are uncertain parameters.

LHS generates a sample matrix P :

$$P = \begin{bmatrix} \theta_1^1 & \theta_2^1 & \dots & \theta_n^1 \\ \theta_1^2 & \theta_2^2 & \dots & \theta_n^2 \\ \vdots & \vdots & \ddots & \vdots \\ \theta_1^N & \theta_2^N & \dots & \theta_n^N \end{bmatrix}, \quad (4.10)$$

where each θ_i^j is sampled as:

$$\theta_i^j = F_i^{-1} \left(\frac{r^j + U^j}{N} \right), \quad j = 1, 2, \dots, N. \quad (4.11)$$

For each parameter set P^j , evaluate:

$$Y^j = f(\theta_1^j, \theta_2^j, \dots, \theta_n^j). \quad (4.12)$$

A high $|\text{PRCC}_i|$ indicates strong sensitivity of Y to θ_i .

Using the baseline values outlined in Table 5, the PRCC and LHS values were calculated for each parameter. These values were subsequently plotted against the model output to evaluate their impact, as shown in Figures 24 through 27.

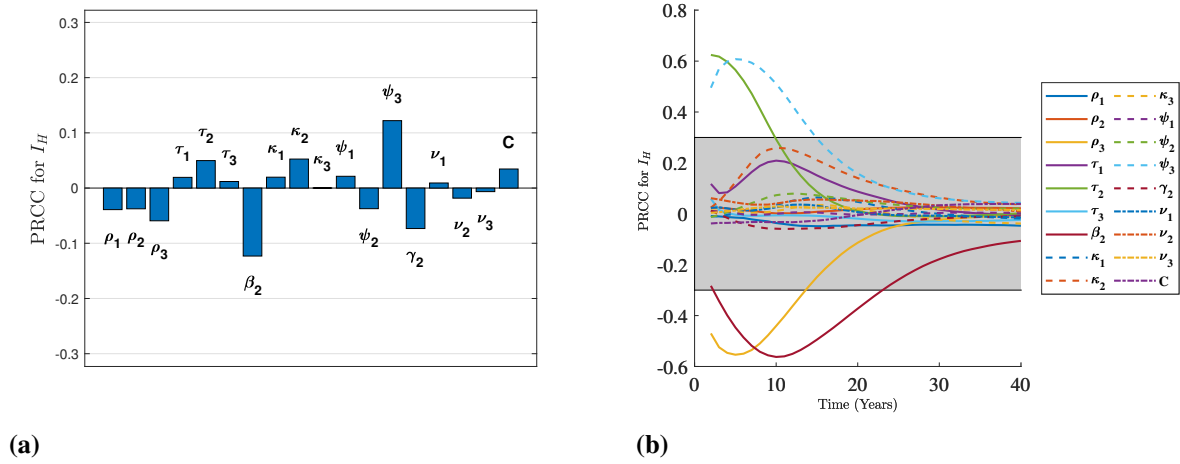
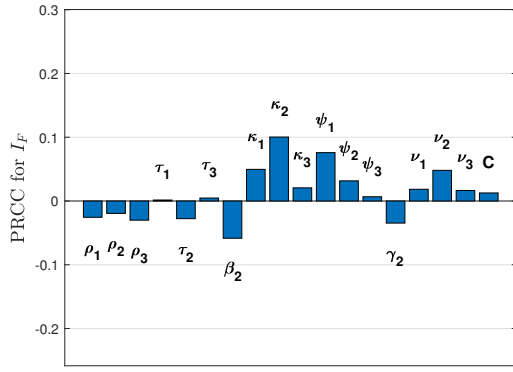
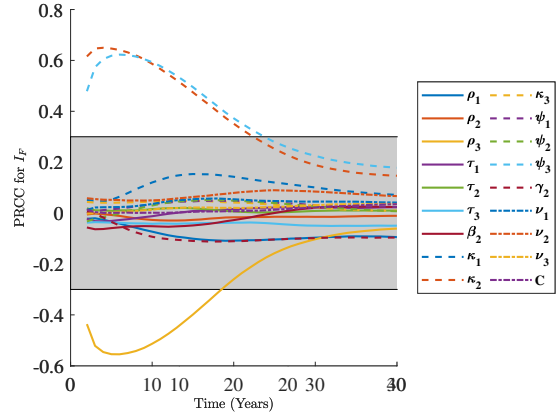


Figure 24: Sensitivity analysis of the model output I_H , showing (a) the time-invariant, and (b) the time-variant profile across the simulation period

The results shown in Fig. 24 indicate that the contact rates τ_1 , τ_2 , and τ_3 between susceptible humans and infectious sources are positively correlated with the number of infected humans (I_H). This correlation suggests that increases in these contact rates by 10% contribute to a higher incidence of human rabies infections. Additionally, factors such as the viral shedding rate (ν_1) and the environmental concentration of the rabies virus (C) play a significant role in maintaining environmental contamination, which facilitates indirect transmission of the virus. In contrast, deterrent parameters ρ_1 , ρ_2 , and ρ_3 , along with the human and domestic dog recovery rate (β_2 and γ_2) respectively, are negatively correlated with I_H , as indicated by their low PRCC values. This implies that increasing these parameters leads to a reduction in the number of I_H . Moreover, host-to-host transmission rates specifically κ_1 , κ_2 , κ_3 , ψ_1 , and ψ_3 have a positive effect on I_H , with the greatest impact coming from free-ranging dogs and domestic dogs (κ_2) as well as contact rate of domestic dogs and the environment containing rabies virus (ψ_3). These findings highlight the essential roles of both direct and indirect transmission pathways in influencing the dynamics of human rabies infections.



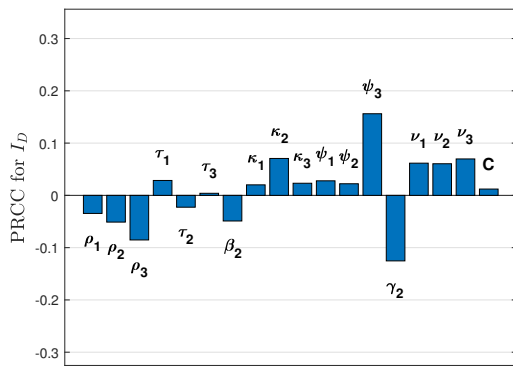
(a)



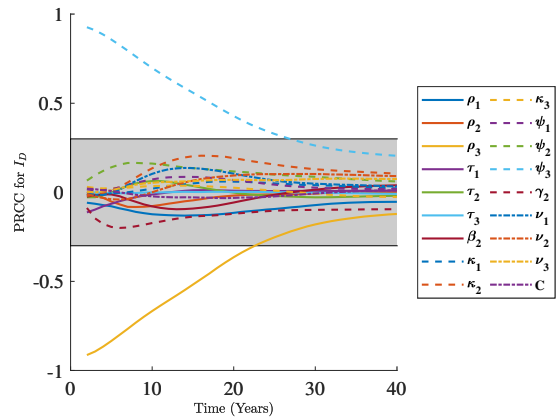
(b)

Figure 25: Sensitivity analysis of the model output I_F , showing (a) the time-invariant, and (b) the time-variant profile across the simulation period

The results shown in Fig. 25 shows that the contact rates κ_1 , κ_2 , and κ_3 between susceptible free range dogs and infectious sources are positively correlated with the number of infected free range dogs (I_F). This correlation suggests that increases in these contact rates by 10% contribute to a higher incidence of Free range rabies infections. Additionally, factors such as the viral shedding rate (ν_1 , ν_2 , ν_3) and the environmental concentration of the rabies virus (C) play a significant role in maintaining environmental contamination, which facilitates indirect transmission of the virus. In contrast, deterrent parameters ρ_1 , ρ_2 , and ρ_3 , along with the recovery rate domestic dog (γ_2) respectively, are negatively correlated with I_F , as indicated by their low PRCC values. This implies that increasing these parameters leads to a reduction in the number of I_F . Moreover, host-to-host transmission rates specifically ψ_1 , ψ_2 and ψ_3 have a positive effect on I_F , with the greatest impact coming from domestic dogs and the environment containing rabies virus (ψ_3).



(a)



(b)

Figure 26: Sensitivity analysis of the model output I_D , showing (a) the time-invariant, and (b) the time-variant profile across the simulation period

The results shown in Fig. 26 indicate that the contact rates ψ_1 , ψ_2 , and ψ_3 between susceptible domestic dogs and infectious sources are positively correlated with the number of infected domestic dogs (I_D). This correlation suggests that a 10% increase in these contact rates contributes to a higher incidence among domestic dogs. Additionally, factors such as the viral shedding rates (v_1 , v_2 , v_3) and the environmental concentration of the rabies virus (C) significantly contribute to environmental contamination, which facilitates the indirect transmission of the virus. In contrast, the deterrent parameters ρ_1 , ρ_2 , and ρ_3 , as well as the recovery rate (γ_2) for domestic dogs, are negatively correlated with I_D , as evidenced by their negative partial rank correlation coefficient (PRCC) values. This implies that increases in these parameters lead to a reduction in the number of infected domestic dogs (I_D). Moreover, the host-to-host transmission rates, specifically κ_1 , κ_2 , and κ_3 , have a positive effect on I_D , with the most significant impact stemming from domestic dogs and the environmental presence of the rabies virus (ψ_3).

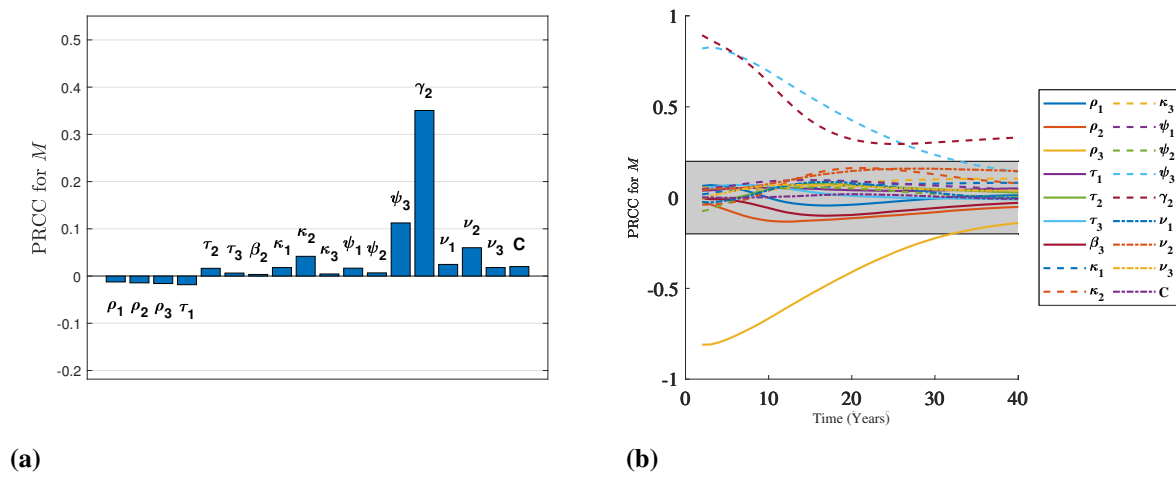


Figure 27: Sensitivity analysis of the model output M , showing (a) the time-invariant, and (b) the time-variant profile across the simulation period

The results shown in Fig. 27 indicate that host-to-host transmission parameters specifically κ_1 , κ_2 , κ_3 , ψ_1 , ψ_2 , and ψ_3 have a positive impact on the environmental concentration of the rabies virus (M). This positive correlation suggests that an increase in these transmission rates leads to higher levels of the rabies virus in the environment, which in turn heightens the risk of indirect transmission among humans and dogs. Additionally, the rates of viral shedding (v_1 , v_2 , v_3) and the environmental concentration parameter (C) significantly contribute to environmental contamination, further promoting the indirect spread of the virus. In contrast, the deterrent parameters ρ_1 , ρ_2 , and ρ_3 show negative partial rank correlation coefficients (PRCCs), suggesting that higher values of these parameters are linked to a reduction in the environmental burden of the rabies virus.

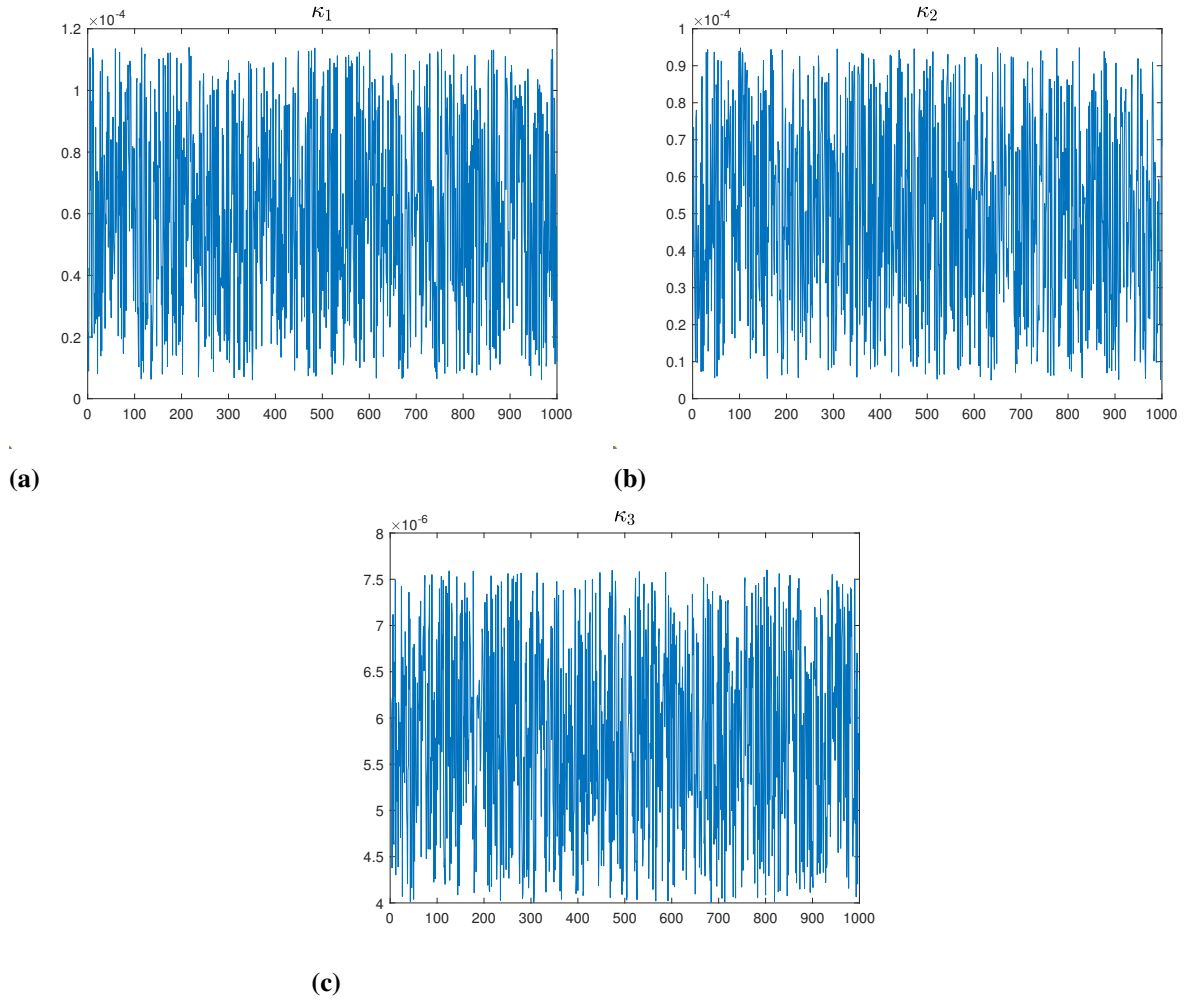


Figure 28: Probability distribution of sampled input parameters for N_F

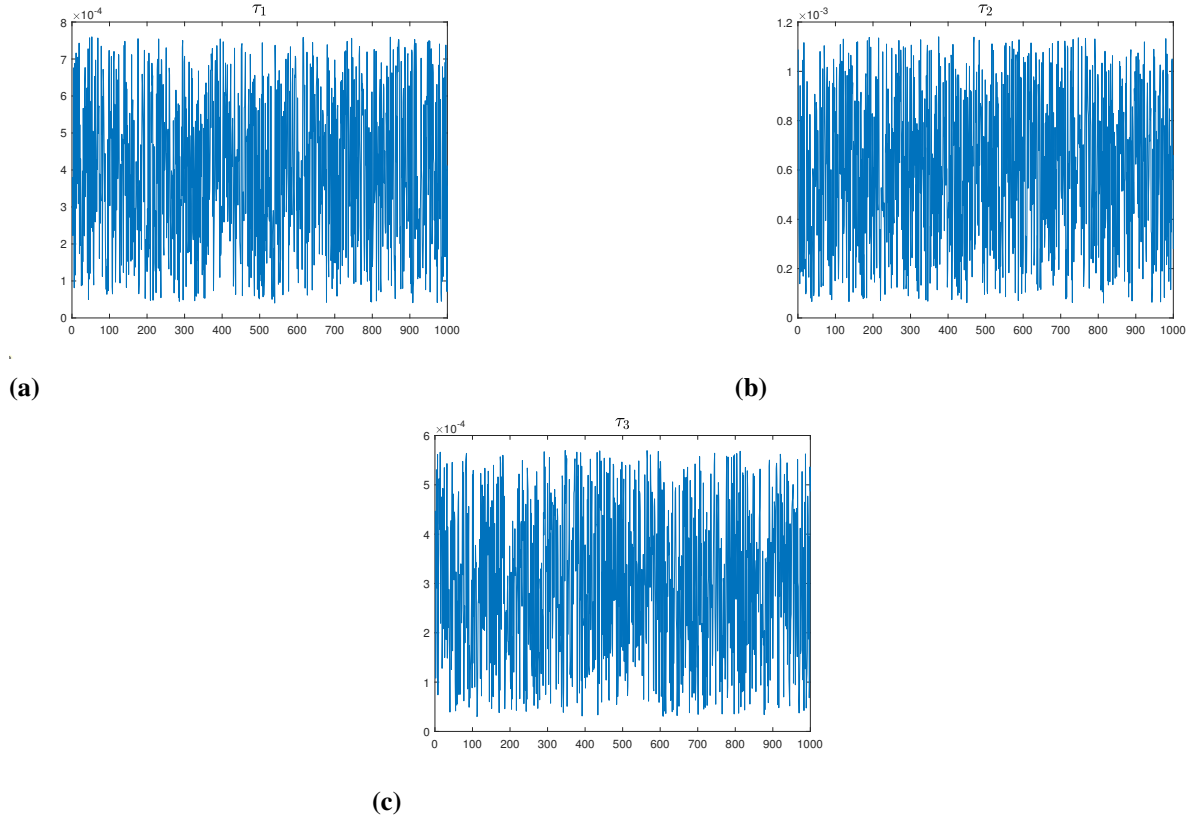


Figure 29: Probability distribution of sampled input parameters for N_H .

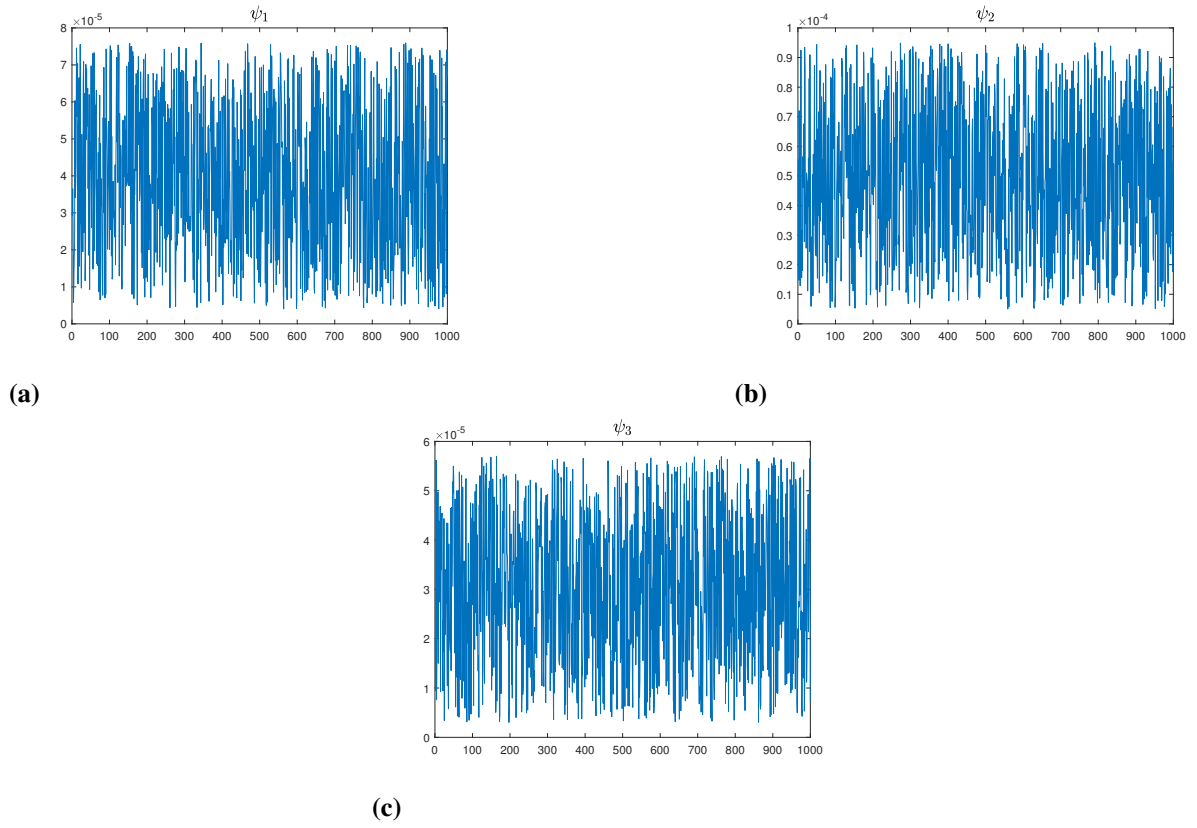


Figure 30: Probability distribution of sampled input parameters for N_D .

4.2 Numerical Results for Deterministic Model

In this section, a model system (3.5) is solved numerically using ODE45 method implemented in MATLAB software. The goal is to showcase the analytical results presented in the previous sections. Parameters used for numerical simulations are presented in Table (5) together with the following initial conditions: $S_H(0) = 36000$, $E_H(0) = 40$, $I_H(0) = 0$, $R_H(0) = 0$, $S_D(0) = 8750$, $E_D(0) = 25$, $I_D(0) = 0$, $R_D(0) = 0$, $S_F(0) = 6720$, $E_F(0) = 20$, $I_F(0) = 0$, $M(0) = 90$. In this context, all trajectories that originate from the region of attraction converge toward the rabies transmission-persistence $\mathcal{E}$ (Fig. 31) and (Fig. 32).

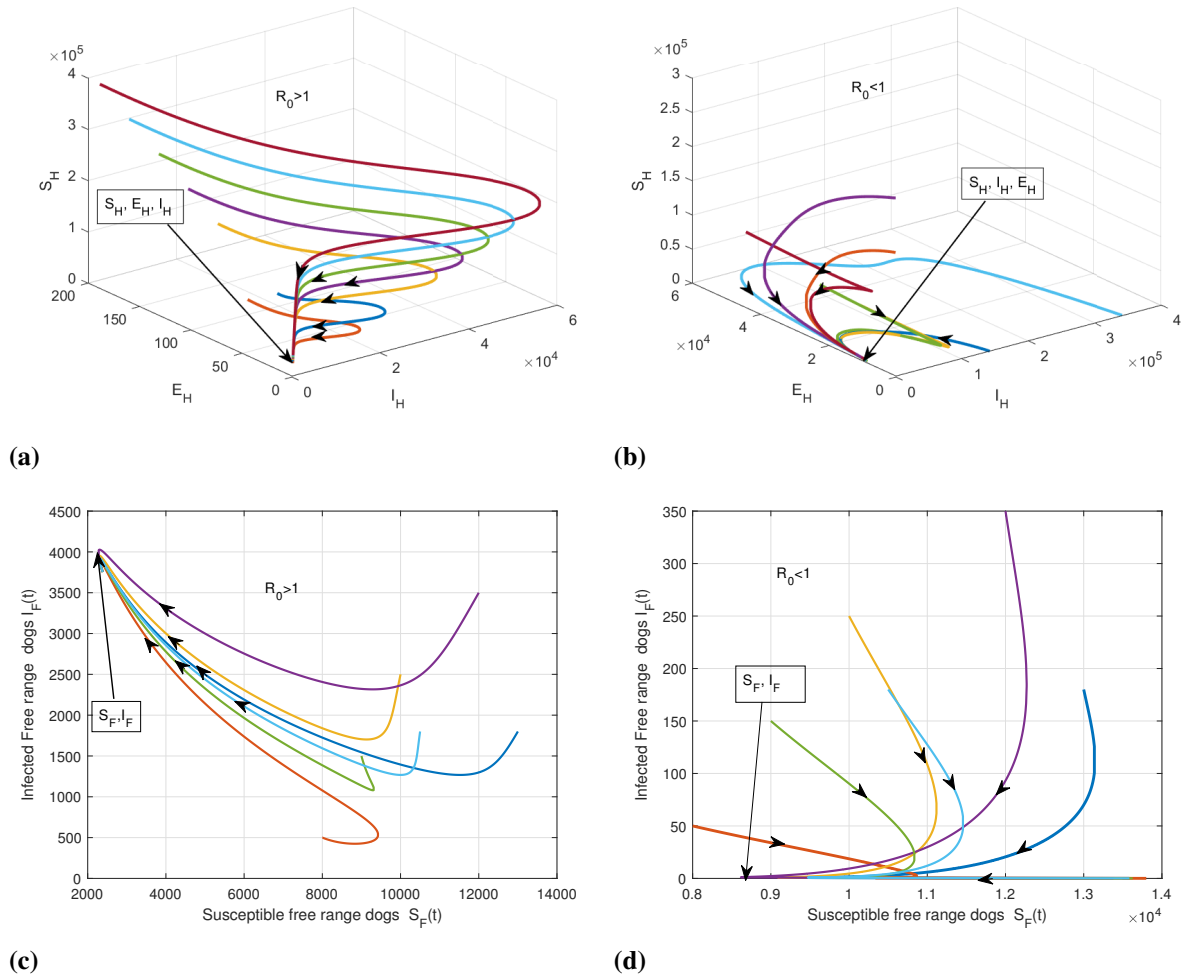


Figure 31: Convergence to the equilibrium point for varying initial values when $R_0 < 1$ and when $R_0 > 1$

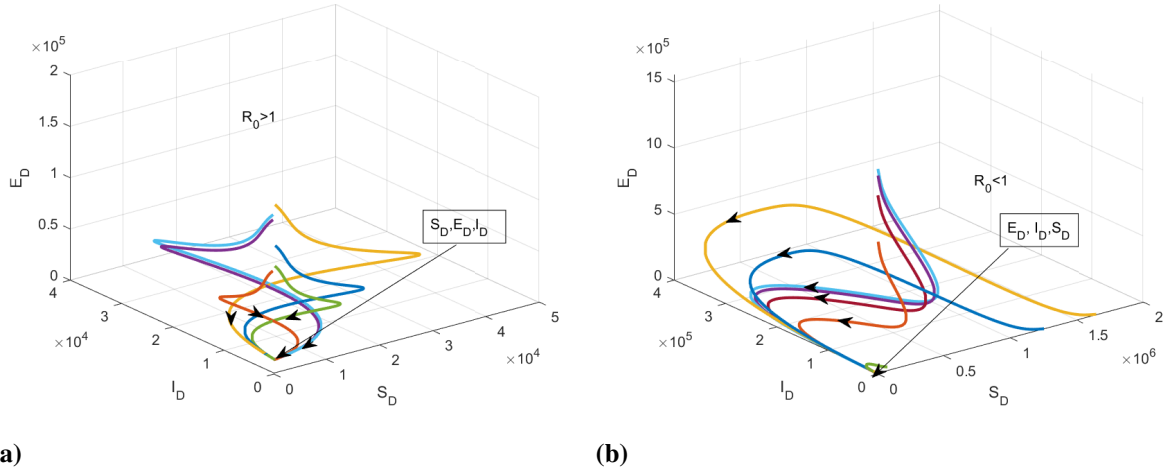


Figure 32: Convergence to the equilibrium point for varying initial values when $R_0 < 1$ and when $R_0 > 1$

Figure (33) (a) depict a rapid decrease in S_H within the first 50 years due to the natural deaths and Infections resulted from I_F , I_D , and M . The decline stabilizes to non-negative constant number after 50 years , implying that S_H does not finish due to continuous recruitment and treatment. The results in Fig. 33 (b) show the increase in number of individuals resulting into the development of infectious individual , leading into the increase on the number of E_H , I_H and R_H within the first 50 years. The results in Fig. 33 (a) coincides with the results in Fig. 33 (b) in the sense that more susceptible individuals get infections. Furthermore the results in Fig. 33 (b) indicates more number of exposed individuals in relation to infected individual because exposed individuals takes proportionality large incubation period before exposing the symptoms. This trend can be attributed to the fact that a greater number of exposed and infected individuals receive immediate PEP following contact with a rabid animal.

Figure (33)(c) illustrates a declining trend in the count of S_F individuals, which eventually stabilizes after a 50-year duration. In contrast, Figure (33) (d) depicts an increase in the number of infected and exposed individuals, which then levels off after 50 years. Notably, the number of infected individuals surpasses that of the exposed ones because a significant portion of free-range dogs does not receive PEP after being exposed to rabid animals.

Figure (33) (e) portrays a rapid decline in the population of S_D to rabies infection, which eventually levels off after a 60-year time frame. In contrast, Figure (33) (f) illustrates an initial increase followed by a decline in the numbers of exposed, infected, and recovered individuals within the first 50 years. This pattern suggests that as the counts of infected, exposed, and recovered individuals rise, the number of susceptible individuals decreases. Moreover, the rate of increase in exposed domestic dogs is lower compared to those in the exposed or infected categories. This phenomenon can be attributed to the fact that a portion of exposed individuals promptly receive PEP following contact with a rabid animal.

Figure (34), depicts that the presence of the rabies virus in the environment increases as the shedding rate rises, eventually reaching a stable level after 40 years. This stabilization of rabies could be indicative of the environment's influence on the transmission dynamics of the rabies disease.

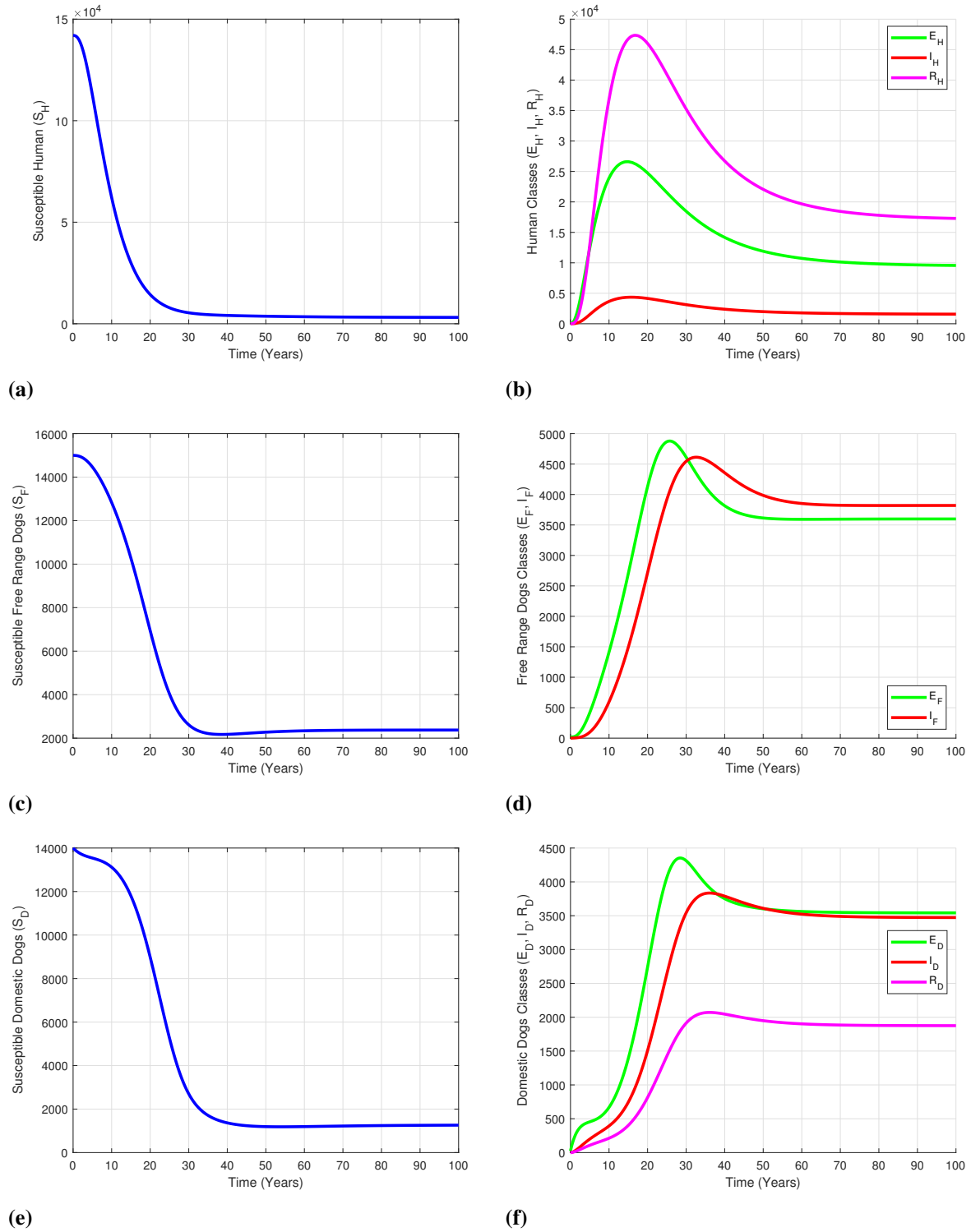


Figure 33: Population dynamics for all Human , Free range and Domestic dogs

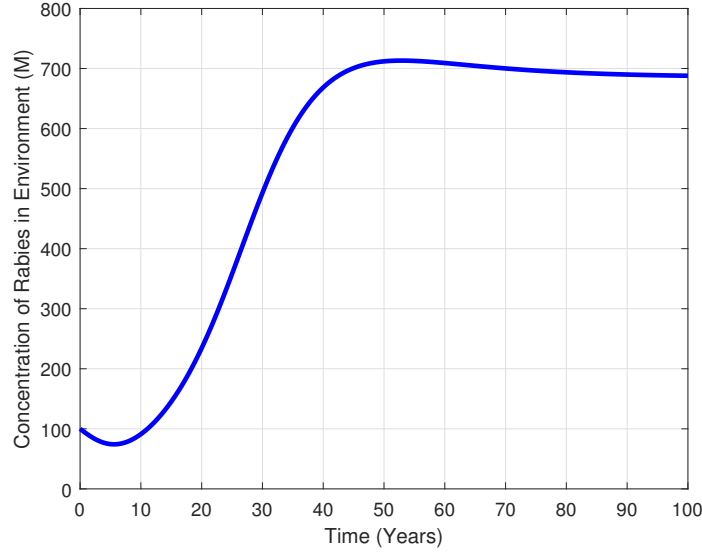


Figure 34: Population dynamics rabies concentration in M

4.2.1 Effect of Varying the Most Sensitive Parameters

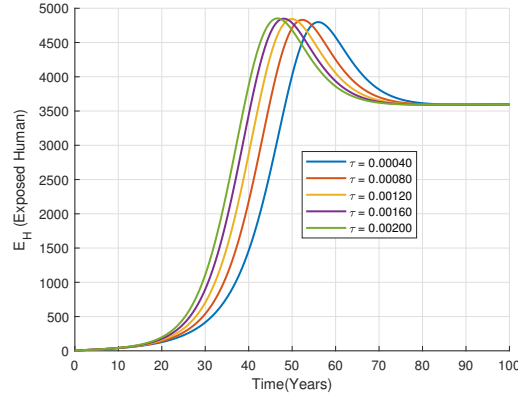
The contact rates β_{ij} govern rabies transmission, with the infection rate given by (4.13)

$$\lambda_i = \sum_j \beta_{ij} I_j, \quad (4.13)$$

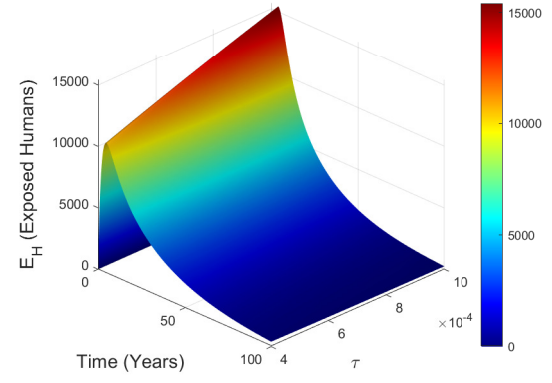
where I_j is the infected population in compartment j . Higher β_{ij} increases λ_i , accelerating disease spread. Mitigation strategies include vaccination (V), population control ($N_i \rightarrow \alpha N_i$, $0 < \alpha < 1$), and contact reduction ($\beta_{ij} \rightarrow \delta \beta_{ij}$, $0 < \delta < 1$). We now examine the impact of β_{ij} between infectious agents and (i) Infected humans, (ii) Infected domestic dogs, and (iii) free-range dogs.

(i) Impact of contact rate on susceptible human

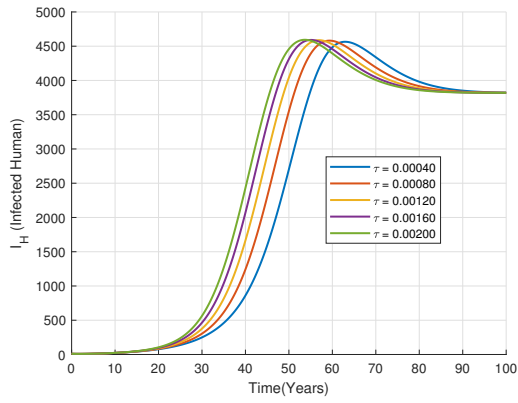
The results presented in Figures (35) (a)-(c) indicate that contact rates (τ) significantly affect the transmission dynamics among susceptible humans. It was noted that the number of exposed individuals (E_H) and infected individuals (I_H) increases as the contact rate (τ) rises, ultimately reaching an equilibrium state after 50 years. This finding underscores the critical importance of education and awareness regarding the interactions between susceptible humans and sources of rabies. Such knowledge is essential for mitigating the transmission of rabies within the human population.



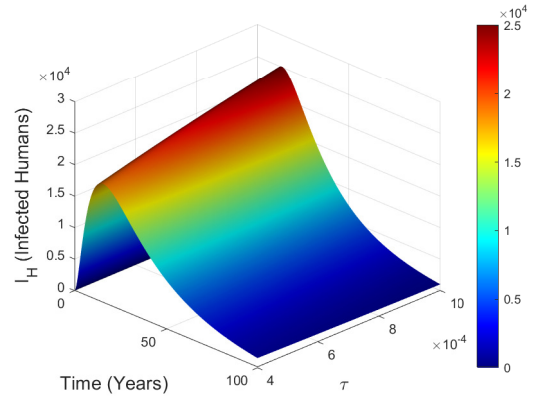
(a)



(b)



(c)

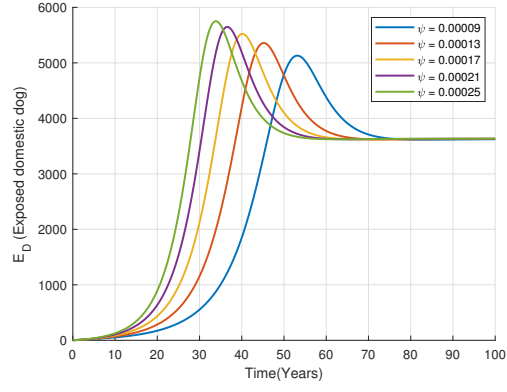


(d)

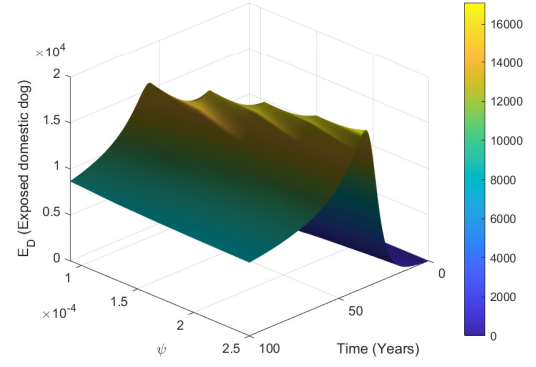
Figure 35: Simulation results of model (3.5) for (a) E_H and (b) I_H with respect to contact rate τ

(ii) Impact of contact rate on susceptible domestic dogs

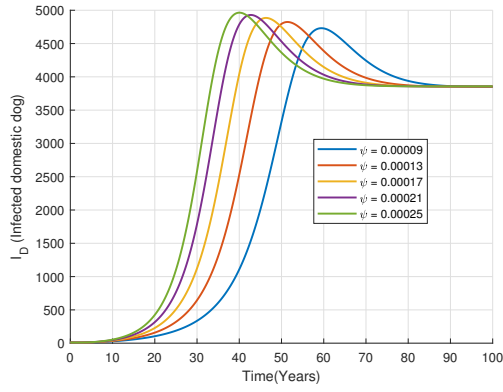
Based on the observations presented in Figure (36)(a)–(d), it is clear that as the contact rates ψ_1 , ψ_2 , and ψ_3 increase, the number of exposed and infected domestic dogs also rises. Notably, after the 50-year mark, the population of infected domestic dogs tends to stabilize. This trend highlights the critical importance of reducing contact between susceptible domestic dogs and those that are infected, as well as limiting interactions with free-ranging dogs and contaminated environmental reservoirs. Such measures are essential to curbing the long-term transmission dynamics of rabies within domestic dog populations.



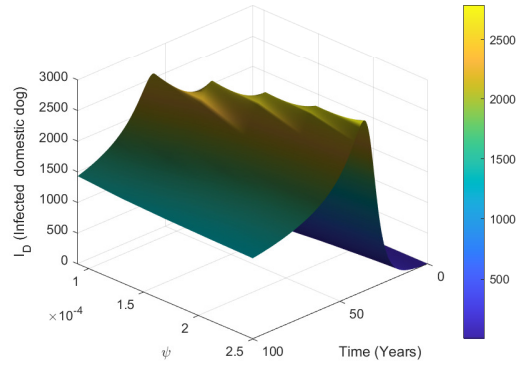
(a)



(b)



(c)



(d)

Figure 36: Simulation results of model (3.5) for (a) E_D and (a) I_D with respect to contact rate ψ

(iii) Impact of contact rate on free range dogs

Figure (37) (a) to (d) illustrates that an increase in the contact rate κ among free-range dogs leads to a higher number of exposed free-range dogs (E_H) and infected free-range dogs (I_H). This indicates that the absence of control measures results in a rise in both carriers and symptomatic infections.

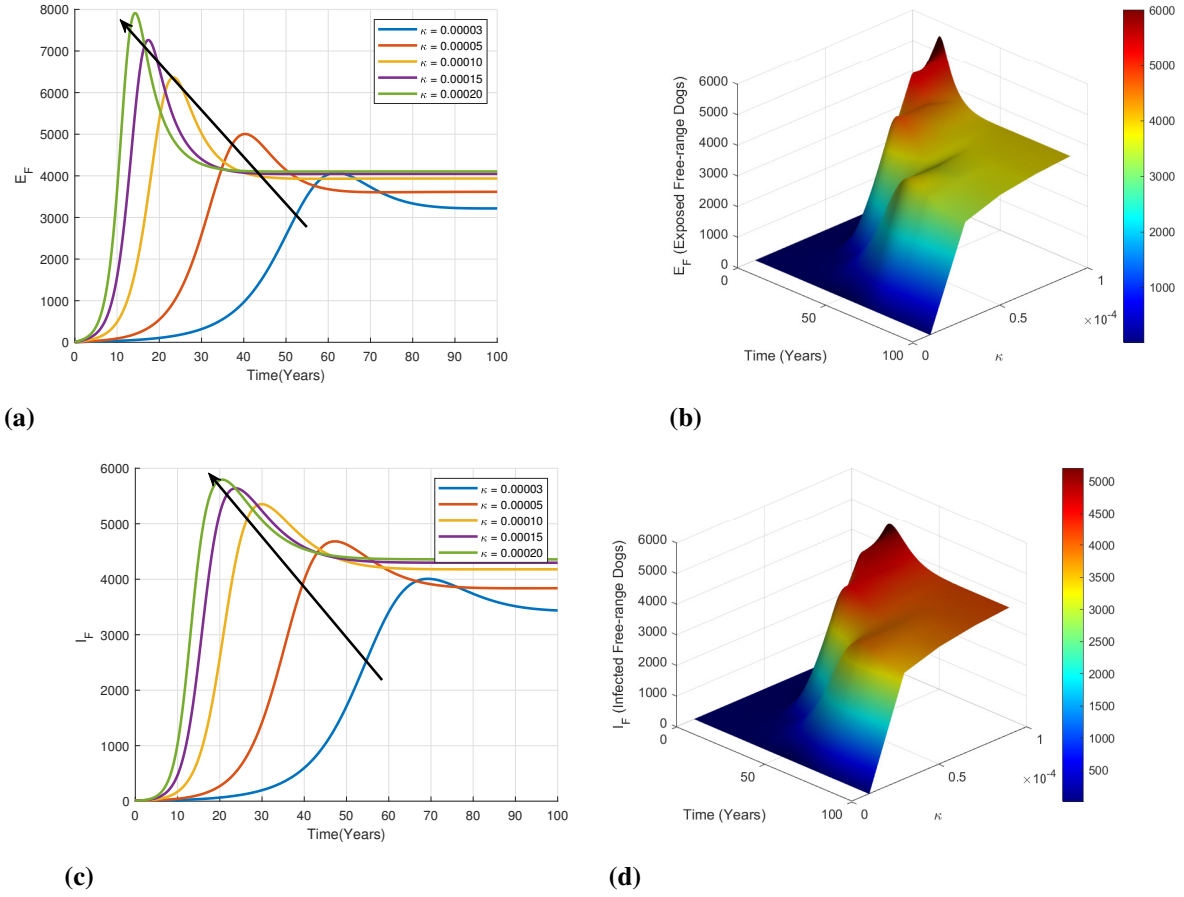


Figure 37: Simulation results of model (3.5) for (a) to (b) for E_F and (c) to (d) for I_F for with respect to contact rate κ

(iv) Effect of deterrent factor (ρ) on the spread of rabies among domestic dogs

Figure 38 indicates that a rise in deterrent factors results in a reduction in the number of domestic dogs that are exposed to or infected by rabies. This implies that deterrent factors are important in ensuring that domestic dog populations do not contract rabies.

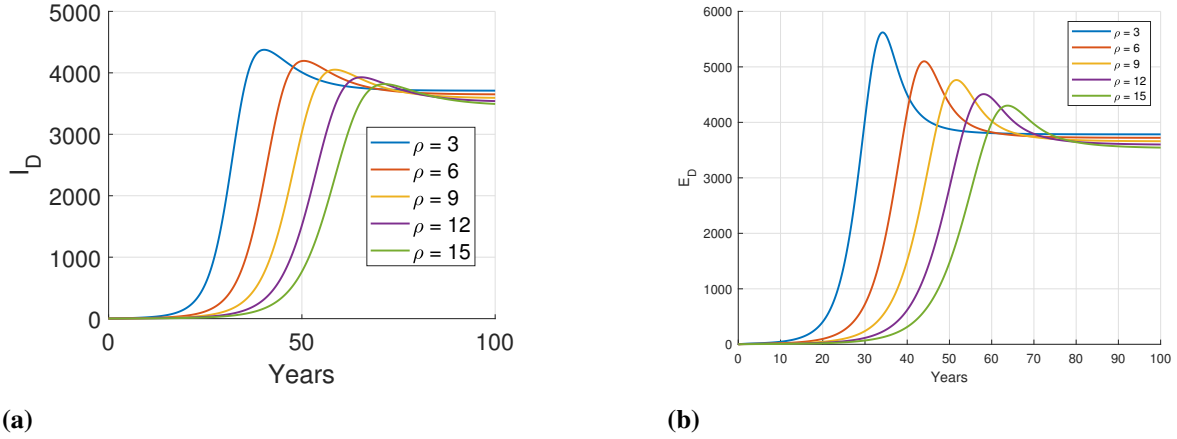


Figure 38: Impact of ρ on the transmission dynamics of rabies for N_D of model (3.5)

4.2.2 The Correlation Coefficients in Rabies Transmission Dynamics

The correlation coefficients quantify the relationships in rabies transmission dynamics. Using MATLAB, the correlation coefficient r from Equation (4.8) was computed and summarized in Table (8) and Fig. 39 through 40. based on Equation (4.14)

$$r = \begin{cases} 1 & \text{Perfect positive correlation.} \\ -1 & \text{Perfect negative correlation.} \\ 0 & \text{No correlation.} \\ 0 < r < 1 & \text{Positive correlation.} \\ -1 < r < 0 & \text{Negative correlation.} \end{cases} \quad (4.14)$$

It was observed that, there is a strong correlation $\text{corr}(I_F, I_D) = 0.97994$ indicates a near-perfect proportionality between infected free-range (I_F) and domestic dogs (I_D), highlighting their interdependence. Similarly, $\text{corr}(I_F, I_H) = 0.79184$ and $\text{corr}(I_D, I_H) = 0.75283$ confirm that human infections (I_H) are primarily driven by dog populations.

Moderate correlations exist between infected dogs and environmental factors, with $\text{corr}(I_D, M) = 0.56781$ and $\text{corr}(I_F, M) = 0.46526$, indicating the environment (M) as a contributing factor to rabies persistence. Additionally, $\text{corr}(I_H, M) = 0.4023$ suggests that environmental contamination plays a secondary role in human infections.

Table 8: Correlation Coefficients of Rabies Transmission Dynamics

Variables	Correlation Coefficient	Mathematical Relation
$\text{corr}(I_F, I_H)$	0.79184	$r_{I_F I_H} \Rightarrow I_F \propto I_H$
$\text{corr}(I_D, I_H)$	0.75283	$r_{I_D I_H} \Rightarrow I_D \propto I_H$
$\text{corr}(I_H, M)$	0.4023	$r_{I_H M} \Rightarrow I_H \propto M$
$\text{corr}(I_F, I_D)$	0.97994	$r_{I_F I_D} \Rightarrow I_F \propto I_D$
$\text{corr}(I_F, M)$	0.46526	$r_{I_F M} \Rightarrow I_F \propto M$
$\text{corr}(I_D, M)$	0.56781	$r_{I_D M} \Rightarrow I_D \propto M$

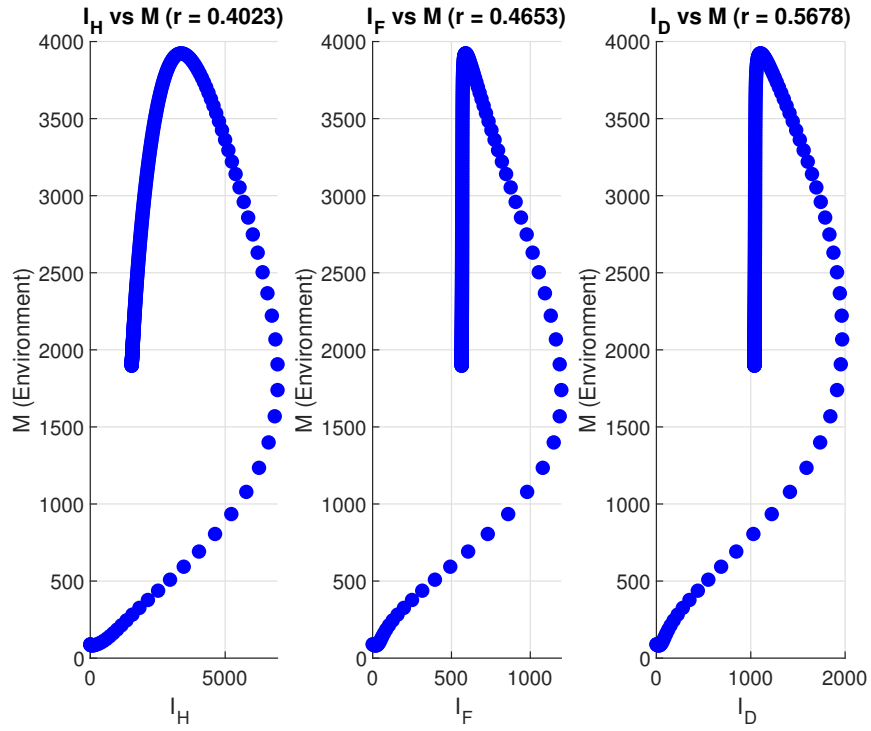


Figure 39: Correlation for I_H, I_F, I_D with M for rabies transmission

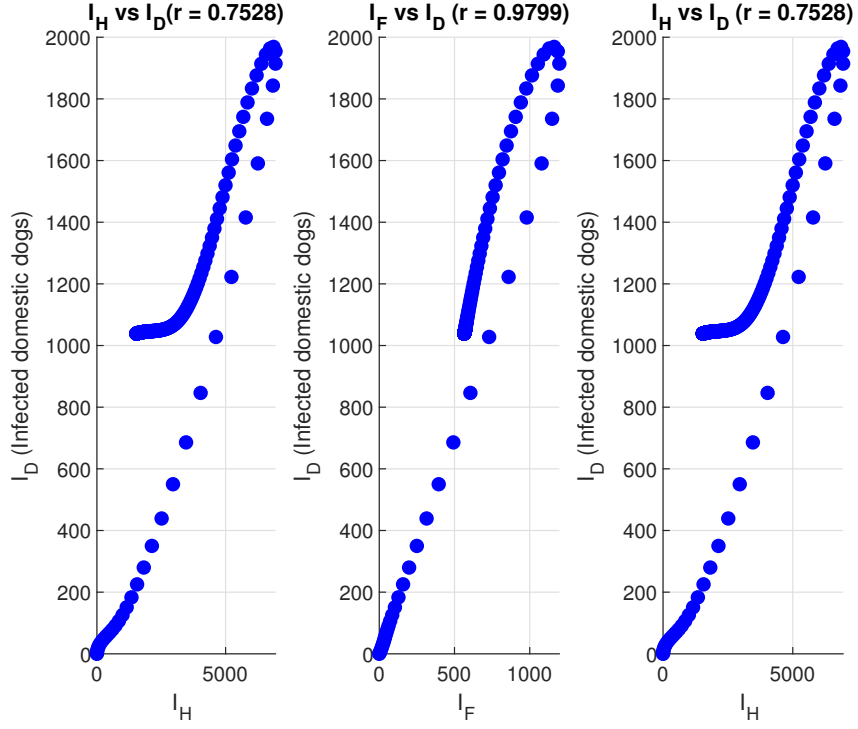


Figure 40: Correlation between I_H , I_F , I_D rabies transmission

4.3 Numerical Results for CTMC Stochastic Model

The simulation for CTMC Stochastic Model performed 10 000 random sample paths, with the results presented graphically alongside the corresponding deterministic numerical solutions for comparative analysis. To conduct the simulations, Euler's method and the Gillespie algorithm are utilized, applying the specified initial conditions $E_H(0) = 10$, $I_H(0) = 5$, $R_H(0) = 0$, $S_H(0) = \frac{\theta_1}{\mu_1} - (E_H(0) + I_H(0) + R_H(0))$, $E_F(0) = 20$, $I_F(0) = 0$, $S_F(0) = \frac{\theta_2}{\mu_2} - (E_F(0) + I_F(0))$, $E_D(0) = 40$, $I_D(0) = 5$, $R_D(0) = 0$, and $S_D(0) = \frac{\theta_3}{\mu_3} - (E_D(0) + I_D(0) + R_D(0))$.

Figures 41–43 illustrate the deterministic and stochastic transmission dynamics of rabies between human and dog populations.

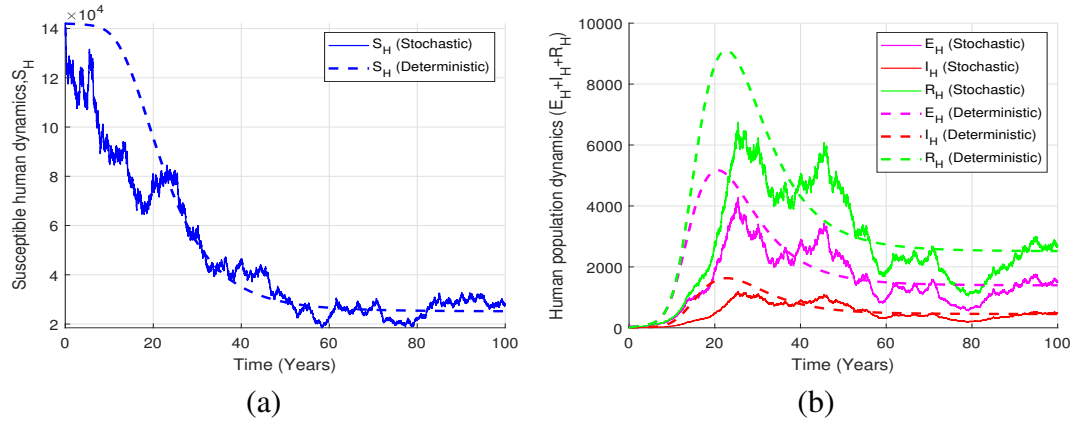


Figure 41: Stochastic and deterministic rabies transmission dynamics in humans

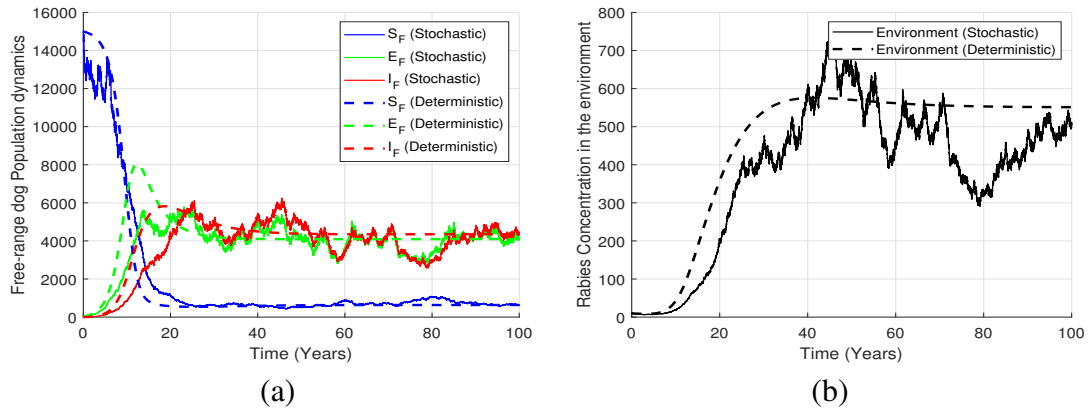


Figure 42: Stochastic and deterministic rabies transmission dynamics in (a) Free range dogs (b) Environment

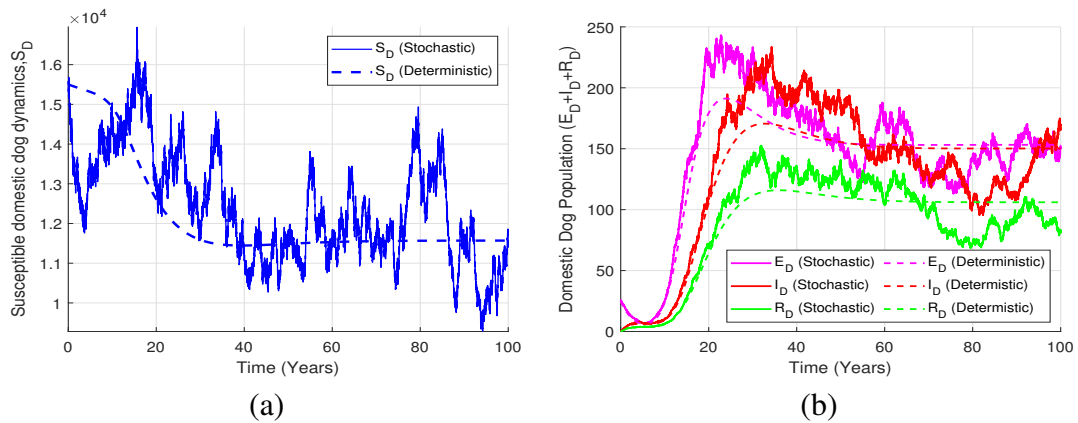


Figure 43: Stochastic and deterministic rabies transmission dynamics in Domestic dogs

Figure 41(b) demonstrates that the number of susceptible humans initially decreases stochastically over the first 40 years, eventually stabilizing to a variable, non-constant level. In contrast, Fig. 41(a) shows an

increase in stochastic fluctuations within the exposed, infected, and recovered human populations during this same period, followed by a decline that approaches a steady, non-zero value. The observed patterns of increase and decrease in the exposed, infected, and recovered populations likely reflect the impact of control interventions, such as the administration of PEP for individuals exposed to rabid animals. Fig. 42(a) illustrates that the number of susceptible free-range dogs initially declines as the population of exposed and infected free-range dogs increases stochastically over the first 20 years, eventually stabilizing at a variable, non-constant level. Meanwhile, Fig. 42(b) shows increasing stochastic fluctuations in the environmental rabies concentration, which subsequently stabilize toward a steady, non-zero level. Fig. 43(a) illustrates that the number of susceptible domestic dogs initially experiences periodic declines as the population of exposed, infected, and recovered domestic dogs in Fig. 43(b) stochastically increases over the first 20 years, eventually stabilizing at a variable, non-constant level.

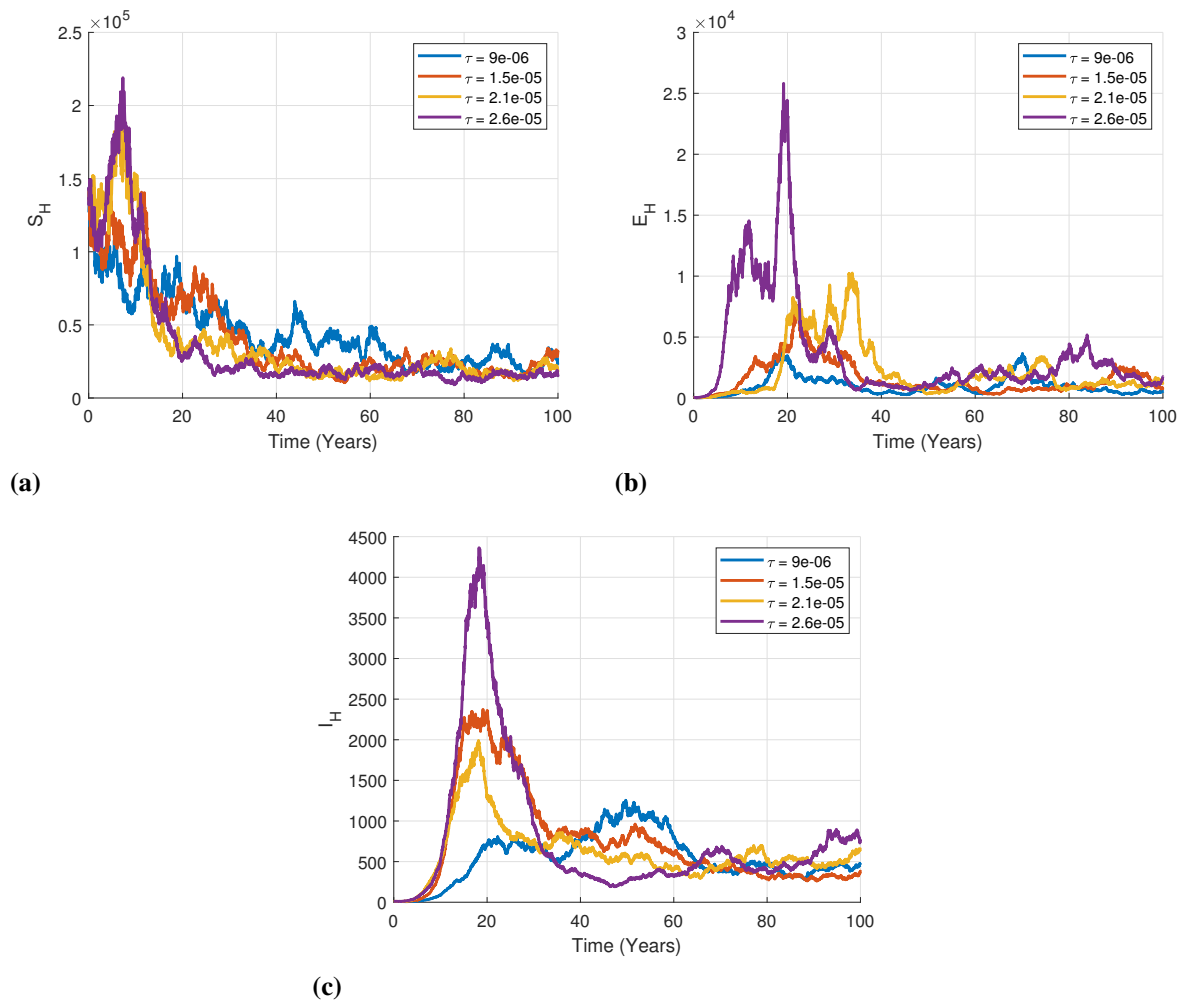


Figure 44: Stochastic trajectory of human due to impact of contact rate τ

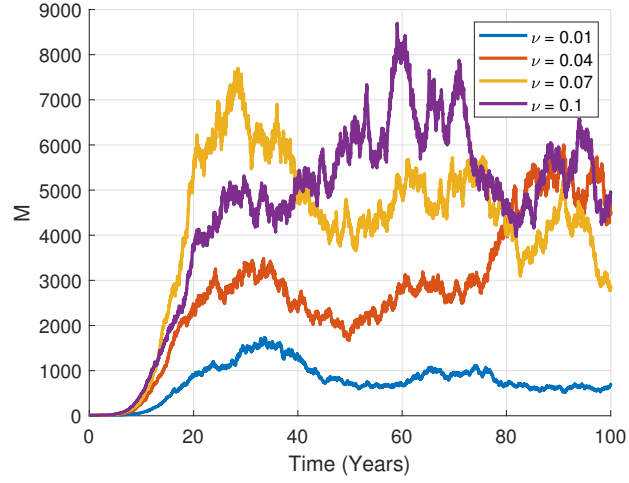
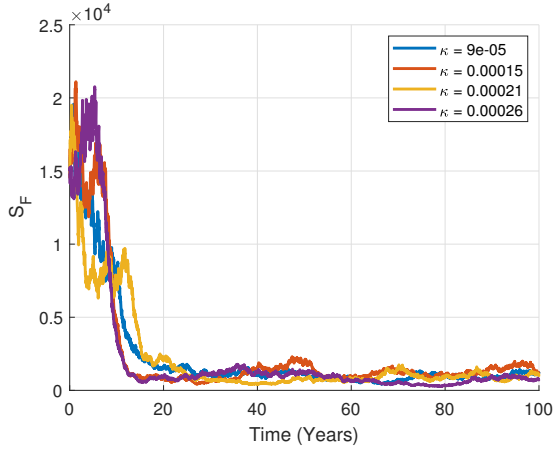
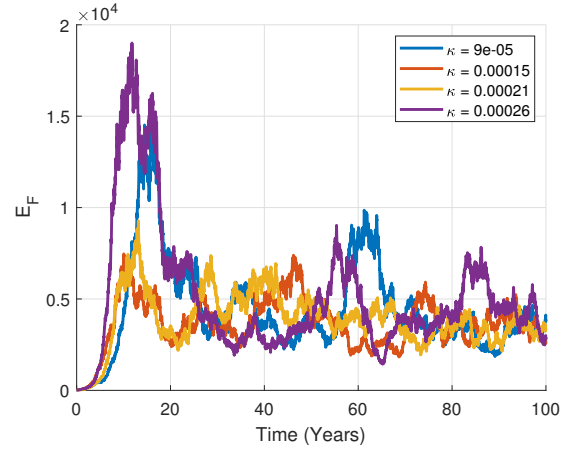


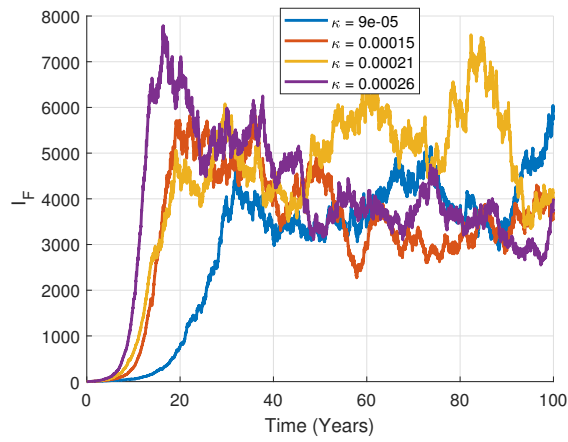
Figure 45: Stochastic trajectory of Environment due to impact of shedding rate ν



(a)



(b)



(c)

Figure 46: Stochastic trajectory of Free range dogs due to impact of contact rate κ

Figures (44) through (47) clearly demonstrate a stochastic increase in the number of infected individuals among humans, domestic animals, and free-range dogs, while there is a decrease in the number of susceptible individuals within the same populations. Additionally, there is a simultaneous stochastic increase in the concentration of rabies in the environment, attributed to variations in contact and shedding rates. These scenarios provide evidence that there is a possibility of rabies outbreak due to movement of free roaming dogs that may introduce new infection.

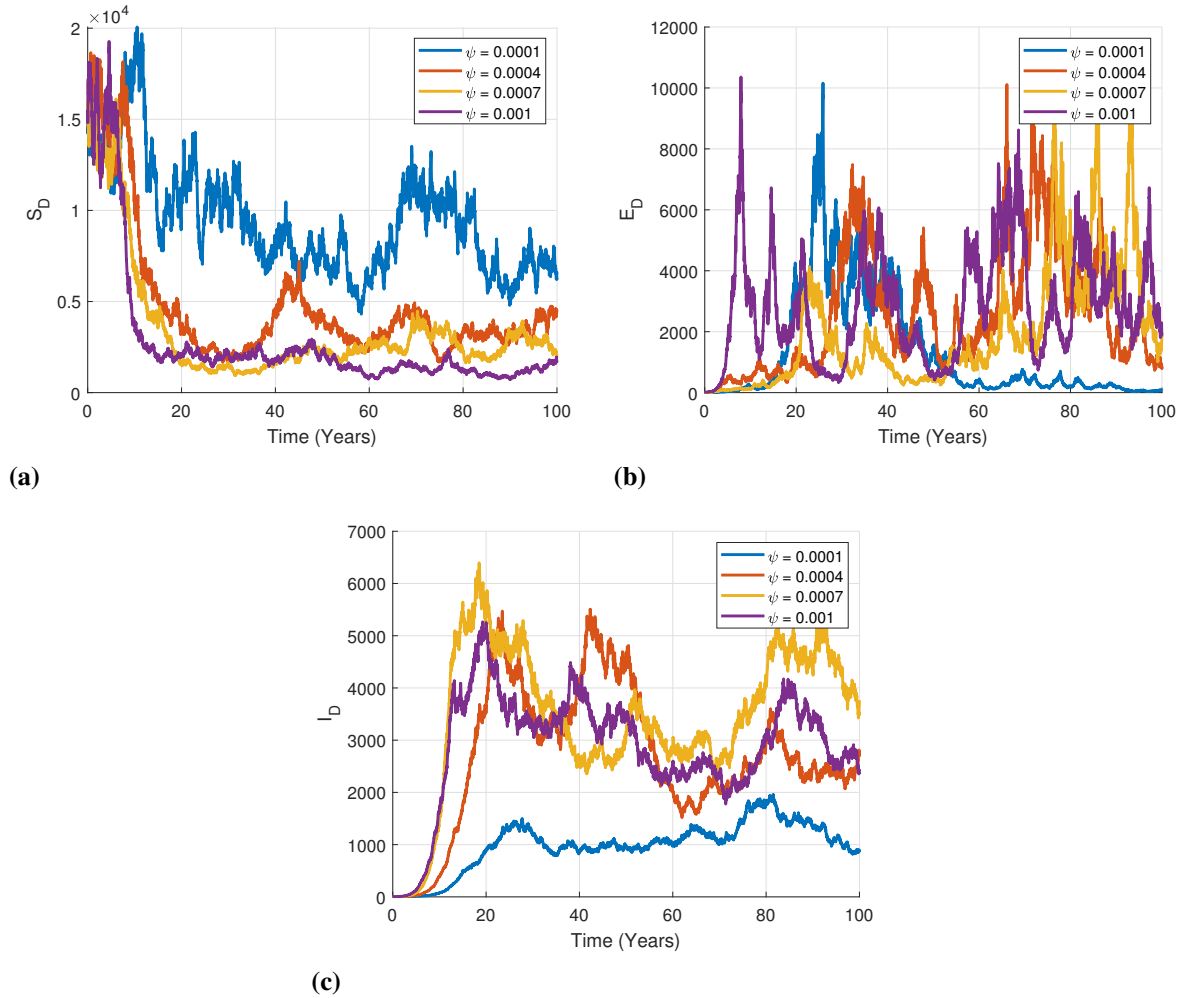


Figure 47: Stochastic trajectory of domestic dogs due to impact of contact rate ψ

Figures 48–52 present both deterministic and CTMC stochastic results, showing a comparable trend in rabies transmission dynamics. These figures illustrate a decrease in the susceptible populations following exposure, infection, and recovery events, stabilizing after approximately 20 to 40 years. Similarly, susceptible humans, free-range dogs, and domestic dogs experience a decline, ultimately reaching a steady state. Both models exhibit a similar general pattern, with deterministic results showing an average trend across CTMC sample paths, while stochastic outputs capture natural fluctuations. The relationship between the susceptible groups and the exposed, infected, and recovered classes is inverse. At first, the populations of exposed, infected, and recovered humans, free-range, and domestic dogs rise, peaking

around the first 20 years, after which they decline gradually and stabilize by year 30. This pattern suggests that initial increases in infections contribute to immunity within populations, ultimately leading to stabilization.

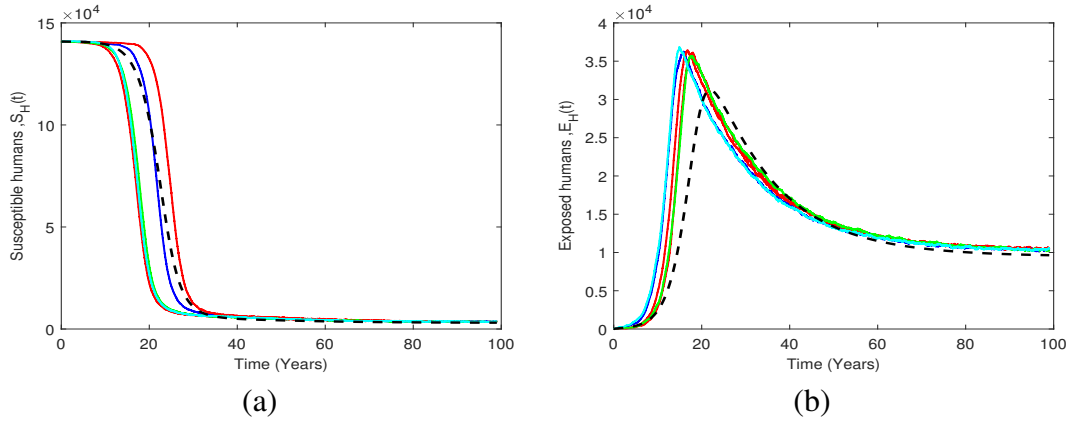


Figure 48: Comparison of Deterministic (dotted lines) and CTMC sample paths for rabies transmission dynamics: (a) susceptible humans and (b) exposed humans

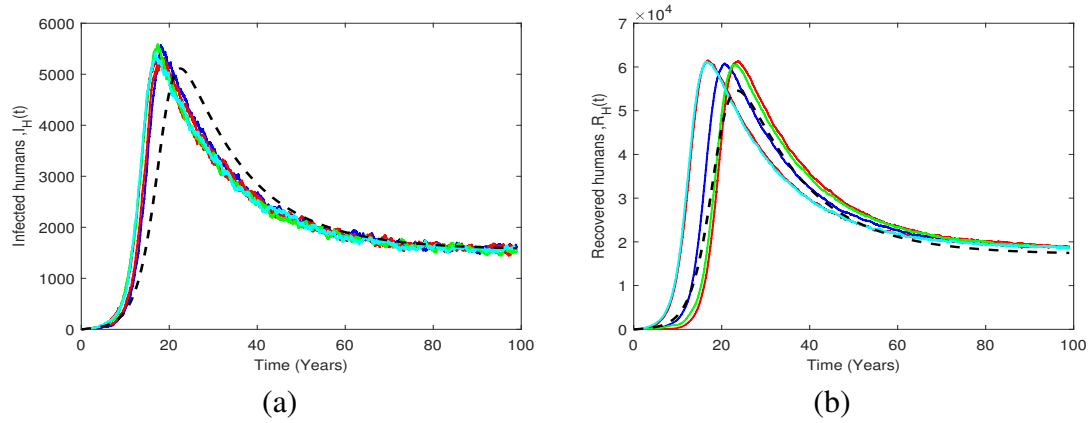


Figure 49: Comparison of deterministic (dotted lines) and CTMC sample paths for rabies transmission dynamics: (a) infected humans and (b) recovered humans

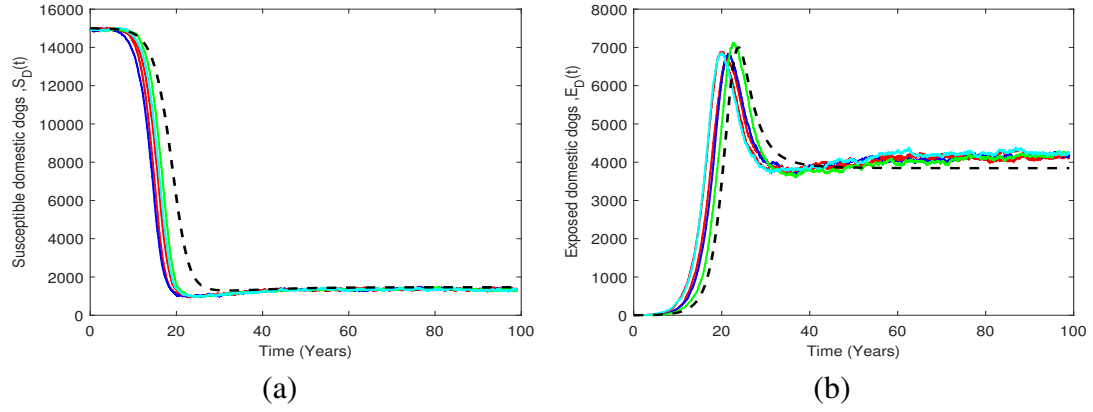


Figure 50: Comparison of deterministic (dotted lines) and CTMC sample paths for rabies transmission dynamics: (a) susceptible domestic dogs and (b) exposed domestic dogs

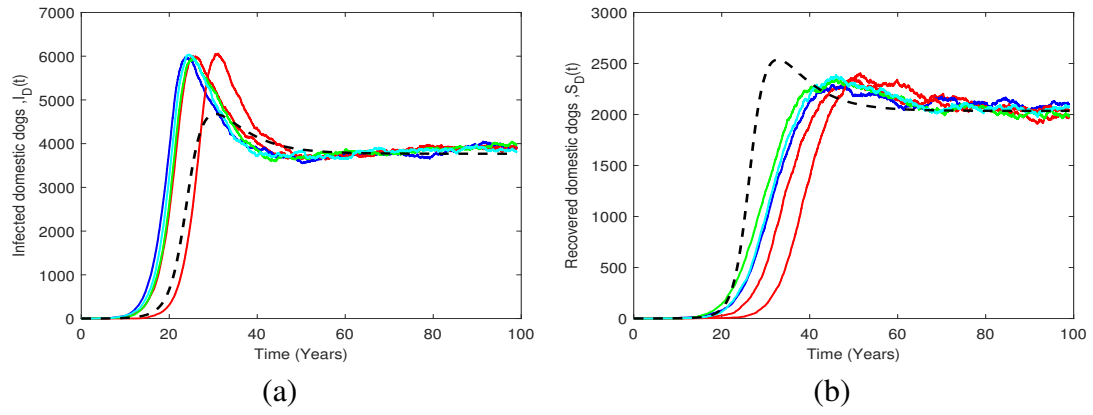


Figure 51: Comparison of deterministic (dotted lines) and CTMC sample paths for rabies transmission dynamics: (a) infected domestic dogs and (b) recovered domestic dogs

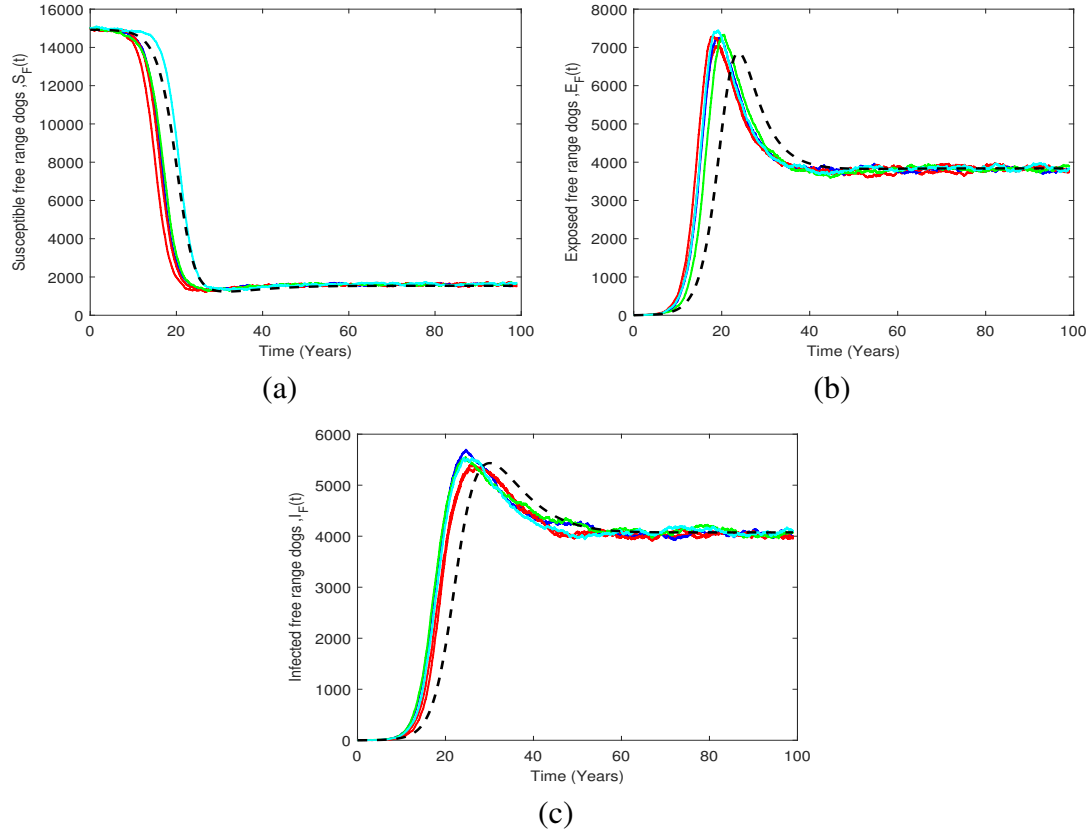


Figure 52: Comparison of deterministic (dotted lines) and CTMC sample paths for rabies transmission dynamics: (a) susceptible free range dogs (b) exposed free range dogs and (c) infected free range dogs

The probabilities of rabies extinction and outbreak, referred to as (Ext. Prob) and (Outb. Prob) respectively, were estimated using a branching process and numerical simulations. The simulation with 10 000 random sample paths of the continuous-time Markov chain (CTMC) epidemic model (3.9), varying the initial values of the infected classes were conducted. The results presented in Table 9 reveals that rabies transmission is highly sensitive to initial infection levels across compartments. High initial infections in humans, free-roaming, and domestic dogs yield a high outbreak probability (0.87), indicating strong persistence potential. In contrast, isolated infections (e.g., only $E_D(0) = 1$) show extinction probability up to 1.00, reflecting limited transmission capability. Environmental presence ($M(0) = 1$) alone yields moderate outbreak probability (0.26), confirming its contributory role. Free-roaming dogs significantly influence dynamics, with $I_F(0) = 1$ leading to outbreak probabilities up to 0.72. These results emphasize the critical need for early intervention and targeted control in high-risk compartments.

Table 9: Probabilities of extinction and outbreak

$E_H(0)$	$I_H(0)$	$E_F(0)$	$I_F(0)$	$E_D(0)$	$I_D(0)$	$M(0)$	Ext. Count	Outb. Count	Ext. Prob	Outb. Prob
0	0	1	1	1	1	1	2743	7166	0.27	0.72
0	0	1	1	0	0	0	4568	5322	0.46	0.53
1	1	1	1	1	1	1	1171	8746	0.12	0.87
1	1	1	1	0	0	0	2056	7852	0.21	0.79
0	0	0	0	1	1	0	9554	436	0.96	0.04
0	0	0	0	0	0	1	7380	2569	0.74	0.26
0	0	1	0	0	0	0	6898	3027	0.69	0.30
0	0	0	1	0	0	0	6575	3362	0.66	0.34
0	0	0	0	1	0	0	10000	0	1.00	0.00
0	0	0	0	0	1	0	9500	487	0.95	0.05
1	1	0	0	0	0	0	4524	5384	0.45	0.54
1	1	0	0	1	1	0	4329	5568	0.43	0.56
0	0	1	1	1	0	0	2671	7220	0.27	0.72
0	0	1	1	1	1	1	2660	7237	0.27	0.72
0	0	0	0	0	1	1	6836	3091	0.68	0.31

4.4 Numerical Simulations for Optimal Control Model

Numerical simulations were conducted using an optimal control framework to examine the transmission dynamics and management of rabies in human populations, as well as in free-range and domestic dog populations. The optimal control solution was derived by solving the optimality system comprising the system of state equations and the corresponding adjoint system formulated in accordance with Theorem 3.9. To obtain the optimal trajectories, the coupled state adjoint system was solved iteratively using a fourth-order Runge–Kutta scheme, with the control variables initialized over the prescribed simulation horizon. This method, with a truncation error of $O(h^5)$, where h represents the step size, ensures high accuracy in each step due to the proportionality of error to h^5 (Sauer, 2012).

In terms of stability, the fourth-order Runge–Kutta method is designed to exhibit stability across a broad spectrum of problems and is widely acknowledged for its stability (Ketcheson *et al.*, 2009). The method’s convergence rate is $O(h^5)$, signifying rapid reduction of error in the numerical solution as the step size diminishes (Ketcheson *et al.*, 2009). For solving the adjoint equations, the backward fourth-order Runge–Kutta scheme is employed, leveraging existing solutions of the state equations in accordance with the transversality conditions (Yusuf *et al.*, 2022). Furthermore, updates to the controls (u_1, u_2, u_3, u_4) are made using a convex combination of the prior controls and values from the characterizations (Yusuf *et al.*, 2022). This iterative process continues until the values of unknowns in subsequent iterations closely approximate those in the current iteration (Sagamiko *et al.*, 2015). The values used for numerical simulation are shown in Table 5. We incorporate four time dependent control variables that are: $u_1(t)$, which measures the effect of promoting good health practice and management (surveillance and monitoring) in reducing the possibility of human and dogs infection with rabies; $u_2(t)$, which represents the control effort due to vaccination of domestic dogs; $u_3(t)$, which measures the impact of educating com-

munities about rabies transmission risks and protective measures to avoid exposure (public awareness and education); $u_4(t)$, which represents the treatment effort to individuals exposed to suspected rabid animals through bites or scratches (Post-Exposure Prophylaxis-PEP). Strategies for eliminating rabies in humans and dog populations have been ranked based on their effectiveness, starting from the least effective and advancing to the most effective as follows.

4.4.1 Comparison between $\mathcal{R}_e$ and $\mathcal{R}_0$

Mesh and contour plots are shown in Fig. 53 and 54. They illustrate the relationship between $\mathcal{R}_e$ and $u_1(t)$, which measures the impact of promoting good health practice and management; $u_2(t)$, which represents control effort due to vaccination of domestic dogs; and $u_4(t)$, which measures the treatment effort given to people bitten or scratched by a potentially rabid animal (PREP and PEP), along with the recovery rate as a result of treating infected individuals. It is observed that the magnitude of $\mathcal{R}_e$ decreases with an increase in $u_1(t)$, $u_2(t)$, and $u_4(t)$. This observation highlights the effectiveness of $u_1(t)$, $u_2(t)$, and $u_4(t)$ interventions in reducing the transmission of rabies across the community.

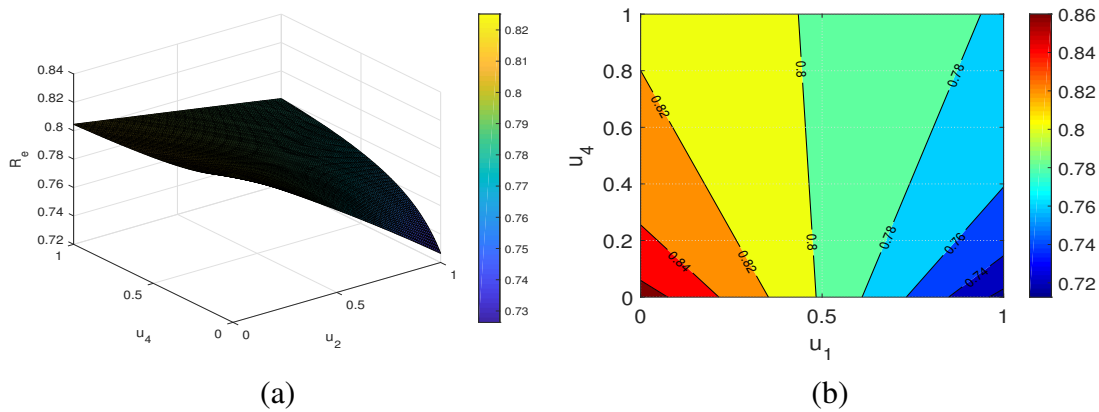


Figure 53: Mesh and contour plots showing the integrated effect of parameters on (a) $\mathcal{R}_e$ in terms of u_2 and u_4 (b) $\mathcal{R}_e$ in terms of u_1 and u_4

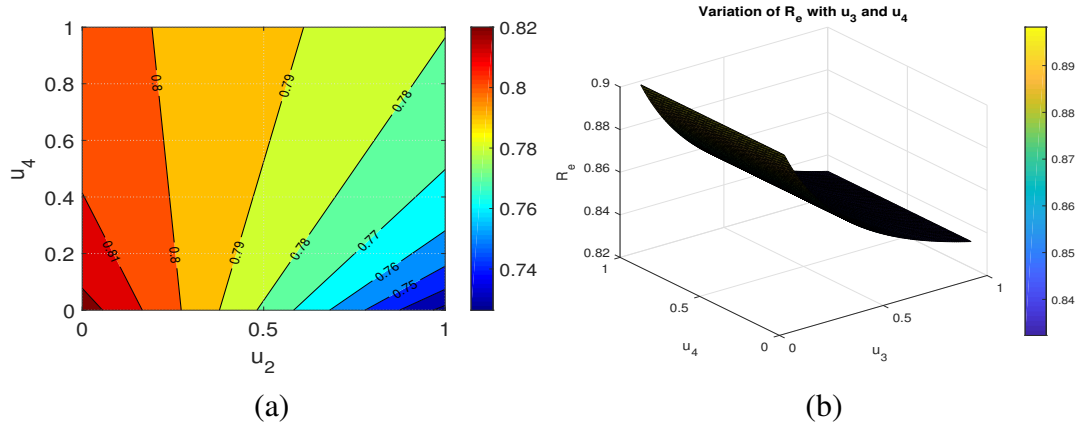


Figure 54: Mesh and contour plots showing the integrated effect of the parameters on (a) R_e in terms of u_2 and u_4 (b) R_e in terms of u_3 and u_4

The mesh and contour plot representing R_e as a function of ψ_1 and ψ_2 , which influence the effective reproduction number in the spread patterns of rabies, are shown in Fig. 55. The results demonstrate that the effectiveness increases along with the values of ψ_1 and ψ_2 . This indicates that increased rates of contact have a positive effect on the rabies transmission in domestic dogs.

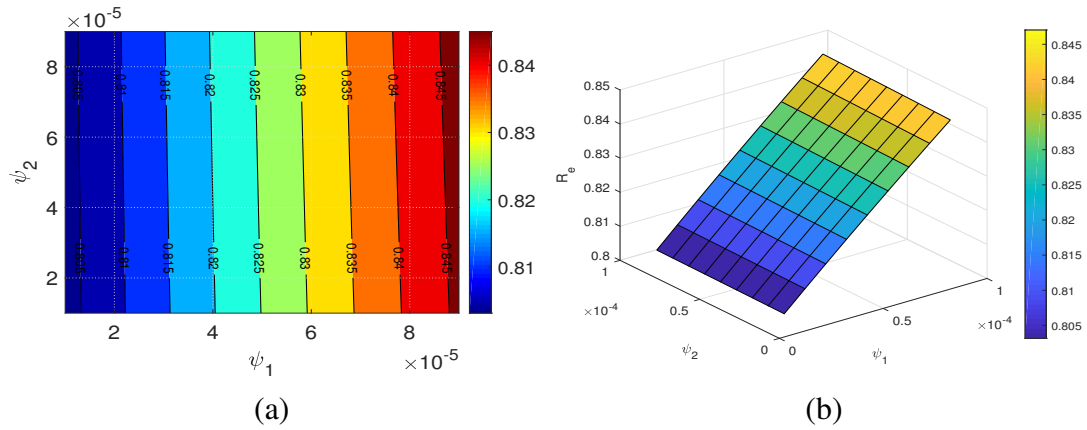


Figure 55: Variations in R_e and R_0 with respect to changes on (a) the rate that S_D gets infection from I_F and ψ_1 ; (b) the rate that S_D gets infection from I_D and ψ_1

Figure (56) shows the contour and mesh graph of R_e as a function of deterrence factors influencing the effective reproduction number in the transmission dynamics of rabies in domestic dogs. The results revealed that as the deterrence factor ρ_1 and ρ_2 increases the effective reproduction number decreases. This results implies that deterrence factors has higher impact on the transmission dynamics of rabies on domestic dogs.

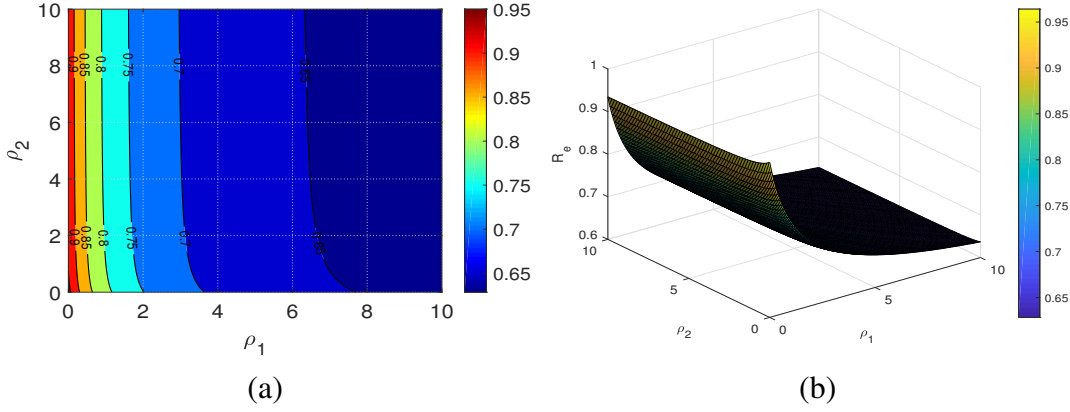


Figure 56: The combined influence of parameters on $\mathcal{R}_e$ (a) $\mathcal{R}_e$ in terms of deterrence factors, ρ_1 and ρ_2 (b) $\mathcal{R}_e$ in terms of ρ_1 and ρ_2

Strategy A: The use of all controls $u_i, i = 1, 2, 3, 4$.

Here we measure the impact of promoting good health practices and management, vaccinating domestic dogs, educating communities, and lastly, providing treatment for those who have been in contact with a potentially rabid animal through bites or scratches (Post-Exposure Prophylaxis-PEP). This strategy aims to eradicate rabies within the community by focusing on four key interventions: promoting good health practices and management, denoted by $u_1(t)$; vaccinating domestic dogs, represented by $u_2(t)$; educating communities, captured by $u_3(t)$; and administering treatment to individuals exposed to potentially rabid animals through Post-Exposure Prophylaxis (PEP), modeled by $u_4(t)$. The results demonstrate that the strategy effectively eliminates rabies within five years (Fig. 57, 58, and 59), with optimal utilization of $u_2(t)$ and $u_4(t)$ at 100% during the first 16 and 6 years respectively, followed by a gradual reduction to non-zero. Meanwhile, $u_1(t)$ and $u_3(t)$ declined over time, reflecting the fact that vaccination does not confer permanent immunity, as illustrated in the control profile.

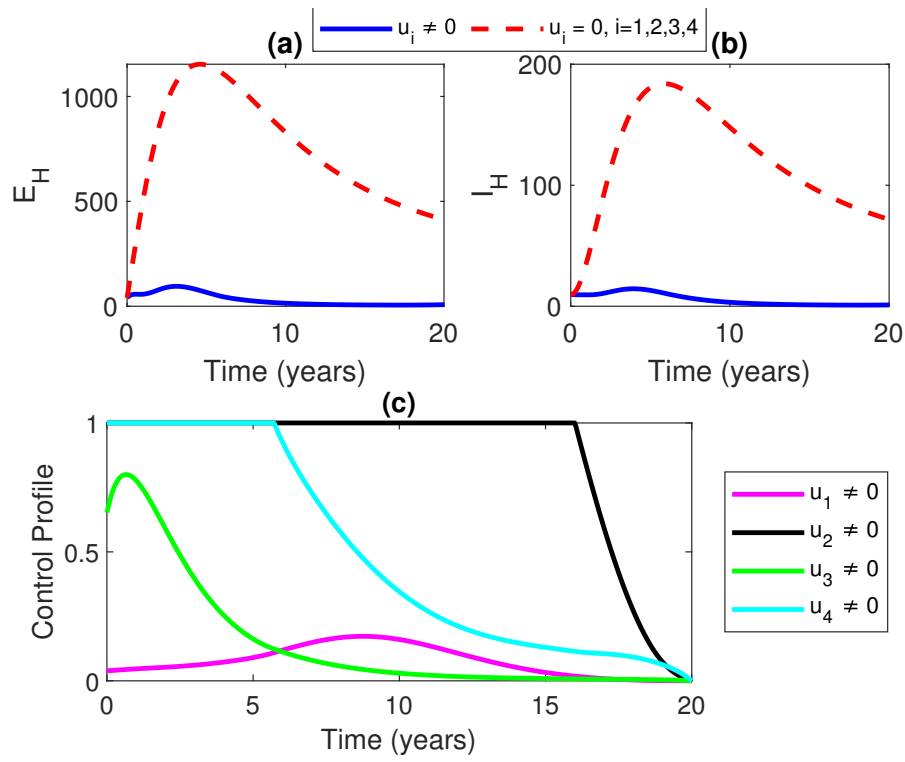


Figure 57: Impact of Strategy A to human population

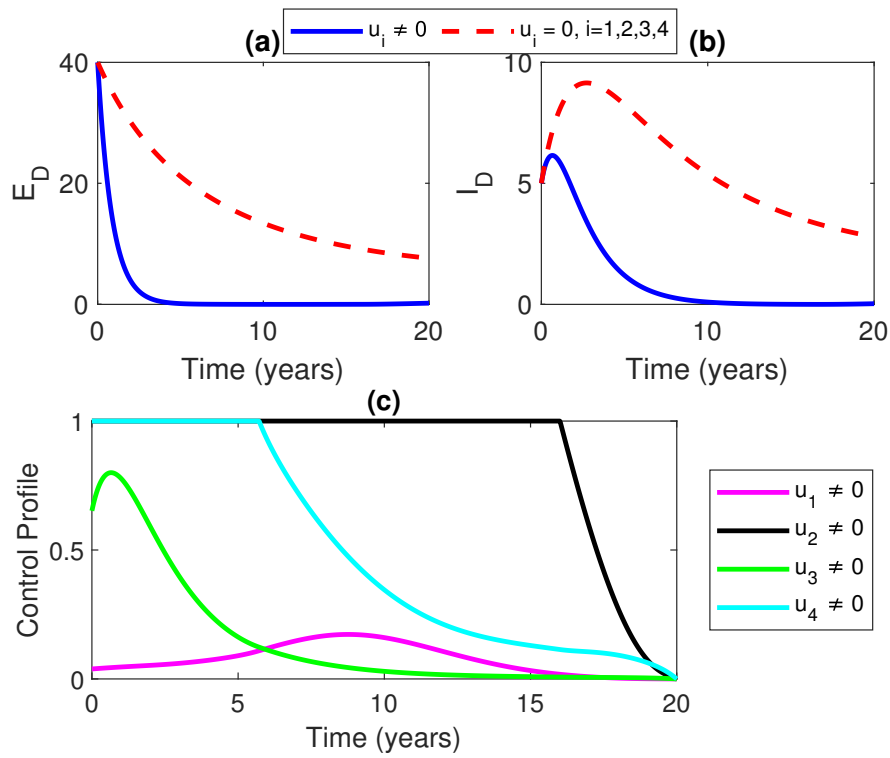


Figure 58: Impact of Strategy A to domestic dogs population

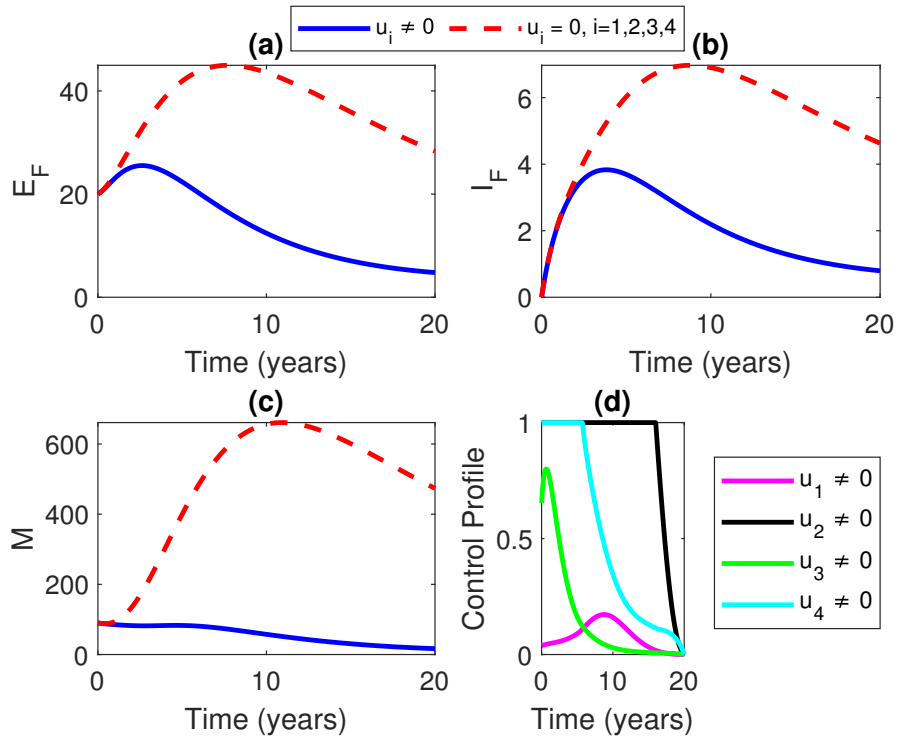


Figure 59: Impact of Strategy A to free-range dogs and rabies virus in the environment

Strategy B: Using controls u_3 and u_4 .

We assess the outcomes of public education and awareness campaigns and the availability of treatment for people exposed to potential rabies via animal bites or scratches (Post-Exposure Prophylaxis-PEP). The results are given in Fig. 60, 61 and 62.

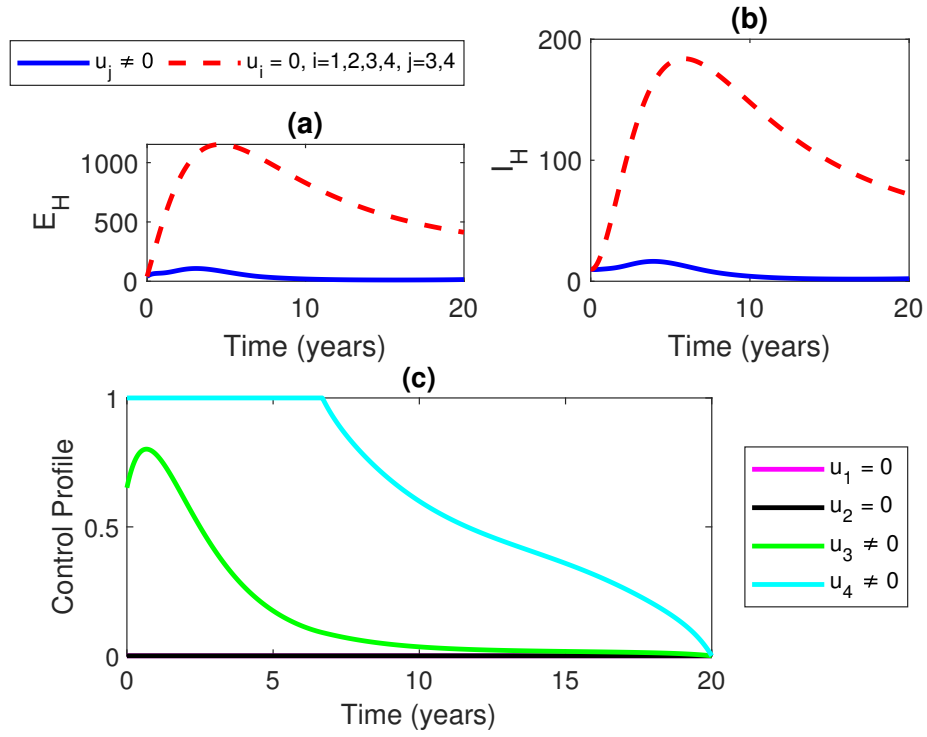


Figure 60: Impact of Strategy B to human population

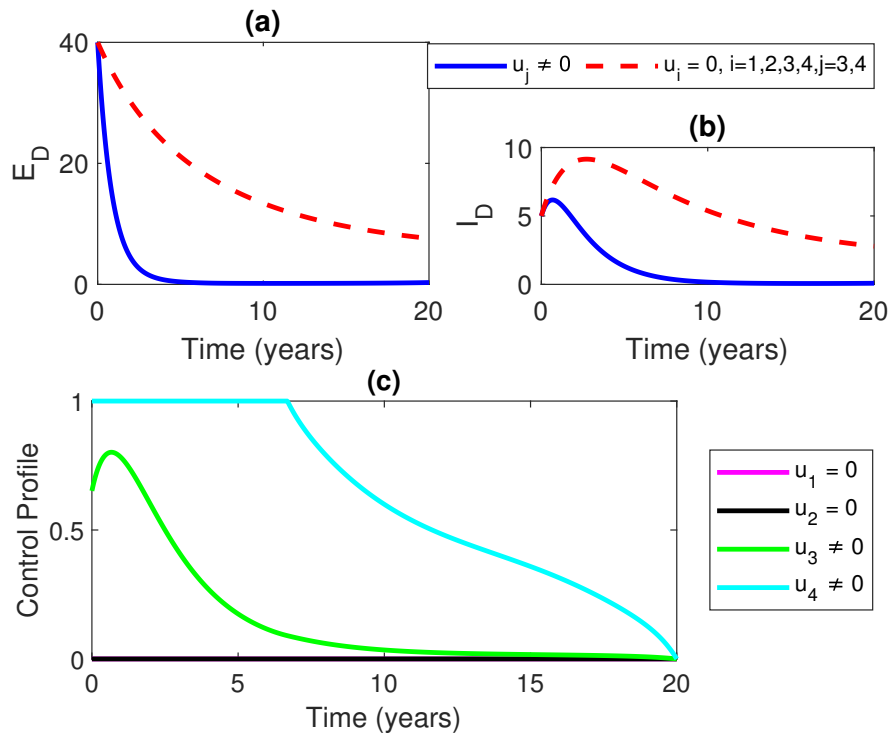


Figure 61: Impact of Strategy B to domestic dogs population

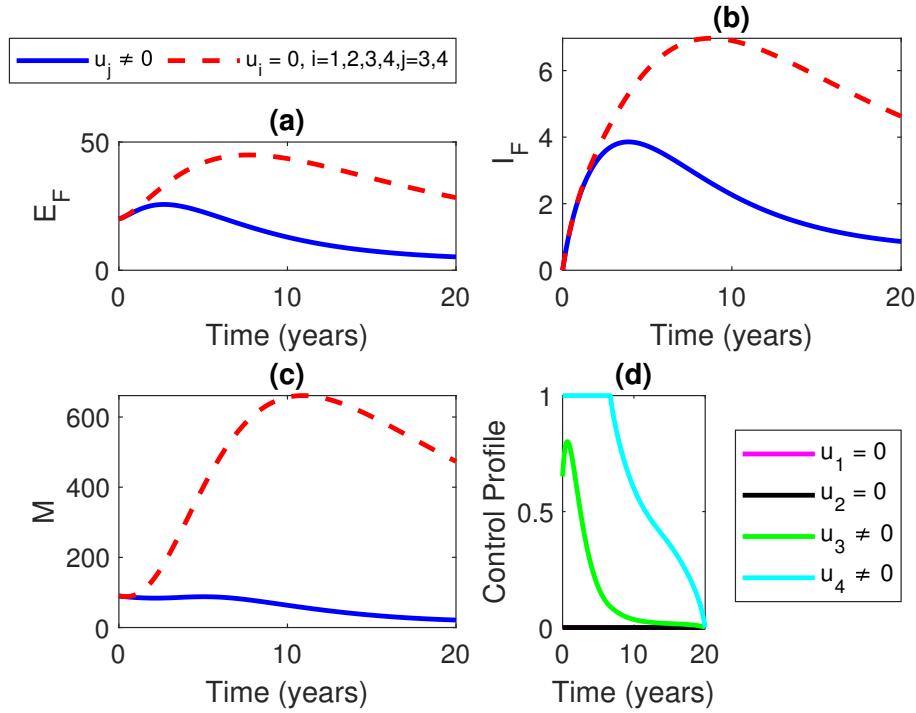


Figure 62: Impact of Strategy B to free-range dogs and rabies virus in the environment

Strategy C: Using only control u_4 .

Now we concentrate on the impact of providing treatment for bitten or scratched individuals by an animal suspected to be rabid (Post-Exposure Prophylaxis-PEP). Figures 63, 64, and 65 illustrate that the timely administration of PEP at 100% effectiveness plays a pivotal role in reducing rabies mortality and curbing disease transmission within the first five years. By treating individuals immediately after exposure, PEP significantly reduces the risk of death and halts secondary transmission, particularly in high-risk areas. The results also reveal that PEP complements long-term rabies control measures, such as vaccination and health education. While these strategies are vital, PEP offers immediate protection, ensuring effective containment of the disease even after exposure.

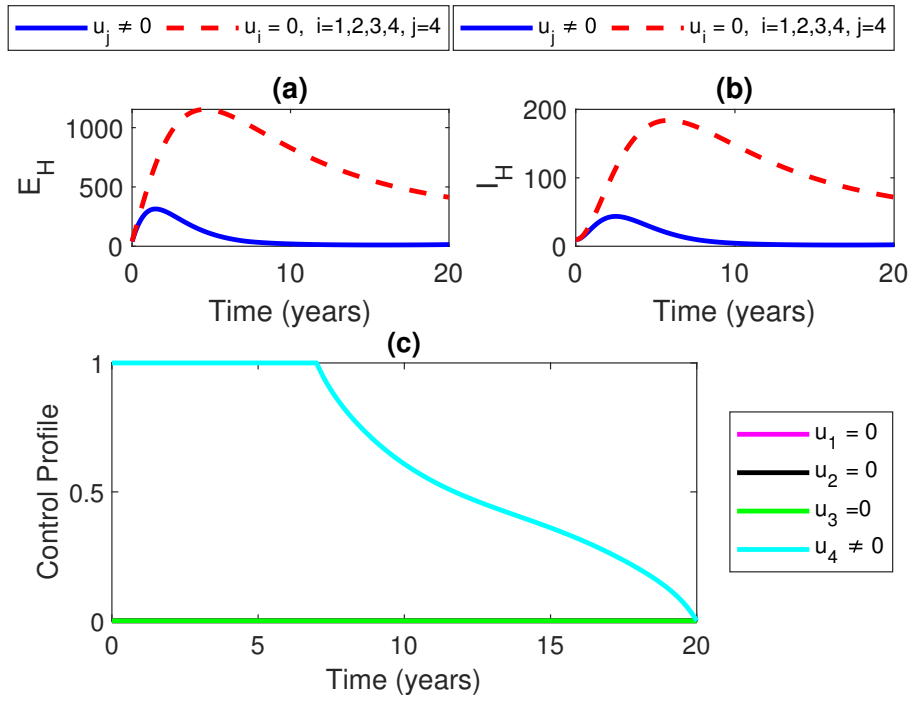


Figure 63: Impact of Strategy C to human population

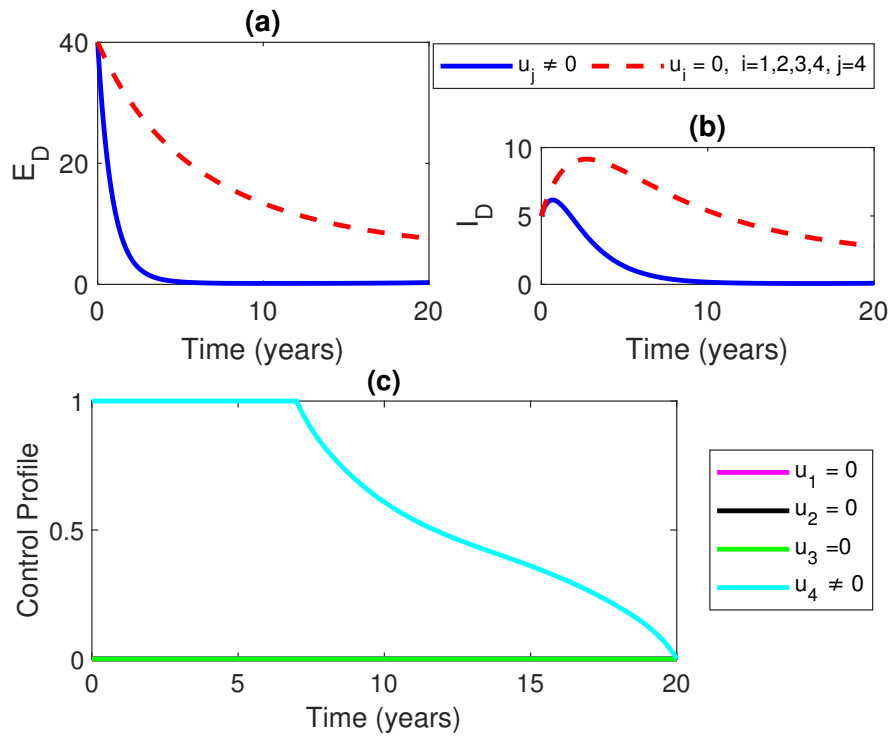


Figure 64: Impact of Strategy C to domestic dogs population

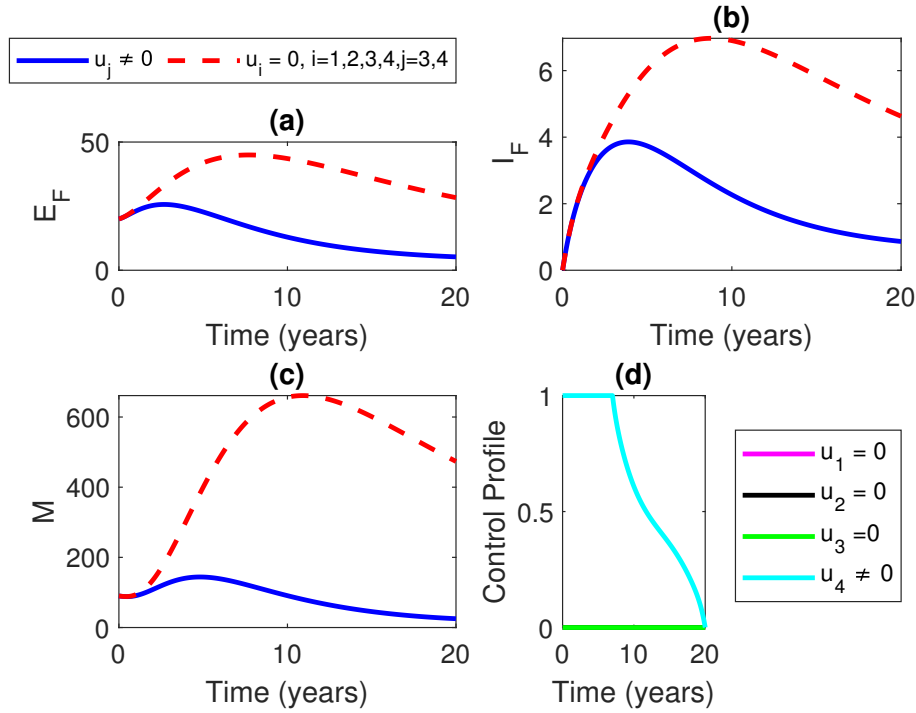


Figure 65: Impact of Strategy C to free-range dogs and rabies virus in the environment

Strategy D: Using controls u_1 and u_2 .

On this strategy, we consider the impact of promoting good health practices and management (surveillance and monitoring) and vaccinating on domestic dogs. This strategy focuses on the combined impact of promoting good health practices and management, including surveillance and monitoring, represented by $u_1(t)$, along with the vaccination of domestic dogs, denoted by $u_2(t)$. The results indicate that the strategy is most effective when both control measures are applied at 100%, as illustrated in Fig. 66, 67, and 68. The effectiveness of the strategy was observed between the 4th and 14th years, followed by a decline after the reduction in control application. These findings suggest that promoting good health practices and vaccination can significantly reduce periodic rabies transmission. However, the data also highlights that vaccinating domestic dogs alone is not sufficient to fully prevent cyclical rabies transmission, primarily due to the movement of free-roaming dogs that may travel to rabies-free areas and initiate new outbreaks.

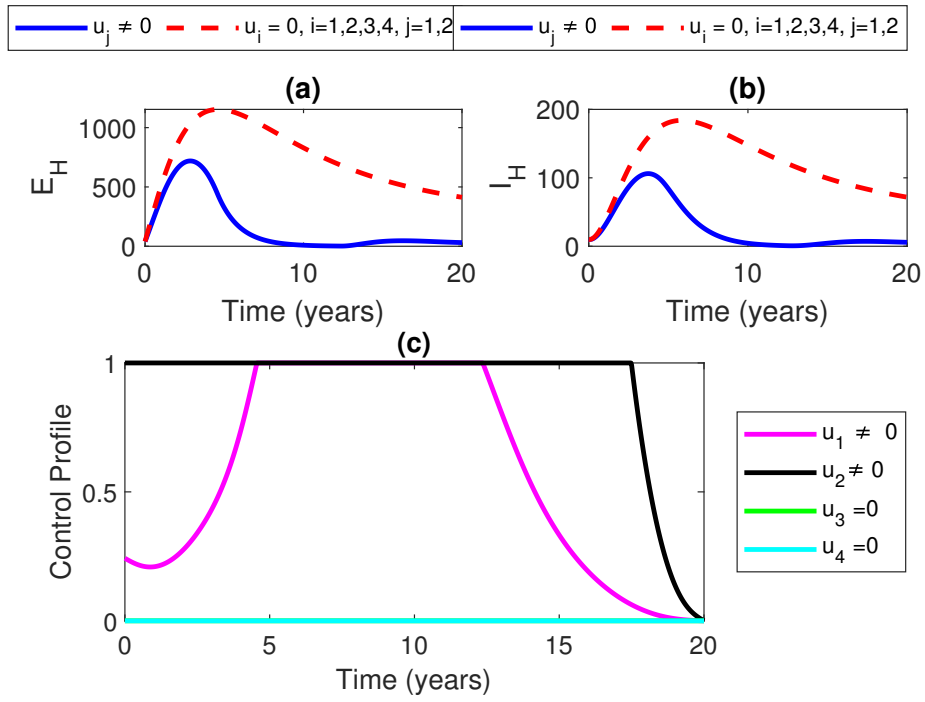


Figure 66: Impact of Strategy D to human population

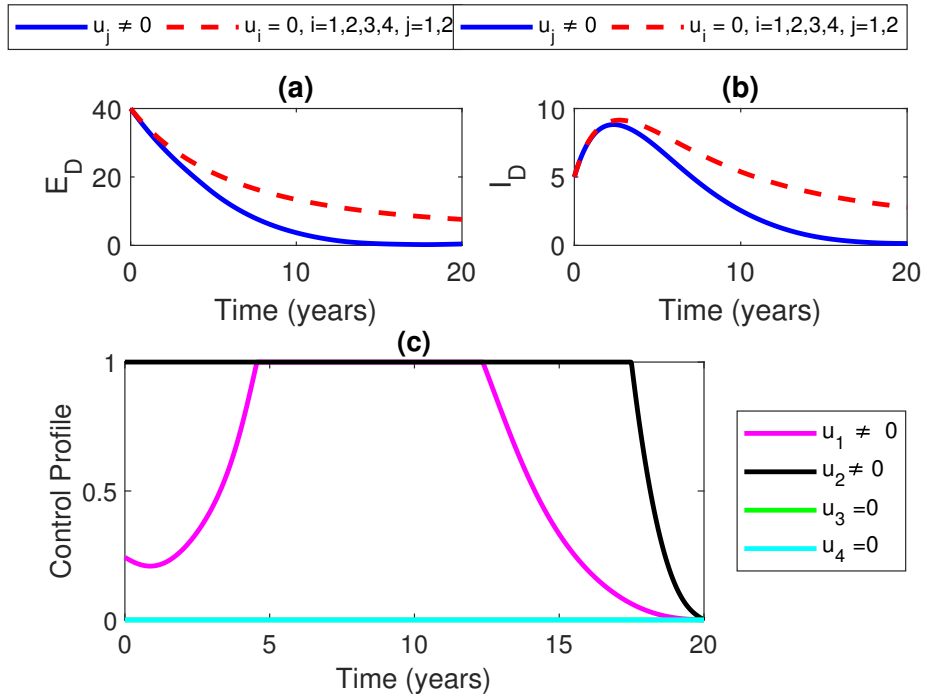


Figure 67: Impact of Strategy D to domestic dogs population

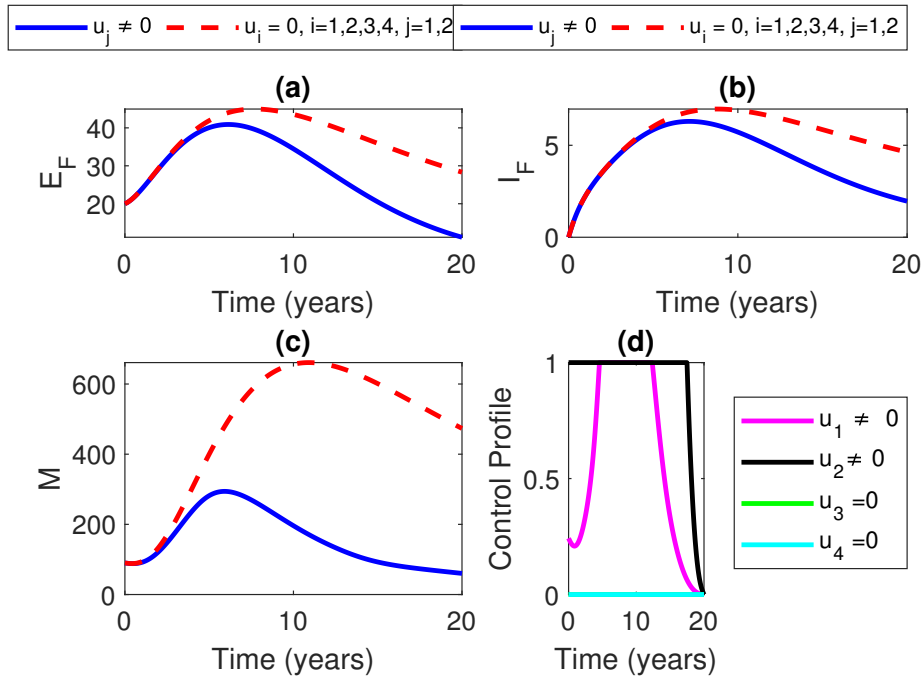


Figure 68: Impact of Strategy D to free-range dogs and rabies virus in the environment

Strategy E: Using controls u_1 and u_4 .

On this strategy, consider the impact of promoting good health practices and management (surveillance and monitoring) and vaccinating on domestic dogs. This strategy focuses on the combined impact of promoting good health practices and management, including surveillance and monitoring, represented by $u_1(t)$, along with Implementation of administration of PEP, denoted by $u_4(t)$. The results indicate that the strategy is most effective when $u_4(t)$ control measure is applied at 100% for the first 6 years and $u_1(t)$ is applied between 10% to 25% , as illustrated in Fig. 69, 70, and 71. The effectiveness of the strategy was observed between the 8th to 10th years followed by a decline after the reduction in control application. These findings suggest that promoting good health practices and administration of PEP can significantly reduce periodic rabies transmission. However, the data also highlights that administration of PEP on domestic dogs and human alone is not sufficient to fully prevent cyclical rabies transmission, primarily due to the movement of free-roaming dogs that may travel to rabies-free areas and initiate new outbreaks.

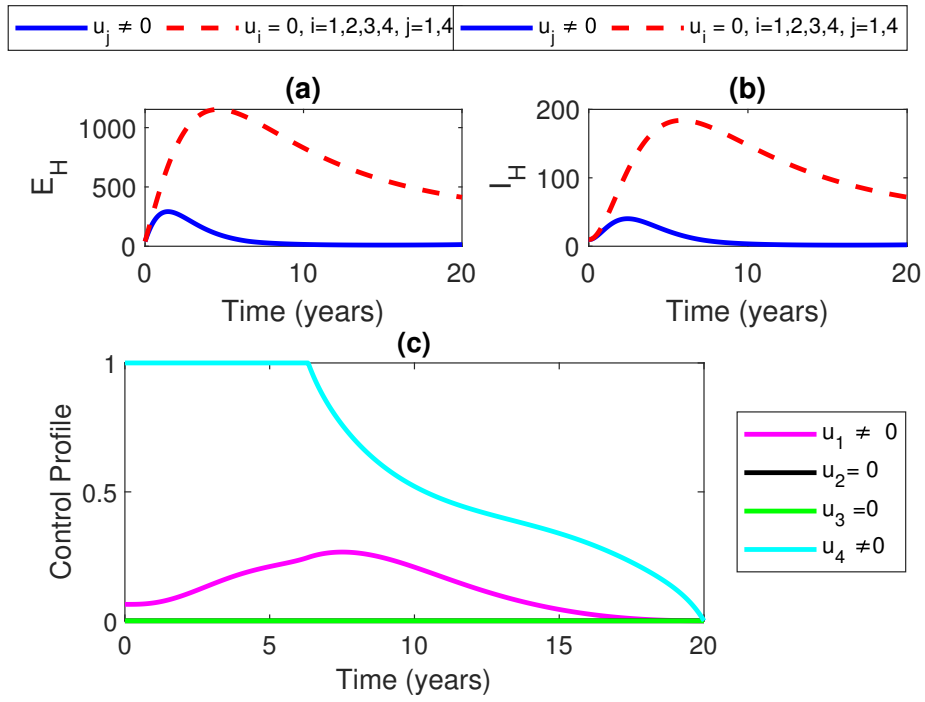


Figure 69: Impact of Strategy E to human population

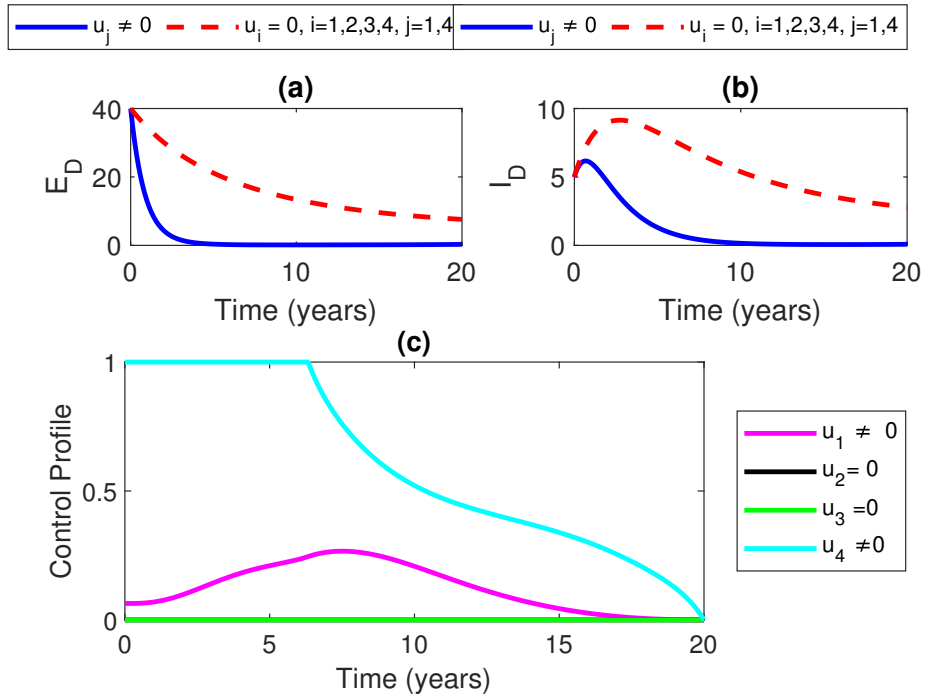


Figure 70: Impact of Strategy E to domestic dogs population

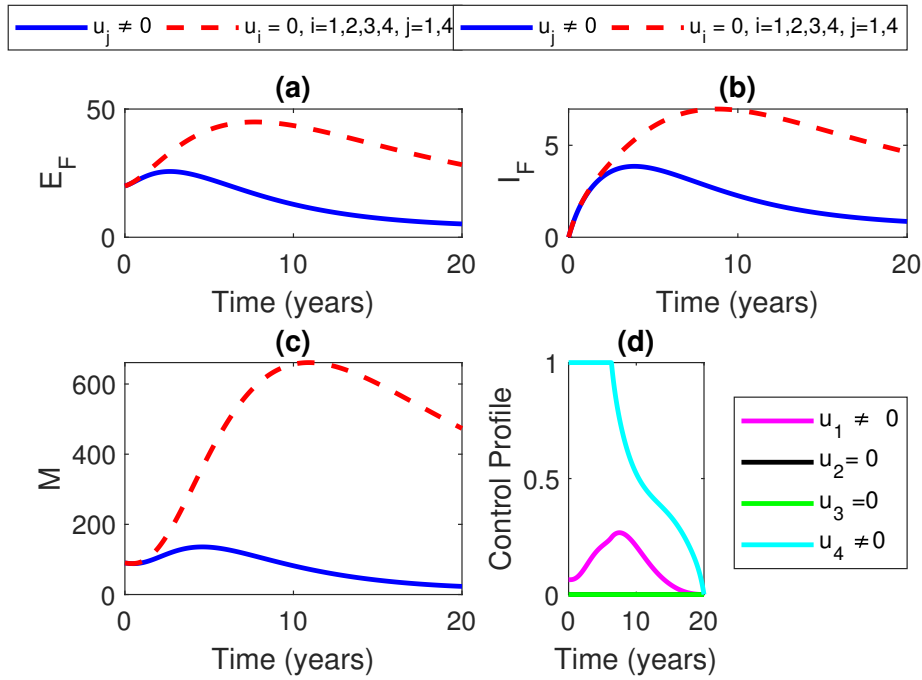


Figure 71: Impact of Strategy E to free-range dogs and rabies virus in the environment

Strategy J: Using controls u_2 and u_3 .

Finally, we consider the impact of combination of vaccination of domestic dogs , public awareness and education with risk associated with rabies. This strategy focuses on the combined impact of combination of vaccination of domestic dogs, represented by $u_2(t)$, along with implementation of public awareness and education with risk associated with rabies denoted by $u_3(t)$. The results indicate that the strategy is most effective when $u_3(t)$ control measure is applied at 100% for the first 10 years and $u_2(t)$ is applied for 100% for 18 years , as illustrated in Fig. 72, 73, and 74. The effectiveness of the strategy was observed between the 1st to 10th years followed by a rise after the reduction in control application. These findings suggest that combination of vaccination of domestic dogs , public awareness and education with risk associated with rabies.

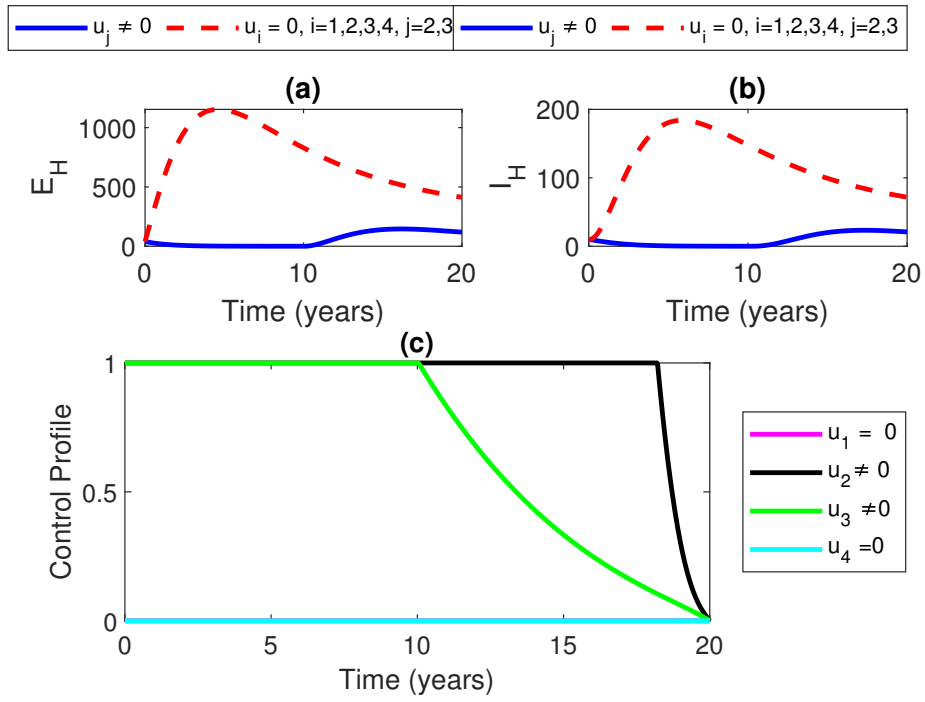


Figure 72: Impact of Strategy J to human population

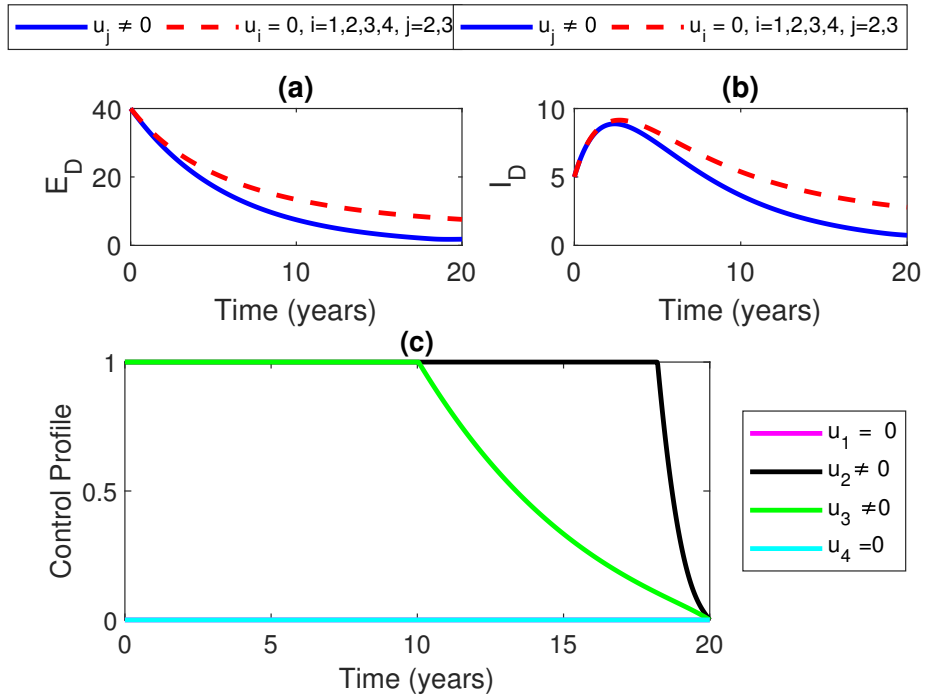


Figure 73: Impact of Strategy J to domestic dogs population

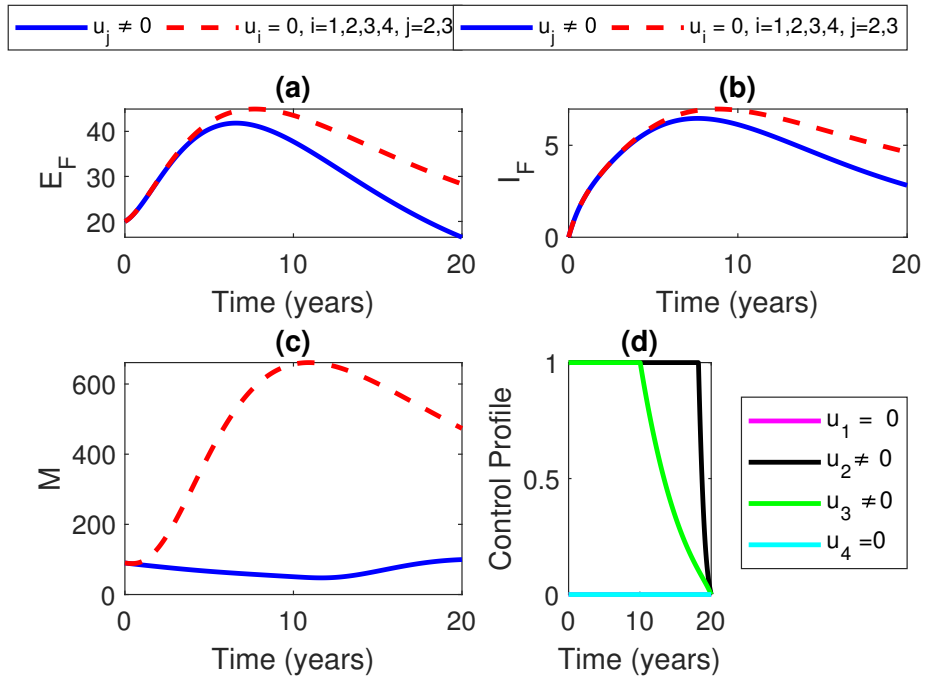


Figure 74: Impact of Strategy J to free-range dogs and rabies virus in the environment

CHAPTER FIVE

CONCLUSION AND RECOMMENDATIONS

5.1 Conclusions

In this study, we developed and analyzed a deterministic model to estimate parameters and assess uncertainty in the transmission dynamics of rabies among humans and dogs. Parameter values were estimated using the non-linear least squares method, based on data generated by adding random Gaussian noise to real-world observations. The basic reproduction number R_0 was derived using the next-generation matrix approach. Stability analysis showed that the rabies-free equilibrium is globally asymptotically stable when $R_0 < 1$, while the rabies-endemic equilibrium is globally stable when $R_0 \geq 1$. Sensitivity analysis, conducted through Latin Hypercube Sampling and Partial Rank Correlation Coefficient methods, identified key parameters influencing model outcomes. Results indicate that increased contact rates between humans and dogs contribute significantly to rabies transmission.

The study further explored the role of environmental factors and contact rates in rabies spread, particularly through dog bites. Stability analysis, employing Jacobian and Metzler matrices, confirmed that higher contact rates (ψ_1 , ψ_2 , ψ_3 , κ_1 , κ_2 , and κ_3) lead to an increase in R_0 , exacerbating disease transmission. These findings provide critical insights into the complexity of rabies dynamics and inform the development of targeted, sustainable prevention and control strategies.

To further assess rabies persistence, we conducted a comparative analysis of stochastic continuous-time Markov chain (CTMC) models and deterministic approaches. Using a multitype branching process, we established the stochastic threshold for rabies persistence, offering insights into the role of randomness in disease extinction. Numerical simulations demonstrated that, while deterministic and stochastic models produce similar outcomes in high-infection scenarios, stochasticity significantly affects low-infection conditions. Unlike deterministic models, which assume fixed transmission rates, stochastic models capture variations due to regional differences, population behaviors, and environmental fluctuations. These insights support the design of dynamic vaccination programs, real-time monitoring, and targeted interventions in high-risk areas.

Moreover, It was noted that a threshold parameter, the reproduction number was qualitatively and quantitatively used to investigate the local and global stability of the model's steady state. For the two steady states the model has (that is the pest-free equilibrium and the pest persistence equilibrium points), it was observed that they were both locally and globally stable. In particular, the pest-free equilibrium point is both locally and globally stable whenever the reproduction number is less than unity. However, when the reproduction number is greater than unity there exists a pest persistence equilibrium point which is also both locally and globally stable.

Finally, we compared the effective reproduction number R_e with the basic reproduction number R_0 , as established in Charles *et al.* (2024), emphasizing the need for robust control measures. Our findings

underscore the effectiveness of vaccination, post-exposure prophylaxis (PEP), and public health education in reducing rabies transmission. However, a single intervention may not suffice for eradication. A comprehensive strategy combining vaccination, treatment, education, and control of free-roaming dog populations proves to be the most effective approach. Sustained efforts, particularly in regions with high contact rates between dogs and humans, are essential for long-term rabies elimination. By integrating vaccination, education, treatment, and deterrence measures, rabies transmission can be significantly reduced, ultimately bringing the effective reproduction number below the critical threshold for eradication.

5.2 Recommendations

Based on the findings of this study, the following recommendations are proposed:

- (i) Governments and health organizations should prioritize mass vaccination campaigns for both domestic and stray dogs to reduce the basic reproduction number R_0 and curb rabies transmission.
- (ii) Public health education programs should be enhanced to raise awareness about rabies prevention, post-exposure prophylaxis (PEP), and responsible pet ownership.
- (iii) Implementation of effective stray dog population control measures, such as sterilization and regulated culling, should be considered to minimize uncontrolled contact between dogs and humans.
- (iv) Active surveillance and real-time monitoring systems should be established to track rabies incidence, detect outbreaks early, and implement rapid intervention strategies.
- (v) Incorporating stochastic modeling approaches in epidemiological studies can provide a more realistic understanding of rabies dynamics, particularly in regions with low infection prevalence.
- (vi) Governments should enforce stricter regulations on dog vaccinations, bite reporting, and movement control to minimize cross-regional transmission risks.

Furthermore, the following future Works are proposed:

- (i) Explore the role of environmental factors and socio-economic conditions in shaping rabies transmission patterns to improve control strategies.
- (ii) Spatial-Temporal Dynamics: Investigate the role of geographic heterogeneity and mobility patterns in rabies transmission.
- (iii) Climate and Environmental Factors: Assess the impact of seasonal variations, urbanization, and habitat changes on disease persistence.
- (iv) Optimized Vaccination Strategies: Develop cost-effective, data-driven vaccination models using optimal control and stochastic approaches.

- (v) Stochastic Threshold Analysis: Examine extinction probabilities under demographic and environmental variability.
- (vi) Socio-Economic and Policy Impact: Evaluate the economic burden of rabies and the effectiveness of public health interventions.
- (vii) Multi-Species Transmission: Extend models to include wildlife reservoirs and interspecies interactions affecting rabies dynamics.
- (viii) Machine Learning Applications: Utilize AI-driven models for outbreak prediction and real-time disease monitoring.

5.2.1 Limitation of the Study

This study was limited to various constraints which need to be acknowledged. The following were the limitations of this study:

- (i) The proposed model were calibrated based on data extracted from literature, however, model validation was not done due to unavailability of observed data.
- (ii) Unavailability of enough funds for travelling to meet my supervisors who were living outside the country for face to face consultation.

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APPENDICES

APPENDIX I: MATLAB Code for Model Without Control

```

1 clear all
2 close all
3 clc
4 global rho_1 rho_2 rho_3 C theta_1 tau_1 tau_2 gamma tau_3 mu_3 beta_1 beta_2
   beta_3 mu_1 sigma_1 theta_2 kappa_1 kappa_2 kappa_3 sigma_2 mu_2 theta_3
   psi_1 psi_2 psi_3 sigma_3 gamma_1 gamma_2 gamma_3 mu_4 nu_1 nu_2 nu_3
5 rho_1=10; %Deterrent coeffiecient of domestic dogs from I_F
6 rho_2=8; %Deterrent coeffiecient of domestic dogs from I_D
7 rho_3=15; %Deterrent coeffiecient of domestic dogs from M
8 theta_1=2000; % recurement rate of susceptible human
9 tau_1=0.0004; %The rate that S_H get infections from I_F
10 tau_2=0.0006; %The rate S_H get infection from I_D %paremeter Estimation
11 tau_3=0.0003; %The rate that S_H get infection from M
12 beta_1=1/6; %progression rate out of E_H to I_H(Incubation period of human)
13 beta_2=1; %Recovery rate of E_H
14 beta_3=0.54; %Rate of immunity loss of human being
15 mu_1=0.0142; %Natural death rate of human
16 sigma_1=1; %Disease induced rate of I_H
17 theta_2=1000; %Recruitment rate of free range dogs
18 gamma=1/6; %Progression rate of E_F to I_F
19 kappa_1=0.00002; %The rate that S_F becomes I_F from I_F
20 kappa_2=0.00008; %The rate that S_F bceomes infection from I_D
21 kappa_3=0.00004; %The rate that S_F gets infection form M
22 sigma_2=0.09; % Disease induced death rate of I_F
23 mu_2=0.067; %Natural Mortality rate of free-range dogs
24 theta_3=1200; %Recruitment rate of domestic dog population
25 psi_1=0.00009; %The rate that S_D get infection from I_F
26 psi_2=0.00007; %The rate that S_D get infection from I_D
27 psi_3=0.00003; %The rate that S_D get infection from M
28 sigma_3= 0.09; %1; %Disease induced death rate for I_D
29 gamma_1=1/6; %The rate at which E_D becomes I_D
30 gamma_2=0.09; %Recovery rate of E_D
31 gamma_3=0.05; %The rate of loss of immunity of E_D
32 mu_3=0.08; % The naural Mortality rate of domestic dogs
33 nu_1=0.002; %Environment shedding rate from I_H
34 nu_2=0.009; %Environment shedding rate from I_F
35 nu_3=0.005; %Environment shedding rate from I_D
36 mu_4=0.08; %Naural removal rate of rabies
37 C=0.003; % Rabies concetration in the environment
38 R_33=(kappa_1*theta_2*gamma)/(mu_2*(mu_2+gamma)*(mu_2+sigma_2));
39 R_35=(kappa_1*theta_2*gamma_1)/(mu_2*(mu_3+gamma_1+gamma_2)*(mu_3+sigma_3));
40 R_53=(psi_1*theta_3*gamma)/(mu_3*(1+rho_1)*(mu_2+gamma)*(mu_2+sigma_2));
41 R_55=(psi_2*theta_3*gamma_1)/((1+rho_2)*(mu_3+gamma_1+gamma_2)*(mu_3+sigma_3));
42 R_0=((R_55+R_33)+sqrt(R_33*(R_33-2*R_55)+4*R_35*R_53+R_55^2))/2
43 tspan=[0:0.0001:100];

```

```

44 y0=[142000,40,0,0,15000,20,0,12500,25,0,0,90];
45 [t,y]=ode45(@(t,y)main_ty(t,y,rho_1,rho_2,rho_3,C,theta_1,tau_1,tau_2,gamma,tau_3
    ,mu_3,beta_1,beta_2,beta_3, mu_1,sigma_1,theta_2,kappa_1,kappa_2,kappa_3,
    sigma_2,mu_2,theta_3,psi_1,psi_2,psi_3,sigma_3,gamma_1,gamma_2,gamma_3,mu_4,
    nu_1,nu_2,nu_3),tspan,y0);
46
47 figure(2)
48 plot(t,y(:,1),'LineWidth',2)
49 grid on
50 xlabel('Time[Years]')
51 ylabel('Susceptible Human')
52 figure(3)
53 plot(t,y(:,2),'LineWidth',2)
54 hold on
55 grid on
56 % grid on
57 plot(t,y(:,3),'LineWidth',2)
58 plot(t,y(:,4),'LineWidth',2)
59 legend('E_H','I_H','R_H')
60 xlabel('Time[Years]')
61 ylabel('Human Population')
62 figure(5)
63 plot(t,y(:,8),'LineWidth',2)
64 %hold on
65 grid on
66 xlabel('Time[Years]')
67 ylabel('Susceptible Domestic dog')
68 figure(6)
69 plot(t,y(:,9),'LineWidth',2)
70 hold on
71 grid on
72 % grid on
73 plot(t,y(:,10),'LineWidth',2)
74 plot(t,y(:,11),'LineWidth',2)
75 legend('E_D','I_D','R_D')
76 xlabel('Time[Years]')
77 ylabel('Domestic dog Population')
78 figure(7)
79 plot(t,y(:,5),'LineWidth',2)
80 grid on
81 xlabel('Time[Years]')
82 ylabel('Susceptible free range dogs')
83 figure(8)
84 plot(t,y(:,6),'LineWidth',2)
85 hold on
86 grid on
87 plot(t,y(:,7),'LineWidth',2)
88 legend('E_F','I_F')
89 xlabel('Time[Years]')

```

```

90 ylabel('Free Range dog Population')
91 figure(9)
92 hold on
93 grid on
94 plot(t,y(:,12),'LineWidth',2)
95
96 xlabel('Time[Years]')
97 ylabel('Rabies in the environment')
98
99 figure(10)
100 bar(t,y(:,12))
101 xlabel('Time[Years]')
102 ylabel('Rabies in the environment')
103 function dy = main_ty(t,y, rho_1,rho_2,rho_3,C,theta_1,tau_1,tau_2,gamma,tau_3,
    mu_3,beta_1,beta_2,beta_3, mu_1,sigma_1,theta_2,kappa_1,kappa_2,kappa_3,
    sigma_2,mu_2,theta_3,psi_1,psi_2,psi_3,sigma_3,gamma_1,gamma_2,gamma_3,mu_4,
    nu_1,nu_2,nu_3)
104 dy=zeros(12,1);
105
106 S_H=y(1);
107 E_H=y(2);
108 I_H=y(3);
109 R_H=y(4);
110 S_F=y(5);
111 E_F=y(6);
112 I_F=y(7);
113 S_D=y(8);
114 E_D=y(9); % assining the model equations
115 I_D=y(10);
116 R_D=y(11);
117 M=y(12);
118 %Model equations
119 dy(1)=theta_1+beta_3*R_H-mu_1*S_H-(tau_1*I_F+tau_2*I_D+tau_3*M/(M+C))*S_H;
120 dy(2)=(tau_1*I_F+tau_2*I_D+tau_3*M/(M+C))*S_H-(mu_1+beta_1+beta_2)*E_H;
121 dy(3)=beta_1*E_H-(sigma_1+mu_1)*I_H;
122 dy(4)=beta_2*E_H-(beta_3+mu_1)*R_H;
123 dy(5)=theta_2-(kappa_1*I_F+kappa_2*I_D+kappa_3*M/(M+C))*S_F-mu_2*S_F;
124 dy(6)=(kappa_1*I_F+kappa_2*I_D+kappa_3*M/(M+C))*S_F-(mu_2+gamma)*E_F;
125 dy(7)=gamma*E_D-(mu_2+sigma_2)*I_F;
126 dy(8)=theta_3-((psi_1/(1+rho_1))*I_F+(psi_2/(1+rho_2))*I_D+(psi_3*M/(1+rho_3))*(M
    +C))*S_D-mu_3*S_D+gamma_3*R_D;
127 dy(9)=((psi_1/(1+rho_1))*I_F+(psi_2/(1+rho_2))*I_D+(psi_3*M/(1+rho_3))*(M+C))*S_D
    -(mu_3+gamma_1+gamma_2)*E_D;
128 dy(10)=gamma_1*E_D-(mu_3+sigma_3)*I_D;
129 dy(11)=gamma_2*E_D-(mu_3+sigma_3)*R_D;
130 dy(12)=(nu_1*I_H+nu_2*I_F+nu_3*I_D)-mu_4*M;
131
132 end

```

APPENDIX II : MATLAB Code for Parameter Estimation

```
1 clc;
2 clear all
3 close all
4 % Parameters
5 par = [10, 8, 15, 2000, 0.0004, 0.0006, 0.0003, 1/6, 1, 0.54, ...
6        0.0142, 1, 1000, 1/6, 0.00002, 0.00008, 0.00004, 0.09, ...
7        0.067, 1200, 0.00009, 0.00007, 0.00003, 0.09, 1/6, 0.09, ...
8        0.05, 0.08, 0.002, 0.009, 0.005, 0.08, 0.003];
9
10 % Time
11 time = linspace(0, 100, 500);
12
13 % Initial conditions
14 y0 = [142000, 40, 0, 0, 15000, 20, 0, 12500, 25, 0, 0, 90];
15 %y0=[142000,40,3,0,15000,10,0,12500,40,5,0,90];
16
17 % ODE solver
18 [t, x] = ode45(@(t,y) model_1(t, y, par), time, y0);
19
20 % Adding noise
21 sigy = 0.05;
22 yy = x.*(1 + randn(size(x)) * sigy);
23
24 % Data structure
25 data.ydata = yy;
26 data.tdata = time;
27 data.y0data = y0;
28
29 % Optimization
30 theta0 = par;
31 opt = optimset('maxiter', 500, 'Display', 'iter');
32 [estimate, ~] = fminsearch(@LSM, theta0, opt, data);
33
34 % Compute the mean and standard deviation for each parameter
35 mean_estimate = mean(estimate);
36 std_dev_estimate = std(estimate);
37
38 % Display the results
39 for i = 1:length(par)
40     disp([sprintf('Parameter %2d:', i), sprintf('%15.6f', estimate(i)), sprintf('
41         %15.6f', mean_estimate), sprintf('%15.6f', std_dev_estimate)]);
42 end
43 % Display the results
44 fprintf('Parameter    Estimated Value        Mean        Std. Deviation\n');
45 fprintf('-----\n');
46 for i = 1:length(par)
47     lower_bound = estimate(i) - std_dev_estimate;
```

```

47     upper_bound = estimate(i) + std_dev_estimate;
48     disp([sprintf('%9d', i), sprintf('%20.6f', estimate(i)), sprintf('%12.6f',
        lower_bound), sprintf('%12.6f', upper_bound)]);
49 end
50
51
52
53 % ODE solver with optimized parameters
54 [~, x_est] = ode45(@(t,y) model_1(t, y, estimate), time, y0);
55
56 % Residuals
57 r = yy - x_est;
58
59
60 figure;
61 variable_labels_humans = {'S_H', 'E_H', 'I_H', 'R_H'};
62 for i = 1:4
63     subplot(2, 2, i);
64     scatter(data.tdata, data.ydata(:, i), 'b', 'filled');
65     hold on;
66     plot(t, x(:, i), 'r', 'LineWidth', 2);
67     plot(t, x_est(:, i), '—k', 'LineWidth', 2);
68     xlabel('Time(Years)');
69     ylabel(sprintf( variable_labels_humans{i})); %'domestic Dogs: %s'
70     legend('Noisy Data', 'Initial Parameter', 'Estimated Parameter');
71     title(sprintf( variable_labels_humans{i})); %'domestic Dogs: %s'
72     grid on;
73     hold off;
74 end
75 figure;
76 variable_labels_free_range_dogs = {'S_F', 'E_F', 'I_F'};
77 for i = 1:3
78     subplot(1, 3, i);
79     scatter(data.tdata, data.ydata(:, i+4), 'b', 'filled');
80     hold on;
81     plot(t, x(:, i+4), 'r', 'LineWidth', 2);
82     plot(t, x_est(:, i+4), '—k', 'LineWidth', 2);
83     xlabel('Time(Years)');
84     ylabel(sprintf( variable_labels_free_range_dogs{i})); %'domestic Dogs: %s'
85     legend('Noisy Data', 'Initial Parameter', 'Estimated Parameter');
86     title(sprintf( variable_labels_free_range_dogs{i})); %'domestic Dogs: %s'
87     grid on;
88     hold off;
89 end
90 figure;
91 variable_labels_domestic_dogs = {'S_D', 'E_D', 'I_D', 'R_D'};
92 for i = 1:4
93     subplot(2, 2, i);
94     scatter(data.tdata, data.ydata(:, i+7), 'b', 'filled');

```



```

95     hold on;
96     plot(t, x(:, i+7), 'r', 'LineWidth', 2);
97     plot(t, x_est(:, i+7), '—k', 'LineWidth', 2);
98     xlabel('Time(Years)');
99     ylabel(sprintf( variable_labels_domestic_dogs{i})); %'domestic Dogs: %s'
100    legend('Noisy Data', 'Initial Parameter', 'Estimated Parameter');
101    title(sprintf( variable_labels_domestic_dogs{i})); %'domestic Dogs: %s'
102    grid on;
103    hold off;
104 end
105
106 % Plot variable M separately
107 figure;
108 %subplot(1, 2, 1);
109 plot(t, x(:, 12), 'r', 'LineWidth', 2);
110 hold on;
111 plot(t, x_est(:, 12), '—k', 'LineWidth', 2);
112 scatter(data.tdata, data.ydata(:, 12), 'b', 'filled');
113 title('M');
114 xlabel('Time (Years)');
115 ylabel('Variable M');
116 legend('Model', 'Fitted', 'Actual');
117 grid on;
118
119 figure;
120 variable_labels = {'S_H', 'E_H', 'I_H', 'R_H'};
121 for i = 1:4
122     subplot(2, 2, i);
123     histogram(r(:, i), 'Normalization', 'probability');
124     title(sprintf(variable_labels{i})); %'Residuals for %s',
125     xlabel('Residual');
126     ylabel('Probability');
127     grid on;
128
129     % Compute histogram values for line plot
130     [counts, edges] = histcounts(r(:, i));
131     centers = (edges(1:end-1) + edges(2:end)) / 2;
132     values = counts / sum(counts);
133
134     % Plot line plot
135     hold on;
136     plot(centers, values, 'm-', 'LineWidth', 2);
137     %legend('Histogram', 'Line Plot');
138 end
139 variable_labels_domestic_dogs = {'S_D', 'E_D', 'I_D', 'R_D'};
140
141 figure;
142 for i = 1:4
143     subplot(2, 2, i);

```

```

144
145 % Plot all three datasets for this variable
146 scatter(data.tdata, data.ydata(:, i+7), 40, 'b', 'filled', 'MarkerFaceAlpha',
147         0.7);
148 hold on;
149 plot(t, x(:, i+7), 'r', 'LineWidth', 2);
150 plot(t, x_est(:, i+7), '—k', 'LineWidth', 2);
151
152 xlabel('Time (Years)');
153 ylabel('Population');
154 title(variable_labels_domestic_dogs{i});
155 legend('Noisy Data', 'Initial Parameter', 'Estimated Parameter', 'Location',
156        'best');
157 grid on;
158 hold off;
159 end
160
161 figure;
162 variable_labels_free_range = {'S_F', 'E_F', 'I_F'};
163 for i = 1:3
164     subplot(1, 3, i);
165     histogram(r(:, i+4), 'Normalization', 'probability');
166     title(sprintf( variable_labels_free_range{i})); %'Residuals for Free Range
167         Dogs: %s',
168     xlabel('Residual');
169     ylabel('Probability');
170     grid on;
171
172     % Compute histogram values for line plot
173     [counts, edges] = histcounts(r(:, i+4));
174     centers = (edges(1:end-1) + edges(2:end)) / 2;
175     values = counts / sum(counts);
176
177     % Plot line plot
178     hold on;
179     plot(centers, values, 'm-', 'LineWidth', 2);
180     %legend('Histogram', 'Line Plot');
181 end
182
183 figure;
184 variable_labels_domestic_dogs = {'S_D', 'E_D', 'I_D', 'R_D'};
185 for i = 1:4
186     subplot(2, 2, i);
187     histogram(r(:, i+7), 'Normalization', 'probability');
188     title(sprintf( variable_labels_domestic_dogs{i})); %'Residuals for Domestic
189         Dogs: %s',
190     xlabel('Residual');
191     ylabel('Probability');

```

```

189     grid on;
190
191     % Compute histogram values for line plot
192     [counts, edges] = histcounts(r(:, i+7));
193     centers = (edges(1:end-1) + edges(2:end)) / 2;
194     values = counts / sum(counts);
195
196     % Plot line plot
197     hold on;
198     plot(centers, values, 'm-', 'LineWidth', 2);
199     %legend('Histogram', 'Line Plot');
200 end
201
202
203 figure;
204 histogram(r(:, 12), 'Normalization', 'probability');
205 title('M');
206 xlabel('Residual');
207 ylabel('Probability');
208 grid on;
209
210 % Compute histogram values for line plot
211 [counts, edges] = histcounts(r(:, 12));
212 centers = (edges(1:end-1) + edges(2:end)) / 2;
213 values = counts / sum(counts);
214
215 % Plot line plot
216 hold on;
217 plot(centers, values, 'm-', 'LineWidth', 2);
218 %legend('Histogram', 'Line Plot');
219 % Compute the posterior distribution
220 posterior = fitdist(r(:), 'Kernel');
221
222 % Plotting the four variables in a subplot
223 % (Existing code for plotting other variables goes here...)
224 % QQ plots for Humans
225 figure;
226 subplot(2, 2, 1); % Subplot for S_H
227 qqplot(r(:, 1)); % QQ plot for susceptible Humans (S_H)
228 title('QQ Plot for Susceptible Humans (S_H)');
229
230 subplot(2, 2, 2); % Subplot for E_H
231 qqplot(r(:, 2)); % QQ plot for Exposed Humans (S_H)
232 title('QQ Plot for Exposed Humans (E_H)');
233
234 subplot(2, 2, 3); % Subplot for I_H
235 qqplot(r(:, 3)); % QQ plot for Infected Humans (I_H)
236 title('QQ Plot for Infected Humans (I_H)');
237

```

```

238 subplot(2, 2, 4); % Subplot for R_H
239 qqplot(r(:, 4)); % QQ plot for Recovered Humans (R_H)
240 title('QQ Plot for Recovered Humans (R_H)');
241
242 % Adjust the layout for better visibility
243 % sgtitle('QQ Plots for Humans');
244
245 % QQ plots for Free-Range Dogs
246 figure;
247
248 subplot(1, 3, 1); % Subplot for S_F
249 qqplot(r(:, 5)); % QQ plot for Susceptible Free-Range (S_F)
250 title('QQ Plot for Susceptible Free-Range Dogs (S_F)');
251
252 subplot(1, 3, 2); % Subplot for E_F
253 qqplot(r(:, 6)); % QQ plot for Susceptible Free-Range (S_F)
254 title('QQ Plot for Exposed Free-Range Dogs (S_F)');
255
256 subplot(1, 3, 3); % Subplot for I_F
257 qqplot(r(:, 7)); % QQ plot for infected Free-Range (E_F)
258 title('QQ Plot for infected Free-Range Dogs (E_F)');
259
260 % Adjust the layout for better visibility
261 % sgtitle('QQ Plots for Free-Range Dogs');
262
263 % QQ plots for Domestic Dogs
264 figure;
265 subplot(2, 2, 1); % Subplot for S_D
266 qqplot(r(:, 8)); % QQ plot for Susceptible domestic dog (S_D)
267 title('QQ Plot for Infected Free-Range Dogs (I_F)');
268
269 subplot(2, 2, 2); % Subplot for E_D
270 qqplot(r(:, 9)); % QQ plot for Exposed Domestic (E_D)
271 title('QQ Plot for Exposed Domestic Dogs (E_D)');
272
273 subplot(2, 2, 3); % Subplot for I_D
274 qqplot(r(:, 10)); % QQ plot for Infected Domestic (I_D)
275 title('QQ Plot for Exposed Domestic Dogs (E_D)');
276
277 subplot(2, 2, 4); % Subplot for R_D
278 qqplot(r(:, 11)); % QQ plot for Recovered Domestic (R_D)
279 title('QQ Plot for Recovered Domestic Dogs (R_D)');
280 figure;
281 qqplot(r(:, 12)); % QQ plot for environment concentration (M)
282 title('QQ Plot for concentrated environment ,M');
283
284 figure;
285 variable_labels = {'S_H', 'E_H', 'I_H', 'R_H'};
286 for i = 1:4

```

```

287     subplot(2, 2, i, 'LineWidth', 2);
288     autocorr(r(:, i));
289     title(sprintf( variable_labels{i})); %'Autocorrelation of Residuals for %s',
290     xlabel('Lag');
291     ylabel('Sample Autocorrelation');
292     grid on;
293 end
294
295 % Plot autocorrelation of residuals for Free Range Dogs variables
296 figure;
297 variable_labels_free_range = {'S_F', 'E_F', 'I_F'};
298 for i = 1:3
299     subplot(1, 3, i, 'LineWidth', 2);
300     autocorr(r(:, i+4));
301     title(sprintf( variable_labels_free_range{i})); %'Autocorrelation of
302     Residuals for Free Range Dogs: %s',
303     xlabel('Lag');
304     ylabel('Sample Autocorrelation');
305     grid on;
306 end
307
308 % Plot autocorrelation of residuals for Domestic Dogs variables
309 figure;
310 variable_labels_domestic_dogs = {'S_D', 'E_D', 'I_D', 'R_D'};
311 for i = 1:4
312     subplot(2, 2, i, 'LineWidth', 2);
313     autocorr(r(:, i+7));
314     title(sprintf(variable_labels_domestic_dogs{i})); %'Autocorrelation of
315     Residuals for Domestic Dogs: %s',
316     xlabel('Lag');
317     ylabel('Sample Autocorrelation');
318     grid on;
319 end
320
321 % Plot autocorrelation of residuals for Variable M
322 figure;
323 autocorr(r(:, 12));
324 title('M'); %Autocorrelation of Residuals for Free Range Dogs: Variable
325 xlabel('Lag');
326 ylabel('Sample Autocorrelation');
327 grid on;
328
329 function dy = model_1(~, y, par)
330     % Parameters
331     rho_1 = par(1); rho_2 = par(2); rho_3 = par(3);
332     theta_1 = par(4); tau_1 = par(5); tau_2 = par(6); tau_3 = par(7);
333     beta_1 = par(8); beta_2 = par(9); beta_3 = par(10); mu_1 = par(11);
334     sigma_1 = par(12); theta_2 = par(13); gamma = par(14);
335     kappa_1 = par(15); kappa_2 = par(16); kappa_3 = par(17);

```

```

334 sigma_2 = par(18); mu_2 = par(19); theta_3 = par(20);
335 psi_1 = par(21); psi_2 = par(22); psi_3 = par(23);
336 sigma_3 = par(24); gamma_1 = par(25); gamma_2 = par(26);
337 gamma_3 = par(27); mu_3 = par(28); nu_1 = par(29);
338 nu_2 = par(30); nu_3 = par(31); mu_4 = par(32); C = par(33);
339
340 % Variables
341 S_H = y(1); E_H = y(2); I_H = y(3); R_H = y(4);
342 S_F = y(5); E_F = y(6); I_F = y(7);
343 S_D = y(8); E_D = y(9); I_D = y(10); R_D = y(11);
344 M = y(12);
345
346 % Model equations
347 dy = zeros(size(y)); % Initialize output vector
348 dy(1) = theta_1 + beta_3 * R_H - mu_1 * S_H - (tau_1 * I_F + tau_2 * I_D +
    tau_3 * M / (M + C)) * S_H;
349 dy(2) = (tau_1 * I_F + tau_2 * I_D + tau_3 * M / (M + C)) * S_H - (mu_1 +
    beta_1 + beta_2) * E_H;
350 dy(3) = beta_1 * E_H - (sigma_1 + mu_1) * I_H;
351 dy(4) = beta_2 * E_H - (beta_3 + mu_1) * R_H;
352 dy(5) = theta_2 - (kappa_1 * I_F + kappa_2 * I_D + kappa_3 * M / (M + C)) *
    S_F - mu_2 * S_F;
353 dy(6) = (kappa_1 * I_F + kappa_2 * I_D + kappa_3 * M / (M + C)) * S_F - (mu_2
    + gamma) * E_F;
354 dy(7) = gamma * E_F - (mu_2 + sigma_2) * I_F;
355 dy(8) = theta_3 - ((psi_1 / (1 + rho_1)) * I_F + (psi_2 / (1 + rho_2)) * I_D
    + (psi_3 * M / (1 + rho_3)) * (M + C)) * S_D - mu_3 * S_D + gamma_3 * R_D
    ;
356 dy(9) = ((psi_1 / (1 + rho_1)) * I_F + (psi_2 / (1 + rho_2)) * I_D + (psi_3 *
    M / (1 + rho_3)) * (M + C)) * S_D - (mu_3 + gamma_1 + gamma_2) * E_D;
357 dy(10) = gamma_1 * E_D - (mu_3 + sigma_3) * I_D;
358 dy(11) = gamma_2 * E_D - (mu_3 + sigma_3) * R_D;
359 dy(12) = (nu_1 * I_H + nu_2 * I_F + nu_3 * I_D) - mu_4 * M;
360 dy = dy(:); % Ensure dy is a column vector
361 end
362
363 function ss = LSM(theta, data)
364 % ODE solver
365 [~, y] = ode45(@(t,y) model_1(t, y, theta), data.tdata, data.y0data);
366
367 % Residuals
368 res = data.ydata - y;
369
370 % Sum of squared residuals
371 ss = sum(sum(res.^2));
372 end

```

APPENDIX III: MATLAB Code for CTMC Model

```
1 %CTCM for transmission dynamics of rabies
2 %By Mfano Charles
3 %College of Busines Education , P.O. BOX 1968 DSM , +255783625429
4 %PhD Student (AMCS) – NM–AIST, Arusha, Tz
5 %21 June, 2024
6 format short
7 clear all
8 close all
9 clc;
10 set(0,'DefaultAxesFontSize', 12)%Increases axes labels
11 set(gca,'fontsize',18);%Changes font size in a figure.
12 %
13 % Parameter values of the model
14 time=99; %time
15 %
16 rho_1=10; %Deterrent coeffiecient of domestic dogs from I_F
17 rho_2=8; %Deterrent coeffiecient of domestic dogs from I_D
18 rho_3=15; %Deterrent coeffiecient of domestic dogs from M
19 theta_1=2000; % recurement of susceptible human
20 tau_1=0.0004; %The rate that S_H get infections from I_F
21 tau_2=0.0006; %The rate S_H get infection from I_D %paremeter Estimation
22 tau_3=0.0003; %The rate that S_H get infection from M
23 beta_1=1/6; %progression rate out of E_H to I_H(Incubation period of human)
24 beta_2=1; %Recovery rate of E_H
25 beta_3=0.54; %Rate of immunity loss of human being
26 mu_1=0.0142; %Natural death rate of human
27 sigma_1=1; %Disease induced rate of I_H
28 theta_2=1000; %Recruitment rate of free range dogs
29 gamma=1/6; %Progression rate of E_F to I_F
30 kappa_1=0.0002; %The rate that S_F becomes I_F from I_F
31 kappa_2=0.0008;%The rate that S_F bceomes infection from I_D
32 kappa_3=0.00004; %The rate that S_F gets infection form M
33 sigma_2=0.09;% Disease induced death rate of I_F
34 mu_2=0.067; %Natural Mortality rate of free-range dogs
35 theta_3=1200;%Recruitment rate of domestic dog population
36 psi_1=0.0009; %The rate that S_D get infection from I_F0.0009
37 psi_2=0.0007; %The rate that S_D get infection from I_D
38 psi_3=0.00003; %The rate that S_D get infection from M
39 sigma_3= 0.09;%1; %Disease induced death rate for I_D
40 gamma_1=1/6; %The rate at which E_D becomes I_D
41 gamma_2=0.09; %Recovery rate of E_D
42 gamma_3=0.05; %The rate of loss of immunity of E_D
43 mu_3=0.08; % The naural Mortality rate of domestic dogs
44 nu_1=0.002; %Environment shedding rate from I_H
45 nu_2=0.009; %Environment shedding rate from I_F
46 nu_3=0.005; %Environment shedding rate from I_D
47 mu_4=0.08; %Naural removal rate of rabies
```

```

48 C=0.003;      % Rabies concetration in the environment
49 %
50 S_H= theta_1/mu_1;
51 S_F= theta_2/mu_2;
52 S_D= theta_3/mu_3;
53 %
54 %X=S_H; X1=E_H; X2=I_H; X3=R_H; P=S_F; P1=E_F; P2=I_F; Y=S_D;Y1=E_D;Y2=I_D;
55 %Y3=R_D; Z=M
56 %
57 %x=S_H; x1=E_H; x2=I_H; x3=R_H; p=S_F; p1=E_F; p2=I_F; y=S_D;y1=E_D;y2=I_D;
58 %y3=R_D; z=M
59 %
60 dt=0.05; tim=time/dt; kk=0:dt:time;
61 %Initial conditions
62 a0=2; b0=1;c0=1; d0=1; e0=2;f0=1;g0=1;q0=2; k0=1; h0=1;
63 m0=1;n0=1;
64 %
65 x(1)=a0;
66 x1(1)=b0;
67 x2(1)=c0;
68 x3(1)=d0;
69 p(1)=e0;
70 p1(1)=f0;
71 p2(1)=g0;
72 y(1)=q0;
73 y1(1)=k0;
74 y2(1)=h0;
75 y3(1)=m0;
76 z(1)=n0;
77 %
78 x(1)=S_H-(b0+c0+d0);
79 p(1)=S_F-(f0+g0);
80 y(1)=S_D-(k0+h0+m0);
81 %
82 for jj=1:tim
83 % HUMAN POPULATION
84 x(jj+1) = x(jj) + dt * (theta_1 + beta_3 * x3(jj) - mu_1 * x(jj) - (tau_1 *
      p2(jj) + tau_2 * y2(jj) + tau_3 * z(jj) / (z(jj) + C)) * x(jj));
85 x1(jj+1) = x1(jj) + dt * ((tau_1 * p2(jj) + tau_2 * y2(jj) + tau_3 * z(jj) /
      (z(jj) + C)) * x(jj) - (mu_1 + beta_1 + beta_2) * x1(jj));
86 x2(jj+1) = x2(jj) + dt * (beta_1 * x1(jj) - (sigma_1 + mu_1) * x2(jj));
87 x3(jj+1) = x3(jj) + dt * (beta_2 * x1(jj) - (beta_3 + mu_1) * x3(jj));
88
89 % FREE RANGE POPULATION
90 p(jj+1) = p(jj) + dt * (theta_2 - (kappa_1 * p2(jj) + kappa_2 * y2(jj) +
      kappa_3 * z(jj) / (z(jj) + C)) * p(jj) - mu_2 * p(jj));
91 p1(jj+1) = p1(jj) + dt * ((kappa_1 * p2(jj) + kappa_2 * y2(jj) + kappa_3 * z(
      jj) / (z(jj) + C)) * p(jj) - (mu_2 + gamma) * p1(jj));
92 p2(jj+1) = p2(jj) + dt * (gamma * p1(jj) - (mu_2 + sigma_2) * p2(jj));

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93
94 % DOMESTIC DOG POPULATION
95 y(jj+1) = y(jj) + dt * (theta_3 - ((psi_1 / (1 + rho_1)) * p2(jj) + (psi_2 /
    (1 + rho_2)) * y2(jj) + (psi_3 * z(jj) / (1 + rho_3)) * (z(jj) + C)) * y(
    jj) - mu_3 * y(jj) + gamma_3 * y3(jj));
96 y1(jj+1) = y1(jj) + dt * (((psi_1 / (1 + rho_1)) * p2(jj) + (psi_2 / (1 +
    rho_2)) * y2(jj) + (psi_3 * z(jj) / (1 + rho_3)) * (z(jj) + C)) * y(jj) -
    (mu_3 + gamma_1 + gamma_2) * y1(jj));
97 y2(jj+1) = y2(jj) + dt * (gamma_1 * y1(jj) - (mu_3 + sigma_3) * y2(jj));
98 y3(jj+1) = y3(jj) + dt * (gamma_2 * y1(jj) - (mu_3 + sigma_3) * y3(jj));
99
100 % RABIES IN THE ENVIRONMENT
101 z(jj+1) = z(jj) + dt * ((nu_1 * x2(jj) + nu_2 * p2(jj) + nu_3 * y2(jj)) -
    mu_4 * z(jj));
102 %
103 end
104 for k=1:4
105 clear t X X1 X2 X3 P P1 P2 Y Y1 Y2 Y3 Z
106 j=1;
107 X(1)=a0;
108 X1(1)=b0;
109 X2(1)=c0;
110 X3(1)=d0;
111 P(1)=e0;
112 P1(1)=f0;
113 P2(1)=g0;
114 Y(1)=q0;
115 Y1(1)=k0;
116 Y2(1)=h0;
117 Y3(1)=m0;
118 Z(1)=n0;
119 X(1)=S_H-(b0+c0+d0);
120 Y(1)=S_F-(e0+f0);
121 P(1)=S_D-(k0+h0+m0);
122 t(1)=0; %Starting at zero
123 %
124 while (X(j)+X1(j)+X2(j)+X3(j)+P(j)+P1(j)+P2(j)+Y1(j)+Y2(j)+Y3(j)+Z(j))>0 && t(j)<
    time
125
126 % Stop if it hits zero or at end time
127 randm1=rand; %Uniform random number
128 randm2=rand; %Uniform random number
129 %
130 NH(j)=X(j)+X1(j)+X2(j)+X3(j);
131 NF(j)=P(j)+P1(j)+P2(j);
132 ND(j)=Y(j)+Y1(j)+Y2(j)+Y3(j);
133 %
134 %Total sum of rates of all possible events at time (t+1)

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135 Total = theta_1 + beta_3.*X3(j) + mu_1.*NH(j) + tau_1.*P2(j).*X(j) + tau_2.*Y2(j)
    .*X(j) + (tau_3.*Z(j))./(Z(j) + C).*X(j) + beta_1.*X1(j) + ...
136 kappa_1.*P2(j).*P(j) + kappa_2.*Y2(j).*P(j) + (kappa_3.*Z(j))./(Z(j) + C).*P(j)
    ) + mu_2.*NF(j) + gamma.*P1(j) + sigma_2.*P2(j) + theta_3 + ...
137 ((psi_1)./(1 + rho_1).*P2(j)).*Y(j) + ((psi_2)./(1 + rho_2)).*Y2(j).*Y(j) +
    ((psi_3.*Z(j))./(1 + rho_3).*(Z(j) + C))).*Y(j)) + ...
138 mu_3.*ND(j) + gamma_3.*Y3(j) + (gamma_1 + gamma_2).*Y1(j) + sigma_3.*Y2(j) +
    nu_1.*X2(j) + nu_2.*P2(j) + nu_3.*Y2(j) + ...
139 mu_4.*Z(j) + beta_2.*X1(j) + sigma_1.*X2(j) + theta_2;
140 %
141 %Defining all possible events at time (t+1)
142 ev1 = theta_1/Total;
143 ev2 = ev1 + mu_1*X(j)/Total;
144 ev3 = ev2 + tau_1*P2(j)*X(j)/Total;
145 ev4 = ev3 + tau_2*Y2(j)*X(j)/Total;
146 ev5 = ev4 + (((Z(j)/(Z(j)+C))*tau_3*X(j)))/Total;
147 ev6 = ev5 + beta_1*X1(j)/Total;
148 ev7 = ev6 + beta_2*X1(j)/Total;
149 ev8 = ev7 + mu_1*X1(j)/Total;
150 ev9 = ev8 + sigma_1*X2(j)/Total;
151 ev10 = ev9 + mu_1*X2(j)/Total;
152 ev11 = ev10 + beta_3*X3(j)/Total;
153 ev12 = ev11 + mu_1*X3(j)/Total;
154 %
155 ev13 = ev12 + theta_2/Total;
156 ev14 = ev13 + mu_2*P(j)/Total;
157 ev15 = ev14 + kappa_1*P2(j)*P(j)/Total;
158 ev16 = ev15 + kappa_2*Y2(j)*P(j)/Total;
159 ev17= ev16 + ((Z(j)/(Z(j)+C))*kappa_3*P(j))/Total;
160 ev18= ev17+ gamma*P1(j)/Total;
161 ev19= ev18 + mu_2*P1(j)/Total;
162 ev20= ev19 + mu_2*P2(j)/Total;
163 ev21= ev20 + sigma_2*P2(j)/Total;
164 %
165 ev22 = ev21 + theta_3/Total;
166 ev23 = ev22 + mu_3*Y(j)/Total;
167 ev24 = ev23 + ((psi_1/(1+rho_1))*P2(j)*Y(j))/Total;
168 ev25 = ev24 + ((psi_2/(1+rho_2))*Y2(j)*Y(j))/Total;
169 ev26= ev25 + ((psi_1/(1+rho_1))*(Z(j)/(Z(j)+C))*P2(j)*Y(j))/Total;
170 ev27= ev26+ gamma_1*Y1(j)/Total;
171 ev28= ev27 + gamma_2*Y1(j)/Total;
172 ev29= ev28 + mu_3*Y1(j)/Total;
173 ev30= ev29 + mu_3*Y2(j)/Total;
174 ev31= ev30 + sigma_3*Y2(j)/Total;
175 ev32= ev31 + mu_3*Y3(j)/Total;
176 ev33 = ev32 + gamma_3*Y3(j)/Total;
177 %
178 ev34= ev33+ nu_1*X2(j)/Total;
179 ev35= ev34+ nu_2*P2(j)/Total;

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180 ev36= ev35+ nu_3*Y2(j)/Total;
181 %
182 t(j+1)=t(j)-log(randm1)/Total; % Time to next event
183 %
184 %Computing probabilities of each event occuring at time (t+1)
185 if randm2<ev1; % Recruitment of SH
186 X(j+1) = X(j)+1;
187 X1(j+1) = X1(j);
188 X2(j+1) = X2(j);
189 X3(j+1) = X3(j);
190 P(j+1) = P(j);
191 P1(j+1) = P1(j);
192 P2(j+1) = P2(j);
193 Y(j+1) = Y(j);
194 Y1(j+1) = Y1(j);
195 Y2(j+1) = Y2(j);
196 Y3(j+1) = Y3(j);
197 Z(j+1) = Z(j);
198 elseif randm2>=ev1 & randm2<ev2 % Natural death of S_H
199 X(j+1) = X(j)-1;
200 X1(j+1) = X1(j);
201 X2(j+1) = X2(j);
202 X3(j+1) = X3(j);
203 P(j+1) = P(j);
204 P1(j+1) = P1(j);
205 P2(j+1) = P2(j);
206 Y(j+1) = Y(j);
207 Y1(j+1) = Y1(j);
208 Y2(j+1) = Y2(j);
209 Y3(j+1) = Y3(j);
210 Z(j+1) = Z(j);
211 elseif randm2>=ev2 & randm2<ev3 % SH contact with IF
212 X(j+1) = X(j)-1;
213 X1(j+1) = X1(j)+1;
214 X3(j+1) = X3(j);
215 X2(j+1) = X2(j);
216 P(j+1) = P(j);
217 P1(j+1) = P1(j);
218 P2(j+1) = P2(j);
219 Y(j+1) = Y(j);
220 Y1(j+1) = Y1(j);
221 Y2(j+1) = Y2(j);
222 Y3(j+1) = Y3(j);
223 Z(j+1) = Z(j);
224 elseif randm2>=ev3 & randm2<ev4 % Inf. of SH from ID.
225 X(j+1) = X(j)-1;
226 X1(j+1) = X1(j)+1;
227 X2(j+1) = X2(j);
228 X3(j+1) = X3(j);

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229 P(j+1) = P(j);
230 P1(j+1) = P1(j);
231 P2(j+1) = P2(j);
232 Y(j+1) = Y(j);
233 Y1(j+1) = Y1(j);
234 Y2(j+1) = Y2(j);
235 Y3(j+1) = Y3(j);
236 Z(j+1) = Z(j);
237 elseif randm2>=ev4 & randm2<ev5 % Inf. of SH from Env.
238 X(j+1) = X(j)-1;
239 X1(j+1) = X1(j)+1;
240 X2(j+1) = X2(j);
241 X3(j+1) = X3(j);
242 P(j+1) = P(j);
243 P1(j+1) = P1(j);
244 P2(j+1) = P2(j);
245 Y(j+1) = Y(j);
246 Y1(j+1) = Y1(j);
247 Y2(j+1) = Y2(j);
248 Y3(j+1) = Y3(j);
249 Z(j+1) = Z(j);
250 elseif randm2>=ev5 & randm2<ev6 % Progression of EH to IH
251 X(j+1) = X(j);
252 X1(j+1) = X1(j)-1;
253 X2(j+1) = X2(j)+1;
254 X3(j+1) = X3(j);
255 P(j+1) = P(j);
256 P1(j+1) = P1(j);
257 P2(j+1) = P2(j);
258 Y(j+1) = Y(j);
259 Y1(j+1) = Y1(j);
260 Y2(j+1) = Y2(j);
261 Y3(j+1) = Y3(j);
262 Z(j+1) = Z(j);
263 elseif randm2>=ev6 & randm2<ev7 % recovery of EH
264 X(j+1) = X(j);
265 X1(j+1) = X1(j)-1;
266 X2(j+1) = X2(j);
267 X3(j+1) = X3(j)+1;
268 P(j+1) = P(j);
269 P1(j+1) = P1(j);
270 P2(j+1) = P2(j);
271 Y(j+1) = Y(j);
272 Y1(j+1) = Y1(j);
273 Y2(j+1) = Y2(j);
274 Y3(j+1) = Y3(j);
275 Z(j+1) = Z(j);
276 elseif randm2>=ev7 & randm2<ev8 % Natural death of EH
277 X(j+1) = X(j);

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278 X1(j+1) = X1(j);
279 X2(j+1) = X2(j)-1;
280 X3(j+1) = X3(j);
281 P(j+1) = P(j);
282 P1(j+1) = P1(j);
283 P2(j+1) = P2(j);
284 Y(j+1) = Y(j);
285 Y1(j+1) = Y1(j);
286 Y2(j+1) = Y2(j);
287 Y3(j+1) = Y3(j);
288 Z(j+1) = Z(j);
289 elseif randm2>=ev8 & randm2<ev9 %Disease Ind. Death of IH
290 X(j+1) = X(j);
291 X1(j+1) = X1(j);
292 X2(j+1) = X2(j)-1;
293 X3(j+1) = X3(j);
294 P(j+1) = P(j);
295 P1(j+1) = P1(j);
296 P2(j+1) = P2(j);
297 Y(j+1) = Y(j);
298 Y1(j+1) = Y1(j);
299 Y2(j+1) = Y2(j);
300 Y3(j+1) = Y3(j);
301 Z(j+1) = Z(j);
302 elseif randm2>=ev9 & randm2<ev10 %Natural death of IH
303 X(j+1) = X(j);
304 X1(j+1) = X1(j);
305 X2(j+1) = X2(j)-1;
306 X3(j+1) = X3(j);
307 P(j+1) = P(j);
308 P1(j+1) = P1(j);
309 P2(j+1) = P2(j);
310 Y(j+1) = Y(j);
311 Y1(j+1) = Y1(j);
312 Y2(j+1) = Y2(j);
313 Y3(j+1) = Y3(j);
314 Z(j+1) = Z(j);
315 elseif randm2>=ev10 & randm2<ev11 %Rate of loss of immunity of RH
316 X(j+1) = X(j)+1;
317 X1(j+1) = X1(j);
318 X2(j+1) = X2(j);
319 X3(j+1) = X3(j)-1;
320 P(j+1) = P(j);
321 P1(j+1) = P1(j);
322 P2(j+1) = P2(j);
323 Y(j+1) = Y(j);
324 Y1(j+1) = Y1(j);
325 Y2(j+1) = Y2(j);
326 Y3(j+1) = Y3(j);

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327 Z(j+1) = Z(j);
328 elseif randm2>=ev11 & randm2<ev12 %Natural death of RH
329 X(j+1) = X(j);
330 X1(j+1) = X1(j);
331 X2(j+1) = X2(j);
332 X3(j+1) = X3(j)-1;
333 P(j+1) = P(j);
334 P1(j+1) = P1(j);
335 P2(j+1) = P2(j);
336 Y(j+1) = Y(j);
337 Y1(j+1) = Y1(j);
338 Y2(j+1) = Y2(j);
339 Y3(j+1) = Y3(j);
340 Z(j+1) = Z(j);
341 %
342 elseif randm2>=ev12 & randm2<ev13 %Recruitment of SF
343 X(j+1) = X(j);
344 X1(j+1) = X1(j);
345 X2(j+1) = X2(j);
346 X3(j+1) = X3(j);
347 P(j+1) = P(j)+1;
348 P1(j+1) = P1(j);
349 P2(j+1) = P2(j);
350 Y(j+1) = Y(j);
351 Y1(j+1) = Y1(j);
352 Y2(j+1) = Y2(j);
353 Y3(j+1) = Y3(j);
354 Z(j+1) = Z(j);
355 elseif randm2>=ev13 & randm2<ev14 %Natural death of SF
356 X(j+1) = X(j);
357 X1(j+1) = X1(j);
358 X2(j+1) = X2(j);
359 X3(j+1) = X3(j);
360 P(j+1) = P(j)-1;
361 P1(j+1) = P1(j);
362 P2(j+1) = P2(j);
363 Y(j+1) = Y(j);
364 Y1(j+1) = Y1(j);
365 Y2(j+1) = Y2(j);
366 Y3(j+1) = Y3(j);
367 Z(j+1) = Z(j);
368 elseif randm2>=ev14 & randm2<ev15 %Conctact of SF and IF
369 X(j+1) = X(j);
370 X1(j+1) = X1(j);
371 X2(j+1) = X2(j);
372 X3(j+1) = X3(j);
373 P(j+1) = P(j)-1;
374 P1(j+1) = P1(j)+1;
375 P2(j+1) = P2(j);

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376 Y(j+1) = Y(j);
377 Y1(j+1) = Y1(j);
378 Y2(j+1) = Y2(j);
379 Y3(j+1) = Y3(j);
380 Z(j+1) = Z(j);
381 elseif randm2>=ev15 & randm2<ev16 %Contact of SF and ID
382 X(j+1) = X(j);
383 X1(j+1) = X1(j);
384 X2(j+1) = X2(j);
385 X3(j+1) = X3(j);
386 P(j+1) = P(j)-1;
387 P1(j+1) = P1(j)+1;
388 P2(j+1) = P2(j);
389 Y(j+1) = Y(j);
390 Y1(j+1) = Y1(j);
391 Y2(j+1) = Y2(j);
392 Y3(j+1) = Y3(j);
393 Z(j+1) = Z(j);
394 elseif randm2>=ev16 & randm2<ev17 %Contact between SF and Env.
395 X(j+1) = X(j);
396 X1(j+1) = X1(j);
397 X2(j+1) = X2(j);
398 X3(j+1) = X3(j);
399 P(j+1) = P(j)-1;
400 P1(j+1) = P1(j)+1;
401 P2(j+1) = P2(j);
402 Y(j+1) = Y(j);
403 Y1(j+1) = Y1(j);
404 Y2(j+1) = Y2(j);
405 Y3(j+1) = Y3(j);
406 Z(j+1) = Z(j);
407 elseif randm2>=ev17 & randm2<ev18 %Progression rate of EF to IF
408 X(j+1) = X(j);
409 X1(j+1) = X1(j);
410 X2(j+1) = X2(j);
411 X3(j+1) = X3(j);
412 P(j+1) = P(j);
413 P1(j+1) = P1(j)-1;
414 P2(j+1) = P2(j)+1;
415 Y(j+1) = Y(j);
416 Y1(j+1) = Y1(j);
417 Y2(j+1) = Y2(j);
418 Y3(j+1) = Y3(j);
419 Z(j+1) = Z(j);
420 elseif randm2>=ev18 & randm2<ev19 %Natural death of EF
421 X(j+1) = X(j);
422 X1(j+1) = X1(j);
423 X2(j+1) = X2(j);
424 X3(j+1) = X3(j);

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425 P(j+1) = P(j);
426 P1(j+1) = P1(j)–1;
427 P2(j+1) = P2(j);
428 Y(j+1) = Y(j);
429 Y1(j+1) = Y1(j);
430 Y2(j+1) = Y2(j);
431 Y3(j+1) = Y3(j);
432 Z(j+1) = Z(j);
433 elseif randm2>=ev19 & randm2<ev20 %Natural death of IF
434 X(j+1) = X(j);
435 X1(j+1) = X1(j);
436 X2(j+1) = X2(j);
437 X3(j+1) = X3(j);
438 P(j+1) = P(j);
439 P1(j+1) = P1(j);
440 P2(j+1) = P2(j)–1;
441 Y(j+1) = Y(j);
442 Y1(j+1) = Y1(j);
443 Y2(j+1) = Y2(j);
444 Y3(j+1) = Y3(j);
445 Z(j+1) = Z(j);
446 elseif randm2>=ev20 & randm2<ev21 %Disease induced of IF
447 X(j+1) = X(j);
448 X1(j+1) = X1(j);
449 X2(j+1) = X2(j);
450 X3(j+1) = X3(j);
451 P(j+1) = P(j);
452 P1(j+1) = P1(j);
453 P2(j+1) = P2(j)–1;
454 Y(j+1) = Y(j);
455 Y1(j+1) = Y1(j);
456 Y2(j+1) = Y2(j);
457 Y3(j+1) = Y3(j);
458 Z(j+1) = Z(j);
459 elseif randm2>=ev21 & randm2<ev22 %Recruitment of SD
460 X(j+1) = X(j);
461 X1(j+1) = X1(j);
462 X2(j+1) = X2(j);
463 X3(j+1) = X3(j);
464 P(j+1) = P(j);
465 P1(j+1) = P1(j);
466 P2(j+1) = P2(j);
467 Y(j+1) = Y(j)+1;
468 Y1(j+1) = Y1(j);
469 Y2(j+1) = Y2(j);
470 Y3(j+1) = Y3(j);
471 Z(j+1) = Z(j);
472 elseif randm2>=ev22 & randm2<ev23 %Natural death of SD
473 X(j+1) = X(j);

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474 X1(j+1) = X1(j);
475 X2(j+1) = X2(j);
476 X3(j+1) = X3(j);
477 P(j+1) = P(j);
478 P1(j+1) = P1(j);
479 P2(j+1) = P2(j);
480 Y(j+1) = Y(j)-1;
481 Y1(j+1) = Y1(j);
482 Y2(j+1) = Y2(j);
483 Y3(j+1) = Y3(j);
484 Z(j+1) = Z(j);
485 elseif randm2>=ev23 & randm2<ev24 %Contact of SD and IF
486 X(j+1) = X(j);
487 X1(j+1) = X1(j);
488 X2(j+1) = X2(j);
489 X3(j+1) = X3(j);
490 P(j+1) = P(j);
491 P1(j+1) = P1(j);
492 P2(j+1) = P2(j);
493 Y(j+1) = Y(j)-1;
494 Y1(j+1) = Y1(j)+1;
495 Y2(j+1) = Y2(j);
496 Y3(j+1) = Y3(j);
497 Z(j+1) = Z(j);
498 elseif randm2>=ev24 & randm2<ev25 % Contact of ID and SD
499 X(j+1) = X(j);
500 X1(j+1) = X1(j);
501 X2(j+1) = X2(j);
502 X3(j+1) = X3(j);
503 P(j+1) = P(j);
504 P1(j+1) = P1(j);
505 P2(j+1) = P2(j);
506 Y(j+1) = Y(j)-1;
507 Y1(j+1) = Y1(j)+1;
508 Y2(j+1) = Y2(j);
509 Y3(j+1) = Y3(j);
510 Z(j+1) = Z(j);
511 elseif randm2>=ev25 & randm2<ev26 % Contact of ID and Env
512 X(j+1) = X(j);
513 X1(j+1) = X1(j);
514 X2(j+1) = X2(j);
515 X3(j+1) = X3(j);
516 P(j+1) = P(j);
517 P1(j+1) = P1(j);
518 P2(j+1) = P2(j);
519 Y(j+1) = Y(j)-1;
520 Y1(j+1) = Y1(j)+1;
521 Y2(j+1) = Y2(j);
522 Y3(j+1) = Y3(j);

```

```

523 Z(j+1) = Z(j);
524 elseif randm2>=ev26 & randm2<ev27 % progression of ED to ID
525 X(j+1) = X(j);
526 X1(j+1) = X1(j);
527 X2(j+1) = X2(j);
528 X3(j+1) = X3(j);
529 P(j+1) = P(j);
530 P1(j+1) = P1(j);
531 P2(j+1) = P2(j);
532 Y(j+1) = Y(j);
533 Y1(j+1) = Y1(j)-1;
534 Y2(j+1) = Y2(j)+1;
535 Y3(j+1) = Y3(j);
536 Z(j+1) = Z(j);
537 elseif randm2>=ev27 & randm2<ev28 % recovery rate of ED
538 X(j+1) = X(j);
539 X1(j+1) = X1(j);
540 X2(j+1) = X2(j);
541 X3(j+1) = X3(j);
542 P(j+1) = P(j);
543 P1(j+1) = P1(j);
544 P2(j+1) = P2(j);
545 Y(j+1) = Y(j);
546 Y1(j+1) = Y1(j)-1;
547 Y2(j+1) = Y2(j);
548 Y3(j+1) = Y3(j)+1;
549 Z(j+1) = Z(j);
550 elseif randm2>=ev28 & randm2<ev29 % Natural death of ED
551 X(j+1) = X(j);
552 X1(j+1) = X1(j);
553 X2(j+1) = X2(j);
554 X3(j+1) = X3(j);
555 P(j+1) = P(j);
556 P1(j+1) = P1(j);
557 P2(j+1) = P2(j);
558 Y(j+1) = Y(j);
559 Y1(j+1) = Y1(j);
560 Y2(j+1) = Y2(j)-1;
561 Y3(j+1) = Y3(j);
562 Z(j+1) = Z(j);
563 elseif randm2>=ev29 & randm2<ev30 % natural death of ID
564 X(j+1) = X(j);
565 X1(j+1) = X1(j);
566 X2(j+1) = X2(j);
567 X3(j+1) = X3(j);
568 P(j+1) = P(j);
569 P1(j+1) = P1(j);
570 P2(j+1) = P2(j);
571 Y(j+1) = Y(j);

```

```

572 Y1(j+1) = Y1(j);
573 Y2(j+1) = Y2(j)-1;
574 Y3(j+1) = Y3(j);
575 Z(j+1) = Z(j);
576 elseif randm2>=ev30 & randm2<ev31 %Induced death of Inf. domestic dogs ID
577 X(j+1) = X(j);
578 X1(j+1) = X1(j);
579 X2(j+1) = X2(j);
580 X3(j+1) = X3(j);
581 P(j+1) = P(j);
582 P1(j+1) = P1(j);
583 P2(j+1) = P2(j);
584 Y(j+1) = Y(j);
585 Y1(j+1) = Y1(j);
586 Y2(j+1) = Y2(j)-1;
587 Y3(j+1) = Y3(j);
588 Z(j+1) = Z(j);
589 elseif randm2>=ev31 & randm2<ev32 % loss of immunity of RD
590 X(j+1) = X(j);
591 X1(j+1) = X1(j);
592 X2(j+1) = X2(j);
593 X3(j+1) = X3(j);
594 P(j+1) = P(j);
595 P1(j+1) = P1(j);
596 P2(j+1) = P2(j);
597 Y(j+1) = Y(j)+1;
598 Y1(j+1) = Y1(j);
599 Y2(j+1) = Y2(j);
600 Y3(j+1) = Y3(j)-1;
601 Z(j+1) = Z(j);
602 elseif randm2>=ev32 && randm2<ev33 % natural death of RD
603 X(j+1) = X(j);
604 X1(j+1) = X1(j);
605 X2(j+1) = X2(j);
606 X3(j+1) = X3(j);
607 P(j+1) = P(j);
608 P1(j+1) = P1(j);
609 P2(j+1) = P2(j);
610 Y(j+1) = Y(j)+1;
611 Y1(j+1) = Y1(j);
612 Y2(j+1) = Y2(j);
613 Y3(j+1) = Y3(j)-1;
614 Z(j+1) = Z(j);
615 elseif randm2>=ev33 & randm2<ev34 % shedding of IF to M
616 X(j+1) = X(j);
617 X1(j+1) = X1(j);
618 X2(j+1) = X2(j);
619 X3(j+1) = X3(j);
620 P(j+1) = P(j);

```

```

621 P1(j+1) = P1(j);
622 P2(j+1) = P2(j)−1;
623 Y(j+1) = Y(j);
624 Y1(j+1) = Y1(j);
625 Y2(j+1) = Y2(j);
626 Y3(j+1) = Y3(j);
627 Z(j+1) = Z(j)+1;
628 elseif randm2>=ev34 & randm2<ev35 % Shedding of ID to M
629 X(j+1) = X(j);
630 X1(j+1) = X1(j);
631 X2(j+1) = X2(j);
632 X3(j+1) = X3(j);
633 P(j+1) = P(j);
634 P1(j+1) = P1(j);
635 P2(j+1) = P2(j);
636 Y(j+1) = Y(j);
637 Y1(j+1) = Y1(j);
638 Y2(j+1) = Y2(j)−1;
639 Y3(j+1) = Y3(j);
640 Z(j+1) = Z(j)+1;
641 elseif randm2>=ev35 & randm2<ev36 % Shedding of IH to M
642 X(j+1) = X(j);
643 X1(j+1) = X1(j);
644 X2(j+1) = X2(j)−1;
645 X3(j+1) = X3(j);
646 P(j+1) = P(j);
647 P1(j+1) = P1(j);
648 P2(j+1) = P2(j);
649 Y(j+1) = Y(j);
650 Y1(j+1) = Y1(j);
651 Y2(j+1) = Y2(j);
652 Y3(j+1) = Y3(j);
653 Z(j+1) = Z(j)+1;
654 else %Removal of rabies in Envi
655 X(j+1) = X(j);
656 X1(j+1) = X1(j);
657 X2(j+1) = X2(j);
658 X3(j+1) = X3(j);
659 P(j+1) = P(j);
660 P1(j+1) = P1(j);
661 P2(j+1) = P2(j);
662 Y(j+1) = Y(j);
663 Y1(j+1) = Y1(j);
664 Y2(j+1) = Y2(j);
665 Y3(j+1) = Y3(j);
666 Z(j+1) = Z(j)−1;
667 end
668 j=j+1;
669 end

```

```

670 figure (1)
671 %subplot(2,3,6)
672 if k==1
673 plot(t,Y,'r-','linewidth',1.5)
674 hold on
675 end
676 if k==2
677 plot(t,Y,'b-','linewidth',1.5)
678 hold on
679 end
680 if k==3
681 plot(t,Y,'g-','linewidth',1.5)
682 hold on
683 end
684 if k==4
685 plot(t,Y,'g-','linewidth',1.5)
686 hold on
687 end
688 if k==5
689 plot(t,Y,'g-','linewidth',1.5)
690 hold on
691 end
692 hold on
693 end
694 %subplot(2,2,1)
695 % figure;
696 plot(kk,y,'k—','LineWidth',2)
697 xlabel('Time (Years)','FontSize',12)
698 ylabel('Susceptible domestic dogs ,S_D(t)','FontSize',12)
699 %axis([0,time,0,120])
700 %

```

APPENDIX IV: MATLAB Code for Optimal Control Model

```
1 %FILE FOR RUNNING (File Name: Control_JAM)
2 %MATLAB code for the Optimal Control of Rabies
3 %By Mfano Charles
4 %mfano.petro@cbe.ac.tz, +255783625429
5 %PhD Student (AMCS) – NM-AIST, Arusha, Tz
6 %24 June 2023
7 clc
8 clear
9 close all
10 format short
11 warning off
12 t0 = 0; tf=20; N=1000;
13 time =linspace(t0,tf,N);
14 %INITIAL POPULATION VALUES
15
16     y0=[142000,40,10,0,15000,20,0,12500,40,5,0,90];
17
18 %SECTION FOR PARAMETERS ASSUMPTION for u1=0
19 Constant=[9.920733 8.116421 14.917005 1993.382113 0.00000405
20           0.0006981326147859037 0.000030701825796768864 0.16668019240094809
21           0.54000001903285143 ...
22 0.54 0.0142 1 1004.12044 1/6 0.00002 0.00008 0.00004 1 ...
23 0.067 1203.844461 0.000009 0.00007 0.00003 0.5 1/6 0.0009 0.00005 0.000008 ...
24 0.9 0.9 0.5 0.2 0.3 10 5 1 10 ...
25 10 0 700000 25 5500 400];
26
27 % definition of variables or terms used
28 rho_1=Constant(1);rho_2 = Constant(2);rho_3= Constant(3);theta_1 = Constant(4);
29 tau_1 = Constant(5);tau_2 = Constant(6);tau_3= Constant(7);beta_1 = Constant(8);
30 beta_2 =Constant(9);beta_3=Constant(10);mu_1= Constant(11);sigma_1=Constant(12);
31 theta_2 = Constant(13);gamma = Constant(14);kappa_1=Constant(15);kappa_2=Constant
32 (16);
33 kappa_3=Constant(17);sigma_2=Constant(18);mu_2=Constant(19);theta_3 = Constant
34 (20);
35 psi_1=Constant(21);psi_2=Constant(22);psi_3=Constant(23);sigma_3=Constant(24);
36 gamma_1= Constant(25);gamma_2 = Constant(26);gamma_3 =Constant(27);mu_3=Constant
37 (28);
38 nu_1=Constant(29);nu_2=Constant(30);nu_3 =Constant(31);mu_4 =Constant(32);C =
39 Constant(33);
40 K1= Constant(34); K2 = Constant(35);K3 =Constant(36);K4 =Constant(37);
41 K5 =Constant(38);K6 =Constant(39);A1=Constant(40);A2 =Constant(41);
42 A3 =Constant(42);A4 =Constant(43);
43 %initionalization
44 lf = [0 0 0 0 0 0 0 0 0 0 0 0];
45 % TEST SECTION
46 init =y0;
47 init2 =lf;
```

```

42 h = (tf-t0)/N;
43 u = linspace(0,0,N+1);
44 u1=u'; u2=u'; u3=u'; u4=u';
45 U = [u1 u2 u3 u4];
46 % IMPLIMENTATION OF THE ALGORITHM
47 %Test 1 stoping condition 1
48 delta = 0.0000001;
49 X=init;
50 i=0; %Initialize iteration counter
51 mm=size(X);
52 NumXX =10e10;
53 Xnew = rand(N+1,mm(2)).*(repmat(X,N+1,1));
54 DenXnew=norm(Xnew);
55 while NumXX/DenXnew>delta
56 Xold = Xnew;
57 oldu = U;
58 %FORWARD RUNGE KUTTA FOR STATES
59 [Tx, X]=rk4foward(@kims,t0, tf,N, init,U,Constant);
60 %BACKWARD RUNGEKUTA FOR COSTATES
61 [Tp, P]=rk4back(@kims_costate,t0,tf,N,init2,U,X,Constant);
62 %UPDATE THE CONTROLS
63 f1 = X(1,:);g = X(2,:);h = X(3,:);j = X(4,:);l = X(5,:);
64 m = X(6,:);p = X(7,:);q = X(8,:);r = X(9,:);s = X(10,:);
65 v= X(11,:);F = X(12,:);
66 L1 = P(1,:); L2 = P(2,:); L3 = P(3,:); L4 = P(4,:); L5 = P(5,:);
67 L6 = P(6,:); L7 = P(7,:); L8 = P(8,:); L9 = P(9,:); L10 = P(10,:);
68 L11 = P(11,:); L12 = P(12,:);
69 %Implementation of all controls
70 %Case1:u1\neq 0, u2\neq 0, u3\neq 0, u4\neq 0,
71 u1 =max(0,min(1,((((((L2-L1).*f1.*((tau_2.*s+tau_1.*p+(F.*tau_3)./(F+C)))+q.*(L9-
L8).*((psi_1.*p)./(1+rho_1)+(psi_2.*s)./(1+rho_2)+(psi_3.*F)./(1+rho_3).*(F+
C))))))))./A1)));
72 u2 =max(0,min(1,((((((q.*(L9-L8).*((psi_1.*p)./(1+rho_1))+((psi_2.*s)./(1+rho_2))
+((psi_3.*F)./(1+rho_3).*(F+C))))))./A2)));
73 u3 =max(0,min(1,((((((p.*tau_1+s.*tau_2+(F.*tau_3)./(F+C)).*f1.*(L2-L1)))))./(A3
))));
74 u4 =max(0,min(1,((((r.*(L9-L11)+(g.*(L2-L4)))./A4))));
75 %Combination of vaccination of domestic dogs , public awareness and
76 %education with risk associated with rabies ,
77 %as well as the administration of post-exposure prophylaxis (PEP).
78 %Case2:u1= 0, u2\neq 0, u3\neq 0, u4\neq 0,
79 % u1 =zeros(1,N+1);
80 % u2 =max(0,min(1,((((((q.*(L9-L8).*((psi_1.*p)./(1+rho_1))+((psi_2.*s)./(1+rho_2))
+((psi_3.*F)./(1+rho_3).*(F+C))))))./A2)));
81 % u3 =max(0,min(1,((((((p.*tau_1+s.*tau_2+(F.*tau_3)./(F+C)).*f1.*(L2-L1)))))./(
A3))));
82 % u4 =max(0,min(1,((((r.*(L9-L11)+(g.*(L2-L4)))./A4))));
83 %Combination of public awareness, and education with risk associated with rabies
,

```

```

84 % as well as the administration of post-exposure prophylaxis (PEP).
85 % Case3:u1= 0, u2=0, u3\neq 0, u4\eq 0,
86 % u1 =zeros(1,N+1);
87 % u2 =zeros(1,N+1);
88 % u3 =max(0,min(1,((((p.*tau_1+s.*tau_2+(F.*tau_3)./(F+C)).*f1.*(L2-L1)))))./(
    A3))));
89 % u4 =max(0,min(1,(((r.*(L9-L11)+(g.*(L2-L4)))./A4))));
90
91 %Implemetation of administration of post-exposure prophylaxis (PEP).
92 % Case4:u1= 0, u2=0, u3=0, u4\neq 0,
93 % u1 =zeros(1,N+1);
94 % u2 =zeros(1,N+1);
95 % u3 =zeros(1,N+1);
96 % u4 =max(0,min(1,(((r.*(L9-L11)+(g.*(L2-L4)))./A4))));
97 %Advocating for the implementation of sound environmental health practices
98 %and effective management through surveillance and monitoring.
99 % Case5:u1\neq 0, u2=0, u3=0, u4=0,
100 % u1 =max(0,min(1,((((L2-L1).*f1.*((tau_2.*s+tau_1.*p+(F.*tau_3)./(F+C)))+q.*(L9
    -L8).*((psi_1.*p)./(1+rho_1)+(psi_2.*s)./(1+rho_2)+(psi_3.*F)./(1+rho_3).*(F
    +C))))))./A1));
101 % u2 =zeros(1,N+1);
102 % u3 =zeros(1,N+1);
103 % u4 =zeros(1,N+1);
104 %Combination of sound environmental health practices
105 % and effective management through surveillance and monitoring as well as
    administration of post-exposure prophylaxis (PEP)
106 % Case6:u1\neq 0, u2= 0, u3= 0, u4 \neq 0,
107 % u1 =max(0,min(1,((((L2-L1).*f1.*((tau_2.*s+tau_1.*p+(F.*tau_3)./(F+C)))+q.*(L9
    -L8).*((psi_1.*p)./(1+rho_1)+(psi_2.*s)./(1+rho_2)+(psi_3.*F)./(1+rho_3).*(F
    +C))))))./A1));
108 % u2 =zeros(1,N+1);
109 % u3 =zeros(1,N+1);
110 % u4 =max(0,min(1,(((r.*(L9-L11)+(g.*(L2-L4)))./A4))));
111 %Combination of implementation of sound environmental health practices
112 %and effective management through surveillance and monitoring as well as
    vaccination of domestic dogs.
113 % Case7:u1\neq 0, u2\neq 0, u3= 0, u4= 0,
114 % u1 =max(0,min(1,((((L2-L1).*f1.*((tau_2.*s+tau_1.*p+(F.*tau_3)./(F+C)))+q.*(L9
    -L8).*((psi_1.*p)./(1+rho_1)+(psi_2.*s)./(1+rho_2)+(psi_3.*F)./(1+rho_3).*(F
    +C))))))./A1));
115 % u2 =max(0,min(1,((((q.*(L9-L8).*((psi_1.*p)./(1+rho_1))+((psi_2.*s)./(1+rho_2)
    )+((psi_3.*F)./((1+rho_3).*(F+C))))))./A2))));
116 % u3 = zeros(1,N+1);
117 % u4=zeros(1,N+1);
118 %Combination of
119 %case 8: u1\neq 0, u2=0,u3\neq 0,u4=0
120 % u1 =max(0,min(1,((((L2-L1).*f1.*((tau_2.*s+tau_1.*p+(F.*tau_3)./(F+C)))+q.*(L9
    -L8).*((psi_1.*p)./(1+rho_1)+(psi_2.*s)./(1+rho_2)+(psi_3.*F)./(1+rho_3).*(F
    +C))))))./A1));

```



```

121 % u2 =zeros(1,N+1);
122 % u3 =max(0,min(1,((((p.*tau_1+s.*tau_2+(F.*tau_3)./(F+C)).*f1.*(L2-L1))))./(
    A3))));
123 % u4 =zeros(1,N+1);
124 %Combination of Vaccination of domestic dogs as well as public awareness ,
125 %and Education (Educating the communities about the risks associated with
126 %rabies )
127 % Case9:u1= 0, u2\neq 0, u3\neq 0, u4= 0,
128 % u1 =zeros(1,N+1);
129 % u2 =max(0,min(1,(((q.*(L9-L8).*((psi_1.*p)./(1+rho_1))+((psi_2.*s)./(1+rho_2)
    )+((psi_3.*F)./((1+rho_3).*(F+C))))))./A2)));
130 % u3 =max(0,min(1,((((p.*tau_1+s.*tau_2+(F.*tau_3)./(F+C)).*f1.*(L2-L1))))./(
    A3))));
131 % u4 =zeros(1,N+1);
132 %Combination of Vaccination of domestic dogs as well as administration of
133 %post-exposure prophylaxis (PEP)
134 % Case10:u1= 0, u2\neq 0, u3= 0, u4\neq 0,
135 % u1 =zeros(1,1+N);
136 % u2 =max(0,min(1,(((q.*(L9-L8).*((psi_1.*p)./(1+rho_1))+((psi_2.*s)./(1+rho_2)
    )+((psi_3.*F)./((1+rho_3).*(F+C))))))./A2)));
137 % u3 =zeros(1,N+1);
138 % u4 =max(0,min(1,(((r.*(L9-L11)+(g.*(L2-L4)))./A4))));
139 %Educating people communities about the risk associated with rabies and
140 %how to prevent from exposure
141 % Case11:u1= 0, u2\neq 0, u3=0, u4=0,
142 % u1 =zeros(1,N+1);
143 % u2 =zeros(1,N+1);
144 % u3 =max(0,min(1,((((p.*tau_1+s.*tau_2+(F.*tau_3)./(F+C)).*f1.*(L2-L1))))./(
    A3))));
145 % u4 =zeros(1,N+1);
146 %Implementation of treatment effort to individual who have been bitten or
    scratched by
147 %potentially rabid animal (post exposure prophylaxis-PEP) only
148 %Case12:u1= 0, u2= 0, u3= 0, u4\neq 0,
149 % u1 =zeros(1,1+N);
150 % u2 =zeros(1,1+N);
151 % u3 =zeros(1,N+1);
152 % u4 =max(0,min(1,(((r.*(L9-L11)+(g.*(L2-L4)))./A4))));
153 %Implementing the Combination vaccination of domestic dogs and administering Post
    -Exposure Prophylaxis (PEP)
154 %to individuals who have come into contact with a rabid animal
155 % Case13:u1= 0, u2=0, u3\neq 0, u4\neq 0,
156 % u1 =zeros(1,N+1);
157 % u2 =zeros(1,N+1);
158 % u3 =max(0,min(1,((((p.*tau_1+s.*tau_2+(F.*tau_3)./(F+C)).*f1.*(L2-L1))))./(
    A3))));
159 % u4 =max(0,min(1,(((r.*(L9-L11)+(g.*(L2-L4)))./A4))));
160
161 %Case14:u1=0,u2\neq 0,u3\neq 0,u4=0

```

```

162 % u1=zeros(1,N+1);
163 % u2 =max(0,min(1,((((q.*(L9-L8).*((psi_1.*p)./(1+rho_1))+((psi_2.*s)./(1+rho_2)
    )+((psi_3.*F)./((1+rho_3).*(F+C))))))./A2)));
164 % u3 =max(0,min(1,((((p.*tau_1+s.*tau_2+(F.*tau_3)./((F+C)).*f1.*(L2-L1)))))./(
    A3))));
165 % u4=zeros(1,N+1);
166
167 Uu=[u1' u2' u3' u4'];
168 U = 0.5*Uu + 0.5*oldu; % Convex combination of the controls
169
170 Xnew = X';
171 NumXX =abs(norm(Xnew-Xold));
172 DenXnew =norm(Xnew);
173 i=i+1; %Update iteration counter
174 end
175 %% PLOTTING
176 X=X';
177 Tx =Tx';
178 XX=X(:,1); YY=X(:,2); VV=X(:,3); ZZ=X(:,4); LL=X(:,5);
179 AA=X(:,6); BB=X(:,7); CC=X(:,8); DD=X(:,9); EE=X(:,10);
180 FF=X(:,11); GG=X(:,12);
181 Up = [0 0 0 0 0];
182 [T,Y] = ode45(@kims,time,y0,[],Up, Constant);
183 %objective function update
184 J =sum((K1.*GG(1:end)+K2.*YY(1:end)+K3.*VV(1:end)+K4.*DD(1:end)+...
185 K5.*EE(1:end)-K6.*CC(1:end))+...
186 ((A1/2)*Uu(:,1).*Uu(:,1)+(A2/2)*Uu(:,2).*Uu(:,2)+...
187 (A3/2)*Uu(:,3).*Uu(:,3)+(A4/2)*Uu(:,4).*Uu(:,4))); %Change to the suitable
    objective function
188 S=[Tx,X];
189 % figure(1)
190 % plot(Tx,X(:,8),'-b',T, Y(:,8),'--r','LineWidth',2);
191 % figure;
192 % plot(Tx,X(:,8),'-b',T, Y(:,8),'--r','LineWidth',2);
193 figure(1)
194 subplot(2,2,1)
195 plot(Tx,X(:,2), '-b',T, Y(:,2), '--r','LineWidth',2);
196 ylim([0 700])
197 ylabel('E_{H}');
198 xlabel('Time (years)');
199 legend('u_1 \neq 0 ,u_2 \neq 0, u_3\neq0,u_4\neq0','u_i=0, i=1,2,3,4')
200 %legend('u_1 \neq 0 ,\newline u_2 \neq 0,u_3\neq0,u_4\neq0','u_i=0, i=1,2,3,4','
    FontSize', 9)
201 % legend('u1= 0 ,u2 = 0,u3\neq0,u4\neq0','u_i=0, i=1,2,3,4')
202 %legend('u1= 0 ,u2 = 0,u3=0,u4\neq0','u_i=0, i=1,2,3,4')
203 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
204 %legend('u1= 0, u2\neq0, u3= 0, u4\neq 0','u_i=0, i=1,2,3,4')
205 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
206 title('a')

```

```

207 subplot(2,2,2)
208 plot(Tx,X(:,3), '-b',T, Y(:,3), '-r', 'LineWidth',2);
209 %ylim([0 700])
210 ylabel('I_{H}');
211 xlabel('Time (years)');
212 legend('u_1 \neq 0 ,u_2 \neq 0, u_3\neq0,u_4\neq0','u_i=0, i=1,2,3,4')
213 % %legend({'$u_1 \neq 0$, $u_2 \neq 0$, \newline $u_3 \neq 0$, $u_4 \neq 0$, ...
214 %         '$u_i = 0$, $i = 1,2,3,4$'}, ...
215 %         'Interpreter', 'latex');
216
217 %legend('u1= 0 ,u2 = 0,u3\neq0,u4\neq0','u_i=0, i=1,2,3,4')
218 %legend('u1= 0 ,u2 = 0,u3=0,u4\neq0','u_i=0, i=1,2,3,4')
219 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
220 %legend('u1= 0, u2\neq0, u3= 0, u4\neq 0','u_i=0, i=1,2,3,4')
221 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
222 title('(b)')
223
224 % subplot(2,2,3)
225 % plot(T,Y(:,4), '-b',Tx, X(:,4), '-r', 'LineWidth',2);
226 % %ylim([250 800])
227 % ylabel('R_{H}');
228 % xlabel('Time (years)');
229 % legend('u1= 0, u2\neq 0, u3= 0, u4\neq 0','u_i=0, i=1,2,3,4')
230 % title('(c)')
231 subplot(2,2,[3,4])
232 plot(Tx,Uu(:,1), '-m',Tx,Uu(:,2), '-k',Tx,Uu(:,3), '-g',...
233 Tx,Uu(:,4), '-c', 'linewidth',2);
234 ylim([0 1])
235 xlim([0 20])
236 ylabel('Control Profile');
237 xlabel('Time (years)');
238 %legend('u1= 0, u2\neq0, u3= 0, u4\neq 0','u_i=0, i=1,2,3,4')
239 legend('u_1\neq0','u_2\neq0','u_3\neq0','u_4\neq0')
240 title('(c)')
241 figure (2)
242 subplot(2,2,1)
243 plot(Tx,X(:,6), '-b',T, Y(:,6), '-r', 'LineWidth',2);
244 %ylim([250 800])
245 ylabel('E_{F}');
246 xlabel('Time (years)');
247 legend('u_1 \neq 0 ,u_2 \neq 0, u_3\neq0,u_4\neq0','u_i=0, i=1,2,3,4')
248 %legend('u_1 \neq 0 ,u_2 \neq 0, \newline u_3\neq0,u_4\neq0','u_i=0, i=1,2,3,4')
249 %legend('u1= 0 ,u2 = 0,u3\neq0,u4\neq0','u_i=0, i=1,2,3,4')
250 %legend('u1= 0 ,u2 = 0,u3=0,u4\neq0','u_i=0, i=1,2,3,4')
251 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
252 %legend('u1= 0, u2\neq0, u3= 0, u4\neq 0','u_i=0, i=1,2,3,4')
253 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
254 title('(a)')
255 subplot(2,2,2)

```

```

256 plot(Tx,X(:,7), '-b',T, Y(:,7), '-r', 'LineWidth',2);
257 ylim([250 800])
258 ylabel('I_{F}');
259 xlabel('Time (years)');
260 legend('u_1 \neq 0 ,u_2 \neq 0, u_3\neq0,u_4\neq0','u_i=0, i=1,2,3,4')
261 %legend('u_1 \neq 0 ,u_2 \neq 0,\newline u_3\neq0,u_4\neq0','u_i=0, i=1,2,3,4')
262 %legend('u1= 0 ,u2 = 0,u3\neq0,u4\neq0','u_i=0, i=1,2,3,4')
263 %legend('u1= 0 ,u2 = 0,u3=0,u4\neq0','u_i=0, i=1,2,3,4')
264 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
265 %legend('u1= 0, u2=0, u3= 0, u4\neq 0','u_i=0, i=1,2,3,4')
266 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
267 title('(b)')
268 subplot(2,2,3)
269 plot(Tx,X(:,12), '-b',T, Y(:,12), '-r', 'LineWidth',2);
270 ylim([250 800])
271 ylabel('M');
272 xlabel('Time (years)');
273 legend('u_1 \neq 0 ,u_2 \neq 0, u_3\neq0,u_4\neq0','u_i=0, i=1,2,3,4')
274 %legend('u_1 \neq 0 ,u_2 \neq 0, \newline u_3\neq0,u_4\neq0','u_i=0, i=1,2,3,4')
275 %legend('u1= 0 ,u2 = 0,u3=0,u4\neq0','u_i=0, i=1,2,3,4')
276 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
277 %legend('u1= 0, u2\neq0, u3= 0, u4\neq 0','u_i=0, i=1,2,3,4')
278 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
279 %legend('u_1\neq0 0','u_2\neq0','u_3\neq0','u_4\neq0')
280 title('(c)')
281 subplot(2,2,4)
282 plot(Tx,Uu(:,1), '-m',Tx,Uu(:,2), '-k',Tx,Uu(:,3), '-g',...
283 Tx,Uu(:,4), '-c', 'linewidth',2);
284 ylim([0 1])
285 xlim([0 20])
286 ylabel('Control Profile');
287 xlabel('Time (years)');
288 %legend('u1= 0, u2\neq0, u3= 0, u4\neq 0','u_i=0, i=1,2,3,4')
289 legend('u_1\neq0','u_2\neq0','u_3\neq0','u_4\neq0')
290 title('(d)')
291 figure(3)
292 subplot(2,2,1)
293 plot(Tx,X(:,9), '-b',T, Y(:,9), '-r', 'LineWidth',2);
294 ylim([250 800])
295 ylabel('E_{D}');
296 xlabel('Time (years)');
297 %legend('u_1 \neq 0 ,u_2 \neq 0, \newline u_3\neq0,u_4\neq0','u_i=0, i=1,2,3,4')
298 legend('u_1 \neq 0 ,u_2 \neq 0, u_3\neq0,u_4\neq0','u_i=0, i=1,2,3,4')
299 %legend('u1= 0 ,u2 = 0,u3\neq0,u4\neq0','u_i=0, i=1,2,3,4')
300 %legend('u1= 0 ,u2 = 0,u3=0,u4\neq0','u_i=0, i=1,2,3,4')
301 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
302 %legend('u1= 0, u2\neq0, u3= 0, u4\neq 0','u_i=0, i=1,2,3,4')
303 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
304 %legend('Location','southoutside')

```

```

305 title('(a)')
306
307 subplot(2,2,2)
308 plot(Tx,X(:,10),'-b',T, Y(:,10),'-r','LineWidth',2);
309 %ylim([250 800])
310 ylabel('I_{D}');
311 xlabel('Time (years)')
312 %legend('u_1\neq 0 ,u_2\neq 0, \newline u_3\neq 0,u_4\neq 0','u_i=0, i=1,2,3,4', '
    FontSize', 9)
313 legend('u_1 \neq 0 ,u_2 \neq 0, u_3\neq 0,u_4\neq 0','u_i=0, i=1,2,3,4')
314
315 %legend('u1= 0 ,u2 = 0,u3\neq 0,u4\neq 0','u_i=0, i=1,2,3,4')
316 %legend('u1= 0 ,u2 = 0,u3=0,u4\neq 0','u_i=0, i=1,2,3,4')
317 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
318 %legend('u1= 0, u2\neq 0, u3= 0, u4\neq 0','u_i=0, i=1,2,3,4')
319 %legend('u1 \neq 0 ,u2 \neq 0,u3=0,u4=0','u_i=0, i=1,2,3,4')
320 %legend('Location','southoutside')
321 title('(b)')
322 % subplot(2,2,3)
323 % plot(Tx,X(:,11),'-b',T, Y(:,11),'-r','LineWidth',2);
324 % %ylim([250 800])
325 % ylabel('R_{D}');
326 % xlabel('Time (years)');
327 % legend('u1= 0, u2\neq 0, u3= 0, u4\neq 0','u_i=0, i=1,2,3,4')
328 % %legend('Location','southoutside')
329 % title('(k)')
330 subplot(2,2,[3,4])
331 plot(Tx,Uu(:,1),'-m',Tx,Uu(:,2),'-k',Tx,Uu(:,3),'-g',...
332 Tx,Uu(:,4),'-c','linewidth',2);
333 ylim([0 1])
334 xlim([0 20])
335 ylabel('Control Profile');
336 xlabel('Time (years)');
337 %legend('u_1=0','u_2\neq 0','u_3=0','u_4\neq 0')
338 legend('u_1\neq 0','u_2\neq 0','u_3\neq 0','u_4\neq 0')
339 %legend('Location','southoutside')
340 title('(c)')
341 % subplot(2,4,8)
342 % plot(Tx,X(:,12),'-c',T, Y(:,10),'-r','LineWidth',1.5);
343 % %ylim([250 800])
344 % ylabel('I_{D}');
345 % xlabel('Time (years)');
346 % title('(l)')
347 %legend('with control','Without Control')
348
349 %Control profile for the implementation of all controls respectively
350 % subplot(2,4,8)
351 % plot(Tx,Uu(:,1),'-b',Tx,Uu(:,2),'-k',Tx,Uu(:,3),'-g',...
352 % Tx,Uu(:,4),'-r','linewidth',2);

```

```

353 % ylim([0 1])
354 % xlim([0 5])
355 % ylabel('Control Profile');
356 % xlabel('Time (years)');
357 % legend('Location','southoutside')
358 % title('(h)')
359
360 %-----
361 % collect all the incidence terms in the ODE
362 X=XX'; % solution of the optimal control
363 U =[0 0 0 0]; % when no control
364 [Tx,Y] = ode45(@kims,time,y0,[],U, Constant);
365 %Inew=sum(Y(:,2))-sum(X(:,2))+sum(Y(:,3))-sum(X(:,3)) %averted
366 Inew=sum(Y(:,2))-sum(X(:,2))+sum(Y(:,3))-sum(X(:,3))+...
367 sum(Y(:,9))-sum(X(:,9))+sum(Y(:,10))-sum(X(:,10))+sum(Y(:,12))-sum(X(:,12))%
    averted
368 Output=[J Inew]
369 Y=(Y);
370 % Defining function that solves the state system with 14 diff.eqns
371 %File Name: kims
372 function ydot = kims(t,yy,U,Constant)
373 %This function solves MU infection on human type three equations
374 f1=yy(1); g=yy(2); h1=yy(3); j=yy(4);l=yy(5);
375 m=yy(6); p=yy(7);q=yy(8); r=yy(9);s=yy(10);
376 v=yy(11);F=yy(12);
377 rho_1=Constant(1);rho_2 = Constant(2);rho_3= Constant(3);theta_1 = Constant(4);
378 tau_1 = Constant(5);tau_2 = Constant(6);tau_3= Constant(7);beta_1 = Constant(8);
379 beta_2 =Constant(9);beta_3=Constant(10);mu_1= Constant(11);sigma_1=Constant(12);
380 theta_2 = Constant(13);gamma = Constant(14);kappa_1=Constant(15);kappa_2=Constant
    (16);
381 kappa_3=Constant(17);sigma_2=Constant(18);mu_2=Constant(19);theta_3 = Constant
    (20);
382 psi_1=Constant(21);psi_2=Constant(22);psi_3=Constant(23);sigma_3=Constant(24);
383 gamma_1= Constant(25);gamma_2 = Constant(26);gamma_3 =Constant(27);mu_3=Constant
    (28);
384 nu_1=Constant(29);nu_2=Constant(30);nu_3 =Constant(31);mu_4 =Constant(32);C =
    Constant(33);
385 K1= Constant(34); K2 = Constant(35);K3 =Constant(36);K4 =Constant(37);
386 K5 =Constant(38);K6 =Constant(39);A1=Constant(40);A2 =Constant(41);
387 A3 =Constant(42);A4 =Constant(43);
388 u1 = U(1); u2=U(2); u3=U(3); u4 =U(4);
389 ydot1=theta_1+beta_3.*j-mu_1.*f1-((1-u1-u3).*(tau_1.*p+tau_2.*s+(tau_3.*F)./(F+C)
    )).*f1;
390 ydot2=((1-u1-u3).*(tau_1.*p+tau_2.*s+(tau_3.*F)/(F+C))).*f1-(mu_1+beta_1+beta_2+
    u4).*g;
391 ydot3=beta_1.*g-(sigma_1+mu_1).*h1;
392 ydot4=(beta_2+u4).*g-(beta_3+mu_1).*j;
393 ydot5=theta_2-(kappa_1.*p+kappa_2.*s+((kappa_3.*F)/(F+C))).*l-mu_2.*l;
394 ydot6=(kappa_1.*p+kappa_2.*s+((kappa_3.*F)/(F+C))).*l-(mu_2+gamma).*m;

```

```

395 ydot7=gamma.*m-(mu_2+sigma_2).*p;
396 ydot8=theta_3-mu_3.*q-(1-u1-u2).*((psi_1.*p)/(1+rho_1)+(psi_2.*s)/(1+rho_2)+(
    psi_3.*F)/((F+C).*(1+rho_3))).*q+gamma_3.*v;
397 ydot9=(1-u1-u2).*((psi_1.*p)/(1+rho_1)+(psi_2.*s)/(1+rho_2)+(psi_3.*F)/((F+C)
    .*(1+rho_3))).*q-(mu_3+gamma_1+gamma_2+u4).*r;
398 ydot10=gamma_1.*r-(mu_3+sigma_3).*s;
399 ydot11=(gamma_2+u4).*r-(mu_3+gamma_3).*v;
400 ydot12=(nu_1.*h1+nu_2.*p+nu_3.*s)-mu_4.*F;
401 ydot= [ydot1;ydot2;ydot3;ydot4;ydot5;ydot6;ydot7;ydot8;ydot9;...
402 ydot10;ydot11;ydot12];
403 end

```

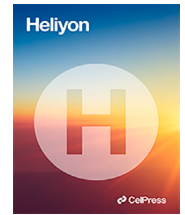
RESEARCH OUTPUTS

(i) Published Papers in Reputable Journals

1. Charles, M., Masanja, V. G., Torres, D. F. M., Mfinanga, S. G., & Lyakurwa, G. A. (2024). Mathematical model to assess the impact of contact rate and environment factor on transmission dynamics of rabies in humans and dogs. *Heliyon*, 10 (11), e32012. <https://doi.org/10.1016/j.heliyon.2024.e32012>
2. Charles, M., Mfinanga, S. G., Lyakurwa, G. A., Torres, D. F. M., & Masanja, V. G. (2024). Parameters estimation and uncertainty assessment in the transmission dynamics of rabies in humans and dogs. *Chaos, Solitons & Fractals*, 189(12), 115633. <https://doi.org/10.1016/j.chaos.2024.115633>
3. Charles, M., Mfinanga, S. G., Lyakurwa, G. A., Torres, D. F. M., & Masanja, V. G. (2025). A Mathematical and Optimal Control Model for Rabies Transmission Dynamics Among Humans and Dogs With Environmental Effects. *Journal of Applied Mathematics*, 2025. <https://doi.org/10.1155/jama/5559842>
4. Charles, M., Mfinanga, S. G., Lyakurwa, G. A., Torres, D. F. M., & Masanja, V. G. (2025). Evaluating the effectiveness of Stochastic CTMC and deterministic models in correlating rabies persistence in human and dog populations. *Franklin Open*, 100397. <https://doi.org/10.1016/j.fraope.2025.100397>

(ii) Poster Presentation

Mathematical Modeling and Analysis of Emerging and Re-emerging of Rabies Transmission Dynamics: Deterministic and Stochastic Approaches with Control Strategies.



Research article

Mathematical model to assess the impact of contact rate and environment factor on transmission dynamics of rabies in humans and dogs

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ABSTRACT

This paper presents a mathematical model to understand how rabies spreads among humans, free-range, and domestic dogs. By analyzing the model, we discovered that there are equilibrium points representing both disease-free and endemic states. We calculated the basic reproduction number, $\mathcal{R}_0$ using the next generation matrix method. When $\mathcal{R}_0 < 1$, the disease-free equilibrium is globally stable, whereas when $\mathcal{R}_0 \geq 1$, the endemic equilibrium is globally stable. To identify the most influential parameters in disease transmission, we used the normalized forward sensitivity index. The simulations revealed that the contact rates between the infectious agent and humans, free-range dogs, and domestic dogs, have the most significant impact on rabies transmission. The study also examines how periodic changes in transmission rates affect the disease dynamics, emphasizing the importance of transmission frequency and amplitude on the patterns observed in rabies spread. To reduce disease sensitivity, one should prioritize effective disease control measures that focus on keeping both free-range and domestic dogs indoors. This is a crucial factor in preventing the spread of disease and should be implemented as a primary disease control measure.

1. Introduction

Rabies is a viral disease that affects mammals, including humans, caused by the rabies virus (*Rabies lyssavirus*) that travels from the site of infection to the brain, causing inflammation and damage to the nervous system [1,2]. Despite that dogs are the primary host of the virus, causing more than 99% of human rabies infections worldwide, other animals such as bats, raccoons, skunks, and foxes can also carry and transmit the virus through bites or scratches [1–3]. The transmission dynamics of rabies are influenced

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by environmental factors such as changes in habitat, land use patterns and wildlife populations which create varied and frequent interactions between infected and susceptible individuals [4].

Symptoms of rabies include fever, headaches, fatigue, restlessness, anxiety, hallucinations, hydrophobia, difficulty swallowing and paralysis that manifest between 20 days and 3 months after exposure. This may vary from 1 week to 1 year after exposure, depending on the location of entry of the virus and the viral load. In rare cases, the incubation period can last up to 7 years, and if left untreated and without appropriate medical care such as vaccination, the disease progresses into a coma state and ultimately leads to death [5–7].

Rabies is responsible for 60,000 human deaths every year globally [8], and the effective management of rabies in low- and middle income countries (LMICs) in Asia and Africa is often hindered by the lack of timely and accurate information on rabies cases in both humans and animals [9,10]. The actual number of fatalities caused by rabies virus (RABV) infections in LMICs is believed to be underestimated and the dynamics of rabid dog populations are poorly understood [10]. A study conducted by [11] revealed that the mortality rate attributed to human rabies in the United Republic of Tanzania was significantly higher than what has been officially reported. By analyzing the active surveillance data on bite incidence, it estimated an annual mortality rate of 1499 deaths, with a confidence interval spanning from 891 to 2238 deaths. This indicates an annual incidence of 4.9 deaths per 100,000 population, ranging from 2.9 to 7.2 deaths per 100,000.

The application of mathematical models has significantly enhanced the understanding and control of contagious diseases, including rabies. This analytical tool has proven instrumental in predicting and analyzing various phenomena, enabling medical experts to structure their approach toward disease management. Numerous mathematical models have been developed and analyzed to investigate the transmission dynamics of rabies in both human and dog populations [12–22]. These studies have identified several factors that influence the dynamics of rabies and have addressed various strategies to control the disease.

None of the studies referred to have comprehensively analyzed the combined impact of environmental factors, human populations, and free range and domestic dog populations on the transmission dynamics of rabies. These factors can result in increased interactions between humans, domestic animals, and wildlife, which together make up a population of approximately 900 million worldwide. This, in turn, increases the risk of rabies transmission [7,23]. The movement of infected animals, whether domestic or wild, also contributes to the spread of the virus. Infected animals can travel long distances, introducing the virus to new areas or re-establishing it in regions where it was previously under control. Studies by [24,25] have documented how these factors can result in increased contact between humans and domestic animals, further facilitating the spread of the virus. As such, this study presents a deterministic mathematical model designed to assess the effect of contact rates and environmental factors on the transmission dynamics of rabies in both human and canine populations.

This paper is structured as follows. Section 2 outlines a deterministic mathematical model describing the dynamics of rabies. Section 3 focuses on analyzing the proposed model. Real-world data is then used in Section 4 for model fitting and parameter estimation, while Section 5 covers the implementation of numerical simulations. Sections 6 and 7 are dedicated to the discussion and conclusion of the study, respectively.

2. Model formulation and model analysis

In this study, we formulated a mathematical model using ordinary differential equations by incorporating the interactions between susceptible individuals, infectious dogs, and the environment [13,17,26,27]. The study used mathematical simulations to examine how rabies spread and persist under different contact rates and environmental impacts.

2.1. Model formulation

We divided the model into three groups; the human population, the dog population and rabies viruses in the environment. The dog population was divided into two subgroups: free-range and domestic dogs.

The free-range dog population defined by N_F was divided into three compartments namely: susceptible free-range dogs (S_F) that were group of free-range dogs free of infection but could get infected after adequate contact with either infected free-range dogs (I_F), infected domestic dogs (I_D) or the environment containing rabies virus (M), exposed free-range dogs (E_F) which included all the free-range dogs who had contracted the disease but could not transmit the disease and had no symptoms of rabies infections, infected free-range dogs (I_F) made up of dogs who had contracted the disease with fully developed rabies symptoms and were infectious.

The domestic dog population defined by N_D was divided into four compartments. First compartment is susceptible domestic dogs denoted by S_D , who were not infected but could get infected after adequate contact with either infected free-range dogs, infected domestic dogs or environment that contains rabies virus. The second compartment were exposed domestic dogs (E_D), who had contracted the disease but could not transmit the disease and did not have disease symptoms. The third compartment is infectious domestic dogs (I_D), who had contracted the disease and were infectious. The fourth compartment is recovered domestic dogs, denoted by R_D , who get post-exposure prophylaxis after contract with rabies infectious agent and develop temporary immunity.

The human population, denoted by N_H , was divided into four compartments; first compartment were humans who had not acquired the rabies infection but could get if they adequately contacted with infectious free-range dogs, domestic dogs or contaminated environment. The second compartment were exposed human (E_H), who had contracted the disease but could not transmit it and

do not have symptoms of the infections. The third compartment is the infected human (I_H), who had contracted the rabies virus and were showing all the symptoms of rabies and were, therefore, infectious. The fourth compartment is recovered human beings, denoted by R_H , who get post-exposure prophylaxis after they contact and developed temporary immunity.

The environment represented the virus causing rabies, that are in the physical object or any other materials in the environment. These virus containing materials are considered as virus transmitting media or the infectious agent denoted as $M(t)$.

2.1.1. Description of model interaction

Susceptible humans are constantly recruited at a rate of θ_1 . When they come into adequate contact with I_F , I_D , or the virus in the environment, individuals in the S_H category become infected at rates τ_1 , τ_2 , and τ_3 , respectively. Thus, the force of infection for human is given by equation (1):

$$\chi_1 = (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H, \quad (1)$$

where

$$\lambda(M) = \frac{M}{M + C}.$$

After contracting rabies infections, susceptible humans become exposed individuals, denoted by E_H , typically for 1 to 3 months. Those in the E_H category who receive post-exposure prophylaxis recover at the rate β_2 . Since post-exposure prophylaxis does not confer permanent immunity, individuals in the R_H category can lose immunity and become susceptible again at the rate β_3 . The remaining proportion of the exposed class progresses to the infectious state I_H at a rate β_1 . Infected humans can die due to the disease at a rate σ_1 . All human compartments experience natural death at a rate of μ_1 .

Free-range dogs that are susceptible to rabies are continually added to the population at a rate of θ_2 . These dogs can contract the disease when they come into contact with infected free-range dogs (I_F), infected domestic dogs (I_D), or the virus in the environment, at rates κ_1 , κ_2 , and κ_3 , respectively. The force of infection for free-range dogs is calculated using equation (2):

$$\chi_2 = (\kappa_1 I_w + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F. \quad (2)$$

After contracting the rabies infection, free-range dogs that are susceptible progress to the exposed state, E_F , which lasts for 1 to 3 months. The exposed free-range dogs then progress to the infectious state, I_F , at a rate of γ . Infected free-range dogs may die due to the disease at a rate of σ_2 . Additionally, all free-range dog compartments experience natural death at a rate of μ_2 .

Susceptible domestic dogs are constantly recruited at a rate of θ_3 . These dogs become infected when they come into adequate contact with I_F , I_D , or the rabies virus in the environment, at the rates ψ_1 , ψ_2 , and ψ_3 , respectively. Since domestic dogs are under human control, they have a lower risk of contracting the disease. Hence, the force of infection for free-range dogs is given by equation (3):

$$\chi_3 = \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right) S_D. \quad (3)$$

After contracting a rabies infection, domestic dogs enter a state E_D , where they stay for several months at a rate of β_1 . If dogs in the E_D category receive post-exposure prophylaxis, they move to the R_D category at a rate of γ_2 . However, post-exposure prophylaxis does not provide permanent immunity, and dogs in the R_D category can lose their immunity and become susceptible again at a rate of γ_3 . Meanwhile, the remaining proportion of the exposed class progresses to the infectious state I_D at a rate of γ_1 . Infected domestic dogs can die from the disease at a rate of σ_3 . All compartments of domestic dogs experience natural death at a rate of μ_3 .

Rabies virus is spread in the environment through shedding by infected free-range and domestic dogs, as well as humans, at rates denoted by v_2 , v_3 , and v_1 , respectively. Equation (4) can be used to estimate the virus recruitment in the environment:

$$\theta_4 = (v_1 I_H + v_2 I_F + v_3 I_D) M. \quad (4)$$

The viruses are removed from the environment at a rate μ_4 .

Fig. 1 illustrates the dynamics of rabies disease among free-range dogs, domestic dogs, humans, and the environment.

Based on the description of the model parameters and their connections to the state variables, we formulate a model in the form of a system of ordinary differential equations, as presented in the model equation (5):

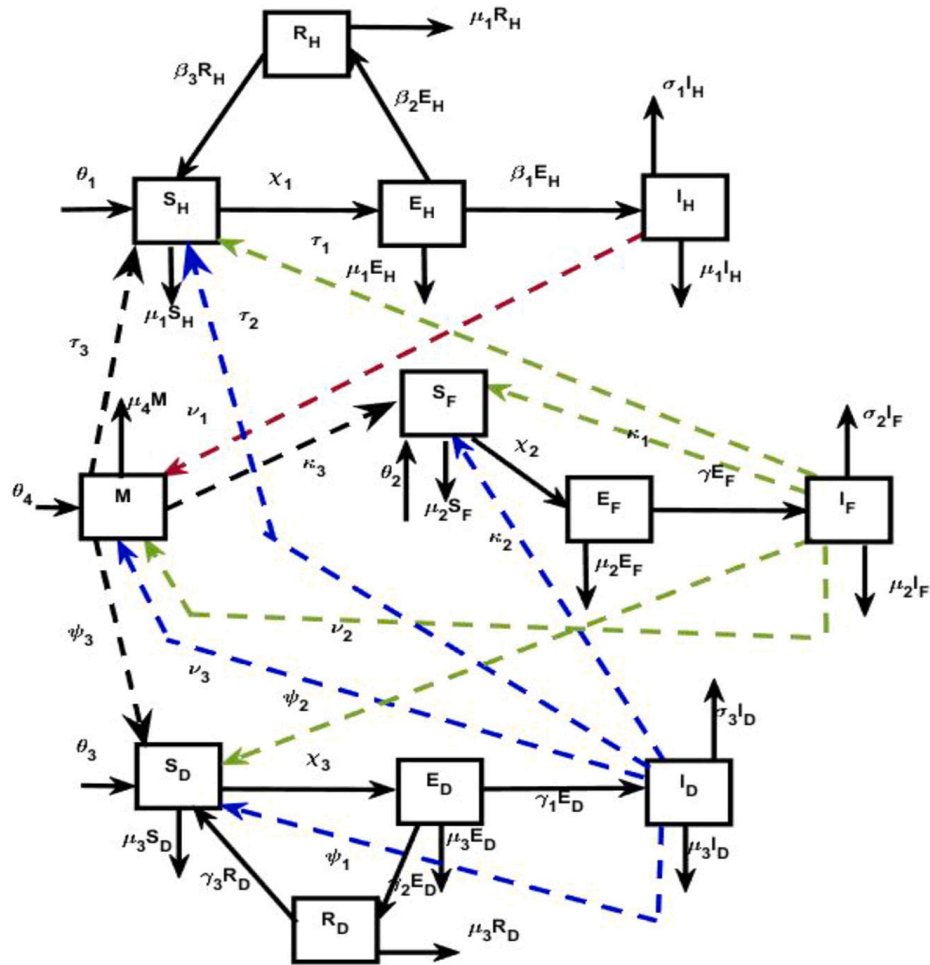


Fig. 1. Flow diagram for rabies transmission among human, free range and domestic dogs with parameters.

$$\left. \begin{aligned}
 \dot{S}_H &= \theta_1 + \beta_3 R_H - \mu_1 S_H - \chi_1, \\
 \dot{E}_H &= \chi_1 - (\mu_1 + \beta_1 + \beta_2) E_H, \\
 \dot{I}_H &= \beta_1 E_H - (\sigma_1 + \mu_1) I_H, \\
 \dot{R}_H &= \beta_2 E_H - (\beta_3 + \mu_1) R_H, \\
 \dot{S}_F &= \theta_2 - \chi_2 - \mu_2 S_F, \\
 \dot{E}_F &= \chi_2 - (\mu_2 + \gamma) E_F, \\
 \dot{I}_F &= \gamma E_F - (\mu_2 + \sigma_2) I_F, \\
 \dot{S}_D &= \theta_3 - \mu_3 S_D - \chi_3 + \gamma_3 R_D, \\
 \dot{E}_D &= \chi_3 - (\mu_3 + \gamma_1 + \gamma_2) E_D, \\
 \dot{I}_D &= \gamma_1 E_D - (\mu_3 + \sigma_3) I_D, \\
 \dot{R}_D &= \gamma_2 E_D - (\mu_3 + \gamma_3) R_D, \\
 \dot{M} &= (\nu_1 I_H + \nu_2 I_F + \nu_3 I_D) - \mu_4 M,
 \end{aligned} \right\} \quad (5)$$

subject to the non-negative conditions

$$\begin{aligned}
 S_H(0) > 0, \quad E_H(0) \geq 0, \quad I_H(0) \geq 0, \quad R_H(0) \geq 0, \quad S_F(0) > 0, \quad E_F(0) \geq 0, \quad I_F(0) \geq 0, \\
 S_D(0) \geq 0, \quad E_D(0) \geq 0, \quad I_D(0) \geq 0, \quad R_D(0) \geq 0.
 \end{aligned} \quad (6)$$

2.2. Model analysis

2.2.1. Model's invariant region

Since the model (5) monitors the human and dog populations, we assume that the model's state variable and parameters are non-negative for $\forall t \geq 0$. Using Theorem 1, we obtain the invariant region of the rabies model.

Theorem 1. Solution of the rabies model system (5) is uniformly bounded if $\Omega \in \mathbb{R}_+^{12}$ and $\Omega = \Omega_H \cup \Omega_D \cup \Omega_F \cup \Omega_M \in \mathbb{R}_+^4 \times \mathbb{R}_+^3 \times \mathbb{R}_+^4 \times \mathbb{R}_+^1$, where

$$\begin{aligned}\Omega_H &= \left\{ (S_H, E_H, I_H, R_H) \in \mathbb{R}_+^4 : 0 \leq N_H \leq \frac{\theta_1}{\mu_1} \right\}, \\ \Omega_F &= \left\{ (S_F, E_F, I_F) \in \mathbb{R}_+^3 : 0 \leq N_F \leq \frac{\theta_2}{\mu_2} \right\}, \\ \Omega_D &= \left\{ (S_D, E_D, I_D, R_D) \in \mathbb{R}_+^4 : 0 \leq N_D \leq \frac{\theta_3}{\mu_3} \right\},\end{aligned}$$

and Ω is the positive invariant region.

Proof. Consider the population of the human. From equation (5) one has

$$\frac{dN_H}{dt} = \frac{dS_H}{dt} + \frac{dE_H}{dt} + \frac{dI_H}{dt} + \frac{dR_H}{dt}. \quad (7)$$

Then, the equation (7) leads to

$$\frac{dN_H}{dt} = \theta_1 - (S_H + E_H + I_H + R_H) \mu_1 - \sigma_1 I_H. \quad (8)$$

Thus, equation (8) becomes

$$\frac{dN_H}{dt} = \theta_1 - (S_H + E_H + I_H + R_H) \mu_1. \quad (9)$$

Given that $N_H = S_H + E_H + I_H + R_H$, the equation (9) results in (10):

$$\frac{dN_H}{dt} = \theta_1 - N_H \mu_1. \quad (10)$$

From the integrating factor we have

$$N_H(t) = e^{\int_0^t \mu_1 dt} = e^{\mu_1 t}. \quad (11)$$

Then, for $t \rightarrow 0$ equation (11) gives

$$N_H(0) \leq \frac{\theta_1}{\mu_1} + Ce^0 \implies N_H(0) - \frac{\theta_1}{\mu_1} \leq C. \quad (12)$$

By simplifying equation (12) and performing simple manipulations, we have

$$\Omega_H = \left\{ (S_H, E_H, I_H, R_H) \in \mathbb{R}_+^4 : 0 \leq N_H \leq \frac{\theta_1}{\mu_1} \right\}.$$

Using the same procedures for free-range and domestic dogs, we have

$$\Omega_F = \left\{ (S_F, E_F, I_F) \in \mathbb{R}_+^3 : 0 \leq N_F \leq \frac{\theta_2}{\mu_2} \right\}, \quad \Omega_D = \left\{ (S_D, E_D, I_D, R_D) \in \mathbb{R}_+^4 : 0 \leq N_D \leq \frac{\theta_3}{\mu_3} \right\}.$$

Regarding the environment that contains the rabies virus, we consider eq. (12) in the model system (5) as

$$\dot{M} = (\nu_1 I_H + \nu_2 I_F + \nu_3 I_D) - \mu_4 M. \quad (13)$$

Since $N_H \leq \frac{\theta_1}{\mu_1}$, $N_F \leq \frac{\theta_2}{\mu_2}$ and $N_D \leq \frac{\theta_3}{\mu_3}$, it follows that $I_H \leq \frac{\theta_1}{\mu_1}$, $I_F \leq \frac{\theta_2}{\mu_2}$ and $I_D \leq \frac{\theta_3}{\mu_3}$.

Thus, equation (13) becomes

$$\dot{M} \leq \left(\frac{\nu_1 \theta_1}{\mu_1} + \frac{\nu_2 \theta_2}{\mu_2} + \frac{\nu_3 \theta_3}{\mu_3} \right) - \mu_4 M. \quad (14)$$

Now, let Y be the unique solution to the initial value problem, such that

$$\left. \begin{aligned} \dot{Y} &\leq \left(\frac{\nu_1 \theta_1}{\mu_1} + \frac{\nu_2 \theta_2}{\mu_2} + \frac{\nu_3 \theta_3}{\mu_3} \right) - \mu_4 M, \text{ for } t > 0, \\ Y(0) &= M(0). \end{aligned} \right\} \quad (15)$$

By using the integration factor, equation (15) becomes

$$M(t) \leq \left(\frac{\nu_1 \theta_1}{\mu_1} + \frac{\nu_2 \theta_2}{\mu_2} + \frac{\nu_3 \theta_3}{\mu_3} \right) \frac{1}{\mu_4} + \left(M(0) - \left(\frac{\nu_1 \theta_1}{\mu_1} + \frac{\nu_2 \theta_2}{\mu_2} + \frac{\nu_3 \theta_3}{\mu_3} \right) \frac{1}{\mu_4} \right). \quad (16)$$

As $t \rightarrow \infty$, the expression $\left(M(0) - \left(\frac{\nu_1 \theta_1}{\mu_1} + \frac{\nu_2 \theta_2}{\mu_2} + \frac{\nu_3 \theta_3}{\mu_3}\right) \frac{1}{\mu_4} e^{\mu_4 t}\right)$ in equation (16) goes to zero, and we have

$$M \leq \left(\frac{\nu_1 \theta_1}{\mu_1} + \frac{\nu_2 \theta_2}{\mu_2} + \frac{\nu_3 \theta_3}{\mu_3}\right) \frac{1}{\mu_4}. \quad (17)$$

Equation (17) gives

$$M(t) \leq \Omega_M = \text{Max} \left\{ \frac{\theta_1 \nu_1}{\mu_1 \mu_4} + \frac{\theta_2 \nu_2}{\mu_2 \mu_4} + \frac{\theta_3 \nu_3}{\mu_3 \mu_4}, M(0) \right\}. \quad (18)$$

Thus, the model system (5) is biologically and mathematically meaningful with its solution relying on the region Ω . $\square$

2.2.2. Positivity of the solution

For the model system (5) to be epidemiologically meaningful and well-posed, we need to prove that the state variables are non-negative for all $t \geq 0$.

Theorem 2. Let $\{S_H(0), E_H(0), I_H(0), R_H(0), S_F(0), E_F(0), I_F(0), S_D(0), E_D(0), I_D(0), R_D(0), M(0)\} \in \mathbb{R}_+^{12}$. Then the set

$$\{S_H(t), E_H(t), I_H(t), R_H(t), S_F(t), E_F(t), I_F(t), S_D(t), E_D(t), I_D(t), R_D(t), M(t)\}$$

of solutions of the model system (5) is positive $\forall t > 0$.

Proof. Consider the human subpopulation of the model system (5). We have

$$\frac{dS_H}{dt} = \theta_1 + \beta_3 R_H - \mu_1 S_H - (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H. \quad (19)$$

Then, equation (19) yields

$$\frac{dS_H}{dt} \geq -(\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M) + \mu_1) S_H. \quad (20)$$

Using integrating techniques on both sides, equation (20) leads to

$$\int \frac{dS_H}{S_H} \geq \int_0^t -(\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M) + \mu_1) ds. \quad (21)$$

Hence, using equation (21), we can derive the following result:

$$S_H \geq S_H(0) e^{\int_0^t -(\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M) + \mu_1) ds} > 0. \quad (22)$$

Thus, S_H is positive for all $t > 0$. Using the same procedure, from equations (19) to (46) we have

$$\begin{aligned} E_H &\geq E_H(0) e^{-(\mu_1 + \beta_1 + \beta_2)t} > 0, \quad I_H \geq I_H(0) e^{-(\sigma_1 + \mu_1)t} > 0, \quad R_H \geq R_H(0) e^{-(\mu_1 + \beta_3)t} > 0, \\ S_F &\geq S_F(0) e^{\int_0^t -((\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M) + \mu_2)) ds} > 0, \quad E_F \geq E_F(0) e^{-(\mu_2 + \gamma)t} > 0, \quad I_F \geq I_F(0) e^{-(\mu_2 + \sigma_2)t} > 0, \\ S_D &\geq S_D(0) e^{\int_0^t -((\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3 \lambda(M) + \mu_3}{1 + \rho_3})) ds} > 0, \quad E_D \geq E_D(0) e^{-(\mu_3 + \gamma_1 + \gamma_2)t} > 0, \\ R_D &\geq R_D(0) e^{-(\mu_3 + \gamma_3)t} > 0, \quad I_D \geq I_D(0) e^{-(\mu_3 + \sigma_3)t} > 0, \quad M \geq M(0) e^{-\mu_4 t} > 0. \end{aligned}$$

Thus, the set of solutions $\{S_H(t), E_H(t), I_H(t), R_H(t), S_F(t), E_F(t), I_F(t), S_D(t), E_D(t), I_D(t), R_D(t), M(t)\}$ of the model system (5) is positive $\forall t > 0$. $\square$

2.2.3. Disease free equilibrium point $\mathcal{E}_0$

The disease-free equilibrium point, denoted as $\mathcal{E}_0$, refers to the point at which there is no disease in a given population. In order to obtain $\mathcal{E}_0$ in the model system (5), we consider all infectious compartments to be equal to zero. Therefore, $\mathcal{E}_0$ can be defined as follows:

$$\mathcal{E}_0 = \left(\frac{\theta_1}{\mu_1}, 0, 0, 0, \frac{\theta_2}{\mu_2}, 0, 0, \frac{\theta_3}{\mu_3}, 0, 0, 0, 0\right).$$

2.2.4. The basic reproduction number, $\mathcal{R}_0$

The $\mathcal{R}_0$ predicts whether rabies will spread across the community or die out. If $\mathcal{R}_0 < 1$, it means that every infectious individual will cause less than one secondary infection, hence the disease will die out in the community. If $\mathcal{R}_0 > 1$, it means that every infectious individual will cause more than one secondary infection, leading to the persistence of rabies in the entire population. In order to determine $\mathcal{R}_0 \geq 1$, the next generation matrix, as applied by [28–30], and the Jacobian Matrix are used, such that

$$\frac{dx_i}{dt} = F_i(x) - (\mathcal{V}_i^+(x) - \mathcal{V}_i^-(x)), \quad (23)$$

where F_i is the new infections in the compartment i while $\mathcal{V}_i^+$ and $\mathcal{V}_i^-$ are the transfer terms in and out of the compartment i , respectively. From equation (5), we define F_i and $\mathcal{V}_i$ by

$$F_i = \begin{pmatrix} (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H \\ 0 \\ (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F \\ 0 \\ \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right) S_D \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V}_i = \begin{pmatrix} (\mu_1 + \beta_1 + \beta_2) E_H \\ (\sigma_1 + \mu_1) I_H - \beta_1 E_H \\ (\mu_2 + \gamma) E_F \\ (\mu_2 + \sigma_2) I_F - \gamma E_F \\ (\mu_3 + \gamma_1 + \gamma_2) E_D \\ (\mu_3 + \delta_3) I_D - \gamma_1 E_D \\ \mu_4 M - (\nu_1 I_H + \nu_2 I_F + \nu_3 I_D) \end{pmatrix}. \quad (24)$$

The Jacobian Matrices F and V at the disease free equilibrium point $\mathcal{E}_0$ are given by equation (25):

$$F = \frac{\partial F_i(\mathcal{E}_0)}{\partial x_j}, \quad V = \frac{\partial \mathcal{V}_i(\mathcal{E}_0)}{\partial x_j}. \quad (25)$$

From equation (24), F and V at $\mathcal{E}_0$ is given by equation (26):

$$\left. \frac{\partial F_i}{\partial x_j} \right|_{\mathcal{E}_0} = F = \begin{pmatrix} 0 & 0 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\psi_1 \theta_3}{(1 + \rho_1) \mu_3} & 0 & \frac{\psi_2 \theta_3}{(1 + \rho_2) \mu_3} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}. \quad (26)$$

In the linearized system (26), the entry F_{ij} represents the rate at which individuals in the infected state j give rise to or develop new infections in individuals in the infected state i , with reference to the infected states indexed by i and j for $i, j \in \{1, 2, 3, 4, 5, 6, 7\}$. As a result, when a person in an infected condition j does not instantly produce any new instances in an infected state i , we have $F_{ij} = 0$. Similarly, V at point $\mathcal{E}_0$ is given by

$$\left. \frac{\partial \mathcal{V}_i}{\partial x_j} \right|_{\mathcal{E}_0} = V = \begin{pmatrix} \mu_1 + \beta_1 + \beta_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\beta_1 & \sigma_1 + \mu_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \mu_2 + \gamma & 0 & 0 & 0 & 0 \\ 0 & 0 & -\gamma & \mu_2 + \sigma_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \mu_3 + \gamma_1 + \gamma_2 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\gamma_1 & \mu_3 + \sigma_3 & 0 \\ 0 & -\nu_1 & 0 & -\nu_2 & 0 & -\nu_3 & \mu_4 \end{pmatrix}. \quad (27)$$

The inverse of matrix V is obtained using Maple software, and its result is given as

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu_1 + \beta_1 + \beta_2} & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_1}{(\sigma_1 + \mu_1)(\mu_1 + \beta_1 + \beta_2)} & \frac{1}{\sigma_1 + \mu_1} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{\mu_2 + \gamma} & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\gamma}{(\mu_2 + \gamma)(\mu_2 + \sigma_2)} & \frac{1}{\mu_2 + \sigma_2} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{\mu_3 + \gamma_1 + \gamma_2} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\gamma_1}{(\mu_3 + \sigma_3)(\mu_3 + \gamma_1 + \gamma_2)} & \frac{1}{\mu_3 + \sigma_3} & 0 \\ \frac{\nu_1 \beta_1}{(\sigma_1 + \mu_1)(\mu_1 + \beta_1 + \beta_2)\mu_4} & \frac{\nu_1}{(\sigma_1 + \mu_1)\mu_4} & \frac{\nu_2 \gamma}{(\mu_2 + \gamma)(\mu_2 + \sigma_2)\mu_4} & \frac{\nu_2}{(\mu_2 + \sigma_2)\mu_4} & \frac{\nu_3 \gamma_1}{(\mu_3 + \sigma_3)(\mu_3 + \gamma_1 + \gamma_2)\mu_4} & \frac{\nu_3}{(\mu_3 + \sigma_3)\mu_4} & \frac{1}{\mu_4} \end{pmatrix}. \quad (28)$$

In the context of computing the basic reproduction number $\mathcal{R}_0$ in epidemiology, the $(V^{-1})_{ij}$ obtained in equation (28) represents the generation matrix. The generation matrix describes the expected number of newly infected individuals that a single infectious individual in each of the different susceptible classes generates. Meanwhile, the diagonal elements represent the rate of leaving the corresponding susceptible class due to other causes such as recoveries caused by administration of post-exposure prophylaxis (PEP). In particular, $\frac{1}{\mu_1 + \beta_1 + \beta_2}$, $\frac{1}{\mu_2 + \gamma}$, and $\frac{1}{\mu_3 + \gamma_1 + \gamma_2}$ represent the average incubation period for rabies in humans, free-range dogs, and domestic dogs, respectively. Meanwhile, $\frac{1}{\mu_1 + \sigma_1}$, $\frac{1}{\mu_2 + \sigma_2}$, and $\frac{1}{\mu_3 + \sigma_3}$ represent the average time spent by an infective human, free-range dog, and domestic dog in the infectious state, respectively, and $\frac{1}{\mu_4}$ is the average time the rabies virus spends in the environment. The next generation Matrix is then calculated by

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{\tau_1 \theta_1 \gamma}{\mu_1 (\mu_2 + \gamma)(\mu_2 + \sigma_2)} & \frac{\tau_1 \theta_1}{\mu_1 (\mu_2 + \sigma_2)} & \frac{\tau_2 \theta_1 \gamma}{\mu_1 (\mu_3 + \gamma_1 + \gamma_2)(\mu_3 + \sigma_3)} & \frac{\tau_2 \theta_1}{\mu_1 (\mu_3 + \sigma_3)} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\kappa_1 \theta_2 \gamma}{\mu_2 (\mu_2 + \gamma)(\mu_2 + \sigma_2)} & \frac{\kappa_1 \theta_2}{\mu_2 (\mu_2 + \sigma_2)} & \frac{\kappa_1 \theta_2 \gamma}{\mu_2 (\mu_3 + \gamma_1 + \gamma_2)(\mu_3 + \sigma_3)} & \frac{\kappa_1 \theta_2}{\mu_2 (\mu_3 + \sigma_3)} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\psi_1 \theta_3 \gamma}{(1 + \rho_1) \mu_3 (\mu_2 + \gamma)(\mu_2 + \sigma_2)} & \frac{\psi_1 \theta_3}{(1 + \rho_1) \mu_3 (\mu_2 + \sigma_2)} & \frac{\psi_2 \theta_3 \gamma}{(1 + \rho_2) \mu_3 (\mu_3 + \gamma_1 + \gamma_2)(\mu_3 + \sigma_3)} & \frac{\psi_2 \theta_3}{(1 + \rho_2) \mu_3 (\mu_3 + \sigma_3)} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}. \quad (29)$$

The expression of matrix FV^{-1} can be presented as

$$FV^{-1} = \begin{pmatrix} 0 & 0 & R_{13} & R_{14} & R_{15} & R_{16} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & R_{33} & R_{34} & R_{35} & R_{36} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & R_{53} & R_{54} & R_{55} & R_{56} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \quad (30)$$

where

Table 1
Sensitivity indices for $\mathcal{R}_0$.

Parameter	Sensitivity Index	Parameter	Sensitivity Index
γ_1	-0.105552	ψ_1	+0.051422
γ_2	-0.056998	ψ_2	+0.005436
κ_1	+0.897120	κ_2	+0.051422
μ_2	-1.616021	ρ_1	-0.046747
μ_3	-0.105358	ρ_2	-0.004832
σ_2	-0.540654	σ_3	-0.05144
θ_2	+0.941420	θ_3	+0.056858

$$\left. \begin{aligned} R_{13} &= \frac{\tau_1 \theta_1 \gamma}{\mu_1 (\mu_2 + \gamma) (\mu_2 + \sigma_2)}, & R_{14} &= \frac{\tau_1 \theta_1}{\mu_1 (\mu_2 + \sigma_2)}, & R_{15} &= \frac{\tau_2 \theta_1 \gamma}{\mu_1 (\mu_3 + \gamma_1 + \gamma_2) (\mu_3 + \sigma_3)}, & R_{16} &= \frac{\tau_2 \theta_1}{\mu_1 (\mu_3 + \sigma_3)}, \\ R_{33} &= \frac{\kappa_1 \theta_2 \gamma}{\mu_2 (\mu_2 + \gamma) (\mu_2 + \sigma_2)}, & R_{34} &= \frac{\kappa_1 \theta_2}{\mu_2 (\mu_2 + \sigma_2)}, & R_{35} &= \frac{\kappa_1 \theta_2 \gamma}{\mu_2 (\mu_3 + \gamma_1 + \gamma_2) (\mu_3 + \sigma_3)}, & R_{36} &= \frac{\kappa_1 \theta_2}{\mu_2 (\mu_3 + \sigma_3)}, \\ R_{53} &= \frac{\psi_1 \theta_3 \gamma}{(1 + \rho_1) (\mu_2 + \gamma) (\mu_2 + \sigma_2) \mu_3}, & R_{54} &= \frac{\psi_1 \theta_3}{(1 + \rho_1) \mu_3 (\mu_2 + \sigma_2)}, & R_{55} &= \frac{\psi_2 \theta_3 \gamma}{(1 + \rho_2) (\mu_3 + \gamma_1 + \gamma_2) (\mu_3 + \sigma_3) \mu_3}, \\ R_{56} &= \frac{\psi_2 \theta_3}{(1 + \rho_2) \mu_3 (\mu_3 + \sigma_3)}. \end{aligned} \right\} \quad (31)$$

From equation (30), we obtained the eigenvalues as

$$\left(0, 0, 0, 0, \frac{1}{2} R_{55} + \frac{1}{2} R_{33} + \frac{1}{2} \sqrt{R_{33}^2 - 2 R_{33} R_{55} + 4 R_{35} R_{53} + R_{55}^2}, \right. \\ \left. \frac{1}{2} R_{55} + \frac{1}{2} R_{33} - \frac{1}{2} \sqrt{R_{33}^2 - 2 R_{33} R_{55} + 4 R_{35} R_{53} + R_{55}^2} \right). \quad (32)$$

The (i, k) element of the FV^{-1} of the next generation matrix represents the expected number of secondary infections in the compartment i caused by individuals in compartment k , assuming that the individual's environments remain consistent throughout the infection. It is worth noting that FV^{-1} Matrix is non-negative, meaning has a non-negative eigenvalue. This non-negative eigenvalue corresponds to a non-negative eigenvector that represents the distribution of infected individuals who generate the highest number of secondary infections per generation, also known as $\mathcal{R}_0$. According to [31], the basic reproduction number $\mathcal{R}_0$ is the largest eigenvalue of the next generation matrix, being given by

$$\mathcal{R}_0 = \rho(FV^{-1}). \quad (33)$$

Therefore, the spectral radius of the next generation Matrix is

$$\rho(FV^{-1}) = \frac{(R_{55} + R_{33}) + \sqrt{R_{33}(R_{33} - 2R_{55}) + 4R_{35}R_{53} + R_{55}^2}}{2}. \quad (34)$$

2.2.5. Local sensitivity analysis

Local sensitivity analysis seeks to determine how each parameter $\mathcal{P}_i$ affects the $\mathcal{R}_0$ and is determined by normalizing the sensitivity indices as approached by [32,33]:

$$\gamma_{\mathcal{P}_i}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \mathcal{P}_i} \times \frac{\mathcal{P}_i}{\mathcal{R}_0}, \quad (35)$$

where $\mathcal{R}_0$ is the rabies basic reproduction number. Therefore, utilizing equation (35) and the parameter values from Table 2, we calculate the sensitivity indices for each parameter, as indicated in Table 1.

The findings from the research presented in Table 1 are significant and provide crucial insights. This study highlights that an increase in the values of the rabies model parameters, including ψ_1 , ψ_2 , κ_1 , and κ_2 , results in a proportional rise in the magnitude of $\mathcal{R}_0$. This indicates that careful consideration of these parameters is essential in predicting the transmission dynamics of the disease. Furthermore, the study also reveals that an increase in the values of parameters such as γ_1 , γ_2 , μ_2 , μ_3 , ρ_1 , ρ_2 , σ_2 , and σ_3 leads to a decrease in the magnitude of $\mathcal{R}_0$. This implies that controlling the spread of the disease can be achieved by adjusting these parameters. It is noteworthy that a 20% increase in any of the parameters ψ_1 , ψ_2 , κ_1 , or κ_2 corresponds exactly to a 20% increase in the value of $\mathcal{R}_0$.

2.3. Existence of the steady state solution

The endemic equilibrium point is the steady state where rabies is present in humans, free-range dogs, and domestic dogs. To find this point, we set the equations of the model system (5) to zero and solve the resulting system simultaneously. The state variables for each compartment are represented by

$$\mathbb{E} (S_H^*, E_H^*, I_H^*, R_H^*, S_F^*, E_F^*, I_F^*, S_D^*, E_D^*, I_D^*, R_D^*, M^*)$$

with

$$\left. \begin{aligned} R_H^* &= \frac{\beta_2 (\sigma_1 + \mu_1) I_H^*}{\beta_1 (\beta_3 + \mu_1)}, \\ I_H^* &= \frac{\beta_1 (\beta_3 + \mu_3) (\sigma_1 + \mu_1)^2 (\beta_1 + \beta_2 + \beta_3) \mu_1 + \beta_1 \beta_3 (\sigma_1 + \mu_1)^2}{(\sigma_1 + \mu_1)^2 ((\beta_1 + \beta_2 + \beta_3) \mu_1 + \beta_1 \beta_3)} \\ &\quad - \frac{\beta_1 (\beta_3 + \mu_3) (\sigma_1 + \mu_1)^2 \beta_3 - \theta_1 (\beta_3 + \mu_3) (\sigma_1 + \mu_1)^2}{(\sigma_1 + \mu_1)^2 ((\beta_1 + \beta_2 + \beta_3) \mu_1 + \beta_1 \beta_3)}, \\ E_H^* &= \frac{(\sigma_1 + \mu_1) I_H^*}{\beta_1}, \\ S_H^* &= \frac{\beta_3 \beta_2 (\sigma_1 + \mu_1) I_H^*}{\beta_1 (\beta_3 + \mu_1) \mu_1} - \frac{(\mu_1 + \beta_1 + \beta_2) (\sigma_1 + \mu_1) I_H^*}{\beta_1 \mu_1} + \frac{\theta_1}{\mu_1}, \end{aligned} \right\}$$

$$\left. \begin{aligned} I_D^* &= \frac{\gamma_1 \psi_1 I_F^* (1 + \rho_2) (1 + \rho_3) M^* + \gamma_1 \psi_3 M^* (1 + \rho_1) (1 + \rho_2)}{(\mu_3 + \gamma_1 + \gamma_2)^2 - \gamma_1 \psi_2 (1 + \rho_1) (1 + \rho_3) M^* (\mu_3 + \gamma_1 + \gamma_2)}, \\ E_D^* &= \frac{(\mu_3 + \sigma_3) I_D^*}{\gamma_1}, \quad R_D^* = \frac{\gamma_2 (\mu_3 + \sigma_3) I_D^*}{\gamma_1 (\mu_3 + \gamma_3)}, \end{aligned} \right\}$$

$$\left. \begin{aligned} S_D^* &= \frac{\gamma_3 (\mu_3 + \sigma_3) I_D^*}{\mu_3 \gamma_1} - \frac{(\mu_3 + \gamma_1 + \gamma_2) \gamma_2 (\mu_3 + \sigma_3) I_D^*}{\gamma_1 (\mu_3 + \gamma_3) \mu_3} + \frac{\theta_3}{\mu_3}, \quad E_F^* = \frac{(\mu_2 + \sigma_2) I_F^*}{\gamma}, \\ I_F^* &= \frac{\gamma E_F^*}{\mu_2 + \sigma_2}, \quad S_F^* = \frac{\theta_2}{\mu_2} - \frac{(\mu_2 + \gamma) (\mu_2 + \sigma_2) I_F^*}{\gamma \mu}, \quad M^* = \frac{\nu_3 I_D^* + \nu_2 I_F^* + \nu_1 I_H^*}{\mu_4}, \end{aligned} \right\}$$

where

$$\left. \begin{aligned} \theta_2 &= \frac{(\mu_2 + \gamma) \mu_2 (1 + (R_0 - 1)) (1 + \rho_1) \mu_3 (\mu_2 + \sigma_2) ((1 + \rho_2) (\mu_3 + \sigma_3) (\mu_3 + \gamma_1 + \gamma_2) (1 + (R_0 - 1)) - \theta_3 \psi_2 \gamma_1)}{(\mu_3 (1 + \rho_2) (1 + \rho_1) (\mu_3 + \sigma_3) (\mu_3 + \gamma_1 + \gamma_2) (1 + (R_0 - 1)) - \theta_3 \gamma_1 (\psi_2 (1 + \rho_1) \mu_3 + \psi_1 (1 + \rho_2))) \gamma \kappa_1}, \\ \theta_3 &= \frac{(-\mu_2 (\mu_2 + \sigma_2) (\mu_2 + \gamma) (1 + (R_0 - 1)) + \gamma \kappa_1 \theta_2) (1 + (R_0 - 1)) (1 + \rho_1) \mu_3 (1 + \rho_2) (\mu_3 + \sigma_3) (\mu_3 + \gamma_1 + \gamma_2)}{((- \mu_2 (\mu_2 + \sigma_2) (\mu_2 + \gamma) (1 + (R_0 - 1)) + \gamma \kappa_1 \theta_2) (1 + \rho_1) \psi_2 \mu_3 + \gamma \kappa_1 \theta_2 \psi_1 (1 + \rho_2)) \gamma_1}. \end{aligned} \right\}$$

The endemic equilibrium point of the rabies disease persists when $I_H, I_F, I_D, M > 0$ and $R_0 \geq 1$, as summarized in Theorem 3.

Theorem 3. The system model (5) has a unique endemic equilibrium if $R_0 \geq 1$ and $I_H, I_F, I_D, M > 0$.

3. Stability analysis

We begin by studying the local stability of the disease free equilibrium (DFE) point (Section 3.1). Then, we investigate its global stability (Section 3.2). The stability of the endemic equilibrium is given in Appendix A.

3.1. Local stability of the disease free equilibrium point

We prove local stability of the disease free equilibrium point with the help of the Routh–Hurwitz criterion.

Theorem 4. The DFE point $\mathcal{E}_0$ is locally asymptotically stable if $R_0 < 1$ and all eigenvalues of the Jacobian matrix $(J(\mathcal{E}_0))$ evaluated at $\mathcal{E}_0$ have negative real parts.

Proof. We need to show that all the eigenvalues of the matrix $J(\mathcal{E}_0)$ in equation (36) of the model system (5) at the DFE point have negative real parts. Subsequently, since the endemic equilibrium exists if, and only if, $R_0 < 1$, we utilize the Jacobian matrix at the disease-free state $J(\mathcal{E}_0)$, which is expressed as

$$J(\mathcal{E}_0) = \begin{pmatrix} -\mu_1 & 0 & 0 & 0 & 0 & 0 & -\frac{\tau_1\theta_1}{\mu_1} & 0 & 0 & -\frac{\tau_2\theta_1}{\mu_1} & 0 & 0 \\ 0 & -a_1 & 0 & 0 & 0 & 0 & \frac{\tau_1\theta_1}{\mu_1} & 0 & 0 & \frac{\tau_2\theta_1}{\mu_1} & 0 & 0 \\ 0 & \beta_1 & -a_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_2 & 0 & -a_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_2 & 0 & -\frac{\kappa_1\theta_2}{\mu_2} & 0 & 0 & -\frac{\kappa_2\theta_2}{\mu_2} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -a_4 & \frac{\kappa_1\theta_2}{\mu_2} & 0 & 0 & \frac{\kappa_2\theta_2}{\mu_2} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma & -a_5 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\psi_1\theta_3}{\mu_3(1+\rho_1)} & -\mu_3 & 0 & -\frac{\psi_2\theta_3}{\mu_3(1+\rho_2)} & \gamma_3 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\psi_1\theta_3}{\mu_3(1+\rho_1)} & 0 & -a_6 & \frac{\psi_2\theta_3}{\mu_3(1+\rho_1)} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma & -a_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_2 & 0 & -a_8 & 0 \\ 0 & 0 & \nu_1 & 0 & 0 & 0 & \nu_2 & 0 & 0 & \nu_3 & 0 & -\mu_4 \end{pmatrix}, \quad (36)$$

where

$$a_1 = \mu_1 + \beta_1 + \beta_2, \quad a_2 = \mu_1 + \sigma_1, \quad a_3 = \mu_3 + \beta_3, \quad a_4 = \mu_2 + \gamma,$$

$$a_5 = \mu_2 + \sigma_2, \quad a_6 = \mu_3 + \gamma_1 + \gamma_2, \quad a_7 = \mu_3 + \sigma_3, \quad a_8 = \mu_3 + \gamma_3.$$

The first, fourth, fifth, eighth, and twelfth columns of the matrix $(J(\mathcal{E}_0))$ in equation (36) contain the diagonal terms. It is obvious from the eigenvalues $\lambda_1 = -\mu_1$, $\lambda_2 = -a_3$, $\lambda_3 = -\mu_2$, $\lambda_4 = -\mu_3$, and $\lambda_5 = -\mu_4$, respectively. Thus, the matrix $(J(\mathcal{E}_0))$ reduces to

$$J(\mathcal{E}_0) = \begin{pmatrix} -a_1 & 0 & 0 & \frac{\tau_1\theta_1}{\mu_1} & 0 & \frac{\tau_2\theta_1}{\mu_1} & 0 \\ \beta_1 & -a_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -a_4 & \frac{\kappa_1\theta_2}{\mu_2} & 0 & \frac{\kappa_2\theta_2}{\mu_2} & 0 \\ 0 & 0 & \gamma & -a_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\psi_1\theta_3}{\mu_3(1+\rho_1)} & -a_6 & \frac{\psi_2\theta_3}{\mu_3(1+\rho_1)} & 0 \\ 0 & 0 & 0 & 0 & \gamma & -a_7 & 0 \\ 0 & 0 & 0 & 0 & \gamma_2 & 0 & -a_8 \end{pmatrix}. \quad (37)$$

Again, the second and seventh columns of the matrix $(J(\mathcal{E}_0))$ in (37) contain the diagonal terms. It is obvious from the eigenvalues $\lambda_6 = -a_2$ and $\lambda_7 = -a_8$. Thus, the matrix $(J(\mathcal{E}_0))$ reduces to

$$J(\mathcal{E}_0) = \begin{pmatrix} -a_1 & 0 & \frac{\tau_1\theta_1}{\mu_1} & 0 & \frac{\tau_2\theta_1}{\mu_1} \\ 0 & -a_4 & \frac{\kappa_1\theta_2}{\mu_2} & 0 & \frac{\kappa_2\theta_2}{\mu_2} \\ 0 & \gamma & -a_5 & 0 & 0 \\ 0 & 0 & \frac{\psi_1\theta_3}{\mu_3(1+\rho_1)} & -a_6 & \frac{\psi_2\theta_3}{\mu_3(1+\rho_1)} \\ 0 & 0 & 0 & \gamma & -a_7 \end{pmatrix}. \quad (38)$$

Again, the first column of the matrix $(J(\mathcal{E}_0))$ in equation (38) contains the diagonal term, and it is obvious from eigenvalues $\lambda_8 = -a_1$. Thus, the matrix $(J(\mathcal{E}_0))$ reduces to

$$J(\mathcal{E}_0) = \begin{pmatrix} -a_4 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} \\ \gamma & -a_5 & 0 & 0 \\ 0 & \frac{\psi_1 \theta_3}{\mu_3(1+\rho_1)} & -a_6 & \frac{\psi_2 \theta_3}{\mu_3(1+\rho_1)} \\ 0 & 0 & \gamma & -a_7 \end{pmatrix}. \quad (39)$$

Computing the eigenvalues of the given matrix $J(\mathcal{E}_0)$ in equation (39) involves solving the characteristic polynomial equation

$$P(\lambda) = \det(J(\mathcal{E}_0) - \lambda I) = 0,$$

where I is the identity matrix and λ represents the eigenvalues. Thus,

$$\lambda^4 + C_1 \lambda^3 + C_2 \lambda^2 + C_3 \lambda + C = 0. \quad (40)$$

Equations (36), (37), and (38) evidence that $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6, \lambda_7$, and λ_8 exhibit negative real parts. By applying the Routh–Hurwitz criterion, the other four eigenvalues of the matrix equation (39) will also have negative real parts if all coefficients in equation (40) are greater than zero. Then,

$$\begin{cases} C_1 = a_7 + a_6 + a_5 + a_4 > 0, \\ C_2 = -\gamma b_1 - \gamma b_4 + a_5 a_4 + a_6 a_4 + a_7 a_4 + a_6 a_5 + a_7 a_5 + a_7 a_6 > 0, \\ C_3 = ((a_6 + a_7)a_5 - \gamma b_4 + a_7 a_6) a_4 + (-\gamma b_4 + a_7 a_6) a_5 - \gamma b_1(a_6 + a_7) > 0, \\ C = \gamma^2 b_1 b_4 - \gamma^2 b_2 b_3 - \gamma a_4 a_5 b_4 - \gamma a_6 a_7 b_1 + a_4 a_5 a_6 a_7 > 0, \end{cases}$$

where

$$\begin{cases} b_1 = \frac{\kappa_1 \theta_2}{\mu_2}, & b_2 = \frac{\psi_1 \theta_3}{\mu_3(1+\rho_1)}, \\ b_3 = \frac{\kappa_2 \theta_2}{\mu_2}, & b_4 = \frac{\psi_2 \theta_3}{\mu_3(1+\rho_1)}. \end{cases}$$

Since all eigenvalues of the Jacobian matrix $(J(\mathcal{E}_0))$ evaluated at $\mathcal{E}_0$ have negative real parts, the model system (5) at the $\mathcal{E}_0$ is locally asymptotically stable if $\mathcal{R}_0 < 1$. $\square$

3.2. Global stability of the DFE point

We prove the global stability of the DFE point $\mathcal{E}_0$ of the rabies model (5) using the theorem described by [38]. To apply the theorem, we write the model system (5) as

$$\left. \begin{aligned} \frac{dY_s}{dt} &= G_0(Y_s - Y(\mathcal{E}_0)) + G_1 Y_i, \\ \frac{dY_i}{dt} &= G_2 Y_i, \end{aligned} \right\}$$

where Y_s is the vector representing the compartments that do not transmit the rabies disease, and Y_i symbolizes the rabies-transmitting vector compartments. In the case of G_2 , if G_2 is a Metzler matrix (i.e., the off-diagonal entries of G_2 are non-negative), and G_0 has real negative eigenvalues, the rabies-free equilibrium is globally asymptotically stable. Based on the model system (5), we have $Y_s = (S_H, R_H, S_F, S_D, R_D)^T$, $Y_i = (E_H, I_H, E_F, I_F, E_D, I_D, M)^T$ and

$$Y_s - Y(\mathcal{E}_0) = \begin{pmatrix} S_H - \frac{\theta_1}{\mu_1} \\ R_H \\ S_F - \frac{\theta_2}{\mu_2} \\ S_D - \frac{\theta_3}{\mu_3} \\ R_D \end{pmatrix},$$

$$G_0 = \begin{pmatrix} -\mu & \beta_3 & 0 & 0 & 0 \\ 0 & -(\beta_3 + \mu_1) & 0 & 0 & 0 \\ 0 & 0 & -\mu_2 & 0 & 0 \\ 0 & 0 & 0 & -\mu_3 & \gamma_3 \\ 0 & 0 & 0 & 0 & -(\mu_3 + \gamma_3) \end{pmatrix}.$$

The eigenvalues of the matrix G_0 are $\lambda_1 = \mu_3$, $\lambda_2 = \mu_2$, $\lambda_3 = \mu_1$, $\lambda_4 = -(\mu_3 + \gamma_3)$, $\lambda_5 = -(\beta_3 + \mu_1)$, while

$$G_1 = \begin{pmatrix} 0 & 0 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ \beta_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} & 0 \\ 0 & 0 & 0 & \frac{\psi_1 \theta_3}{\mu_3 (1 + \rho_1)} & 0 & \frac{\psi_2 \theta_3}{\mu_3 (1 + \rho_2)} & 0 \\ 0 & 0 & 0 & 0 & \gamma_2 & 0 & 0 \end{pmatrix},$$

$$G_2 = \begin{pmatrix} -\mu_1 - \beta_1 - \beta_2 & 0 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ \beta_1 & -\sigma_1 - \mu_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\mu_2 - \gamma & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} & 0 \\ 0 & 0 & \gamma & -\mu_2 - \sigma_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\psi_1 \theta_3}{\mu_3 (1 + \rho_1)} & -\mu_3 - \gamma_1 - \gamma_2 & \frac{\psi_2 \theta_3}{\mu_3 (1 + \rho_1)} & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\mu_3 - \sigma_3 & 0 \\ 0 & v_1 & 0 & v_2 & 0 & v_3 & -\mu_4 \end{pmatrix}.$$

Since the eigenvalues of the G_0 are negative and the off-diagonal entries of the Metzler matrix G_2 are non-negative, then the rabies DFE point is globally asymptotically stable.

4. Model fitting and parameter estimation

After conducting model analysis of the dynamics and qualitative outcomes of the rabies model, it is essential to accurately determine the model's parameters for making quantitative predictions within a limited time frame using real-world data [39]. In this study, we employed the non-linear least squares method (NLSM) to estimate the parameters of model equation (5). To achieve this, we generated synthetic data that represented the expected disease spread patterns at various time points, denoted as t_i [40]. These patterns were computed by numerically solving equation (5) with a fifth-order Runge–Kutta method in the MATLAB environment, initializing the parameters with values from literature denoted as Θ_i and initial condition for the number of $S_H(0) = 142000$, $E_H(0) = 40$, $I_H(0) = 0$, $R_H(0) = 0$, $S_D(0) = 15000$, $E_D(0) = 25$, $I_D(0) = 0$, $R_D(0) = 0$, $S_F(0) = 12500$, $E_F(0) = 20$, $I_F(0) = 0$, and $M(0) = 90$. In order to generate the rabies dataset $RD(t_i, \Theta_i)$ we added random Gaussian noise $\eta_i(t_i, \Theta_i)$ measurements to the data, simulating real-world dynamics where measurement errors are common. Thus the observed/actual dependent data were given as

$$Y_i = RD(t_i, \Theta_i) + \eta_i(t_i, \Theta_i) \text{ for each time } t_i \in [1, n].$$

The parameter values $\overline{Y\overline{Y}}$ of Table 2 were determined by minimizing the sum of squared residuals expressed as

$$\overline{Y\overline{Y}}(\Theta) = \min \sum_{k=1}^n (Y_i - Y)^2$$

between the model solutions (Y) obtained through solving the rabies (5) model using the real parameters from the generated data and the synthetic data (Y_i) generated by introducing random Gaussian noise to the model output $RD(t_i, \Theta_i)$ [39]. The estimated parameter values were then used to fit the data (Y_i), and the resulting best fits are depicted in Fig. 2(a)–(d) and the resulting estimated parameters given in Table 2.

5. Numerical simulations

In this section, we employed the `ode45` method available in MATLAB software to numerically solve a model system (5) using parameters presented in Table 2 along with the initial conditions $S_H(0) = 142000$, $E_H(0) = 40$, $I_H(0) = 0$, $R_H(0) = 0$, $S_D(0) =$

Table 2
Model parameters, their description and values.

Parameters	Description	Value (Year ⁻¹)	Source
θ_1	Recruitment rate S_H	2000	(Estimated)
τ_1	The rate that S_H gets infection from I_F	0.0004	[10]
τ_2	The rate that S_H gets infection from I_D	0.0004	[10]
τ_3	The rate that S_H gets infection from M	[0.0003 0.0100]	(Estimated)
β_1	Progression rate out of E_H to I_H	$\frac{1}{6}$	[10,32]
β_2	Recovery rate of E_H	[0.54 1]	[12,32]
β_3	Rate of immunity loss of humans	1	(Estimated)
μ_1	Natural death rate of humans	0.0142	[34,35]
σ_1	Disease induced death rate for I_H	1	[12,32]
θ_2	Recruitment rate of free-range dogs	1000	(Estimated)
κ_1	The rate that S_F gets infection from I_F	0.00006	(Estimated)
κ_2	The rate that S_F gets infection from I_D	0.00005	(Estimated)
κ_3	The rate that S_F gets infection from M	[0.00001 0.00003]	(Estimated)
γ	The rate that S_F gets infection from I_F	$\frac{1}{6}$	[10,12,32]
σ_2	Disease induced death rate of I_F	0.09	[32,36]
μ_2	Natural mortality rate of free-range dogs	0.067	(Estimated)
θ_3	Recruitment rate of domestic dog population	1200	(Estimated)
ψ_1	The rate that S_D gets infection from I_D	0.0004	[24,36]
ψ_2	The rate that S_D gets infection from I_F	0.0004	[7]
ψ_3	The rate that S_D gets infection from M	0.0003	(Estimated)
μ_3	Natural death rate for domestic dog population	0.067	(Estimated)
σ_3	Disease induced death rate for I_D	0.08	[32]
γ_1	The rate at which E_D becomes I_D	$\frac{1}{6}$	[10,32]
γ_2	Recovery rate of E_D	0.09	[32]
γ_3	Rate of loss of temporary immunity for R_D	0.05	(Estimated)
v_1	Environmental virus shedding rate from I_H	0.001	(Estimated)
v_2	Environmental virus shedding rate from I_F	0.006	(Estimated)
v_3	Environmental virus shedding rate from I_D	0.001	(Estimated)
μ_4	Natural removal rate of rabies from the environment	0.08	(Estimated)
ρ_1	The deterrent coefficient of domestic dog from I_F	10	[37]
ρ_2	The deterrent coefficient of domestic dog from I_D	8	(Estimated)
ρ_3	The deterrent coefficient of domestic dog from M	15	(Estimated)
C	Concentration of rabies in the environment	0.003 (PFU)/mL	(Estimated)

15000, $E_D(0) = 25$, $I_D(0) = 0$, $R_D(0) = 0$, $S_F(0) = 12500$, $E_F(0) = 20$, $I_F(0) = 0$, and $M(0) = 90$. The objective is to illustrate the analytical findings discussed in earlier sections.

5.1. Impact of the periodic infection rate on the occurrence of rabies outbreaks

The periodic effect of rabies on bite incidence describes the cyclic variation in the number of dog bites within a population due to recurrent outbreaks of rabies in dogs [41]. These outbreaks stem from the viral infection's influence on dog behavior, causing increased aggression and a propensity to bite. This cyclic pattern arises as rabies outbreaks occur intermittently, influenced by factors like seasonal fluctuations, vaccination efforts, and animal movement. To investigate on the effect of periodic infection for bite incidence, we employed the formula described as

$$\text{Bite Incidence}(t) = \beta_{\text{mean}} (1 + A \sin(2\pi f t + \phi)) SI, \quad (41)$$

where β_{mean} is the infection rate, A is the amplitude,

$$f = \frac{t}{\text{period of control of outbreak}} = \frac{t}{T} \text{ is the frequency of sinusoidal variation, and } \phi \text{ is the phase shift of } S, I.$$

Therefore, to incorporate and unify the changing dynamics of rabies, transmission rates τ_1 , τ_2 , τ_3 , ψ_1 , ψ_2 , ψ_3 , κ_1 , κ_2 , κ_3 , v_1 , v_2 and v_3 , as applied by [42], are considered as

$$\begin{aligned} \tau_i &= \tau_{(\text{mean})} \left(1 + A_i \sin \left(\frac{2\pi t}{T} + \phi \right) \right), & \psi_i &= \psi_{(\text{mean})} \left(1 + A_i \sin \left(\frac{2\pi t}{T} + \phi \right) \right), \\ \kappa_i &= \kappa_{(\text{mean})} \left(1 + A_i \sin \left(\frac{2\pi t}{T} + \phi \right) \right), & v_i &= v_{(\text{mean})} \left(1 + A_i \sin \left(\frac{2\pi t}{T} + \phi \right) \right), \end{aligned}$$

for $i = 1, 2$ and 3 .

The results of bite incidence are presented in Fig. 3(a)–(b), Fig. 4(a)–(b), Fig. 5(a)–(b), and Fig. 6(a)–(b).

Fig. 3(a)–(b), Fig. 4(a)–(b), and Fig. 5(a)–(b) illustrate that an increase in bite incidents leads to a rise in the number of exposed and infected individuals while reducing the number of susceptible individuals in both human and dog populations. Furthermore, all these figures demonstrate that the rabies outbreak, driven by a higher infection rate, remains active within the first 20 years and,

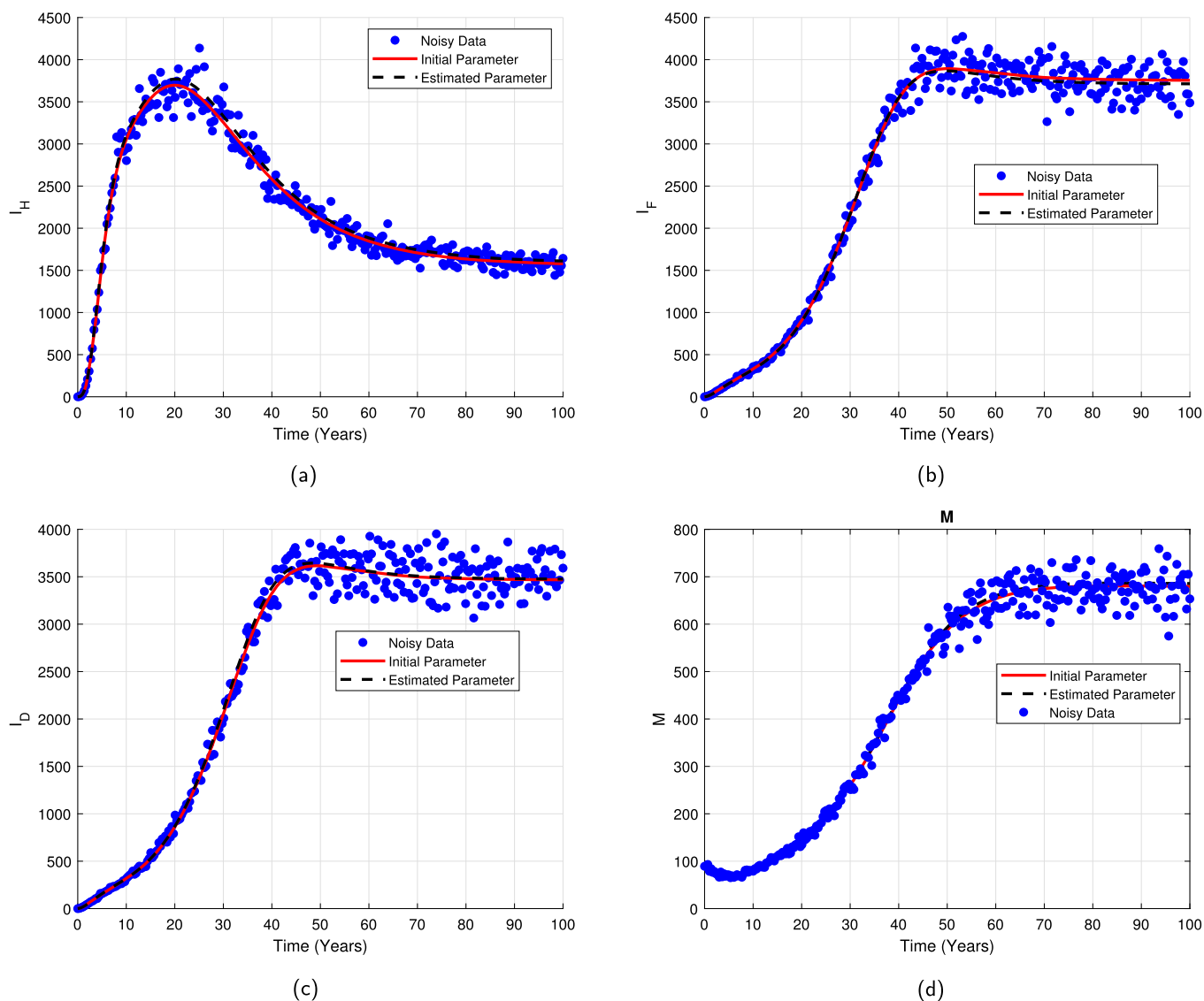


Fig. 2. Scatter estimated with standard deviation of 0.05 and numerical simulation (sold) with confidence interval of 95%.

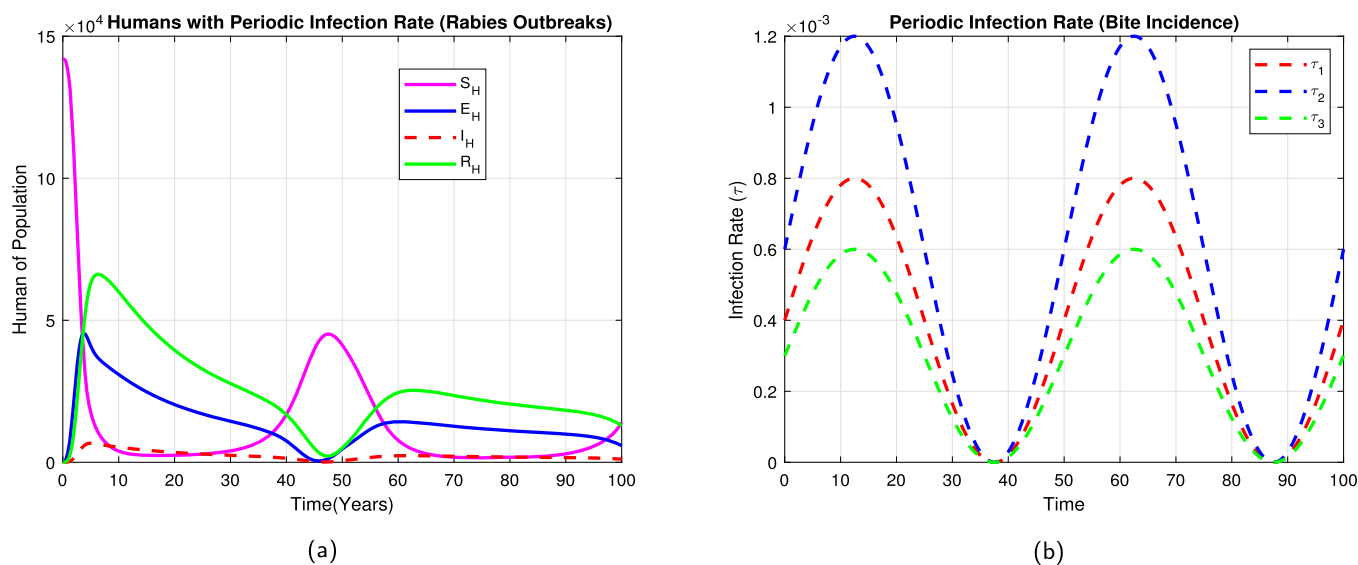


Fig. 3. The impact of human population bite incidence on the occurrence of periodic rabies outbreaks.

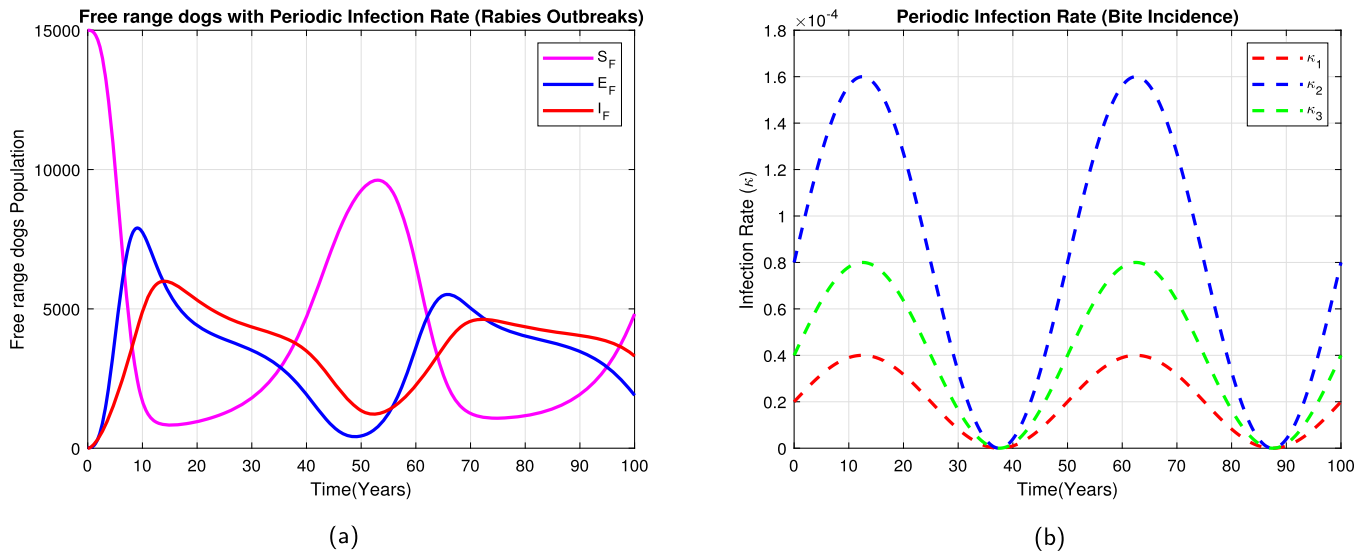


Fig. 4. The impact of free range dogs bite incidence on the occurrence of periodic rabies outbreaks.

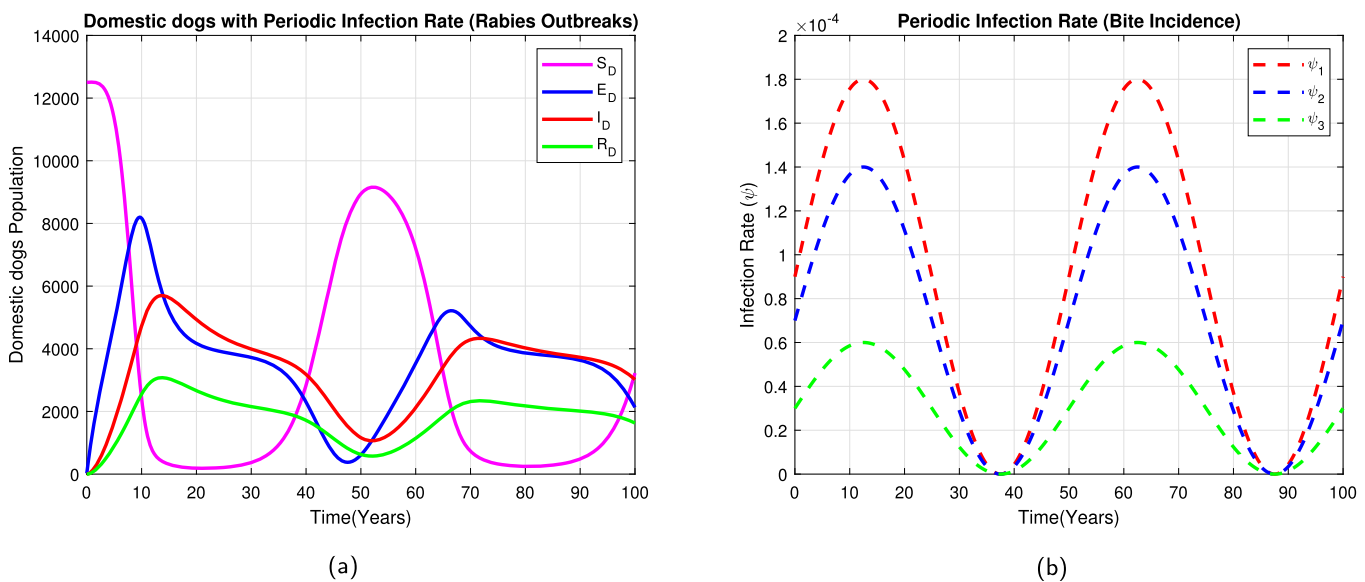


Fig. 5. The impact of domestic dogs bite incidence on the occurrence of periodic rabies outbreaks.

subsequently, exhibits periodic declines. This decline in the number of infections is attributed to the decrease in the infection rate in both populations. On the other hand, Fig. 6(a)–(b) shows that an increase in the rate of shedding into the environment results in periodic rises in rabies contamination within the environment. These scenarios highlight the significance of effective vaccination campaigns, responsible pet ownership, and timely post-exposure prophylaxis for individuals who have been bitten. These measures are essential for managing the public health impact of this periodic phenomenon, underscoring the importance of rabies control strategies.

The results presented in Fig. 7(a)–(b) demonstrate that the parameters ψ_1 , ψ_2 , κ_1 , and κ_2 have a positive impact on the basic reproductive number, $\mathcal{R}_0$. The study reveals that changes in these parameters generate varying effects on $\mathcal{R}_0$, ranging from 1.8 to 2.0 and 1.2 to 2.4, respectively. These findings support the estimates provided by [18] and suggest that intervention strategies can have a significant impact on the incidence of rabies in a given population. Furthermore, the parameter values outlined in Table 2 indicate that increasing κ_1 , κ_2 , ψ_1 , and ψ_2 corresponds to an increase in $\mathcal{R}_0$.

5.2. Effect of varying the most sensitive parameters

Now we investigate the impact of the contact rate between infectious agent and: (i) susceptible human; (ii) susceptible domestic dogs; and (iii) free range dogs.

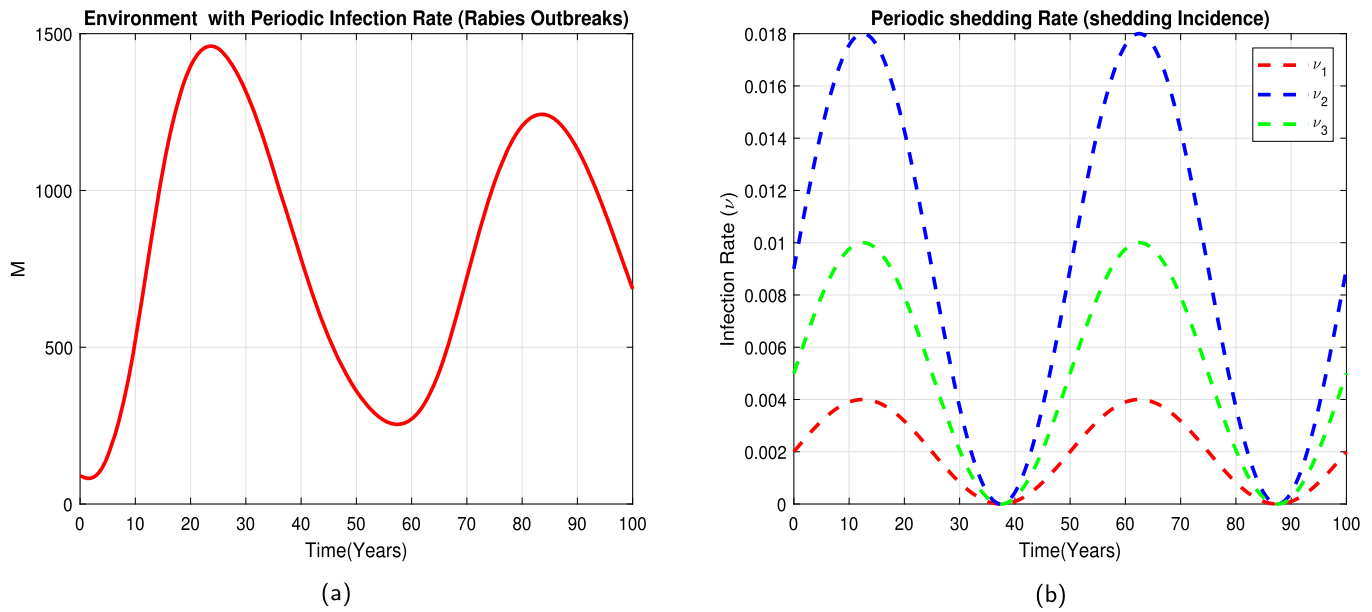


Fig. 6. The impact of environment shedding incidence on the occurrence of periodic rabies outbreaks.

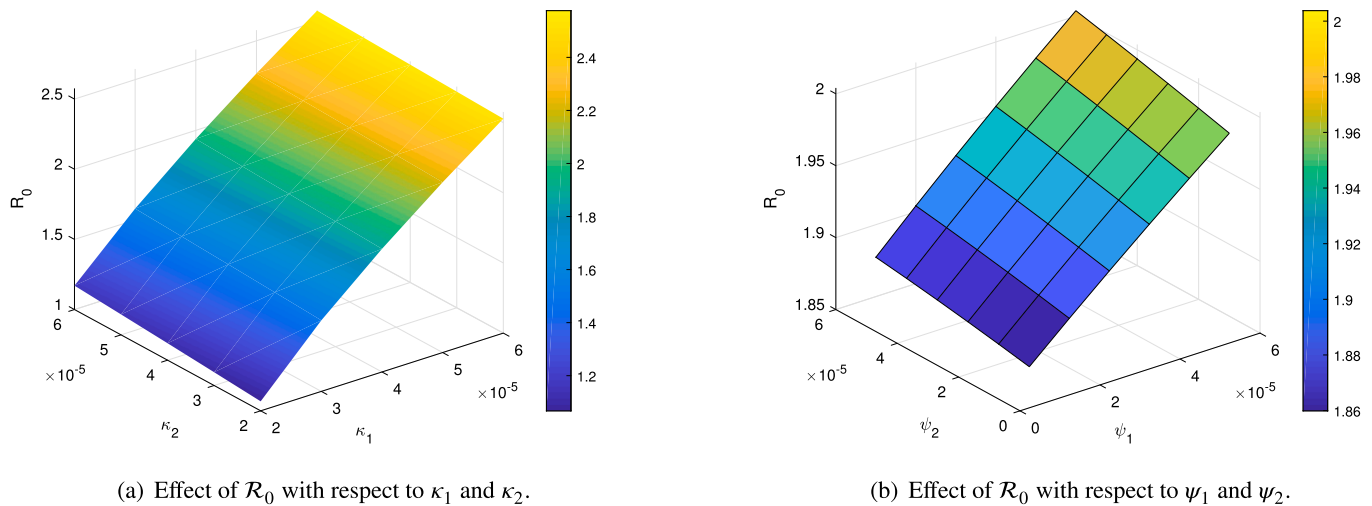


Fig. 7. Effect of R_0 with respect to κ_1 , κ_2 , ψ_1 , and ψ_2 .

5.2.1. Impact of contact rate between infectious agent and susceptible human

The findings presented in Fig. 8(a)–(c) demonstrate that the contact rates τ_1 , τ_2 , and τ_3 exert a significant influence on the transmission dynamics among susceptible humans, infected free-range and domestic dogs, and the environment. Nevertheless, it is noted that these dynamics ultimately reach a stable state after 80 years. This underscores the crucial role of education and awareness in mitigating the transmission of rabies among the human population by reducing contact between susceptible humans and sources carrying the rabies virus.

5.2.2. Impact of contact rate between infectious agent and susceptible domestic dogs

The findings presented in Fig. 9(a)–(c) reveal that an increase in contact rates, denoted as ψ_1 , ψ_2 , and ψ_3 , results in a higher prevalence of rabies in domestic dogs. After approximately 50 years, the number of infected dogs reaches a steady state, implying that mitigating the contact between susceptible, infected, and free-range dogs and the environment carrying the rabies virus is critical to reduce the transmission of the disease.

5.2.3. Impact of contact rate between infectious agent and free range dogs

Fig. 10(a) presents the finding that an increase in the contact rate κ_1 results in a higher number of susceptible free-range dogs becoming infected, which suggests an inadequacy of control measures. Conversely, Fig. 10(b) portrays that a rise in the contact rate κ_2 with free-range dogs leads to an increase in carriers and symptomatic infections. In addition, Fig. 10(c) indicates that an increase in the contact rate κ_3 between free-range dogs and the environment yields a slight upsurge in the number of infectious individuals or carriers.

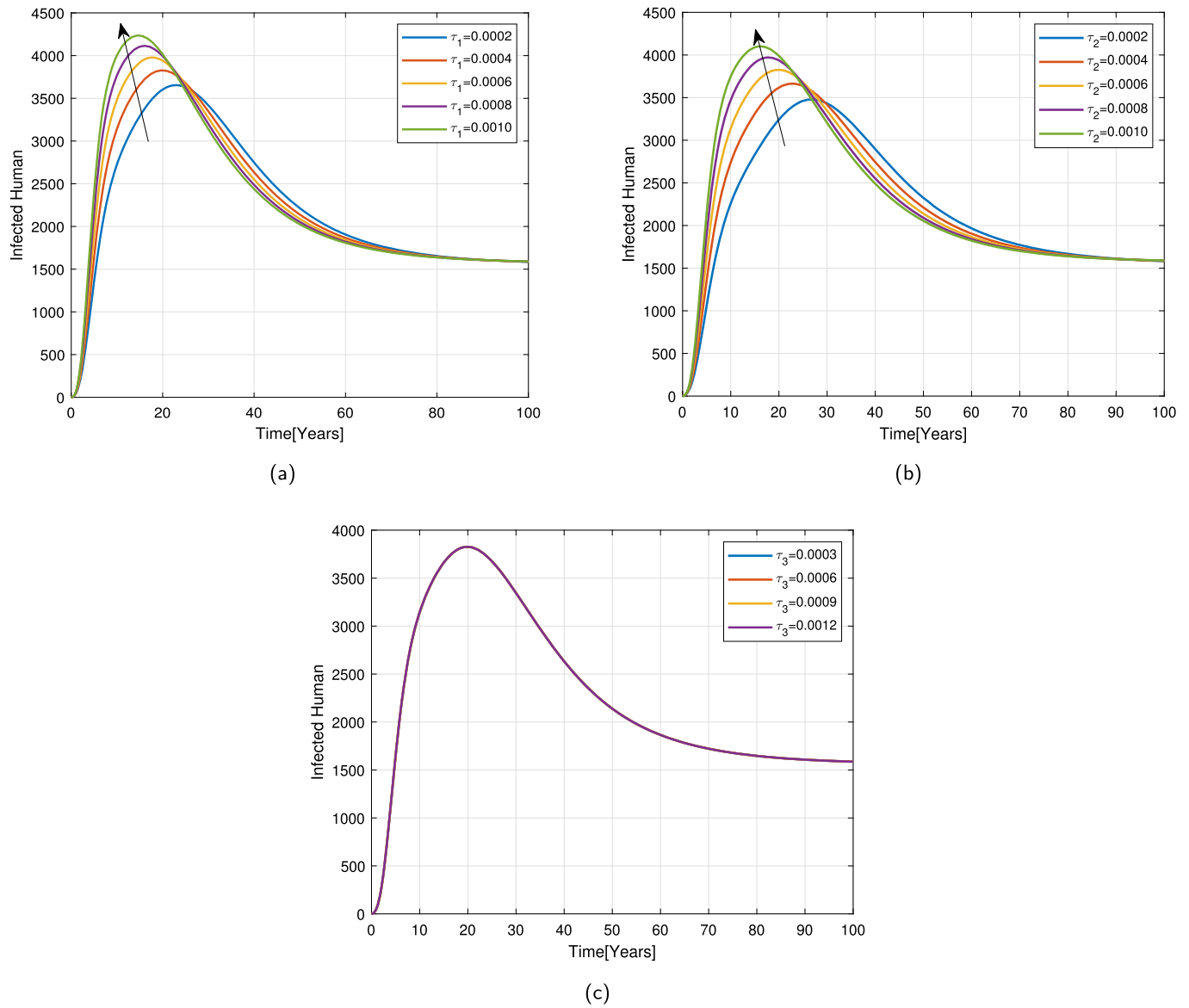


Fig. 8. Simulation results of model (5) for I_H with respect to τ_1 , τ_2 , and τ_3 .

6. Discussion

Rabies is a fatal viral disease that can easily spread from an infected animal to a human, making it a significant public health concern worldwide. Dogs are the primary reservoir and transmitter of rabies to humans, causing most human cases. To understand the transmission dynamics of rabies and develop effective prevention and control strategies, a study was conducted. The study aimed to create a deterministic model to investigate how changes in contact rates and environmental conditions impact the spread of rabies. Mathematical tools such as Jacobian and Metzler matrices were used to conduct stability analyses and uncover the underlying dynamics of rabies transmission. The study also aimed to determine the relationship between contact rates, environmental factors, and the basic reproduction number $\mathcal{R}_0$, which is a crucial indicator of disease spread. By gaining insights into the complex dynamics of rabies transmission, the study hopes to contribute to the development of targeted and sustainable strategies for its prevention and control.

7. Conclusion

The transmission of rabies among humans and dogs is influenced by their contact rate and environmental factors. A deterministic model was created to investigate how these factors affect the spread of rabies that results from dog bites. Stability analysis was conducted using Jacobian and Metzler matrices. The study's numerical simulations showed that the transmission of rabies from dog bites has serious consequences for both human and dog populations. Furthermore, the study found that an increase in the contact rate (such as ψ_1 , ψ_2 , ψ_3 , κ_1 , κ_2 , and κ_3) leads to a rise in the basic reproduction number $\mathcal{R}_0$. By examining the relationship between

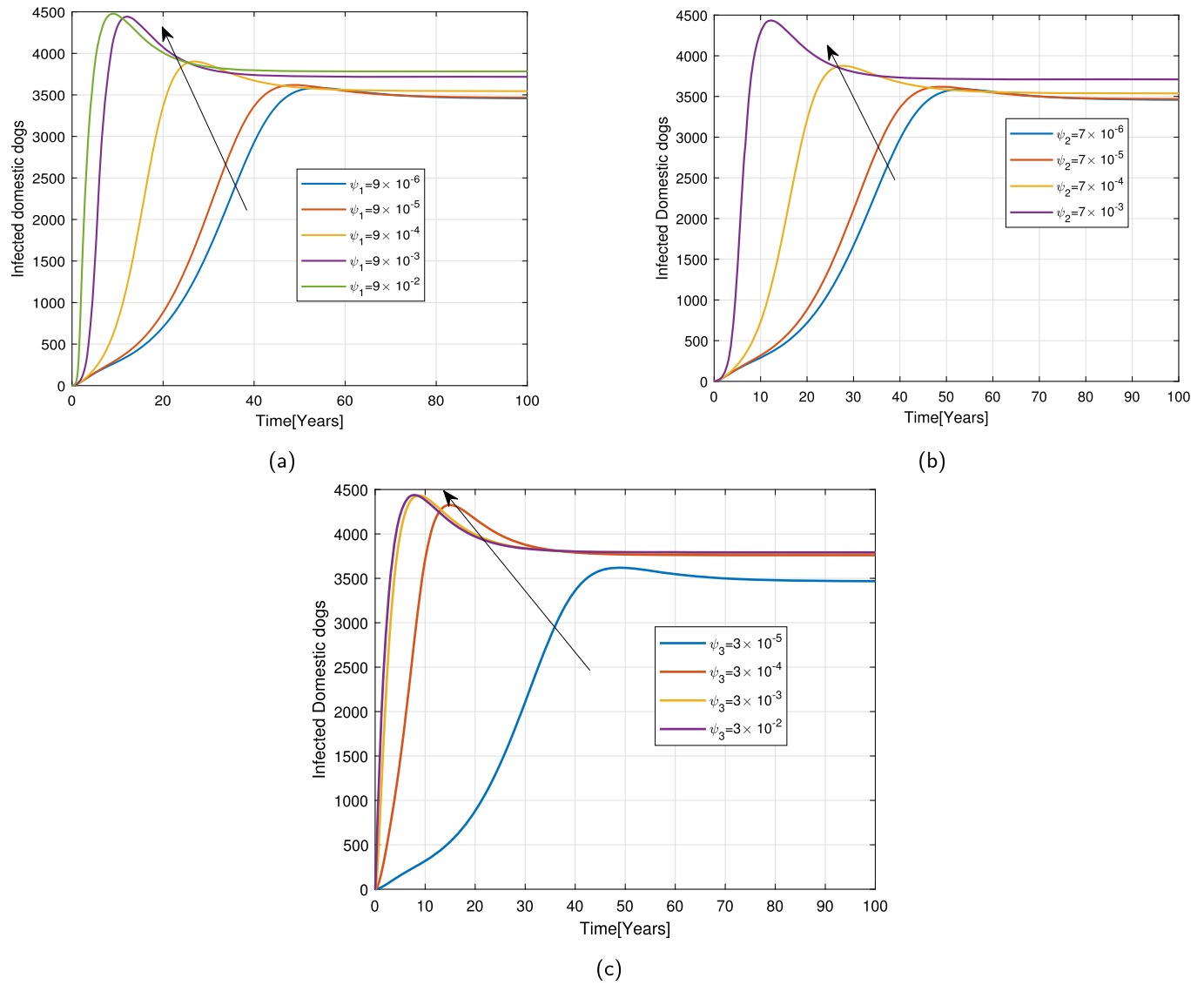


Fig. 9. Simulation results of model (5) for I_D with respect to ψ_1 , ψ_2 , and ψ_3 .

contact rate, environmental impact, and the transmission dynamics of rabies, this study provides insights into the complexity of rabies transmission. Ultimately, it contributes to the development of targeted, sustainable strategies for preventing and controlling the spread of rabies.

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CRediT authorship contribution statement

Mfano Charles: Writing – original draft, Visualization, Validation, Software, Methodology, Formal analysis, Conceptualization. **Verdiana G. Masanja:** Writing – review & editing, Supervision. **Delfim F.M. Torres:** Writing – review & editing, Supervision. **Sayoki G. Mfinanga:** Writing – review & editing, Supervision. **G.A. Lyakurwa:** Writing – review & editing, Supervision.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

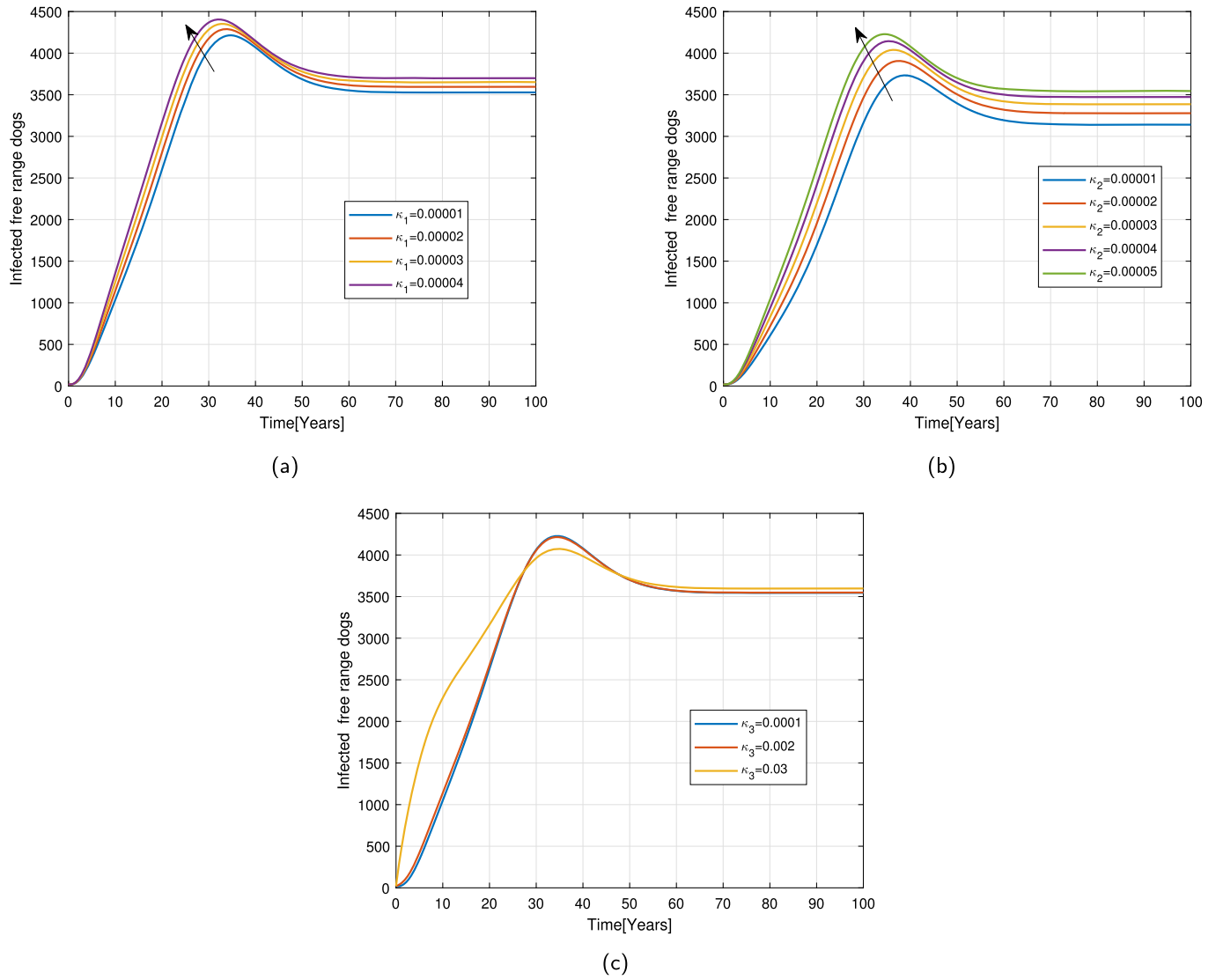


Fig. 10. Simulation results of model (5) for I_F with respect to τ_1 , τ_2 , and τ_3 .

Data availability

The data used in this study is available from the corresponding author upon request.

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Appendix A. Global stability of the endemic equilibrium $\mathbb{E}^*$

Here we prove the global stability of the endemic equilibrium characterized in Section 2.3.

Theorem 5. *The endemic equilibrium point $\mathbb{E}^*$ of the rabies model (5) is globally asymptotically stable whenever $\mathcal{R}_0 \geq 1$.*

Proof. To prove Theorem 5, we adopt the approach of [19,43,44] by constructing a Lyapunov function of the form

$$\mathcal{L} = \sum_{i=1}^n G_i \left(y_i - y_i^* + y_i^* \ln \left(\frac{y_i}{y_i^*} \right) \right), G_i > 0 \text{ for } i = 1, 2, 3, \dots, n,$$

where G_i represents a positive constant that needs to be determined, y_i stands for the population variable at compartment i , and y_i^* denotes the equilibrium point of the rabies model at compartment i for $i \in \{1, 2, 3, \dots, 12\}$. Therefore, we define the Lyapunov $\mathcal{L}$ for model system (5) as follows:

$$\begin{aligned} \mathcal{L} = & G_1 \left(S_H - S_H^* + S_H \ln \left(\frac{S_H}{S_H^*} \right) \right) + G_2 \left(E_H - E_H^* + E_H \ln \left(\frac{E_H}{E_H^*} \right) \right) + G_3 \left(I_H - I_H^* + I_H \ln \left(\frac{I_H}{I_H^*} \right) \right) \\ & + G_4 \left(R_H - R_H^* + R_H \ln \left(\frac{R_H}{R_H^*} \right) \right) + G_5 \left(S_F - S_F^* + S_F \ln \left(\frac{S_F}{S_F^*} \right) \right) + G_6 \left(E_F - E_F^* + E_F \ln \left(\frac{E_F}{E_F^*} \right) \right) \\ & + G_7 \left(I_F - I_F^* + I_F \ln \left(\frac{I_F}{I_F^*} \right) \right) + G_8 \left(S_D - S_D^* + S_D \ln \left(\frac{S_D}{S_D^*} \right) \right) + G_9 \left(E_D - E_D^* + E_D \ln \left(\frac{E_D}{E_D^*} \right) \right) \\ & + G_{10} \left(I_D - I_D^* + I_D \ln \left(\frac{I_D}{I_D^*} \right) \right) + G_{11} \left(R_D - R_D^* + R_D \ln \left(\frac{R_D}{R_D^*} \right) \right) + G_{12} \left(M - M^* + M \ln \left(\frac{M}{M^*} \right) \right). \end{aligned} \quad (42)$$

Evaluating equation (42) at the endemic equilibrium point $\mathbb{E}^*$ gives

$$\mathcal{L} = \mathbb{E}^* (S_H^*, E_H^*, I_H^*, R_H^*, S_F^*, E_F^*, I_F^*, S_D^*, E_D^*, I_D^*, R_D^*, M^*) = 0.$$

Then, using the time derivative of the Lyapunov function $\mathcal{L}$ in equation (42) gives

$$\begin{aligned} \frac{d\mathcal{L}}{dt} = & G_1 \left(1 - \frac{S_H^*}{S_H} \right) \frac{dS_H}{dt} + G_2 \left(1 - \frac{E_H^*}{E_H} \right) \frac{dE_H}{dt} + G_3 \left(1 - \frac{I_H^*}{I_H} \right) \frac{dI_H}{dt} + G_4 \left(1 - \frac{R_H^*}{R_H} \right) \frac{dR_H}{dt} \\ & + G_5 \left(1 - \frac{S_F^*}{S_F} \right) \frac{dS_F}{dt} + G_6 \left(1 - \frac{E_F^*}{E_F} \right) \frac{dE_F}{dt} + G_7 \left(1 - \frac{I_F^*}{I_F} \right) \frac{dI_F}{dt} + G_8 \left(1 - \frac{S_D^*}{S_D} \right) \frac{dS_D}{dt} \\ & + G_9 \left(1 - \frac{E_D^*}{E_D} \right) \frac{dE_D}{dt} + G_{10} \left(1 - \frac{I_D^*}{I_D} \right) \frac{dI_D}{dt} + G_{11} \left(1 - \frac{R_D^*}{R_D} \right) \frac{dR_D}{dt} + G_{12} \left(1 - \frac{M^*}{M} \right) \frac{dM}{dt}. \end{aligned} \quad (43)$$

Consider the endemic equilibrium point (EEP), $\mathbb{E}^*$ of equation (5) such that

$$\begin{aligned} \theta_1 = & (\tau_1 I_F^* + \tau_2 I_D^* + \tau_3 \lambda(M^*)) S_H^* + \mu_1 S_H^* - \beta_3 R_H^*, \quad \mu_1 + \beta_1 + \beta_2 = \frac{(\tau_1 I_F^* + \tau_2 I_D^* + \tau_3 \lambda(M^*)) S_H^*}{E_H^*}, \\ \sigma_1 + \mu_1 = & \frac{\beta_1 E_H^*}{I_H^*}, \quad \beta_3 + \mu_1 = \frac{\beta_2 E_H^*}{R_H^*}, \quad \theta_2 = (\kappa_1 I_F^* + \kappa_2 I_D^* + \kappa_3 \lambda(M^*)) S_F^* + \mu_2 S_F^*, \\ \mu_2 + \gamma = & \frac{(\kappa_1 I_F^* + \kappa_2 I_D^* + \tau_3 \lambda(M^*)) S_F^*}{E_F^*}, \quad \sigma_2 + \mu_2 = \frac{\gamma E_F^*}{I_F^*}, \\ \theta_3 = & \left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} \frac{\psi_3 \lambda(M^*)}{1 + \rho_3} \right) S_D^* + \mu_3 S_D^* - \gamma_3 R_D^*, \quad \mu_3 + \gamma_1 + \gamma_2 = \frac{\left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} \frac{\psi_3 \lambda(M^*)}{1 + \rho_3} \right) S_D^*}{E_D^*}, \\ \sigma_3 + \mu_3 = & \frac{\gamma_1 E_D^*}{I_D^*}, \quad \gamma_3 + \mu_3 = \frac{\gamma_2 E_D^*}{R_D^*}, \quad \mu_4 = \frac{(\nu_1 I_H^* + \nu_2 I_F^* + \nu_3 I_D^*)}{M^*}. \end{aligned} \quad (44)$$

Then, by substituting (5) into (43), we have

$$\left. \begin{aligned} \frac{d\mathcal{L}}{dt} = & G_1 \left(1 - \frac{S_H^*}{S_H} \right) (\theta_1 + \beta_3 R_H - \mu_1 S_H - \chi_1) + G_2 \left(1 - \frac{E_H^*}{E_H} \right) (\chi_1 - (\mu_1 + \beta_1 + \beta_2) E_H) \\ & + G_3 \left(1 - \frac{I_H^*}{I_H} \right) (\beta_1 E_H - (\sigma_1 + \mu_1) I_H) + G_4 \left(1 - \frac{R_H^*}{R_H} \right) (\beta_2 E_H - (\beta_3 + \mu_1) R_H) \\ & + G_5 \left(1 - \frac{S_F^*}{S_F} \right) (\theta_2 - \chi_2 - \mu_2 S_F) + G_6 \left(1 - \frac{E_F^*}{E_F} \right) (\chi_2 - (\mu_2 + \gamma) E_F) \\ & + G_7 \left(1 - \frac{I_F^*}{I_F} \right) (\gamma E_F - (\mu_2 + \sigma_2) I_F) + G_8 \left(1 - \frac{S_D^*}{S_D} \right) (\theta_3 - \mu_3 S_D - \chi_3 + \gamma_3 R_D) \\ & + G_9 \left(1 - \frac{E_D^*}{E_D} \right) (\chi_3 - (\mu_3 + \gamma_1 + \gamma_2) E_D) + G_{10} \left(1 - \frac{I_D^*}{I_D} \right) (\gamma_1 E_D - (\mu_3 + \delta_3) I_D) \\ & + G_{11} \left(1 - \frac{R_D^*}{R_D} \right) (\gamma_2 E_D - (\mu_3 + \gamma_3) R_D) + G_{12} \left(1 - \frac{M^*}{M} \right) ((v_1 I_H + v_2 I_F + v_3 I_D) - \mu_4 M). \end{aligned} \right\} \quad (45)$$

Using the endemic equilibrium point (EEP) described in equation (44), we simplify the equation (45) as

$$\left. \begin{aligned} \frac{d\mathcal{L}}{dt} = & G_1 \left(1 - \frac{S_H^*}{S_H} \right) \left((\tau_1 I_F^* + \tau_2 I_D^* + \tau_3 \lambda(M^*)) S_H^* + \mu_1 S_H^* - \beta_3 R_H^* - \mu_1 S_H \right. \\ & \left. - (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H + \beta_3 R_H \right) \\ & + G_2 \left(1 - \frac{E_H^*}{E_H} \right) \left((\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H - \frac{(\tau_1 I_F^* + \tau_2 I_D^* + \tau_3 \lambda(M^*)) S_H^* E_H}{E_H^*} \right) \\ & + G_3 \left(1 - \frac{I_H^*}{I_H} \right) \left(\beta_1 E_H - \frac{\beta_1 E_H^* I_H}{I_H^*} \right) + G_4 \left(1 - \frac{R_H^*}{R_H} \right) \left(\beta_2 E_H - \frac{\beta_2 E_H^* R_H}{R_H^*} \right) \\ & + G_5 \left(1 - \frac{S_F^*}{S_F} \right) \left((\kappa_1 I_F^* + \kappa_2 I_D^* + \kappa_3 \lambda(M^*)) S_F^* + \mu_2 S_F^* - \mu_2 S_F \right. \\ & \left. - (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F \right) \\ & + G_6 \left(1 - \frac{E_F^*}{E_F} \right) \left((\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F - \frac{(\tau_1 I_F^* + \kappa_2 I_D^* + \kappa_3 \lambda(M^*)) S_F^* E_F}{E_F^*} \right) \\ & + G_7 \left(1 - \frac{I_F^*}{I_F} \right) \left(\gamma E_F - \frac{\gamma E_F^* I_F}{I_F^*} \right) \\ & + G_8 \left(1 - \frac{S_D^*}{S_D} \right) \left(\left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} \frac{\psi_3 \lambda(M^*)}{1 + \rho_3} \right) S_D^* + \mu_3 S_D^* - \gamma_3 R_D^* \right. \\ & \left. - \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} \frac{\psi_3 \lambda(M)}{1 + \rho_3} \right) S_D - \mu_3 S_D + \gamma_3 R_D \right) \\ & + G_9 \left(1 - \frac{E_D^*}{E_D} \right) \left(\left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} \frac{\psi_3 \lambda(M)}{1 + \rho_3} \right) S_D - \frac{\left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} \frac{\psi_3 \lambda(M^*)}{1 + \rho_3} \right) S_D^* E_D}{E_D^*} \right) \\ & + G_{10} \left(1 - \frac{I_D^*}{I_D} \right) \left(\gamma_1 E_D - \frac{\gamma_1 E_D^* I_D}{I_D^*} \right) + G_{11} \left(1 - \frac{R_D^*}{R_D} \right) \left(\gamma_2 E_D - \frac{\gamma_2 E_D^* R_D}{R_D^*} \right) \\ & + G_{12} \left(1 - \frac{M^*}{M} \right) \left(v_1 I_H + v_2 I_F + v_3 I_D - \frac{(v_1 I_H^* + v_2 I_F^* + v_3 I_D^*) M}{M^*} \right). \end{aligned} \right\} \quad (46)$$

Then, equation (46) can be expressed as follows:

$$\begin{aligned}
\frac{d\mathcal{L}}{dt} = & -G_1\mu_1 S_H \left(1 - \frac{S_H^*}{S_H}\right)^2 + G_1\tau_1 S_H I_F \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{I_F^* S_H^*}{I_F S_H} - 1\right) + G_1\tau_2 S_H I_D \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{I_D^* S_H^*}{I_D S_H} - 1\right) \\
& + G_1\tau_3 S_H \lambda(M) \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{\lambda(M^*) S_H^*}{\lambda(M) S_H} - 1\right) + G_1\beta_3 R_H \left(1 - \frac{S_H^*}{S_H}\right) \left(1 - \frac{R_H^*}{R_H}\right) \\
& + G_2\tau_1 S_H I_F \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{I_F^* S_H^* E_H}{I_F S_H E_H^*}\right) + G_2\tau_2 S_H I_D \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{I_D^* S_H^* E_H}{I_D S_H E_H^*}\right) \\
& + G_2\tau_3 S_H \lambda(M) \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{\lambda(M^*) S_H^* E_H}{\lambda(M) S_H E_H^*}\right) \\
& + G_3\beta_1 E_H \left(1 - \frac{I_H^*}{I_H}\right) \left(1 - \frac{E_H^* I_H}{E_H I_H^*}\right) + G_4\beta_2 E_H \left(1 - \frac{R_H^*}{R_H}\right) \left(-\frac{E_H^* R_H}{E_H R_H^*}\right) \\
& - G_5\mu_2 S_F \left(1 - \frac{S_F^*}{S_F}\right)^2 + G_5\kappa_1 S_F I_F \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{I_F^* S_F^*}{I_F S_F} - 1\right) + G_5\kappa_2 S_F I_D \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{I_D^* S_F^*}{I_D S_F} - 1\right) \\
& + G_5\kappa_3 S_F \lambda(M) \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{\lambda(M^*) S_F^*}{\lambda(M) S_F} - 1\right) + G_6\kappa_1 S_F I_F \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{I_F^* S_F^* E_F}{I_F S_F E_F^*}\right) \\
& + G_6\kappa_2 S_F I_D \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{I_D^* S_F^* E_F}{I_D S_F E_F^*}\right) \\
& + G_6\kappa_3 S_F \lambda(M) \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{\lambda(M^*) S_F^* E_F}{\lambda(M) S_F E_F^*}\right) + G_7\gamma E_F \left(1 - \frac{I_F^*}{I_F}\right) \left(1 - \frac{E_F^* I_F}{E_F I_F^*}\right) \\
& - G_8\mu_3 S_D \left(1 - \frac{S_D^*}{S_D}\right)^2 + \frac{\psi_1 S_D I_F G_8}{(1 + \rho_1)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{I_F^* S_D^*}{I_F S_D} - 1\right) + \frac{\psi_2 S_D I_F G_8}{(1 + \rho_2)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{I_D^* S_D^*}{I_D S_D} - 1\right) \\
& + \frac{\psi_3 S_D \lambda(M) G_8}{(1 + \rho_3)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{\lambda(M^*) S_D^*}{\lambda(M) S_D} - 1\right) + G_8\gamma_3 R_D \left(1 - \frac{S_D^*}{S_D}\right) \left(1 - \frac{R_D^*}{R_D}\right) \\
& + \frac{\psi_1 S_D I_F G_9}{(1 + \rho_1)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{I_F^* S_D^* E_D}{I_F S_D E_D^*}\right) + \frac{\psi_2 S_D I_F G_9}{(1 + \rho_2)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{I_D^* S_D^* E_D}{I_D S_D E_D^*}\right) \\
& + \frac{\psi_3 S_D I_F G_9}{(1 + \rho_3)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{\lambda(M^*) S_D^* E_D}{\lambda(M) S_D E_D^*}\right) \\
& + G_{10}\gamma_1 E_D \left(1 - \frac{I_D^*}{I_D}\right) \left(1 - \frac{E_D^* I_D}{E_D I_D^*}\right) + G_{11}\gamma_2 E_D \left(1 - \frac{R_D^*}{R_D}\right) \left(-\frac{E_D^* R_D}{E_D R_D^*}\right) \\
& + G_{12}\nu_1 I_H \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_H^* M}{I_H M^*}\right) + G_{12}\nu_2 I_F \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_F^* M}{I_F M^*}\right) + G_{12}\nu_3 I_D \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_D^* M}{I_D M^*}\right).
\end{aligned} \tag{47}$$

Equation (47) can be written as

$$\frac{d\mathcal{L}}{dt} = Q + \mathcal{R},$$

where

$$\mathcal{R} = -G_1\mu_1 S_H \left(1 - \frac{S_H^*}{S_H}\right)^2 - G_5\mu_2 S_F \left(1 - \frac{S_F^*}{S_F}\right)^2 - G_8\mu_3 S_D \left(1 - \frac{S_D^*}{S_D}\right)^2$$

and

$$\begin{aligned}
Q = & G_1 \tau_1 S_H I_F \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{I_F^* S_H^*}{I_F S_H} - 1\right) + G_1 \tau_2 S_H I_D \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{I_D^* S_H^*}{I_D S_H} - 1\right) \\
& + G_1 \tau_3 S_H \lambda(M) \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{\lambda(M^*) S_H^*}{\lambda(M) S_H} - 1\right) + G_1 \beta_3 R_H \left(1 - \frac{S_H^*}{S_H}\right) \left(1 - \frac{R_H^*}{R_H}\right) \\
& + G_2 \tau_1 S_H I_F \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{I_F^* S_H^* E_H^*}{I_F S_H E_H^*}\right) + G_2 \tau_2 S_H I_D \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{I_D^* S_H^* E_H^*}{I_D S_H E_H^*}\right) \\
& + G_2 \tau_3 S_H \lambda(M) \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{\lambda(M^*) S_H^* E_H^*}{\lambda(M) S_H E_H^*}\right) \\
& + G_3 \beta_1 E_H \left(1 - \frac{I_H^*}{I_H}\right) \left(1 - \frac{E_H^* I_H^*}{E_H I_H^*}\right) + G_4 \beta_2 E_H \left(1 - \frac{R_H^*}{R_H}\right) \left(-\frac{E_H^* R_H^*}{E_H R_H^*}\right) \\
& + G_5 \kappa_1 S_F I_F \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{I_F^* S_F^*}{I_F S_F} - 1\right) + G_5 \kappa_2 S_F I_D \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{I_D^* S_F^*}{I_D S_F} - 1\right) \\
& + G_5 \kappa_3 S_F \lambda(M) \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{\lambda(M^*) S_F^*}{\lambda(M) S_F} - 1\right) + G_6 \kappa_1 S_F I_F \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{I_F^* S_F^* E_F^*}{I_F S_F E_F^*}\right) \\
& + G_6 \kappa_2 S_F I_D \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{I_D^* S_F^* E_F^*}{I_D S_F E_F^*}\right) \\
& + G_6 \kappa_3 S_F \lambda(M) \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{\lambda(M^*) S_F^* E_F^*}{\lambda(M) S_F E_F^*}\right) + G_7 \gamma E_F \left(1 - \frac{I_F^*}{I_F}\right) \left(1 - \frac{E_F^* I_F^*}{E_F I_F^*}\right) \\
& + \frac{\psi_1 S_D I_F G_8}{(1 + \rho_1)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{I_F^* S_D^*}{I_F S_D} - 1\right) + \frac{\psi_2 S_D I_F G_8}{(1 + \rho_2)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{I_D^* S_D^*}{I_D S_D} - 1\right) \\
& + \frac{\psi_3 S_D \lambda(M) G_8}{(1 + \rho_3)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{\lambda(M^*) S_D^*}{\lambda(M) S_D} - 1\right) + G_8 \gamma_3 R_D \left(1 - \frac{S_D^*}{S_D}\right) \left(1 - \frac{R_D^*}{R_D}\right) \\
& + \frac{\psi_1 S_D I_F G_9}{(1 + \rho_1)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{I_F^* S_D^* E_D^*}{I_F S_D E_D^*}\right) + \frac{\psi_2 S_D I_F G_9}{(1 + \rho_2)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{I_D^* S_D^* E_D^*}{I_D S_D E_D^*}\right) \\
& + \frac{\psi_3 S_D I_F G_9}{(1 + \rho_3)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{\lambda(M^*) S_D^* E_D^*}{\lambda(M) S_D E_D^*}\right) \\
& + G_{10} \gamma_1 E_D \left(1 - \frac{I_D^*}{I_D}\right) \left(1 - \frac{E_D^* I_D^*}{E_D I_D^*}\right) + G_{11} \gamma_2 E_D \left(1 - \frac{R_D^*}{R_D}\right) \left(-\frac{E_D^* R_D^*}{E_D R_D^*}\right) \\
& + G_{12} \nu_1 I_H \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_H^* M^*}{I_H M^*}\right) + G_{12} \nu_2 I_F \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_F^* M^*}{I_F M^*}\right) + G_{12} \nu_3 I_D \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_D^* M^*}{I_D M^*}\right).
\end{aligned} \tag{48}$$

To simplify (48), let

$$\begin{aligned}
a = \frac{S_H}{S_H^*}, b = \frac{E_H}{E_H^*}, c = \frac{I_H}{I_H^*}, d = \frac{R_H}{R_H^*}, e = \frac{S_F}{S_F^*}, f = \frac{E_F}{E_F^*}, g = \frac{I_F}{I_F^*}, \\
h = \frac{S_D}{S_D^*}, r = \frac{E_D}{E_D^*}, n = \frac{I_D}{I_D^*}, m = \frac{\lambda(M)}{\lambda(M^*)}, l = \frac{R_D}{R_D^*}, \text{ and } k = \frac{M}{M^*}.
\end{aligned}$$

One gets from (48) that

$$\begin{aligned}
Q = & \tau_1 S_H I_F \left(1 - \frac{1}{a}\right) \left(\frac{1}{ab} - 1\right) + \tau_2 S_H I_D \left(1 - \frac{1}{a}\right) \left(\frac{1}{an} - 1\right) + \tau_3 S_H \lambda(M) \left(1 - \frac{1}{a}\right) \left(\frac{1}{am} - 1\right) \\
& + \beta_3 R_H \left(1 - \frac{1}{a}\right) \left(1 - \frac{1}{d}\right) + \tau_1 S_H I_F \left(1 - \frac{1}{b}\right) \left(1 - \frac{1}{bf}\right) + \tau_2 S_H I_D \left(1 - \frac{1}{b}\right) \left(1 - \frac{1}{bn}\right) \\
& + \tau_3 S_H \lambda(M) \left(1 - \frac{1}{b}\right) \left(1 - \frac{1}{bm}\right) + \beta_1 E_H \left(1 - \frac{1}{c}\right) \left(1 - \frac{1}{c}\right) + \beta_2 E_H \left(1 - \frac{1}{d}\right) \left(1 - \frac{1}{d}\right) \\
& + \kappa_1 S_F I_F \left(1 - \frac{1}{e}\right) \left(\frac{1}{ef} - 1\right) + \kappa_2 S_F I_D \left(1 - \frac{1}{e}\right) \left(\frac{1}{ne} - 1\right) + \kappa_3 S_F \lambda(M) \left(1 - \frac{1}{e}\right) \left(\frac{1}{me} - 1\right) \\
& + \kappa_1 S_F I_F \left(1 - \frac{1}{f}\right) \left(1 - \frac{1}{fn}\right) + \kappa_2 S_F I_D \left(1 - \frac{1}{f}\right) \left(1 - \frac{1}{fn}\right) + \kappa_3 S_F \lambda(M) \left(1 - \frac{1}{f}\right) \left(1 - \frac{1}{fn}\right) \\
& + \gamma E_F \left(1 - \frac{1}{f}\right) \left(1 - \frac{1}{fg}\right) + \frac{\psi_1 S_D I_F}{(1 + \rho_1)} \left(1 - \frac{1}{h}\right) \left(\frac{1}{hg} - 1\right) + \frac{\psi_2 S_D I_F}{(1 + \rho_2)} \left(1 - \frac{1}{h}\right) \left(\frac{1}{hn} - 1\right) \\
& + \frac{\psi_3 S_D \lambda(M)}{(1 + \rho_3)} \left(1 - \frac{1}{h}\right) \left(\frac{1}{mh} - 1\right) + \gamma_3 R_D \left(1 - \frac{1}{h}\right) \left(1 - \frac{1}{l}\right) + \frac{\psi_1 S_D I_F}{(1 + \rho_1)} \left(1 - \frac{1}{r}\right) \left(1 - \frac{1}{rg}\right) \\
& + \frac{\psi_2 S_D I_F}{(1 + \rho_2)} \left(1 - \frac{1}{r}\right) \left(1 - \frac{1}{rn}\right) + \frac{\psi_3 S_D I_F}{(1 + \rho_3)} \left(1 - \frac{1}{r}\right) \left(1 - \frac{1}{rm}\right) + \gamma_1 E_D \left(1 - \frac{1}{n}\right) \left(1 - \frac{1}{n}\right) \\
& + \gamma_2 E_D \left(1 - \frac{1}{l}\right) \left(1 - \frac{1}{l}\right) + \nu_1 I_H \left(1 - \frac{1}{k}\right) \left(1 - \frac{1}{k}\right) + \nu_2 I_F \left(1 - \frac{1}{k}\right) \left(1 - \frac{1}{k}\right) + \nu_3 I_D \left(1 - \frac{1}{k}\right) \left(1 - \frac{1}{k}\right).
\end{aligned} \tag{49}$$

We express the equation (49) as

$$\left. \begin{aligned} Q = & \tau_1 S_H I_F \left(1 - \frac{b}{ac} + \frac{1}{ac} - \frac{1}{b}\right) + \tau_2 S_H I_D \left(\frac{1}{an} - 1 + \frac{1}{a^2 n} + \frac{1}{a}\right) + \tau_3 S_H \lambda(M) \left(\frac{1}{am} - 1 + \frac{1}{a^2 m} + \frac{1}{a}\right) \\ & + \beta_3 R_H \left(1 - \frac{1}{d} - \frac{1}{a} + \frac{1}{ad}\right) + \tau_1 S_H I_F \left(1 - \frac{b}{af} - \frac{1}{b} + \frac{1}{af}\right) + \tau_2 S_H I_D \left(1 - \frac{b}{an} - \frac{1}{b} + \frac{1}{an}\right) \\ & + \tau_3 S_H \lambda(M) \left(1 - \frac{b}{af} - \frac{1}{b} + \frac{1}{af}\right) + \tau_2 S_H I_D \left(1 - \frac{b}{am} - \frac{1}{b} + \frac{1}{am}\right) + \beta_1 E_H \left(1 - \frac{b}{c} - \frac{1}{c} + \frac{b}{c^2}\right) \\ & + \beta_2 E_H \left(1 - \frac{b}{d} - \frac{1}{d} + \frac{b}{d^2}\right) + \kappa_1 S_F I_F \left(\frac{1}{ef} - 1 - \frac{1}{e^2 f} + \frac{1}{e}\right) + \kappa_2 S_F I_D \left(\frac{1}{en} - 1 - \frac{1}{e^2 n} + \frac{1}{e}\right) \\ & + \kappa_3 S_F \lambda(M) \left(\frac{1}{em} - 1 - \frac{1}{e^2 m} + \frac{1}{e}\right) + \kappa_1 S_F I_F \left(1 - \frac{f}{en} - \frac{1}{f} + \frac{1}{en}\right) + \kappa_2 S_F I_D \left(1 - \frac{f}{en} - \frac{1}{f} + \frac{1}{en}\right) \\ & + \kappa_3 S_F \lambda(M) \left(1 - \frac{f}{me} - \frac{1}{f} + \frac{1}{me}\right) + \gamma E_F \left(1 - \frac{g}{f} - \frac{1}{f} + \frac{g}{f^2}\right) + \frac{\psi_1 S_D I_F}{(1 + \rho_1)} \left(1 - \frac{1}{h} - \frac{1}{h^2 g} + \frac{1}{h}\right) \\ & + \frac{\psi_2 S_D I_F}{(1 + \rho_2)} \left(1 - \frac{1}{h} - \frac{1}{h^2 g} + \frac{1}{h}\right) + \frac{\psi_3 S_D \lambda(M)}{(1 + \rho_3)} \left(\frac{1}{mh} - 1 - \frac{1}{h^2 m} + \frac{1}{h}\right) + \gamma_3 R_D \left(1 - \frac{1}{l} - \frac{1}{h} + \frac{1}{hl}\right) \\ & + \frac{\psi_1 S_D I_F}{(1 + \rho_1)} \left(1 - \frac{r}{hg} - \frac{1}{r} + \frac{1}{hg}\right) + \frac{\psi_2 S_D I_F}{(1 + \rho_2)} \left(1 - \frac{r}{hn} - \frac{1}{r} + \frac{1}{hn}\right) + \frac{\psi_3 S_D I_F}{(1 + \rho_3)} \left(1 - \frac{r}{hm} - \frac{1}{r} + \frac{1}{hm}\right) \\ & + \gamma_1 E_D \left(1 - \frac{l}{r} - \frac{1}{l} + \frac{1}{r}\right) + \gamma_2 E_D \left(1 - \frac{r}{hg} - \frac{1}{r} + \frac{1}{hg}\right) + v_1 I_H \left(1 - \frac{k}{c} - \frac{1}{k} + \frac{1}{c}\right) + v_2 I_F \left(1 - \frac{k}{g} - \frac{1}{k} + \frac{1}{g}\right) \\ & + v_3 I_D \left(1 - \frac{k}{n} - \frac{1}{k} + \frac{1}{n}\right). \end{aligned} \right\} \quad (50)$$

Now we make use of the following basic inequality.

Proposition 6. If $\epsilon(y) = 1 - y + \ln y$, then $\epsilon(y) \leq 0$ such that $1 - y \leq -\ln y$ if and only if $y > 0$.

From equation (50), we have

$$1 - \frac{1}{d} - \frac{1}{a} + \frac{1}{ad} = \left(1 - \frac{1}{d}\right) + \left(1 - \frac{1}{a}\right) - \left(1 - \frac{1}{ad}\right). \quad (51)$$

Using Proposition 6 and the concept of geometric mean, equation (51) can be written as

$$\begin{aligned} \left(1 - \frac{1}{d}\right) + \left(1 - \frac{1}{a}\right) - \left(1 - \frac{1}{ad}\right) & \leq -\ln\left(\frac{1}{d}\right) - \ln\left(\frac{1}{a}\right) + \ln\left(\frac{1}{ad}\right) \\ & \leq \ln\left(a \times d \times \frac{1}{ad}\right) = \ln(1) = 0. \end{aligned} \quad (52)$$

Following similar procedures in (52), we get

$$1 - \frac{c}{b} - \frac{1}{c} + \frac{1}{b} \leq 0, \quad 1 - \frac{p}{m} - \frac{1}{p} + \frac{1}{m} \leq 0, \quad 1 - \frac{d}{b} - \frac{1}{d} + \frac{1}{b} \leq 0.$$

From equation (47), the global stability holds only if $\frac{d\mathcal{L}}{dt} \leq 0$. Now, if $\mathcal{R} < \mathcal{Q}$, then $\frac{d\mathcal{L}}{dt}$ will be negative definite, which implies that $\frac{d\mathcal{L}}{dt} < 0$ and $\frac{d\mathcal{L}}{dt} = 0$ only at the endemic equilibrium point $\mathbb{E}^*$. Hence, by LaSalle's invariance principle [28], the only invariant set in $\{(S_H(t), E_H(t), I_H(t), R_H(t), S_F(t), E_F(t), I_F(t), S_D(t), E_D(t), I_D(t), R_H(t)) \in \mathbb{R}_+^{12}\} : \{(S_H(t), E_H(t), I_H(t), R_H(t), S_F(t), E_F(t), I_F(t), S_D(t), E_D(t), I_D(t), R_H(t)) \rightarrow \mathbb{E}^*\}$ is the singleton endemic point $\mathbb{E}^*$. Thus, any solution to the rabies model (5) which intersect the interior $\mathbb{R}_+^{12}$ limits to $\mathbb{E}^*$ is globally asymptotically stable whatever $\mathcal{R}_0 > 1$. $\square$

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Parameters estimation and uncertainty assessment in the transmission dynamics of rabies in humans and dogs

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ABSTRACT

Rabies remains a pressing global public health issue, demanding effective modeling and control strategies. This study focused on developing a mathematical model using ordinary differential equations (ODEs) to estimate parameters and assess uncertainties related to the transmission dynamics of rabies in humans and dogs. To determine model parameters and address uncertainties, next-generation matrices were utilized to calculate the basic reproduction number R_0 . Furthermore, the Partial Rank Correlation Coefficient was used to identify parameters that significantly influence model outputs. The analysis of equilibrium states revealed that the rabies-free equilibrium is globally asymptotically stable when $R_0 < 1$, whereas the endemic equilibrium is globally asymptotically stable when $R_0 \geq 1$. To reduce the severity of rabies and align with the Global Rabies Control (GRC) initiative by 2030, the study recommends implementing control strategies targeting indoor domestic dogs.

1. Introduction

Rabies is a fatal and neglected viral disease that affects mammals, including humans. The disease is primarily spread through bites and scratches by infected animals [1–5]. The disease is caused by rabies virus, a member of the family Rhabdoviridae and the genus Lyssavirus. The disease typically presents with symptoms such as inflammation, fever, headache, and malaise. These initial symptoms can progress to more specific manifestations including anxiety, confusion, hallucinations, dysphagia, muscle spasms, and paralysis, therefore, immediate medical attention is essential in the event of a bite or scratch from animals such as dogs and cats [6–9]. Prevention of rabies primarily involves vaccinating humans and domestic dogs, and avoiding contact with wild animals that are carriers of rabies virus [2,10]. In many countries, vaccination programs have been successful in reducing the incidence of rabies in humans and domestic dogs [11,12]. Post-exposure prophylaxis (PEP) is a vaccine recommended for individuals who have been bitten or scratched by an animal who are carrier of rabies virus. PEP comprises a series of injections with both rabies

vaccine and rabies immune globulin to prevent the virus from causing an infection [3].

Mathematical models are fundamental tools for comprehending and addressing the transmission dynamics of rabies. They aid in describing and predicting epidemiological phenomena, allocating resources, and structuring control strategies. Researchers in different countries have developed several mathematical models to study the transmission dynamics of rabies in dog populations [13–18], as well as interactions between dog and human populations [19–21], and even among dog, human, and other animal populations [22–24]. These studies have identified several factors that can shape the dynamics of rabies in different countries and ways of controlling the disease. However, due to the complex nature of disease transmission dynamics and the inherent variability in real-world data, parameter estimation poses significant challenges. Estimating these parameters accurately is essential for designing effective control measures and interventions to prevent the spread of rabies. One common approach to parameter estimation involves using the least square method and validating the model by fitting

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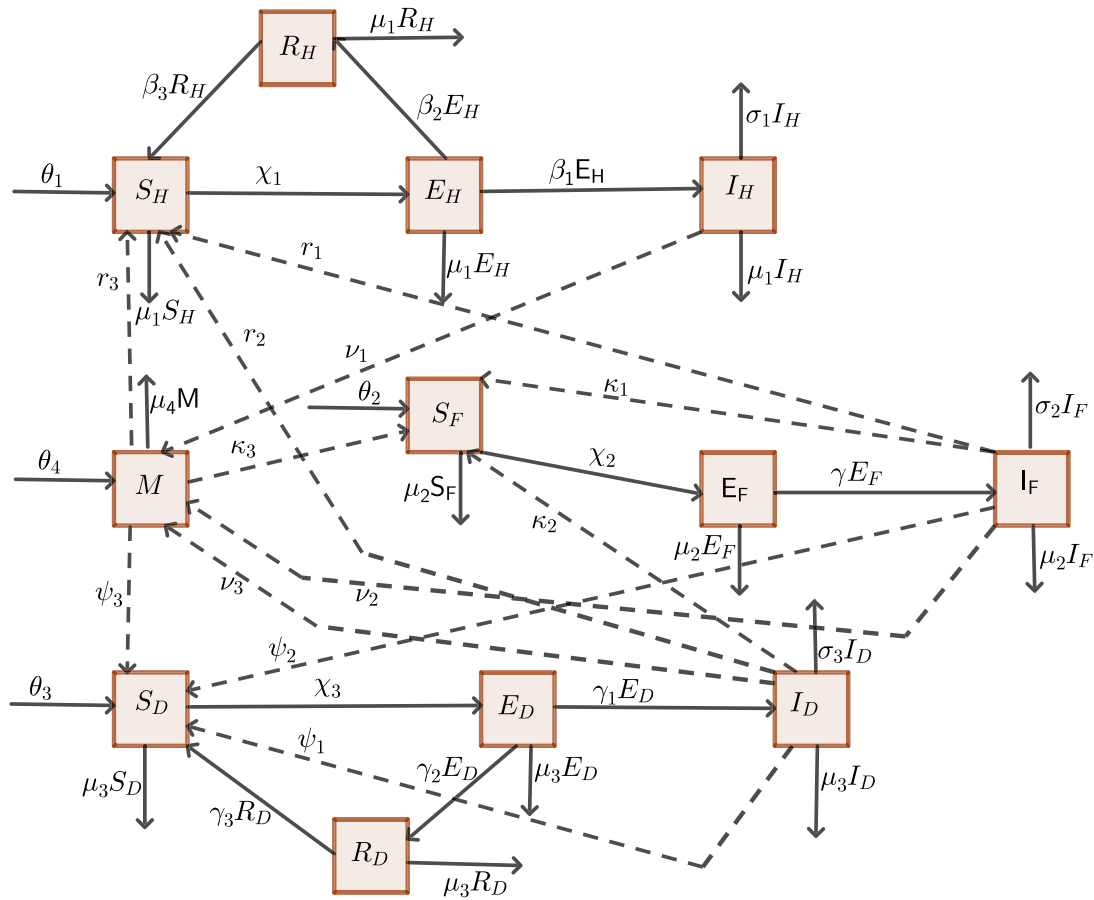


Fig. 1. Schematic diagram for the flow of transmission dynamics of rabies.

real-world data to the proposed model. It is crucial to assess uncertainty in parameter estimates to understand the reliability of model predictions and make informed decisions about disease control strategies. Uncertainty can arise from various sources, including variability in data, model structure, and parameter estimation techniques.

To address this issue, statistical techniques such as Latin Hypercube Sampling (LHS) and Partial Rank Correlation Coefficient (PRCC) are utilized for uncertainty and sensitivity analysis of input parameters [25]. LHS generates random samples within the parameter space, while PRCC assesses the correlation between model output and each input parameter [25,26]. LHS enables efficient sampling of multi-dimensional parameter space, while PRCC computes and ranks the correlation coefficient between the model inputs (parameters) and the model outputs (state variables). It gauges the degree of a non-linear and monotonic relationship between model inputs (parameters) and model outputs. Therefore, this paper discusses the transmission dynamics of rabies, the parameter estimation, and the role of statistical techniques such as LHS and PRCC in evaluating the impact of uncertainties on model predictions.

The structure of this paper is organized with sections dedicated to formulating the mathematical model (Section 2), qualitative analysis (Section 3), quantitative analysis (Section 4), and discussion and concluding the findings (Sections 5 and 6, respectively).

2. Model formulation

The model for rabies transmission is divided into four distinctive components. The first component pertains to the human population,

which is represented by $N_H = S_H + E_H + R_H + I_H$. The second component is the environmental virus reservoir, represented by M . The third component is comprised of free-range dogs, represented by $N_F = S_F + E_F + I_F$, which includes both stray and feral dogs. The fourth component represents domestic dogs, represented by $N_D = S_D + E_D + R_D + I_D$. Susceptible humans (S_H) are individuals who can be infected. They are recruited into the population at a rate of θ_1 . Exposure happens upon contact with infected free-range dogs (I_F), domestic dogs (I_D), or the virus (M) at rates of τ_1 , τ_2 , or τ_3 , respectively. The latency period for exposed individuals is 1–3 months. Thus the force of infection for human is given by

$$\chi_1 = (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H, \quad (1)$$

where

$$\lambda(M) = \frac{M}{M + C}.$$

Exposed individuals who receive post-exposure prophylaxis recover at a rate of β_2 . Vaccinated individuals lose their protective immunity and become susceptible at a rate of β_3 . In some cases, exposed individuals vaccinated with less efficacious vaccine may progress to infectious state I_H at the transition rate of β_1 . Infected individuals may suffer to induced deaths at a rate of σ_1 . Susceptible free-range dogs S_F become infected upon contact with infected individuals I_F , I_D , or the rabies virus in the environment at rates of κ_1 , κ_2 , or κ_3 , respectively. The force of infection of free range dogs is given by

$$\chi_2 = (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F. \quad (2)$$

Upon exposure to rabies, susceptible free-range dogs transition to the latent state E_F , where they remain for 1 to 3 months. Subsequently, they advance to the infectious state I_F at a rate γ . Infected free-range dogs face mortality at a rate σ_2 , while all free-range dogs experience natural death at a rate of μ_2 .

Susceptible domestic dogs (S_D) are continually recruited at a rate θ_3 and become infected through contact with either infected individuals (I_F , I_D , M) or the virus in the environment at rates ψ_1 , ψ_2 , or ψ_3 , respectively, and the force of infection is defined as

$$\chi_3 = \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right) S_D. \quad (3)$$

Following exposure to rabies, susceptible domestic dogs transition to the latent state E_D , where they remain for a period of time at a rate β_1 . Those in E_D who receive post-exposure prophylaxis move to the recovered state R_D at rate γ_2 . However, as post-exposure prophylaxis does not provide permanent immunity, individuals in R_D can lose immunity and become susceptible again at rate γ_3 . The remaining portion of E_D progresses to the infectious state I_D at rate γ_1 . Infected domestic dogs face mortality due to the disease at rate σ_3 . Additionally, all domestic dogs experience natural death at a rate of μ_3 . The rabies virus in the environment is introduced through shedding from infectious free-range dogs, domestic dogs, and humans at rates v_2 , v_3 , and v_1 , respectively, at the general rate defined as

$$\theta_4 = (v_1 I_H + v_2 I_F + v_3 I_D) M. \quad (4)$$

The viruses are eliminated from the environment at a rate of μ_4 .

Using the details provided about the model parameters and their associations with the state variables, we develop a model represented as a set of ordinary differential equations, as shown in Eq. (5),

$$\begin{cases} \dot{S}_H = \theta_1 + \beta_3 R_H - \chi_1 - \mu_1 S_D, \\ \dot{E}_H = \chi_2 - (\mu_1 + \beta_1 + \beta_2) E_H, \\ \dot{I}_H = \beta_1 E_H - (\sigma_1 + \mu_1) I_H, \\ \dot{R}_H = \beta_2 E_H - (\beta_3 + \mu_1) R_H, \\ \dot{S}_F = \theta_2 - \chi_2 - \mu_2 S_F, \\ \dot{E}_F = \chi_2 - (\mu_2 + \gamma) E_F, \\ \dot{I}_F = \gamma E_F - (\mu_2 + \sigma_2) I_F, \\ \dot{S}_D = \theta_3 - \chi_3 - \mu_3 S_D + \gamma_3 R_D, \\ \dot{E}_D = \chi_3 - (\mu_3 + \gamma_1 + \gamma_2) E_D, \\ \dot{I}_D = \gamma_1 E_D - (\mu_3 + \sigma_3) I_D, \\ \dot{R}_D = \gamma_2 E_D - (\mu_3 + \gamma_3) R_D, \\ \dot{M} = (v_1 I_H + v_2 I_F + v_3 I_D) - \mu_4 M, \end{cases} \quad (5)$$

subject to the following non-negative conditions:

$$\begin{aligned} S_H(0) > 0, E_H(0) \geq 0, I_H(0) \geq 0, R_H(0) \geq 0, S_F(0) > 0, E_F(0) \geq 0, \\ I_F(0) \geq 0, \\ S_D(0) \geq 0, E_D(0) \geq 0, I_D(0) \geq 0, R_D(0) \geq 0. \end{aligned} \quad (6)$$

The schematic diagram corresponding to (5) is given in Fig. 1.

3. Qualitative analysis

In this section we prove existence of a positive solution, its boundedness, we compute the Rabies free equilibrium and the basic reproduction number.

3.1. Positivity of the solution

For the model system (5) to be epidemiologically meaningfully and well-posed, we need to prove that the state variables are non-negative $\forall t \geq 0$.

Lemma 1. All solution of the system in the region (5) that start in $\Omega \subset \mathbb{R}_+^{12}$ remain positive all the time.

Proof. To prove for the existence of model solution (5), we consider initial conditions and apply the integral operator $\int_0^t (\cdot) ds$ to all equations in the model Eq. (5), to obtain:

$$\begin{cases} S_H(t) - S_H(0) = \int_0^t (\theta_1 + \beta_3 R_H - \mu_1 S_H - \chi_1) ds, \\ E_H(t) - E_H(0) = \int_0^t (\chi_1 - (\mu_1 + \beta_1 + \beta_2) E_H) ds, \\ I_H(t) - I_H(0) = \int_0^t (\beta_1 E_H - (\sigma_1 + \mu_1) I_H) ds, \\ R_H(t) - R_H(0) = \int_0^t (\beta_2 E_H - (\beta_3 + \mu_1) R_H) ds, \\ S_F(t) - S_F(0) = \int_0^t (\theta_2 - \chi_2 - \mu_2 S_F) ds, \\ E_F(t) - E_F(0) = \int_0^t (\chi_2 - (\mu_2 + \gamma) E_F) ds, \\ I_F(t) - I_F(0) = \int_0^t (\gamma E_F - (\mu_2 + \sigma_2) I_F) ds, \\ S_D(t) - S_D(0) = \int_0^t (\theta_3 - \mu_3 S_D - \chi_3 + \gamma_3 R_D) ds, \\ E_D(t) - E_D(0) = \int_0^t (\chi_3 - (\mu_3 + \gamma_1 + \gamma_2) E_D) ds, \\ I_D(t) - I_D(0) = \int_0^t (\gamma_1 E_D - (\mu_3 + \sigma_3) I_D) ds, \\ R_D(t) - R_D(0) = \int_0^t (\gamma_2 E_D - (\mu_3 + \gamma_3) R_D) ds, \\ M(t) - M(0) = \int_0^t ((v_1 I_H + v_2 I_F + v_3 I_D) - \mu_4 M) ds. \end{cases} \quad (7)$$

For convenience, from Eq. (7) we define the following functions:

$$\begin{cases} f_1(t, S_H) = \theta_1 + \beta_3 R_H - \mu_1 S_H - \chi_1, \\ f_2(t, E_H) = \chi_1 - (\mu_1 + \beta_1 + \beta_2 + \mu_4) E_H, \\ f_3(t, E_H) = \beta_1 E_H - (\sigma_1 + \mu_1) I_H, \\ f_4(t, R_H) = (\beta_2 + \mu_4) E_H - (\beta_3 + \mu_1) R_H, \\ f_5(t, S_F) = \theta_2 - \chi_2 - \mu_2 S_F, \\ f_6(t, E_F) = \chi_2 - (\mu_2 + \gamma) E_F, \\ f_7(t, I_F) = \gamma E_F - (\mu_2 + \sigma_2) I_F, \\ f_8(t, S_D) = \theta_3 - \mu_3 S_D - \chi_3 + \gamma_3 R_D, \\ f_9(t, E_D) = \chi_3 - (\mu_3 + \gamma_1 + \gamma_2) E_D, \\ f_{10}(t, I_D) = \gamma_1 E_D - (\mu_3 + \sigma_3) I_D, \\ f_{11}(t, R_D) = \gamma_2 E_D - (\mu_3 + \gamma_3) R_D, \\ f_{12}(t, M) = (v_1 I_H + v_2 I_F + v_3 I_D) - \mu_4 M. \end{cases} \quad (8)$$

Since $S_H, E_H, I_H, R_H, S_F, E_F, I_F, S_D, E_D, I_D, R_D, M$ are positive and bounded in the region $\Omega \subset \mathbb{R}_+^{12}$, there exist non-negative values $\mathbb{L}_i, i = 1, 2, \dots, 12$, such that

$$\begin{aligned} \|f_1(t, S_H(t)) - f_1(t, S_H(t_1))\| &\leq \|(\theta_1 + \beta_3 R_H(t) - \mu_1 S_H(t) - \chi_1) - \\ &\quad - (\theta_1 + \beta_3 R_H(t_1) - \mu_1 S_H(t_1) - \chi_1)\| \\ &\leq \mu_1 \|S_H(t_1) - S_H(t)\| + \chi_1 \|S_H(t_1) - S_H(t)\| \\ &\leq (\mu_1 + \chi_1) \|S_H(t_1) - S_H(t)\| \\ &\leq \epsilon_1 \|S_H(t_1) - S_H(t)\|. \end{aligned} \quad (9)$$

Using similar approach as used in Eq. (9) we obtain for functions f_i , $i = 2, 3, \dots, 12$, that

$$\left\{ \begin{array}{l} \|f_2(t, E_H(t)) - f_2(t, E_H(t_1))\| \leq \epsilon_2 \|E_H(t_1) - E_H(t)\|, \\ \|f_3(t, I_H(t)) - f_3(t, I_H(t_1))\| \leq \epsilon_3 \|I_H(t_1) - I_H(t)\|, \\ \|f_4(t, R_H(t)) - f_4(t, R_H(t_1))\| \leq \epsilon_4 \|R_H(t_1) - R_H(t)\|, \\ \|f_5(t, S_F(t)) - f_5(t, S_F(t_1))\| \leq \epsilon_5 \|S_F(t_1) - S_F(t)\|, \\ \|f_6(t, E_F(t)) - f_6(t, E_F(t_1))\| \leq \epsilon_6 \|E_F(t_1) - E_F(t)\|, \\ \|f_7(t, I_F(t)) - f_7(t, I_F(t_1))\| \leq \epsilon_7 \|I_F(t_1) - I_F(t)\|, \\ \|f_8(t, S_D(t)) - f_8(t, S_D(t_1))\| \leq \epsilon_8 \|S_D(t_1) - S_D(t)\|, \\ \|f_9(t, E_D(t)) - f_9(t, E_D(t_1))\| \leq \epsilon_9 \|E_D(t_1) - E_D(t)\|, \\ \|f_{10}(t, I_D(t)) - f_{10}(t, I_D(t_1))\| \leq \epsilon_{10} \|I_D(t_1) - I_D(t)\|, \\ \|f_{11}(t, S_D(t)) - f_{11}(t, R_D(t_1))\| \leq \epsilon_{11} \|R_D(t_1) - R_D(t)\|, \\ \|f_{12}(t, M(t)) - f_{12}(t, M(t_1))\| \leq \epsilon_{12} \|M(t_1) - M(t)\|, \end{array} \right. \quad (10)$$

where $\epsilon_1 = \mu_1 + \chi_1$, $\epsilon_2 = \mu_1 + \beta_1 + \beta_2 + u_4$, $\epsilon_3 = \sigma_1 + \mu_1$, $\epsilon_4 = \beta_3 + \mu_1$, $\epsilon_5 = \mu_2 + \chi_2$, $\epsilon_6 = \mu_2 + \gamma$, $\epsilon_7 = \mu_2 + \sigma_1$, $\epsilon_8 = \mu_3 + \chi_3$, $\epsilon_9 = \mu_3 + \gamma_1 + \gamma_2 + u_4$, $\epsilon_{10} = \mu_3 + \sigma_3$, $\epsilon_{11} = \mu_3 + \gamma_3$, $\epsilon_{12} = \mu_4$. Since the Lipschitz condition is satisfied, then a solution to our model (5) exists. $\square$

3.2. Boundedness of the model solution

Theorem 2. The solution of the rabies model system (5) is uniformly bounded if $\Omega \in \mathbb{R}_+^{12}$ and $\Omega = \Omega_H \cup \Omega_D \cup \Omega_F \cup \Omega_M \in \mathbb{R}_+^4 \times \mathbb{R}_+^3 \times \mathbb{R}_+^4 \times \mathbb{R}_+^1$, where

$$\begin{aligned} \Omega_H &= \left\{ (S_H, E_H, I_H, R_H) \in \mathbb{R}_+^4 : 0 \leq N_H \leq \frac{\theta_1}{\mu_1} \right\}, \\ \Omega_F &= \left\{ (S_F, E_F, I_F) \in \mathbb{R}_+^3 : 0 \leq N_F \leq \frac{\theta_2}{\mu_2} \right\}, \\ \Omega_D &= \left\{ (S_D, E_D, I_D, R_D) \in \mathbb{R}_+^4 : 0 \leq N_D \leq \frac{\theta_3}{\mu_3} \right\}, \\ \Omega_M &= \max \left\{ \frac{\theta_1 v_1}{\mu_1 \mu_4} + \frac{\theta_2 v_2}{\mu_2 \mu_4} + \frac{\theta_3 v_3}{\mu_3 \mu_4}, M(0) \right\}, \end{aligned}$$

and Ω is the positive invariant region.

Proof. Since the model (5) monitors both human and dog populations, we assume that the model's state variables and parameters are non-negative for all $t \geq 0$. By utilizing Theorem 2, we derive the invariant region of the rabies model as follows. Consider the population of the human from Eq. (5) as

$$\frac{dN_H}{dt} = \frac{dS_H}{dt} + \frac{dE_H}{dt} + \frac{dI_H}{dt} + \frac{dR_H}{dt}. \quad (11)$$

Then, Eq. (11) implies to Eq. (12) as follows:

$$\frac{dN_H}{dt} = \theta_1 - (S_H + E_H + I_H + R_H) \mu_1 - \sigma_1 I_H. \quad (12)$$

But if $N_H = S_H + E_H + I_H + R_H$, then Eq. (12) can be written as

$$\frac{dN_H}{dt} = \theta_1 - N_H \mu_1, \quad (13)$$

and by utilizing the integrating factor in Eq. (13) and $t \rightarrow 0$, we have

$$N_H(0) \leq \frac{\theta_1}{\mu_1} + Ce^0 \implies N_H(0) - \frac{\theta_1}{\mu_1} \leq C. \quad (14)$$

By simplifying Eq. (14) and after simple manipulations, it follows that

$$\Omega_H = \left\{ (S_H, E_H, I_H, R_H) \in \mathbb{R}_+^4 : 0 \leq N_H \leq \frac{\theta_1}{\mu_1} \right\}.$$

Employing the same procedures for free range and domestic dog populations, we obtain:

$$\begin{aligned} \Omega_F &= \left\{ (S_F, E_F, I_F) \in \mathbb{R}_+^3 : 0 \leq N_F \leq \frac{\theta_2}{\mu_2} \right\}, \\ \Omega_D &= \left\{ (S_D, E_D, I_D, R_D) \in \mathbb{R}_+^4 : 0 \leq N_D \leq \frac{\theta_3}{\mu_3} \right\}. \end{aligned} \quad (15)$$

Again, from the environment that contains rabies virus,

$$\dot{M} = (v_1 I_H + v_2 I_F + v_3 I_D) - \mu_4 M. \quad (16)$$

Since $N_H \leq \frac{\theta_1}{\mu_1}$, $N_F \leq \frac{\theta_2}{\mu_2}$, and $N_D \leq \frac{\theta_3}{\mu_3}$, it follows that $I_H \leq \frac{\theta_1}{\mu_1}$, $I_F \leq \frac{\theta_2}{\mu_2}$, and $I_D \leq \frac{\theta_3}{\mu_3}$. Therefore, Eq. (16) can be expressed as

$$\dot{M} \leq \left(\frac{v_1 \theta_1}{\mu_1} + \frac{v_2 \theta_2}{\mu_2} + \frac{v_3 \theta_3}{\mu_3} \right) - \mu_4 M. \quad (17)$$

Now, let Y be the solution which is unique to the initial value problem such that

$$\left\{ \begin{array}{l} \dot{Y} \leq \left(\frac{v_1 \theta_1}{\mu_1} + \frac{v_2 \theta_2}{\mu_2} + \frac{v_3 \theta_3}{\mu_3} \right) - \mu_4 M, \text{ for } t > 0 \\ Y(0) = M(0). \end{array} \right. \quad (18)$$

By using integration factor Eq. (18) as $t \rightarrow \infty$, we get the expression

$$\left(M(0) - \left(\frac{v_1 \theta_1}{\mu_1} + \frac{v_2 \theta_2}{\mu_2} + \frac{v_3 \theta_3}{\mu_3} \right) \frac{1}{\mu_4} e^{\mu_4 t} \right) \quad (19)$$

and as Eq. (19) goes to zero, we have

$$M(t) \leq \Omega_M. \quad (20)$$

Thus, the model system (5) is biologically and mathematically meaningful such that their solution relies in the region Ω . $\square$

3.3. Rabies free equilibrium ($\mathbb{E}_0$) and the basic reproduction number $\mathcal{R}_0$

In order to achieve a rabies-free equilibrium ($\mathbb{E}_0$) in both humans, free-range, and domestic dog populations, we equate all infectious compartments in Eq. (5) to zero, which leads to

$$\mathbb{E}_0 = \left(\frac{\theta_1}{\mu_1}, 0, 0, 0, \frac{\theta_2}{\mu_2}, 0, 0, \frac{\theta_3}{\mu_3}, 0, 0, 0, 0 \right).$$

In order to determine the basic reproduction $\mathcal{R}_0$, the next generation matrix method, as applied by [27–29], is adopted as follows:

$$\frac{dx_i}{dt} = F_i(x) - (\mathcal{V}_i^+(x) - \mathcal{V}_i^-(x)), \quad (21)$$

where F_i is the matrix new infections in the compartment i while $\mathcal{V}_i^+$ and $\mathcal{V}_i^-$ are matrices of the transfer terms in and out of the compartment i , respectively. From Eq. (21), we define F_i and $\mathcal{V}_i$ by

$$F_i = \begin{pmatrix} (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H \\ 0 \\ (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F \\ 0 \\ \left(\frac{\psi_1 I_F}{1+\rho_1} + \frac{\psi_2 I_D}{1+\rho_2} + \frac{\psi_3 \lambda(M)}{1+\rho_3} \right) S_D \\ 0 \\ 0 \end{pmatrix}, \quad (22)$$

$$\mathcal{V}_i = \begin{pmatrix} (\mu_1 + \beta_1 + \beta_2) E_H \\ (\sigma_1 + \mu_1) I_H - \beta_1 E_H \\ (\mu_2 + \gamma) E_F \\ (\mu_2 + \sigma_2) I_F - \gamma E_F \\ (\mu_3 + \gamma_1 + \gamma_2) E_D \\ (\mu_3 + \delta_3) I_D - \gamma_1 E_D \\ \mu_4 M - (v_1 I_H + v_2 I_F + v_3 I_D) \end{pmatrix}.$$

The Jacobian matrices F and V at the disease free equilibrium point $\mathbb{E}_0$ are given by the expression of matrix FV^{-1} which can be presented

as

$$FV^{-1} = \begin{pmatrix} 0 & 0 & R_{13} & R_{14} & R_{15} & R_{16} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & R_{33} & R_{34} & R_{35} & R_{36} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & R_{53} & R_{54} & R_{55} & R_{56} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \quad (23)$$

where

$$\left. \begin{aligned} R_{13} &= \frac{\tau_1 \theta_1 \gamma}{\mu_1 (\mu_2 + \gamma) (\mu_2 + \sigma_2)}, & R_{14} &= \frac{\tau_1 \theta_1}{\mu_1 (\mu_2 + \sigma_2)}, \\ R_{15} &= \frac{\tau_2 \theta_1 \gamma}{\mu_1 (\mu_3 + \gamma_1 + \gamma_2) (\mu_3 + \sigma_3)}, \\ R_{33} &= \frac{\kappa_1 \theta_2 \gamma}{\mu_2 (\mu_2 + \gamma) (\mu_2 + \sigma_2)}, & R_{34} &= \frac{\kappa_1 \theta_2}{\mu_2 (\mu_2 + \sigma_2)}, \\ R_{35} &= \frac{\kappa_1 \theta_2 \gamma}{\mu_2 (\mu_3 + \gamma_1 + \gamma_2) (\mu_3 + \sigma_3)}, \\ R_{53} &= \frac{\psi_1 \theta_3 \gamma}{(1 + \rho_1) (\mu_2 + \gamma) (\mu_2 + \sigma_2) \mu_3}, & R_{54} &= \frac{\psi_1 \theta_3}{(1 + \rho_1) \mu_3 (\mu_2 + \sigma_2)}, \\ R_{16} &= \frac{\tau_2 \theta_1}{\mu_1 (\mu_3 + \sigma_3)}, \\ R_{55} &= \frac{\psi_2 \theta_3 \gamma}{(1 + \rho_2) (\mu_3 + \gamma_1 + \gamma_2) (\mu_3 + \sigma_3) \mu_3}, \\ R_{56} &= \frac{\psi_2 \theta_3}{(1 + \rho_2) \mu_3 (\mu_3 + \sigma_3)}, & R_{36} &= \frac{\kappa_1 \theta_2}{\mu_2 (\mu_3 + \sigma_3)}. \end{aligned} \right\} \quad (24)$$

From Eq. (23), we obtain the eigenvalues as

$$\left(0, 0, 0, 0, \frac{1}{2} R_{55} + \frac{1}{2} R_{33} + \frac{1}{2} \sqrt{R_{33}^2 - 2R_{33}R_{55} + 4R_{35}R_{53} + R_{55}^2}, \right. \\ \left. \frac{1}{2} R_{55} + \frac{1}{2} R_{33} - \frac{1}{2} \sqrt{R_{33}^2 - 2R_{33}R_{55} + 4R_{35}R_{53} + R_{55}^2} \right). \quad (25)$$

This non-negative eigenvalue corresponds to a non-negative eigenvector that represents the distribution of infected individuals who generate the highest number of secondary infections per generation, also known as R_0 . According to [30], the basic reproduction number R_0 is the largest eigenvalue of the next generating matrix given by

$$R_0 = \rho(FV^{-1}). \quad (26)$$

Therefore, the spectral radius of the next generation matrix is given by

$$R_0 = \rho(FV^{-1}) = \frac{(R_{55} + R_{33}) + \sqrt{R_{33}^2 - 2R_{33}R_{55} + 4R_{35}R_{53} + R_{55}^2}}{2}. \quad (27)$$

3.3.1. Global stability of the rabies free equilibrium

The model behavior at the disease free equilibrium point, $\mathbb{E}_0$, is investigated using a Metzler matrix as applied by Castillo-Chavez et al. [31]. Let X_s denote non-transmitting class, X_m be transmitting class and X_{DFE} be the Disease Free equilibrium, whereby

$$\left. \begin{aligned} \frac{dX_s}{dt} &= B(X_s - X_{DFE}) + B_1 X_m \\ \frac{dX_m}{dt} &= B_2 X_m \end{aligned} \right\}. \quad (28)$$

Then, from the model system (5), the equations in Box 1 can be deduced.

The non-negative out-diagonal entries of matrix B_2 indicate that it is the Metzler matrix. To prove the stability of matrix B_2 using the Metzler matrix block form, we need to demonstrate that it satisfies the conditions for a Metzler matrix and has eigenvalues with negative real parts. We adopt the idea of a stable Metzler matrix and apply the following Lemma 3.

Lemma 3 (See [32]). Let N be a square Metzler matrix written in block form

$$N = \begin{bmatrix} A & B \\ C & D \end{bmatrix}, \quad (29)$$

where A , B and D are rectangular matrices while C is a square matrix. Then N is Metzler stable if, and only if, matrices A and $D - CA^{-1}B$ are Metzler stable.

In our case one has

$$A = \begin{bmatrix} -\mu_1 - \beta_1 - \beta_2 & 0 & 0 \\ \beta_1 & -\sigma_1 - \mu_1 & 0 \\ 0 & 0 & -\mu_2 - \gamma \end{bmatrix},$$

$$B = \begin{bmatrix} \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ 0 & 0 & 0 & 0 \\ \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 \\ 0 & 0 & 0 & 0 \\ \frac{\psi_1 \theta_3}{\mu_3(1+\rho_1)} & 0 & \frac{\psi_2 \theta_3}{\mu_3(1+\rho_1)} & 0 \\ 0 & 0 & 0 & 0 \\ v_1 & 0 & v_2 & 0 \end{bmatrix},$$

$$C = \begin{bmatrix} 0 & 0 & 0 \\ \gamma & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix},$$

and

$$D = \begin{bmatrix} -\mu_2 - \sigma_2 & 0 & 0 \\ 0 & -\mu_3 - \gamma_1 - \gamma_2 & 0 \\ 0 & \gamma & -\mu_3 - \sigma_3 \end{bmatrix}.$$

To show that matrices A and $D - CA^{-1}B$ are Metzler stable, we need to demonstrate that all their off-diagonal elements are non-negative. Since the off-diagonal elements of A are β_1 and 0, both of which are non-negative, we conclude that matrix A is Metzler stable.

Next, matrix $D - CA^{-1}B$ can be calculated as follows:

$$CA^{-1}B = \begin{bmatrix} \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} \\ 0 & 0 & 0 \\ 0 & \gamma & -\mu_2 - \sigma_2 \end{bmatrix}.$$

Subtracting $CA^{-1}B$ from D gives

$$D - CA^{-1}B = \begin{bmatrix} 0 & 0 & 0 \\ 0 & -\mu_1 - \sigma_1 & 0 \\ 0 & 0 & -\mu_2 - \gamma - \sigma_2 \end{bmatrix}.$$

Since the eigenvalues of the Matrix B are $\lambda_1 = -\mu_3$, $\lambda_2 = -\mu_2$, $\lambda_3 = -\mu_1$, $\lambda_4 = -(\mu_3 + \gamma_3)$, and $\lambda_5 = -(\beta_3 + \mu_1)$ and the off-diagonal elements of $D - CA^{-1}B$ are all 0, which are non-negative, thus matrix $D - CA^{-1}B$ is also Metzler stable. Since both matrices A and $D - CA^{-1}B$ have non-negative off-diagonal elements, they are Metzler stable. Thus, B_2 is also Metzler stable. Therefore, the rabies free equilibrium point of the model system (5) is globally asymptotically stable if $R_0 < 1$ and unstable otherwise.

3.3.2. Rabies Persistence Equilibrium Point (RPEP)

The endemic equilibrium point is the steady state where rabies is present in humans, free-range dogs, and domestic dogs. To find this point, we set the equations of the model system (5) to zero and solve the resulting system simultaneously. The state variables for each compartment are represented by

$$RPEP(S_H^*, E_H^*, I_H^*, R_H^*, S_F^*, E_F^*, I_F^*, S_D^*, E_D^*, I_D^*, R_D^*, M^*)$$

$$\begin{aligned}
X_s &= (S_H, R_H, S_F, S_D, R_D)^T, \\
X_m &= (E_H, I_H, E_D, I_D, M)^T, \\
X_s - X_{DFE} &= \begin{bmatrix} S_H - \frac{\theta_1}{\mu_1} \\ R_H \\ S_F - \frac{\theta_2}{\mu_2} \\ S_D - \frac{\theta_3}{\mu_3} \\ R_D \end{bmatrix}, \\
B &= \begin{bmatrix} -\mu & \beta_3 & 0 & 0 & 0 \\ 0 & -(\beta_3 + \mu_1) & 0 & 0 & 0 \\ 0 & 0 & -\mu_2 & 0 & 0 \\ 0 & 0 & 0 & -\mu_3 & \gamma_3 \\ 0 & 0 & 0 & 0 & -(\mu_3 + \gamma_3) \end{bmatrix}, \\
B_1 &= \begin{bmatrix} 0 & 0 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ \beta_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} & 0 \\ 0 & 0 & 0 & \frac{\psi_1 \theta_3}{\mu_3 (1 + \rho_1)} & 0 & \frac{\psi_2 \theta_3}{\mu_3 (1 + \rho_2)} & 0 \\ 0 & 0 & 0 & 0 & \gamma_2 & 0 & 0 \end{bmatrix}, \text{ and} \\
B_2 &= \begin{bmatrix} -\mu_1 - \beta_1 - \beta_2 & 0 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ \beta_1 & -\sigma_1 - \mu_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\mu_2 - \gamma & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} & 0 \\ 0 & 0 & \gamma & -\mu_2 - \sigma_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\psi_1 \theta_3}{\mu_3 (1 + \rho_1)} & -\mu_3 - \gamma_1 - \gamma_2 & \frac{\psi_2 \theta_3}{\mu_3 (1 + \rho_1)} & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\mu_3 - \sigma_3 & 0 \\ 0 & \nu_1 & 0 & \nu_2 & 0 & \nu_3 & -\mu_4 \end{bmatrix}.
\end{aligned}$$

Box I.

such that

$$\left. \begin{aligned}
R_H^* &= \frac{\beta_2 (\sigma_1 + \mu_1) I_H^*}{\beta_1 (\beta_3 + \mu_1)}, \\
I_H^* &= \frac{\beta_1 (\beta_3 + \mu_3) (\sigma_1 + \mu_1)^2 (\beta_1 + \beta_2 + \beta_3) \mu_1 + \beta_1 \beta_3 (\sigma_1 + \mu_1)^2}{(\sigma_1 + \mu_1)^2 ((\beta_1 + \beta_2 + \beta_3) \mu_1 + \beta_1 \beta_3)} \\
&\quad - \frac{\beta_1 (\beta_3 + \mu_3) (\sigma_1 + \mu_1)^2 \beta_3 - \theta_1 (\beta_3 + \mu_3) (\sigma_1 + \mu_1)^2}{(\sigma_1 + \mu_1)^2 ((\beta_1 + \beta_2 + \beta_3) \mu_1 + \beta_1 \beta_3)}, \\
E_H^* &= \frac{(\sigma_1 + \mu_1) I_H^*}{\beta_1}, \\
S_H^* &= \frac{\beta_3 \beta_2 (\sigma_1 + \mu_1) I_H^*}{\beta_1 (\beta_3 + \mu_1) \mu_1} - \frac{(\mu_1 + \beta_1 + \beta_2) (\sigma_1 + \mu_1) I_H^*}{\beta_1 \mu_1} + \frac{\theta_1}{\mu_1}
\end{aligned} \right\}.$$

Re-writing Eq. (25) as

$$(R_0 - 1) (R_0 - R_{33} - R_{55} + 1) + (1 - R_{33}) (1 - R_{55}) - R_{35} R_{53} = 0, \quad (30)$$

where R_{33} , R_{55} , R_{35} , and R_{53} are defined in Eq. (24), we get

$$\left. \begin{aligned}
I_D^* &= \frac{\gamma_1 \psi_1 I_F^* (1 + \rho_2) (1 + \rho_3) M^* + \gamma_1 \psi_3 M^* (1 + \rho_1) (1 + \rho_2)}{(\mu_3 + \gamma_1 + \gamma_2)^2 - \gamma_1 \psi_2 (1 + \rho_1) (1 + \rho_3) M^* (\mu_3 + \gamma_1 + \gamma_2)}, \\
E_D^* &= \frac{(\mu_3 + \sigma_3) I_D^*}{\gamma_1}, \quad R_D^* = \frac{\gamma_2 (\mu_3 + \sigma_3) I_D^*}{\gamma_1 (\mu_3 + \gamma_3)}, \\
S_D^* &= \frac{\gamma_3 (\mu_3 + \sigma_3) I_D^*}{\mu_3 \gamma_1} - \frac{(\mu_3 + \gamma_1 + \gamma_2) \gamma_2 (\mu_3 + \sigma_3) I_D^*}{\gamma_1 (\mu_3 + \gamma_3) \mu_3} + \frac{\theta_3}{\mu_3}, \\
E_F^* &= \frac{(\mu_2 + \sigma_2) I_F^*}{\gamma}, \\
S_F^* &= \frac{\theta_2}{\mu_2} - \frac{(\mu_2 + \gamma) (\mu_2 + \sigma_2) I_F^*}{\gamma \mu}, \quad M^* = \frac{\nu_3 I_D^* + \nu_2 I_F^* + \nu_1 I_H^*}{\mu_4},
\end{aligned} \right\}$$

where θ_2 and θ_3 are given in Box II.

The endemic equilibrium point of the rabies disease persists when $I_H, I_F, I_D, M > 0$ and $R_0 \geq 1$, as summarized in Theorem 4.

Theorem 4. The system (5) has a unique endemic equilibrium RPEP if $R_0 \geq 1$ and $I_H, I_F, I_D, M > 0$.

$$\theta_2 = \frac{(\mu_2 + \gamma)\mu_2(1 + (R_0 - 1))(1 + \rho_1)\mu_3(\mu_2 + \sigma_2)}{[\mu_3(1 + \rho_2)(1 + \rho_1)(\mu_3 + \sigma_3)(\mu_3 + \gamma_1 + \gamma_2)(1 + (R_0 - 1)) - \theta_3\gamma_1(\psi_2(1 + \rho_1)\mu_3 + \psi_1(1 + \rho_2))] \gamma\kappa_1} \\ \times [(1 + \rho_2)(\mu_3 + \sigma_3)(\mu_3 + \gamma_1 + \gamma_2)(1 + (R_0 - 1)) - \theta_3\psi_2\gamma_1], \\ \theta_3 = \frac{[-\mu_2(\mu_2 + \sigma_2)(\mu_2 + \gamma)(1 + (R_0 - 1)) + \gamma\kappa_1\theta_2](1 + (R_0 - 1))(1 + \rho_1)\mu_3(1 + \rho_2)(\mu_3 + \sigma_3)(\mu_3 + \gamma_1 + \gamma_2)}{[(-\mu_2(\mu_2 + \sigma_2)(\mu_2 + \gamma)(1 + (R_0 - 1)) + \gamma\kappa_1\theta_2)(1 + \rho_1)\psi_2\mu_3 + \gamma\kappa_1\theta_2\psi_1(1 + \rho_2)] \gamma_1}.$$

Box II.

4. Quantitative analysis

In this study, we create artificial data sets that represents the dynamics of infectious diseases and estimate model parameters using the least squares technique. Baseline parameter values θ_i of the rabies model from the literature are used to numerically solve a non-linear deterministic model (5) in the Matlab environment, generating synthetic datasets at each time t_i . Initial conditions for the number of susceptible, exposed infected, and recovered humans, free range and Domestic dogs are also provided.

4.1. Parameter estimation and model fitting

In order to generate the rabies model-predicted number of infected individuals $RD(t_i; \theta_i)$ dataset at time t_i for a given parameter vector θ_i , we added random Gaussian noise $\eta_i(t_i, \theta_i)$ measurements to the data simulate real-world dynamics, where measurement errors are common. Thus, the observed dependent data were given as

$$I(t_i; \theta_i) = RD(t_i, \theta_i) + \eta_i(t_i, \theta_i) \text{ for each time } t_i \in [1, n], \quad (31)$$

where n is the number of data points. The objective function quantifies the difference between the model's predictions $I(t_i; \theta_i)$ and the estimated data $I_{\text{estimated},i}$. We define the objective function $J(\theta_i)$ as the sum of squared differences between model predictions and estimated dataset as

$$J(\theta_i) = \sum_{i=1}^{33} a_i (I(t_i; \theta_i) - I_{\text{estimated},i})^2, \quad (32)$$

where $I_{\text{estimated},i}$ is the estimated number of infected rabies individuals at time t_i . The term $(I(t_i; \theta_i) - I_{\text{estimated},i})^2$ represents the squared difference between the model-predicted value $I(t_i; \theta_i)$ and the estimated value $I_{\text{estimated},i}$ at time t_i ; a_i typically represents the weighting or importance assigned to each term in the summation. The goal is to find the parameter vector $\hat{\theta}_i$ that minimizes the objective function $J(\theta_i)$ given by

$$\hat{\theta}_i = \arg \min J(\theta_i). \quad (33)$$

The expression indicates that $\hat{\theta}_i$ is the parameter vector that optimally fits the model to the estimated data by minimizing the objective function $J(\theta_i)$. The symbol $\arg \min$ is used to find the argument (in this case, θ_i) that minimizes the function $J(\theta_i)$. To find the parameter value θ_i , we apply partial derivatives on Eq. (5) with respect to each of the 33 parameters represented by θ_i such that

$$\frac{\partial J(\theta_i)}{\partial \theta_i} = \sum_{i=1}^{33} 2 (I(t_i; \theta_i) - I_{\text{estimated},i}) \frac{\partial (I(t_i; \theta_i))}{\partial \theta_i} = 0 \text{ for } i = 1, 2, \dots, 33, \quad (34)$$

where (see Table 1)

$$\theta_i = \rho_1, \rho_2, \rho_3, \theta_1, \tau_1, \tau_2, \tau_3, \beta_1, \beta_2, \beta_3, \mu_1, \sigma_1, \theta_2, \gamma, \kappa_1, \kappa_2, \kappa_3, \sigma_2, \mu_2, \theta_3, \psi_1, \psi_2, \psi_3, \sigma_3, \gamma_1, \gamma_2, \gamma_3, \mu_3, v_1, v_2, v_3, \mu_4, C.$$

4.2. Parameter sensitivity analysis

For the modeling uncertainty and parameter estimation of rabies transmission global sensitivity analysis (GSA), we employed LHS-PRCC to analyze uncertainty, specifically in the context of a model with 33 parameters (referred to as model (5)). The aim is to identify the key factors influencing the transmission of a new infection under a specific intervention, following the approach outlined in Ref. [36]. This approach involved using LHS to sample the parameters that contribute to the variable R_0 and calculating Partial Rank Correlation Coefficients (PRCC) for these parameters, as defined in Eq. (35). We conducted a total of 1000 simulations for each LHS run. Our parameter sampling was based on a uniform distribution, treating the model parameters as input variables, and R_0 as the output variable. Larger absolute PRCC values indicate a more substantial influence of a parameter on R_0 . If the p -value exceeds 0.05, it is assumed that a parameter is not statistically significant for R_0 . Positive values denote positive correlations, negative values signify negative correlations, and values within the range of -0.2 to $+0.2$ represent weak or negligible correlations. PRCC values greater than $+0.6$ or less than -0.6 indicate strong positive or strong negative correlations, while values within the range of $0.2 < \text{PRCC} < 0.6$ or $-0.6 < \text{PRCC} < -0.2$ represent moderate positive or moderate negative correlations, respectively.

The PRCC between parameter y_i and output x_j , controlling for the effects of other variables Z , is then computed as

$$\text{PRCC}(y_i, x_j | Z) = \frac{\text{cov}(X, Y)}{\sigma_X \sigma_Y} = \frac{\text{Corr}(y_i, x_j | Z)}{\sqrt{\text{Corr}(y_i, y_i | Z) \times \text{Corr}(x_j, x_j | Z)}}, \quad (35)$$

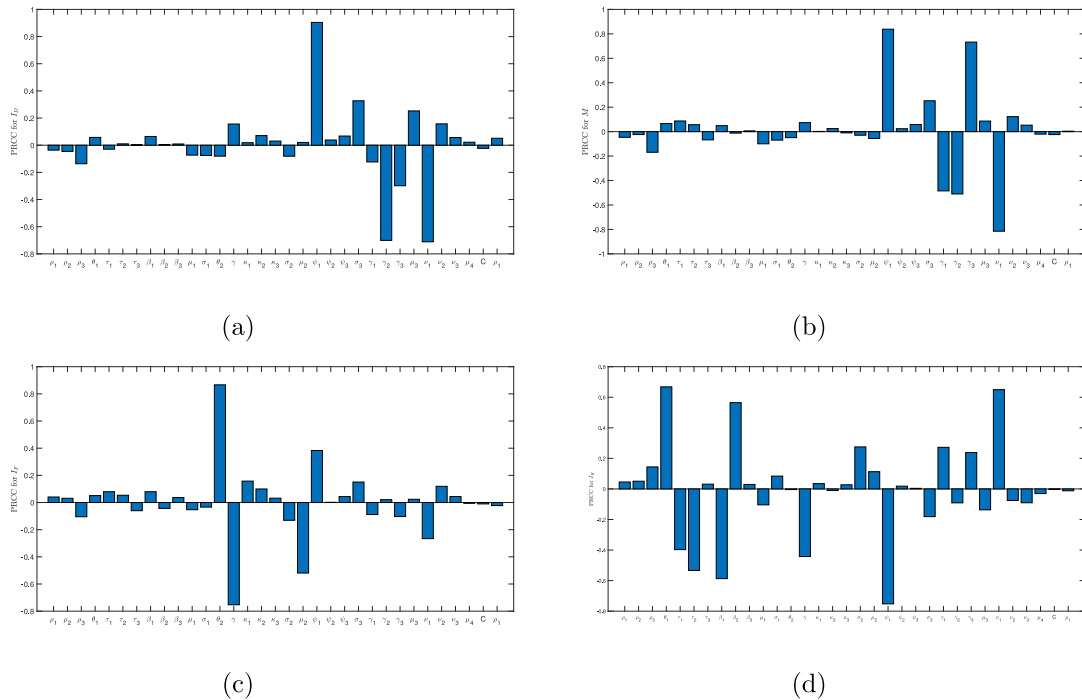
where X is the matrix of model outputs (e.g., disease incidence or prevalence), Y is the matrix of model parameters (e.g., transmission rates, recovery rates), Z is the matrix of other relevant variables, $\text{cov}(X, Y)$ is the covariance between the model output (X) and the input parameter (Y), and σ_X and σ_Y are the standard deviations of X and Y , respectively.

Once PRCC and LHS values are computed for each parameter, they were plotted against the model output to visualize their impact. This visualization helps identify which parameters have the most significant influence on the model output and how changes in those parameters affect the model behavior. Additionally, these techniques aid in obtaining solutions by guiding parameter estimation processes and improving model calibration and validation.

The PRCC results provide insights into the impact of each parameter on the model and its associated uncertainty. Parameters with PRCC values close to zero, or equal to zero, are considered statistically insignificant. Fig. 2 indicates that $\psi_1, \psi_2, \psi_3, \tau_1, \tau_2, \tau_3, \kappa_1, \kappa_2$, and κ_3 have positive correlation with the transmission dynamics of rabies based on the model solution computed in Eq. (35), while $\rho_1, \rho_2, \rho_3, \beta_1, \beta_2, \beta_3, \mu_1, \sigma_1, \sigma_2, \mu_2, \theta_3, \psi_1, \psi_2, \psi_3, \sigma_3, \gamma_1, \gamma_2, \gamma_3, \mu_3$, and C have negative correlation with the transmission dynamics of rabies. Fig. 3 illustrates the sensitivity of PRCC values across the entire time interval of the model simulation. It assesses significance and demonstrates how the sensitivity of each parameter influences the dynamics of the model system. This suggests that, to control rabies in humans and dogs, more efforts should be directed to reduce the rate of infections by intervening the transmission and control of the contact rate.

Table 1Estimated model parameters (Year⁻¹), initial guess for parameters (Year⁻¹) and their respective source.

Parameters	Baseline value	Source	Estimated value	Mean (μ) and std (σ)
θ_1	2000	(Estimated)	1993.382113	$\mathcal{N}(1996.691056 \ 4.4679553)$.
τ_1	0.0004	[33]	0.000405	$\mathcal{N}(0.000402 \ 4 \times 10^{-6})$.
τ_2	0.0004	[33]	0.000604	$\mathcal{N}(0.000502 \ 1.44 \times 10^{-4})$.
τ_3	[0.0003 0.0100]	(Estimated)	0.000303	$\mathcal{N}(0.000302 \ 2 \times 10^{-6})$.
β_1	$\frac{1}{6}$	[33,34]	0.165581	$\mathcal{N}(0.166124 \ 7.68 \times 10^{-4})$.
β_2	[0.54 1]	[13,34]	0.540487	$\mathcal{N}(0.5402435 \ 3.7815 \times 10^{-4})$.
β_3	1	(Estimated)	0.999301	$\mathcal{N}(0.9996505 \ 1.6521 \times 10^{-4})$.
μ_1	0.0142	[35,36]	0.014417	$\mathcal{N}(0.014309 \ 1.53 \times 10^{-4})$.
σ_1	1	[13,34]	1.006332	$\mathcal{N}(1.03166 \ 4.47 \times 10^{-3})$.
θ_2	1000	(Estimated)	1004.12044	$\mathcal{N}(1002.060222 \ 2.913594)$.
κ_1	0.00006	(Estimated)	0.000020	$\mathcal{N}(0.000040 \ 2.8 \times 10^{-5})$.
κ_2	0.00005	(Estimated)	0.000081	$\mathcal{N}(0.000066 \ 2.2 \times 10^{-5})$.
κ_3	[0.00001 0.00003]	(Estimated)	0.000040	$\mathcal{N}(0.000025 \ 2.1 \times 10^{-5})$.
γ	$\frac{1}{6}$	[13,33,34]	0.166374	$\mathcal{N}(0.166520 \ 2.07 \times 10^{-4})$.
σ_2	0.09	[34,37]	0.089556	$\mathcal{N}(0.089778 \ 3.14 \times 10^{-4})$.
μ_2	0.067	(Estimated)	0.066268	$\mathcal{N}(0.066634 \ 1.58 \times 10^{-4})$.
θ_3	1200	(Estimated)	1203.844461	$\mathcal{N}(1201.922230 \ 2.718444)$.
ψ_1	0.0004	[37,38]	0.000077	$\mathcal{N}(0.000238 \ 2.28 \times 10^{-4})$.
ψ_2	0.0004	[39]	0.000066	$\mathcal{N}(0.000233 \ 2.36 \times 10^{-4})$.
ψ_3	0.0003	(Estimated)	0.000030	$\mathcal{N}(0.0003 \ 1.91 \times 10^{-4})$.
μ_3	0.067	(Estimated)	0.080129	$\mathcal{N}(0.073565 \ 8.056 \times 10^{-3})$.
σ_3	0.08	[34]	0.091393	$\mathcal{N}(0.085697 \ 8.056 \times 10^{-3})$.
γ_1	$\frac{1}{6}$	[33,34]	0.172489	$\mathcal{N}(0.169578 \ 4.117 \times 10^{-3})$.
γ_2	0.09	[34]	0.090308	$\mathcal{N}(0.090154 \ 2.18 \times 10^{-4})$.
γ_3	0.05	(Estimated)	0.050128	$\mathcal{N}(0.050128 \ 9.1 \times 10^{-5})$.
v_1	0.001	(Estimated)	0.001958	$\mathcal{N}(0.001479 \ 6.77 \times 10^{-4})$.
v_2	0.006	(Estimated)	0.008971	$\mathcal{N}(0.007485 \ 2.101 \times 10^{-3})$.
v_3	0.001	(Estimated)	0.005735	$\mathcal{N}(0.003367 \ 3.3348 \times 10^{-3})$.
μ_4	0.08	(Estimated)	0.080625	$\mathcal{N}(0.080313 \ 4.42 \times 10^{-4})$.
ρ_1	10	[40]	9.920733	$\mathcal{N}(9.960366 \ 5.605 \times 10^{-2})$.
ρ_2	8	(Estimated)	8.116421	$\mathcal{N}(8.058211 \ 8.2322 \times 10^{-2})$.
ρ_3	15	(Estimated)	14.917005	$\mathcal{N}(14.958502 \ 5.8686 \times 10^{-2})$.
C	0.003 (PFU)/mL	(Estimated)	0.003011	$\mathcal{N}(0.003005 \ 8.0000 \times 10^{-6})$.

**Fig. 2.** The sensitivity analysis of Rabies model dynamics involved 1000 simulations employing Latin Hypercube Sampling (LHS). The analysis evaluated Partial Rank Correlation Coefficients (PRCCs) concerning (a) domestic dogs, (b) Environment, (c) Free-range dogs, and (d) Human population, respectively.

4.3. Numerical simulation

Due to the resource-intensive nature of individually solving for 33 parameters, we employed the MATLAB built-in function `fminsearch`, which leverages the Nelder–Mead simplex algorithm [41] to identify

local minima for the residual sum of squares as presented in Eq. (34). Our choice of initial parameter values was guided by the criteria established during the qualitative analysis along with the initial conditions $S_H(0) = 142000$, $E_H(0) = 40$, $I_H(0) = 0$, $R_H(0) = 0$, $S_D(0) = 15000$, $E_D(0) = 25$, $I_D(0) = 0$, $R_D(0) = 0$, $S_F(0) = 12500$, $E_F(0) = 20$,

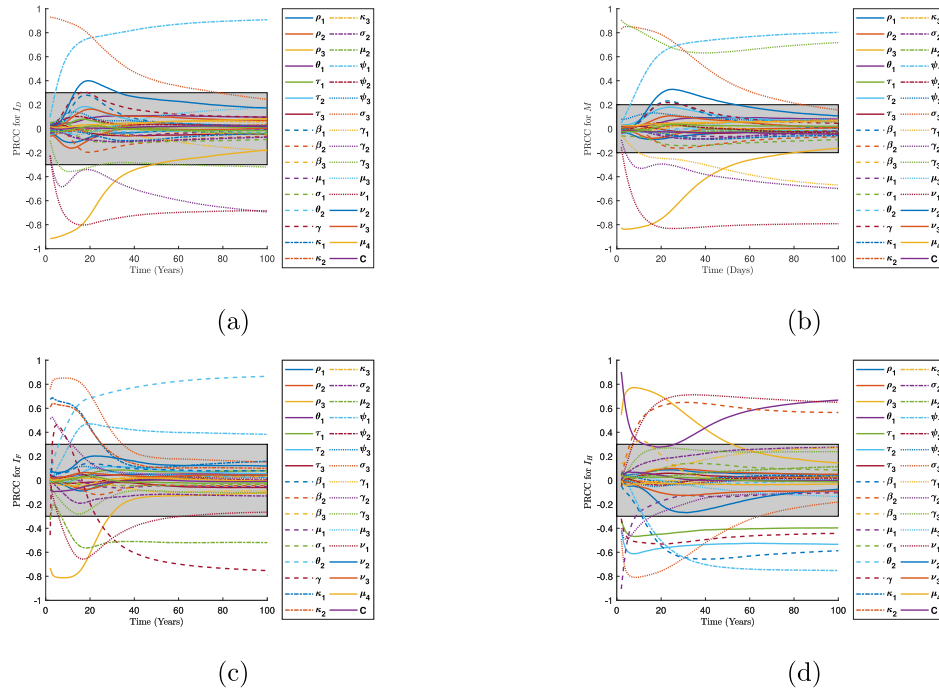


Fig. 3. A visual representation showcasing the evolution of parameter sensitivity throughout the progression of the system dynamics. PRRC values over time span of 100 years with respect to (a) domestic dogs, (b) Environment, (c) Free-range dogs, and (d) Human population.

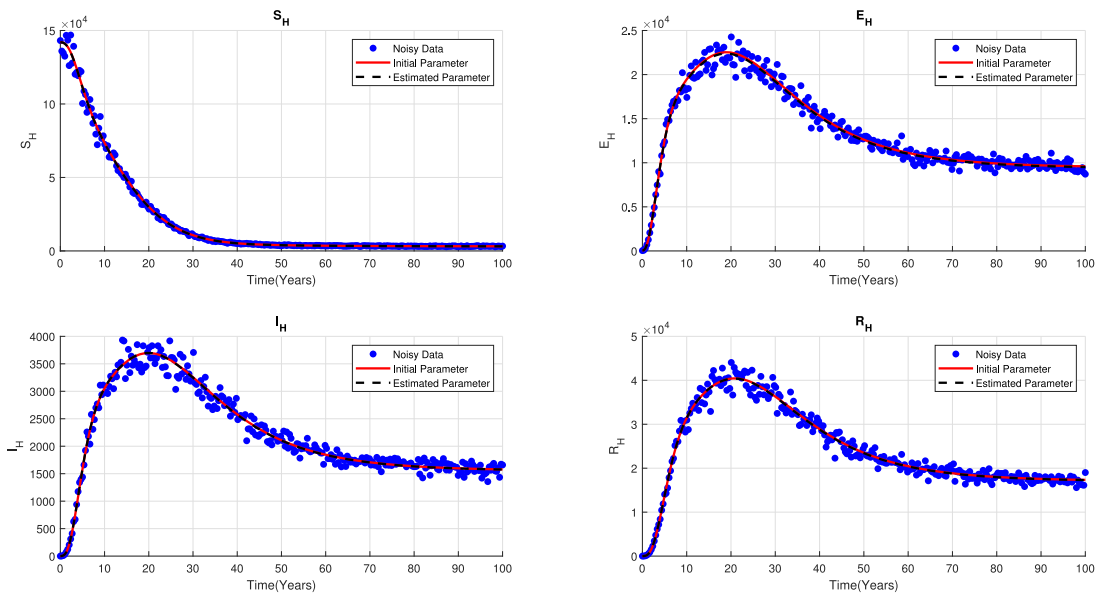


Fig. 4. Model fitting and scatter plot with corresponding parameter estimation with standard deviation $\sigma = 0.05$ and Confidence interval (C.I.) = 95% for human population.

$I_F(0) = 0$, $M(0) = 90$. The estimated parameter values in Table 1 were then applied to model the data, and the outcomes are presented in Figs. 4–7.

5. Discussion

Figs. 8–10 illustrate the histogram of residuals, which provide insight into the consistency between the proposed model fitting and the estimated parameter values, indicating that they stem from the same probability distribution function. By examining the distribution of residuals, which represent the differences between observed data and model predictions, we can assess how effectively the model captures the underlying dynamics of rabies transmission. Meanwhile, Figs. 11–13

show an autocorrelation between 0.2 and -0.2 , implying a moderate to weak correlation between consecutive residuals. This suggests that the errors in the model predictions are relatively independent of each other over time, which can be interpreted as a desirable property indicating that the model adequately captures the underlying dynamics without systematic biases or trends in the residuals.

However, the spread of infectious diseases among human and dog populations often results in a dynamic interplay between the number of susceptible individuals and the number of infected individuals. In particular, Figs. 4–13 show trends for humans, free-range and domestic dog populations. The data presented in these figures suggest that as the number of infections increases, the number of susceptible individuals decreases towards a non-negative constant value. This phenomenon

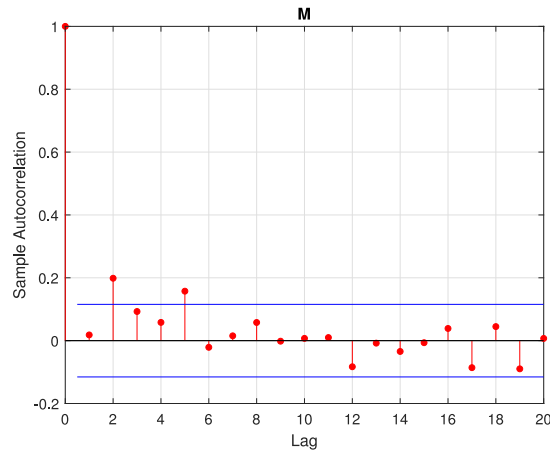


Fig. 5. The sample autocorrelation of the residuals in relation to environment indicating the lack of significance at the 5% level.

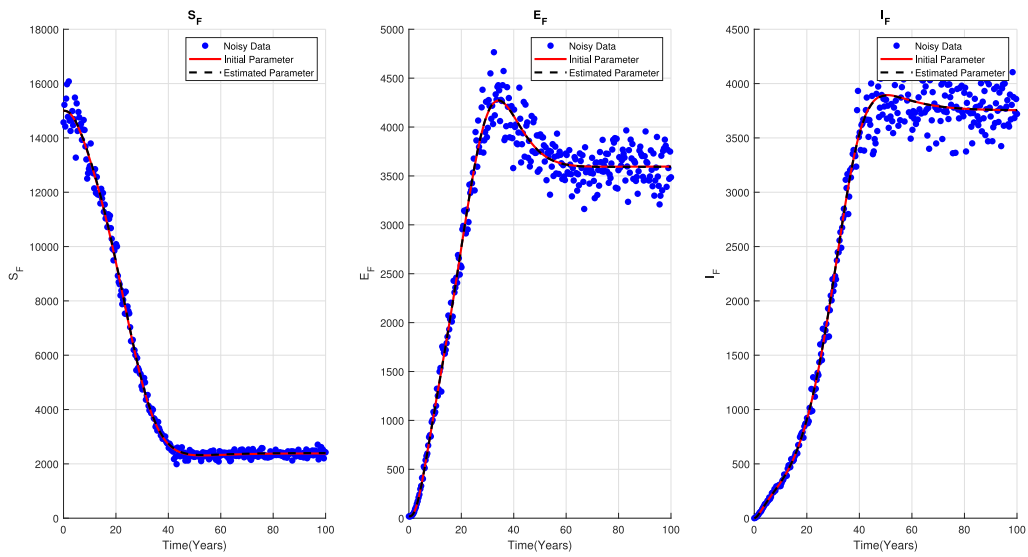


Fig. 6. Model fitting and scatter plot with corresponding parameter estimation with standard deviation $\sigma = 0.05$ and Confidence interval (C.I.) = 95% for free range dogs.

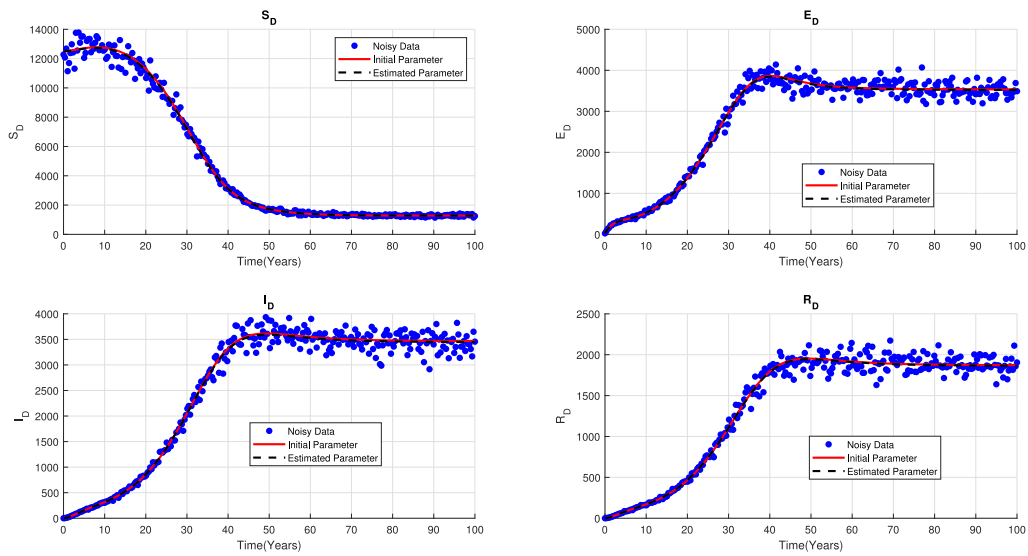


Fig. 7. Model fitting and scatter plot with corresponding parameter estimation with standard deviation $\sigma = 0.05$ and Confidence interval (C.I.) = 95% for domestic dogs.

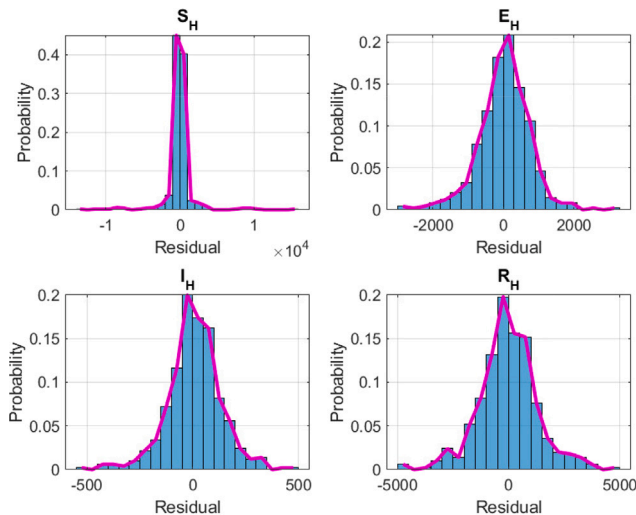


Fig. 8. Normal distribution of the rabies model variable with standard deviation $\sigma = 0.05$ and Confidence interval (C.I.) = 95% for human population.

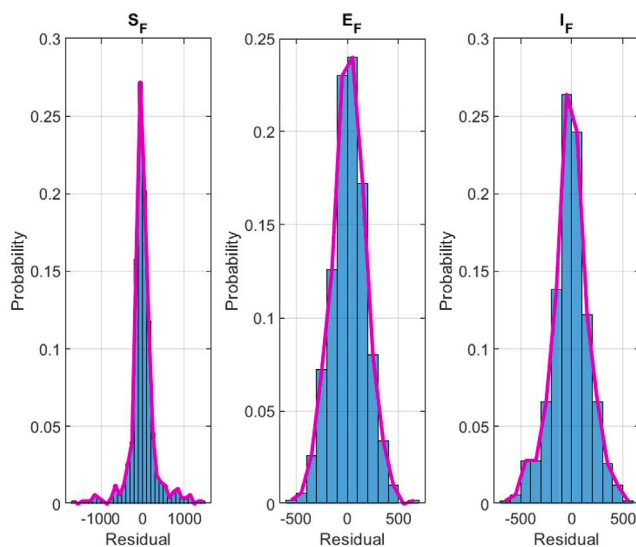


Fig. 9. Normal distribution of the rabies model variable with standard deviation $\sigma = 0.05$ and Confidence interval (C.I.) = 95% for free-range dogs population.

can be explained by the increased contact rate between infectious and susceptible compartments.

6. Conclusion

In this study, we developed and analyzed a deterministic model to estimate parameters and assess uncertainty in the transmission dynamics of rabies in humans and dogs. Data were generated by adding random Gaussian noise to a real world data, and parameter values were estimated using the non-linear least squares method. The basic reproduction number $\mathcal{R}_0$ was derived using the next-generation matrix approach. We determined model equilibria and found that the rabies-free equilibrium is globally asymptotically stable when $\mathcal{R}_0$ is less than one, and the rabies-endemic equilibrium is globally stable when $\mathcal{R}_0$ is greater than or equal to one. Latin Hypercube and Partial Rank Correlation Coefficient analyses were employed to identify which parameters positively or negatively affect model outputs. Sensitivity analysis revealed that contact rates between humans and dogs contribute to an increase in average new rabies infections in the entire population.

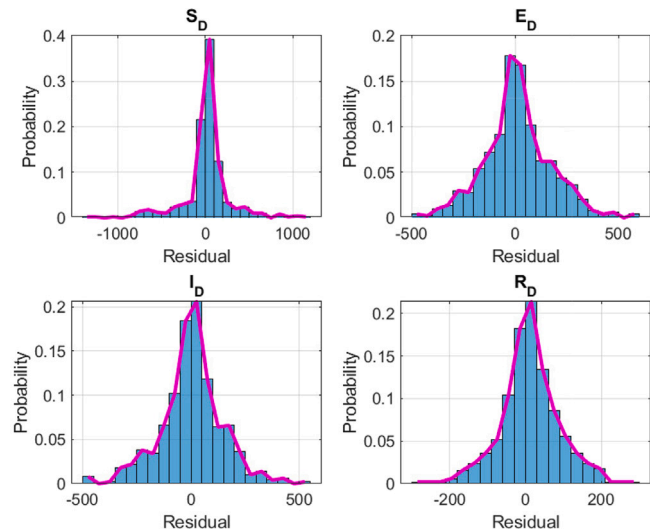


Fig. 10. Normal distribution of the rabies model variable with standard deviation $\sigma = 0.05$ and Confidence interval (C.I.) = 95% for domestic dogs population.

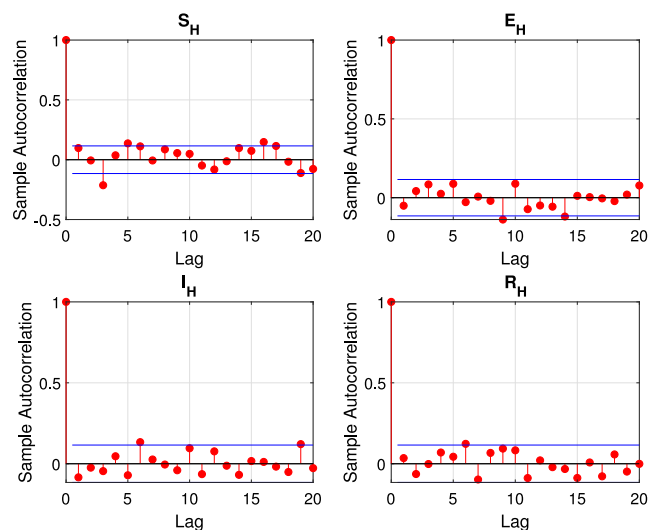


Fig. 11. The sample autocorrelation of the residuals in relation to human population indicating the lack of significance at the 5% level.

CRedit authorship contribution statement

Mfano Charles: Writing – original draft, Software, Methodology, Formal analysis, Conceptualization. **Sayoki G. Mfinanga:** Writing – review & editing, Supervision, Methodology, Data curation, Conceptualization. **G.A. Lyakurwa:** Writing – review & editing, Supervision. **Delfim F.M. Torres:** Writing – review & editing, Supervision, Methodology. **Verdiana G. Masanja:** Writing – review & editing.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

The data used in this study are included in the manuscript.

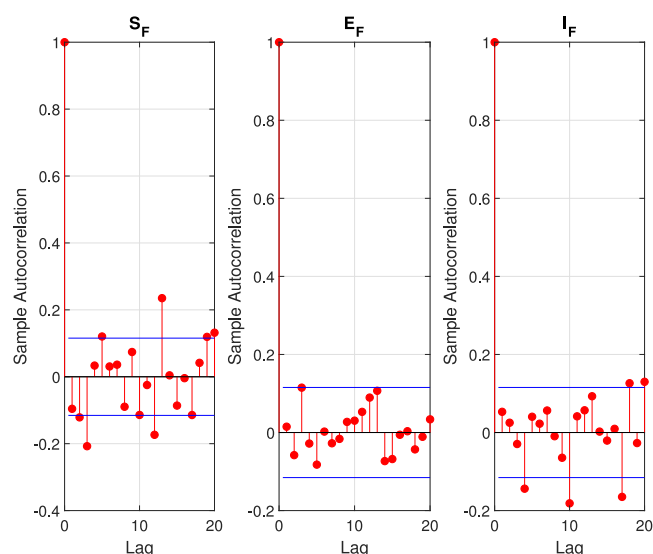


Fig. 12. The sample autocorrelation of the residuals in relation to domestic dogs indicating the lack of significance at the 5% level.

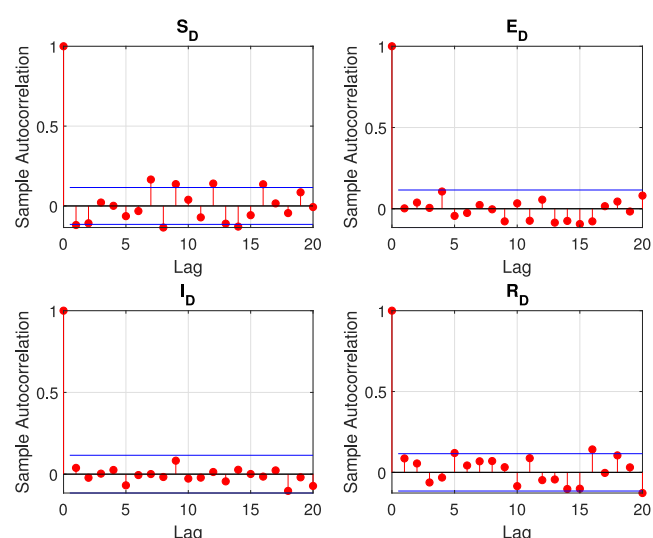
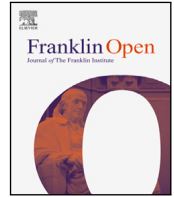


Fig. 13. The sample autocorrelation of the residuals in relation to free range indicating the lack of significance at the 5% level.

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Evaluating the effectiveness of stochastic CTMC and deterministic models in correlating rabies persistence in human and dog populations

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ABSTRACT

Rabies continues to pose a significant zoonotic threat, particularly in areas with high populations of domestic dogs that serve as viral reservoirs. This study conducts a comparative analysis of Stochastic Continuous-Time Markov Chain (CTMC) and deterministic models to gain insights into rabies persistence within human and canine populations. By employing a multitype branching process, the stochastic threshold for rabies persistence was determined, revealing important insights into how stochasticity influences extinction probabilities. The stochastic model utilized 10,000 sample paths to estimate the probabilities of rabies outbreaks, offering a rigorous assessment of the variability in disease occurrences. Additionally, the study introduces a novel mathematical formulation of rabies transmission dynamics, which includes environmental reservoirs, free-ranging dogs, and domestic dogs as essential transmission factors. The basic reproduction number (R_0) was derived and analyzed within stochastic frameworks, effectively bridging the gap between these two modeling approaches. Numerical simulations confirmed that the results from the stochastic model closely aligned with those from the deterministic model, while also highlighting the importance of stochasticity in scenarios with low infection rates. Ultimately, the study advocates for a comprehensive approach to rabies control that integrates both the predictable trends identified through deterministic models and the impact of random events emphasized by stochastic models.

1. Introduction

The ongoing presence of rabies among interconnected populations of dogs and humans poses a significant public health challenge, particularly in regions where access to medical resources is limited [1]. Although rabies is preventable through vaccination, its continued prevalence in areas such as sub-Saharan Africa and parts of Asia underscores the persistent gaps in disease control [2].

One of the primary challenges in addressing rabies transmission in these regions is the underreporting of cases, particularly in rural and remote communities where healthcare infrastructure is minimal [3]. This underreporting creates a critical gap in understanding the true extent of the disease, hindering effective surveillance, prevention, and intervention strategies. The situation is further complicated by environmental factors such as climate change and seasonality, which can

exacerbate the spread of rabies and affect the interaction rates between dogs and humans. Variations in temperature and precipitation patterns can influence the behavior of rabies-carrying wildlife and domestic animals, contributing to unpredictable outbreaks. Additionally, the dynamics of dog populations in specific regions where free-roaming dogs are common and may not receive regular vaccinations serve as a significant reservoir for the virus, complicating efforts to control its transmission [4,5].

Understanding the persistence of rabies in dog populations, along with the associated risks of human infection, necessitates the integration of both stochastic and deterministic modeling frameworks [6]. A key strength of Stochastic Continuous-Time Markov Chain (CTMC) models is their ability to capture the inherent randomness of disease transmission, which is particularly relevant in contexts characterized by

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low numbers of infected individuals [7]. For any given compartment, each random event results in either the exit of an individual from the compartment or the entrance of an individual, unless the compartment is not involved. Therefore, for each process $\mathbb{W}_i^\theta$, at a given time t such that $\mathbb{W}_i^\theta(t) = w_i \in \mathbb{N}$, a transition is always of the form $w_i \rightarrow w_i + \varepsilon_i$, where

$$\varepsilon_i = \begin{cases} -1 & \text{if an individual leaves compartment } i, \\ +1 & \text{if an individual enters compartment } i, \\ 0 & \text{otherwise.} \end{cases}$$

Consequently, the process $\mathbb{W}^\theta$ is stochastic such that at each time t , $\mathbb{W}^\theta(t) \in \mathbb{N}^d$. This probabilistic framework makes Continuous-time Markov Chain (CTMC) models particularly effective for understanding the dynamics of rabies outbreaks and identifying the key factors contributing to persistent trends of the virus [8,9]. In contrast, deterministic models offer a reliable approximation of overall disease dynamics and are instrumental in formulating large-scale intervention strategies, such as determining the vaccination coverage necessary to achieve herd immunity [10,11]. Nevertheless, deterministic models may fail to account for the influence of random events that can significantly alter disease dynamics, especially in situations where population sizes are small or where intervention efforts fluctuate over time [11, 12]. Despite these limitations, deterministic models remain essential tools for grasping overarching epidemiological trends and serve as a foundation for assessing the effectiveness of control measures. This comparison is vital for developing control strategies that address both predictable trends and the inherent variability of the transmission process, thereby ensuring a comprehensive approach to managing rabies outbreaks.

The structure of this paper is organized with sections dedicated to formulating the mathematical model (Section 2), qualitative analysis (Section 3), and quantitative analysis (Section 4). We end with Section 5 of discussion and conclusion.

2. Model formulation

2.1. A deterministic model

A deterministic model describing the transmission dynamics of rabies among humans, free-range dogs, domestic dogs, and the environment is formulated, grounded on the following assumptions.

- (i) Rabies transmission occurs exclusively through effective contact between a susceptible host and an infectious host, or via contaminated environmental media (fomites, carcasses, or inanimate objects harboring the virus).
- (ii) All infectious individuals are subject to both natural and disease-induced mortality, whereas non-infectious individuals experience only natural mortality.
- (iii) Domestic dogs exhibit reduced susceptibility to infection due to human-provided protective measures, while free-range dogs receive neither PEP nor PrEP. Upon confirmed exposure, humans and domestic dogs receive effective PEP.
- (iv) Recruitment rates in each population exceed corresponding natural mortality rates, ensuring persistence in the absence of disease, with recruitment assumed constant and unaffected by seasonal or stochastic variation.
- (v) Populations are homogeneously mixed, with uniform contact probabilities between individuals, regardless of spatial, social, or behavioral heterogeneity.

- (vi) Humans and domestic dogs acquire temporary immunity following recovery, with immunity waning at a constant rate over time.

Model limitations

- (i) Parameter values are assumed constant, neglecting seasonal variability, and rare events such as outbreak fade-outs, which may limit realism in small populations.
- (ii) Uniform contact rates overlook heterogeneity due to spatial segregation, social hierarchies, or human-mediated interactions, and the absence of age structure may omit key transmission dynamics.
- (iii) Immunity decay is modeled as a constant-rate process, disregarding inter-individual variability in immune responses.
- (iv) Environmental contamination is simplified, with limited representation of viral decay rates, persistence, or spatial clustering.
- (v) Assumes PEP is universally effective and accessible, which may not hold in resource-limited or rural settings.
- (vi) Wildlife reservoirs and alternative host species are excluded, focusing solely on human–dog transmission dynamics.
- (vii) The CTMC stochastic framework, although more representative of random processes, is computationally intensive, difficult to parameterize, and requires probability distributions that may not fully align with empirical data.

2.2. Description of model interaction

Susceptible humans are recruited at rate θ_1 and become infected through contact with I_F , I_D , or the environment at rates τ_1 , τ_2 , and τ_3 , respectively. The infection rate is

$$\chi_1 = (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H, \quad \lambda(M) = \frac{M}{M + C}.$$

Exposed humans (E_H) progress to I_H at rate β_1 or recover with post-exposure prophylaxis at rate β_2 . Immunity can wane at rate β_3 , and the disease-induced death rate is σ_1 . All human compartments experience a natural death rate of μ_1 . Free-range dogs are recruited at rate θ_2 and become infected through contact with I_F , I_D , or the environment at rates κ_1 , κ_2 , and κ_3 , respectively, with infection rate

$$\chi_2 = (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F.$$

Exposed free-range dogs (E_F) become I_F at rate γ , with disease and natural death rates σ_2 and μ_2 . Domestic dogs are recruited at rate θ_3 and infected at rates ψ_1 , ψ_2 , and ψ_3 . Their infection rate is

$$\chi_3 = \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right) S_D.$$

Exposed domestic dogs (E_D) progress to I_D at rate γ_1 or recover at γ_2 , with possible immunity loss at γ_3 , disease-induced death at σ_3 , and natural death at μ_3 . Virus shedding in the environment occurs from I_H , I_F , and I_D at rates v_1 , v_2 , and v_3 :

$$\theta_4 = (v_1 I_H + v_2 I_F + v_3 I_D) M, \text{ with removal at rate } \mu_4.$$

The flow diagram presented in Fig. 1 illustrates the dynamics of rabies transmission, incorporating model assumptions, variable definitions, and parameter specifications.

By adopting a stochastic approach, the model captures the inherent randomness and variability in the transmission process, recognizing that real-world outcomes often deviate from average trends due to chance events. This extension is particularly useful for understanding the unpredictable nature of transmission, especially in smaller populations, where random fluctuations can result in unexpected outbreaks or

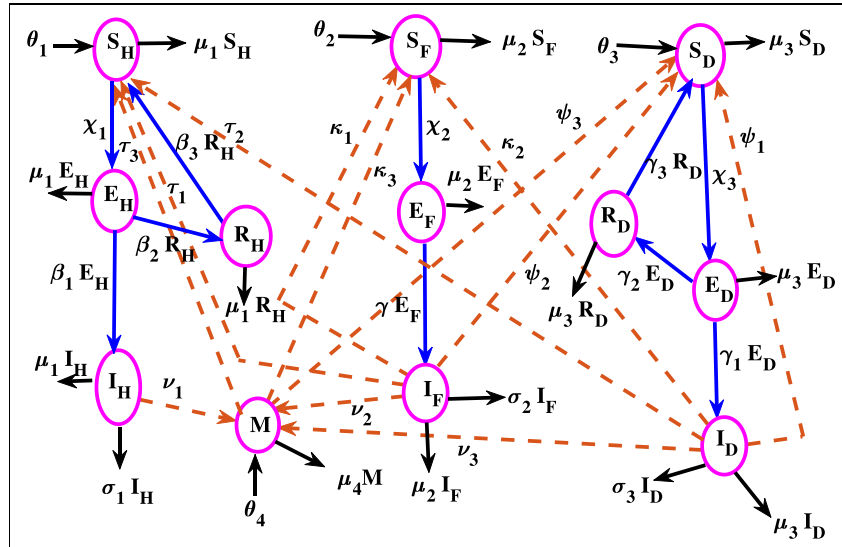


Fig. 1. Flow diagram for rabies transmission among humans, free-range dogs, and domestic dogs.

even the elimination of the disease. A Deterministic Model of the rabies is described by system (1) as

$$\begin{cases}
 \dot{S}_H = \theta_1 + \beta_3 R_H - \mu_1 S_H - \chi_1, \\
 \dot{E}_H = \chi_1 - (\mu_1 + \beta_1 + \beta_2) E_H, \\
 \dot{I}_H = \beta_1 E_H - (\sigma_1 + \mu_1) I_H, \\
 \dot{R}_H = \beta_2 E_H - (\beta_3 + \mu_1) R_H, \\
 \\
 \dot{S}_F = \theta_2 - \chi_2 - \mu_2 S_F, \\
 \dot{E}_F = \chi_2 - (\mu_2 + \gamma) E_F, \\
 \dot{I}_F = \gamma E_F - (\mu_2 + \sigma_2) I_F, \\
 \\
 \dot{S}_D = \theta_3 - \mu_3 S_D - \chi_3 + \gamma_3 R_D, \\
 \dot{E}_D = \chi_3 - (\mu_3 + \gamma_1 + \gamma_2) E_D, \\
 \dot{I}_D = \gamma_1 E_D - (\mu_3 + \sigma_3) I_D, \\
 \dot{R}_D = \gamma_2 E_D - (\mu_3 + \gamma_3) R_D, \\
 \\
 \dot{M} = (\nu_1 I_H + \nu_2 I_F + \nu_3 I_D) - \mu_4 M,
 \end{cases} \quad (1)$$

subject to initial non-negative conditions

$$\begin{aligned}
 S_H(0) > 0, E_H(0) \geq 0, I_H(0) \geq 0, R_H(0) \geq 0, S_F(0) > 0, E_F(0) \geq 0, I_F(0) \geq 0, \\
 S_D(0) \geq 0, E_D(0) \geq 0, I_D(0) \geq 0, R_D(0) \geq 0.
 \end{aligned}$$

3. Qualitative analysis

In this section, we begin by proving the positivity and boundedness of the solutions of system (1) (Lemma 1 and Theorem 2), necessary conditions for the existence of a unique endemic equilibrium (Theorem 3), and global stability of the rabies disease-free equilibrium point (Theorem 4). Then, we formulate a nonlinear continuous-time Markov chain (CTMC) stochastic model for rabies transmission dynamics and analyze its behavior employing the theory of multitype branching processes near the rabies disease-free equilibrium point.

3.1. Positivity of the solutions and boundedness of the system (1)

We begin by proving existence and positivity.

Lemma 1. System (1) admits a solution. Moreover, all solutions of the system (1) that start in the region $\Omega \subset \mathbb{R}_+^{12}$ remain positive all the time.

Proof. To prove the existence of a solution to model (1), we consider initial conditions and apply the integral operator $\int_0^t (\cdot) ds$ on each compartment of the model Eq. (1) as

$$\dot{S}_H = \theta_1 + \beta_3 R_H - \mu_1 S_H - (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H. \quad (2)$$

Integrating (2) both sides over $[0, t]$, we get that

$$\int_0^t \dot{S}_H dt = \int_0^t (\theta_1 + \beta_3 R_H - \mu_1 S_H - (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H) dt. \quad (3)$$

Then, the left-hand side of (3) leads to $S_H(t) - S_H(0)$ and the right-hand side of the same equation leads to

$$\int_0^t \theta_1 dt + \int_0^t \beta_3 R_H dt - \int_0^t \mu_1 S_H dt - \int_0^t (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H dt. \quad (4)$$

By simplifying each integral in (4), we have

$$\begin{aligned}
 & \int_0^t f(s) ds \\
 &= \begin{cases} \theta_1 t, & \text{if } f(s) = \theta_1, \\ \beta_3 \int_0^t R_H(s) ds, & \text{if } f(s) = \beta_3 R_H(s), \\ \mu_1 \int_0^t S_H(s) ds, & \text{if } f(s) = \mu_1 S_H(s), \\ \int_0^t (\tau_1 I_F(s) + \tau_2 I_D(s) + \tau_3 \lambda(M)) S_H(s) ds, & \text{for disease dynamics.} \end{cases} \quad (5)
 \end{aligned}$$

By combining and rearranging the results in (5), it follows that

$$\begin{aligned}
 S_H(t) &= S_H(0) + \theta_1 t + \beta_3 \int_0^t R_H(s) ds - \mu_1 \int_0^t S_H(s) ds \\
 &\quad - \int_0^t (\tau_1 I_F(s) + \tau_2 I_D(s) + \tau_3 \lambda(M)) S_H(s) ds. \quad (6)
 \end{aligned}$$

Thus, we conclude with the non-negativity of the integral terms

$$\begin{aligned}
 S_H(t) &> S_H(0) + \theta_1 t + \beta_3 \int_0^t R_H(s) ds - \mu_1 (\tau_1 I_F(s) + \tau_2 I_D(s) \\
 &\quad + \tau_3 \lambda(M)) \int_0^t S_H(s) ds, \quad (7)
 \end{aligned}$$

since

$$S_H(0) > 0, \quad \theta_1 \geq 0, \quad \beta_3 R_H(s) \geq 0, \quad s \in [0, t].$$

It follows that $S_H(t) > 0$ for all $t \geq 0$. Similarly, we prove the positivity of $I_H(t)$ by considering

$$\dot{I}_H = \beta_1 E_H - (\sigma_1 + \mu_1) I_H. \quad (8)$$

Rearranging Eq. (8) and applying the integrating factor $\mu(t)$, it results in Eq. (9):

$$e^{(\sigma_1 + \mu_1)t} I_H = I_H(0) + \int_0^t \beta_1 e^{(\sigma_1 + \mu_1)s} E_H(s) ds. \quad (9)$$

By solving for $I_H(t)$ in Eq. (9), we obtain

$$I_H(t) = e^{-(\sigma_1 + \mu_1)t} \left(I_H(0) + \int_0^t \beta_1 e^{(\sigma_1 + \mu_1)s} E_H(s) ds \right).$$

Since $I_H(0) \geq 0$ and $E_H(s) \geq 0$ for $s \geq 0$, it follows that

$$I_H(t) \geq 0 \quad \forall t \geq 0. \quad (10)$$

Using the same procedure, we conclude that

$$S_H(t) > 0, E_H(t) \geq 0, I_H(t) \geq 0, R_H(t) \geq 0, S_F(t) > 0, E_F(t) \geq 0, I_F(t) \geq 0,$$

$$S_D(t) \geq 0, E_D(t) \geq 0, I_D(t) \geq 0, R_D(t) \geq 0, \forall t \geq 0,$$

and the proof is complete. $\square$

Theorem 2. All solutions of system (1) starting in $\mathbb{R}^{12+}$ are uniformly bounded.

Proof. The model system (1) can be divided in the subsection of human population, free-range, and domestic dogs, as follows:

$$\begin{aligned} \frac{d(S_H + E_H + I_H + R_H)}{dt} &= \theta_1 + \beta_3 R_H - \mu_1 S_H - (\mu_1 + \beta_1 + \beta_2 + \mu_4) E_H \\ &\quad + \beta_1 E_H - (\sigma_1 + \mu_1) I_H + \beta_2 E_H - (\beta_3 + \mu_1) R_H. \end{aligned} \quad (11)$$

Since the total number of human is given by $S_H + E_H + I_H + R_H = N_H$, Eq. (11) becomes

$$\frac{dN_H}{dt} = \theta_1 - (S_H + E_H + I_H + R_H) \mu_1 - \sigma_1 I_H. \quad (12)$$

We now recall the integrating factor on (12) as

$$N_H(t) = e^{\int_0^t \mu_1 dt} = e^{\mu_1 t} \quad (13)$$

and, for $t \rightarrow 0$, Eq. (13) is simplified as

$$N_H(0) \leq \frac{\theta_1}{\mu_1} + C e^0 \rightarrow N_H(0) - \frac{\theta_1}{\mu_1} \leq C. \quad (14)$$

By simplifying Eq. (14) with simple manipulation, we have

$$\Omega_H = \left\{ (S_H, E_H, I_H, R_H) \in \mathbb{R}_+^4 : 0 \leq S_H + E_H + I_H + R_H \leq \frac{\theta_1}{\mu_1} \right\}. \quad (15)$$

So, using the same procedure, it can be concluded that

$$\begin{aligned} \Omega_F &= \left\{ (S_F, E_F, I_F) \in \mathbb{R}_+^3 : 0 \leq S_F + E_F + I_F \leq \frac{\theta_2}{\mu_2} \right\}, \\ \Omega_D &= \left\{ (S_D, E_D, I_D, R_D) \in \mathbb{R}_+^4 : 0 \leq S_D + E_D + I_D + R_D \leq \frac{\theta_3}{\mu_3} \right\}, \\ M(t) &= \Omega_M = \max \left\{ \frac{\theta_1 v_1}{\mu_1 \mu_4} + \frac{\theta_2 v_2}{\mu_2 \mu_4} + \frac{\theta_3 v_3}{\mu_3 \mu_4}, M(0) \right\}, \end{aligned}$$

and solutions are biologically and mathematically meaningful: any solution relies in the region Ω . $\square$

3.2. Rabies persistent equilibrium point E^*

The point E^* denotes the steady-state condition at which rabies persists concurrently within the human population, free-range dog population, and domestic dog population. This equilibrium is determined

by setting the right-hand sides of the governing equations in system (1) to zero and solving the resulting system of nonlinear equations simultaneously. The endemic equilibrium state is expressed as

$$E^* (S_H^*, E_H^*, I_H^*, R_H^*, S_F^*, E_F^*, I_F^*, S_D^*, E_D^*, I_D^*, R_D^*, M^*),$$

where the components are given by

$$\left. \begin{aligned} R_H^* &= \frac{\beta_2(\sigma_1 + \mu_1)I_H^*}{\beta_1(\beta_3 + \mu_1)}, \\ I_H^* &= \frac{\beta_1(\beta_3 + \mu_3)(\sigma_1 + \mu_1)^2(\beta_1 + \beta_2 + \beta_3)\mu_1 + \beta_1\beta_3(\sigma_1 + \mu_1)^2}{(\sigma_1 + \mu_1)^2((\beta_1 + \beta_2 + \beta_3)\mu_1 + \beta_1\beta_3)} \\ &\quad - \frac{\beta_1(\beta_3 + \mu_3)(\sigma_1 + \mu_1)^2\beta_3 - \theta_1(\beta_3 + \mu_3)(\sigma_1 + \mu_1)^2}{(\sigma_1 + \mu_1)^2((\beta_1 + \beta_2 + \beta_3)\mu_1 + \beta_1\beta_3)}, \\ E_H^* &= \frac{(\sigma_1 + \mu_1)I_H^*}{\beta_1}, \\ S_H^* &= \frac{\beta_3\beta_2(\sigma_1 + \mu_1)I_H^*}{\beta_1(\beta_3 + \mu_1)\mu_1} - \frac{(\mu_1 + \beta_1 + \beta_2)(\sigma_1 + \mu_1)I_H^*}{\beta_1\mu_1} + \frac{\theta_1}{\mu_1}, \\ I_D^* &= \frac{\gamma_1\psi_1 I_F^*(1 + \rho_2)(1 + \rho_3)M^* + \gamma_1\psi_3 M^*(1 + \rho_1)(1 + \rho_2)}{(\mu_3 + \gamma_1 + \gamma_2)^2 - \gamma_1\psi_2(1 + \rho_1)(1 + \rho_3)M^*(\mu_3 + \gamma_1 + \gamma_2)}, \\ E_D^* &= \frac{(\mu_3 + \sigma_3)I_D^*}{\gamma_1}, \quad R_D^* = \frac{\gamma_2(\mu_3 + \sigma_3)I_D^*}{\gamma_1(\mu_3 + \gamma_3)}, \\ S_D^* &= \frac{\gamma_3(\mu_3 + \sigma_3)I_D^*}{\mu_3\gamma_1} - \frac{(\mu_3 + \gamma_1 + \gamma_2)\gamma_2(\mu_3 + \sigma_3)I_D^*}{\gamma_1(\mu_3 + \gamma_3)\mu_3} + \frac{\theta_3}{\mu_3}, \\ E_F^* &= \frac{(\mu_2 + \sigma_2)I_F^*}{\gamma}, \\ S_F^* &= \frac{\theta_2}{\mu_2} - \frac{(\mu_2 + \gamma)(\mu_2 + \sigma_2)I_F^*}{\gamma\mu_2}, \\ M^* &= \frac{v_3 I_D^* + v_2 I_F^* + v_1 I_H^*}{\mu_4}. \end{aligned} \right\}$$

Here, the auxiliary parameters θ_2 and θ_3 are given by Box I

The endemic equilibrium exists when $I_H > 0$, $I_F > 0$, $I_D > 0$, $M > 0$, and $R_0 \geq 1$, as summarized in Theorem 3.

Theorem 3. The model system (1) possesses a unique endemic equilibrium E^* if $R_0 \geq 1$ and $E_H, E_F, E_D, M > 0$.

The proof that the endemic equilibrium point E^* of the rabies model (1) is globally asymptotically stable whenever $R_0 \geq 1$ is given in Appendix.

3.3. Global stability of the rabies disease free equilibrium point E^0

To obtain E^0 , the left hand side of equation in the model system (1) is set to zero, such that

$$E^0 = \left(\frac{\theta_1}{\mu_1}, 0, 0, 0, \frac{\theta_2}{\mu_2}, 0, 0, \frac{\theta_3}{\mu_3}, 0, 0, 0, 0 \right).$$

Theorem 4. The rabies disease free equilibrium point E^0 is globally asymptotically stable when $R_0 < 1$ and unstable otherwise.

Proof. The analysis of the equilibrium behavior E^0 of the model described in (1) employs the Metzler matrix, as demonstrated by [5,13]. In this context, U_s represent the compartments that do not transmit rabies, and U_i represent the rabies-transmitting compartments. If A_2 is a Metzler matrix (with non-negative off-diagonal entries) and A_0 has real negative eigenvalues, then the rabies-free equilibrium is globally

$$\left. \begin{aligned} \theta_2 &= \frac{(\mu_2 + \gamma)\mu_2(1 + (R_0 - 1))(1 + \rho_1)\mu_3(\mu_2 + \sigma_2)((1 + \rho_2)(\mu_3 + \sigma_3)(\mu_3 + \gamma_1 + \gamma_2)(1 + (R_0 - 1)) - \theta_3\psi_2\gamma_1)}{(\mu_3(1 + \rho_2)(1 + \rho_1)(\mu_3 + \sigma_3)(\mu_3 + \gamma_1 + \gamma_2)(1 + (R_0 - 1)) - \theta_3\gamma_1(\psi_2(1 + \rho_1)\mu_3 + \psi_1(1 + \rho_2)))\gamma\kappa_1} \\ \theta_3 &= \frac{(-\mu_2(\mu_2 + \sigma_2)(\mu_2 + \gamma)(1 + (R_0 - 1)) + \gamma\kappa_1\theta_2)(1 + (R_0 - 1))(1 + \rho_1)\mu_3(1 + \rho_2)(\mu_3 + \sigma_3)(\mu_3 + \gamma_1 + \gamma_2)}{((- \mu_2(\mu_2 + \sigma_2)(\mu_2 + \gamma)(1 + (R_0 - 1)) + \gamma\kappa_1\theta_2)(1 + \rho_1)\psi_2\mu_3 + \gamma\kappa_1\theta_2\psi_1(1 + \rho_2))\gamma_1} \end{aligned} \right\}$$

Box I.

$$A_2 = \begin{pmatrix} -\mu_1 - \beta_1 - \beta_2 & 0 & 0 & \frac{\tau_1\theta_1}{\mu_1} & 0 & \frac{\tau_2\theta_1}{\mu_1} & 0 \\ \beta_1 & -\sigma_1 - \mu_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\mu_2 - \gamma & \frac{\kappa_1\theta_2}{\mu_2} & 0 & \frac{\kappa_2\theta_2}{\mu_2} & 0 \\ 0 & 0 & \gamma & -\mu_2 - \sigma_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\psi_1\theta_3}{\mu_3(1+\rho_1)} & -\mu_3 - \gamma_1 - \gamma_2 & \frac{\psi_2\theta_3}{\mu_3(1+\rho_1)} & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\mu_3 - \sigma_3 & 0 \\ 0 & \nu_1 & 0 & \nu_2 & 0 & \nu_3 & -\mu_4 \end{pmatrix},$$

Box II.

asymptotically stable. Then, the model Eq. (1) is decomposed to

$$\begin{cases} \frac{dU_s}{dt} = A_0(U_s - U(E^0)) + A_1U_i, \\ \frac{dU_i}{dt} = A_2U_i, \end{cases} \quad (16)$$

where

$$U_s - U(E^0) = \begin{pmatrix} S_H - \frac{\theta_1}{\mu_1} \\ R_H \\ S_F - \frac{\theta_2}{\mu_2} \\ S_D - \frac{\theta_3}{\mu_3} \\ R_D \end{pmatrix},$$

$$A_0 = \begin{pmatrix} -\mu & \beta_3 & 0 & 0 & 0 \\ 0 & -(\beta_3 + \mu_1) & 0 & 0 & 0 \\ 0 & 0 & -\mu_2 & 0 & 0 \\ 0 & 0 & 0 & -\mu_3 & \gamma_3 \\ 0 & 0 & 0 & 0 & -(\mu_3 + \gamma_3) \end{pmatrix},$$

$$A_1 = \begin{pmatrix} 0 & 0 & 0 & \frac{\tau_1\theta_1}{\mu_1} & 0 & \frac{\tau_2\theta_1}{\mu_1} & 0 \\ \beta_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\kappa_1\theta_2}{\mu_2} & 0 & \frac{\kappa_2\theta_2}{\mu_2} & 0 \\ 0 & 0 & 0 & \frac{\psi_1\theta_3}{\mu_3(1+\rho_1)} & 0 & \frac{\psi_2\theta_3}{\mu_3(1+\rho_2)} & 0 \\ 0 & 0 & 0 & 0 & \gamma_2 & 0 & 0 \end{pmatrix},$$

and A_2 is as in Box II. Given that the eigenvalues of the matrix A_0 are negative and the off-diagonal entries of the Metzler matrix A_2 are non-negative, it follows that the rabies equilibrium point E^0 is globally asymptotically stable. $\square$

3.4. Rabies CTMC stochastic model formulation

Continuous Time Markov Chain (CTMC) stochastic models utilize Galton–Watson Branching Processes to delineate the probabilities of

various events, offering valuable insights into dynamics, control strategies, prediction of expected case numbers, extinction time, and the assessment of surveillance and response system effectiveness. While deterministic models rely on the basic reproduction number (R_0) to determine whether a disease persists or diminishes in a population, stochastic models view R_0 as a stochastic threshold, recognizing that the disease can still cease to exist even if the threshold exceeds one, contingent upon the initial number of infectives introduced into a susceptible population.

A continuous time Markov chain (CTMC) stochastic model for rabies transmission dynamics has been developed based on the assumptions employed in the stochastic model (1). For the sake of simplicity, the same notations and parameters as those used in the deterministic model have been adopted. Let $S_H, E_H, I_H, R_H, S_F, E_F, I_F, S_D, E_D, I_D, R_D, M$, denote the discrete random variable for susceptible humans, exposed humans, infectious humans, recovered humans, susceptible free-range dogs, exposed free-range dogs, infectious free-range dogs, susceptible domestic dogs, exposed domestic dogs, infectious domestic animals, recovered domestic dogs, and environment respectively. Let

$$\mathbf{X}(t) = (S_H, E_H, I_H, R_H, S_F, E_F, I_F, S_D, E_D, I_D, R_D, M)^T$$

be the associated random vector for all discrete random variables $S_H, E_H, I_H, R_H, S_F, E_F, I_F, S_D, E_D, I_D, R_D$, and M . Given the time-homogeneous nature of the Continuous-Time Markov Chain (CTMC) model and its adherence to the Markov property, it is established that the future state of the process at $(t + \Delta t)$ hinges entirely upon the current state at time t . As a result, the interval between events follows an exponential distribution characterized by a specific parameter:

$$\left\{ \begin{aligned} \Psi(\mathbf{X}) &= \theta_1 + \beta_3 R_H + \mu_1 N_H + \tau_1 I_F S_H + \tau_2 I_D S_H + \left(\frac{\tau_3 M}{M + C} \right) S_H + \beta_1 E_H \\ &\quad + \kappa_1 I_F S_F + \kappa_2 I_D S_F + \left(\frac{\kappa_3 M}{M + C} \right) S_F + \mu_2 N_F + \gamma E_F + \sigma_2 I_F + \theta_3 \\ &\quad + \left(\frac{\psi_1}{1 + \rho_1} \right) I_F S_D + \left(\frac{\psi_2}{1 + \rho_2} \right) I_D S_D + \left(\frac{\psi_3 M}{(1 + \rho_3)(M + C)} \right) S_D \\ &\quad + \mu_3 N_D + \gamma_3 R_D + \gamma_1 E_D + \gamma_2 E_D + \sigma_3 I_D + \nu_1 I_H + \nu_2 I_F + \nu_3 I_D \\ &\quad + \mu_4 M + \beta_2 E_H + \sigma_1 I_H + \theta_2, \end{aligned} \right. \quad (17)$$

Table 1
State transitions and rates of occurrence for the CTMC.

Event	Rate, r	Transition $\Delta Z(t)$
Recruitment of S_H	θ_1	(1, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)
Natural death of S_H	$\mu_1 S_H$	(-1, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0)
Contact of S_H and I_F	$\tau_1 I_F S_H$	(-1, 1, 0, 0, 0, 0, 0, 0, 0, 0, 0)
Contact of S_H and I_D	$\tau_2 I_D S_H$	(-1, 1, 0, 0, 0, 0, 0, 0, 0, 0, 0)
Contact of S_H and M	$\frac{M\tau_3}{M+C} S_H$	(-1, 1, 0, 0, 0, 0, 0, 0, 0, 0, 0)
Progression from E_H to I_H	$\beta_1 E_H$	(0, -1, 1, 0, 0, 0, 0, 0, 0, 0, 0)
Recovery of E_H	$\beta_2 E_H$	(0, -1, 0, 1, 0, 0, 0, 0, 0, 0, 0)
Natural death of E_H	$\mu_1 E_H$	(0, -1, 0, 0, 0, 0, 0, 0, 0, 0, 0)
Disease induced death of I_H	$\sigma_1 I_H$	(0, 0, -1, 0, 0, 0, 0, 0, 0, 0, 0)
Natural death of I_H	$\mu_1 I_H$	(0, 0, -1, 0, 0, 0, 0, 0, 0, 0, 0)
Natural death of R_H	$\mu_1 R_H$	(0, 0, 0, -1, 0, 0, 0, 0, 0, 0, 0)
Rate of immunity loss of R_H	$\beta_3 R_H$	(1, 0, 0, -1, 0, 0, 0, 0, 0, 0, 0)
Recruitment of S_F	θ_2	(0, 0, 0, 0, 1, 0, 0, 0, 0, 0, 0)
Natural death of S_F	$\mu_2 S_F$	(0, 0, 0, 0, -1, 0, 0, 0, 0, 0, 0)
Contact of S_F and I_F	$\kappa_1 I_F S_F$	(0, 0, 0, 0, -1, 1, 0, 0, 0, 0, 0)
Contact of S_F and I_D	$\kappa_2 I_D S_F$	(0, 0, 0, 0, -1, 1, 0, 0, 0, 0, 0)
Contact of S_F and M	$\frac{M\kappa_3}{M+C} S_F$	(0, 0, 0, 0, -1, 1, 0, 0, 0, 0, 0)
Progression of E_F to I_F	γE_F	(0, 0, 0, 0, 0, -1, 1, 0, 0, 0, 0)
Natural death of E_F	$\mu_2 E_F$	(0, 0, 0, 0, 0, -1, 0, 0, 0, 0, 0)
Natural death of I_F	$\mu_2 I_F$	(0, 0, 0, 0, 0, 0, -1, 0, 0, 0, 0)
Disease induced death of I_F	$\sigma_2 I_F$	(0, 0, 0, 0, 0, 0, -1, 0, 0, 0, 0)
Recruitment of S_D	θ_3	(0, 0, 0, 0, 0, 0, 0, 1, 0, 0, 0)
Natural death of S_D	$\mu_3 S_D$	(0, 0, 0, 0, 0, 0, 0, -1, 0, 0, 0)
Contact of S_D and I_F	$\frac{\psi_1 I_F}{1+\rho_1} S_D$	(0, 0, 0, 0, 0, 0, 0, -1, 1, 0, 0)
Contact of S_D and I_D	$\frac{\psi_2 I_D}{1+\rho_2} S_D$	(0, 0, 0, 0, 0, 0, 0, -1, 1, 0, 0)
Contact of S_D and M	$\frac{\psi_3 M}{(1+\rho_3)(C+M)} S_D$	(0, 0, 0, 0, 0, 0, 0, -1, 1, 0, 0)
Progression of E_D to I_D	$\gamma_1 E_D$	(0, 0, 0, 0, 0, 0, 0, 0, -1, 1, 0)
Recovery of E_D	$\gamma_2 E_D$	(0, 0, 0, 0, 0, 0, 0, 0, -1, 0, 1)
Disease induced death of I_D	$\sigma_3 I_D$	(0, 0, 0, 0, 0, 0, 0, 0, 0, -1, 0)
Natural death of E_D	$\mu_3 E_D$	(0, 0, 0, 0, 0, 0, 0, 0, 0, -1, 0)
Natural death of I_D	$\gamma_3 R_D$	(0, 0, 0, 0, 0, 0, 0, 0, 0, -1, 0)
Natural death of R_D	$\mu_3 R_D$	(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, -1)
Removal of rabies in the environment	$\mu_4 M$	(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, -1)
Shedding of I_H to M	$\nu_1 I_H$	(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 1)
Shedding of I_F to M	$\nu_2 I_F$	(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 1)
Shedding of I_D to M	$\nu_3 I_D$	(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 1)

where

$$N_H = S_H + E_H + I_H + R_H, \quad N_F = S_F + E_F + I_F \quad \text{and} \quad N_D = S_D + E_D + I_D + R_D.$$

For modeling the transmission dynamics of rabies between humans and dogs using a Continuous Time Markov Chain (CTMC) model, event transitions and their corresponding rates are typically derived from the deterministic model. These transitions occur as individuals move between compartments due to recruitment or movement, assuming an initial presence of only one individual while other sub-populations are not yet established. Table 1 summarizes the events and their associated transition rates, where the values 1, -1, and 0 represent an increase by 1, a decrease by 1, and no change in state, respectively, for the variable from time t to $t + \Delta t$.

3.5. Multitype branching process

The multitype branching process theory is employed to analyze the behavior of the nonlinear Continuous-Time Markov Chain (CTMC) near the Rabies-free equilibrium point $\mathbb{E}_0$. This theory is crucial for determining the probabilities of disease extinction or outbreak under various initial conditions. In CTMC models, the branching process can either grow exponentially or diminish to zero, particularly when the initial number of infectives is minimal at the onset of a disease outbreak. In order for the multitype branching process to be applicable, it necessitates a sufficiently large initial susceptible population. As per the parameters of this study, we have established the initial susceptible populations as follows: $S_H(0) = \frac{\theta_1}{\mu_1}$, $S_F(0) = \frac{\theta_2}{\mu_2}$, and $S_D(0) = \frac{\theta_3}{\mu_3}$. We assume that infectives of type i , I_i , produce infectives of type

j , I_j , and the number of offspring produced by an individual of type I_i is independent of the number of offspring produced by either type I_i or type I_j , where $j \neq i$. The term ‘birth’ describes the infection transmission between susceptible humans, infected humans, susceptible domestic dogs, infected domestic dogs, susceptible free-range dogs, infected free-range dogs, and the rabies in the environment. Since the multitype branching process is linear near the disease-free equilibrium, the numbers of deaths and births are independent. We define probability generating functions (pgfs) for the births and deaths of rabies in the environment, infected humans, domestic and free-range dogs, which are essential for determining the probability of rabies extinction or outbreak in humans and dogs.

Let $\{Y_{ji}\}_{j=1}^n$ be the offspring random variable for type i , where $i = 1, 2, \dots, n$ infectious hosts. Here, Y_{ji} represents the number of offspring of type j produced by an infective of type i . The offspring probability generating function (pgf) for the infectious population I_i is defined under the condition that there is initially one infectious host at the beginning of the disease outbreak, i.e., $I_i(0) = 1$, and all other types are zero: $I_j = 0$. The offspring pgf $f_i : [0, 1]^n \rightarrow [0, 1]$ for type i individuals, given $I_i(0) = 1$ and $I_j(0) = 0$ for $j \neq i$, is expressed as

$$f_i(u_1, u_2, \dots, u_n) = \sum_{\ell_1=0}^{\infty} \sum_{k_2=0}^{\infty} \dots \sum_{\ell_n=0}^{\infty} P_i(\ell_1, \ell_2, \dots, \ell_n) u_1^{\ell_1} u_2^{\ell_2} \dots u_n^{\ell_n}, \quad (18)$$

where

$$P_i(\ell_1, \ell_2, \dots, \ell_n) = \text{Prob}\{Y_{1j} = \ell_1, Y_{2j} = \ell_2, \dots, Y_{nj} = \ell_n\} \quad (19)$$

is the probability that a single infectious individual of type i will produce k offspring of type j . Eq. (18) is utilized to establish an $n \times n$

non-negative and irreducible expectation matrix $\mathcal{M}_1 = [m_{ji}]$, where m_{ji} denotes the expected number of offspring of type j generated by an infected individual of type i . The elements of matrix $\mathcal{M}_1$ are calculated by differentiating f_i with respect to u_j and then evaluating all u variables at 1 [14,15], meaning that

$$m_{ji} = \left. \frac{\partial f_i}{\partial u_j} \right|_{u=1} < \infty. \quad (20)$$

The probability of disease extinction or outbreak is determined by the size of the spectral radius of the expectation matrix $\mathcal{M}_1$, $\rho(\mathcal{M}_1)$. If $\rho(\mathcal{M}_1) \leq 1$, then the probability of disease extinction is one, that is,

$$\mathbb{P}_0 = \lim_{t \rightarrow \infty} \text{Prob}\{\tilde{I}(t) = \bar{0}\} = 1, \quad (21)$$

and if $\rho(\mathcal{M}_1) > 1$, then there exists a positive probability such that the probability of disease extinction is given by

$$\mathbb{P}_0 = \lim_{t \rightarrow \infty} \text{Prob}\{\tilde{I}(t) = \bar{0}\} = q_1^{i_1} q_2^{i_2} \dots q_k^{i_k} < 1, \quad (22)$$

where $(q_1, q_2, \dots, q_k)$ is the unique fixed point of the k offspring pgf, $f_i(q_1, q_2, \dots, q_k) = q_i$, and $0 < q_i < 1$, $i = 1, 2, \dots, k$ [14,16,17]. The probability of disease outbreak is

$$1 - \mathbb{P}_0 = 1 - q_1^{i_1} q_2^{i_2} \dots q_k^{i_k}, \quad (23)$$

where $\mathbb{P}_0$ is the probability of extinction or outbreak [14,15]. Predictions concerning disease extinction and the occurrence of outbreaks can be made using stochastic epidemic theory, which focuses on the number of infectious individuals within each group. If a disease originates from an infectious group with a reproduction number ($\mathcal{R}_0 > 1$), and i infective individuals are introduced into an entirely susceptible population, the probability of a significant outbreak is approximately $1 - \left(\frac{1}{\mathcal{R}_0}\right)^i$. Conversely, the probability of the disease becoming extinct is approximately $\left(\frac{1}{\mathcal{R}_0}\right)^i$. In the early stages of a rabies outbreak, with only a few infected dogs, there is a limited potential for generating infectious humans and dogs. Exposed humans and both free-range and domestic dogs can progress to infectious classes. The offspring probability generating function for the exposed class E_H , given that $E_H(0) = 1$, $I_H(0) = 0$, $E_F(0) = 0$, $I_F(0) = 0$, $E_D(0) = 0$, $I_D(0) = 0$, and $M(0) = 0$, is given by

$$f_1(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\beta_1 u_2 + \beta_2 + \mu_1}{\beta_1 + \beta_2 + \mu_1}. \quad (24)$$

The expression $\beta_1/(\beta_1 + \beta_2 + \mu_1)$ denotes the probability of exposed individual progressing to the infectious class I_H . The term $\beta_2/(\beta_1 + \beta_2 + \mu_1)$ represents the probability of exposed individuals recovering as a result of Rabies Postexposure Prophylaxis (PEP), while $\mu_1/(\beta_1 + \beta_2 + \mu_1)$ indicates the probability of exposed individuals naturally dying before transitioning to the infected class.

If $E_H(0) = 0$, $I_H(0) = 1$, $E_F(0) = 0$, $I_F(0) = 0$, $E_D(0) = 0$, $I_D(0) = 0$, and $M(0) = 0$, then the offspring probability generating function for I_H is given by

$$f_2(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\nu_1 u_2 u_7 + \sigma_1 + \mu_1}{\nu_1 + \sigma_1 + \mu_1}. \quad (25)$$

In pgf (25), the term $\nu_1/(\mu_1 + \nu_1 + \sigma_1)$ represents the probability of infected humans shedding the rabies virus in the environment; $\mu_1/(\mu_1 + \nu_1 + \sigma_1)$ signifies the probability of infected humans dying naturally; and the term $\sigma_1/(\mu_1 + \nu_1 + \sigma_1)$ refers to the probability of infected humans dying due to the disease.

The offspring probability generating function for E_F , such that $E_H(0) = 0$, $I_H(0) = 0$, $E_F(0) = 1$, $I_F(0) = 0$, $E_D(0) = 0$, $I_D(0) = 0$, and $M(0) = 0$, is given by

$$f_3(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\gamma u_4 + \mu_2}{\gamma + \mu_2}, \quad (26)$$

where $\gamma/(\gamma + \mu_2)$ denotes the probability of the exposed free range dogs class progressing to the infected class, and $\mu_2/(\gamma + \mu_2)$ represents the probability of exposed free-range dogs dying naturally before progressing to the infected class.

The offspring probability for I_F , given that $E_H(0) = 0$, $I_H(0) = 0$, $E_F(0) = 0$, $I_F(0) = 1$, $E_D(0) = 0$, $I_D(0) = 0$, and $M(0) = 0$, is given by

$$f_4(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\hat{\lambda}_1 u_1 u_4 + \hat{\lambda}_2 u_4 u_5 + \hat{\lambda}_3 u_3 u_4 + \mu_2 + \sigma_2 + \nu_2 u_4 u_7}{\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + \nu_2},$$

for $\hat{\lambda}_1 = \tau_1 S_H^0$, $\hat{\lambda}_2 = \frac{\psi_1}{1 + \rho_1} S_D^0$, $\hat{\lambda}_3 = \kappa_1 S_F^0$. (27)

In pgf (27), the term $\hat{\lambda}_1/(\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + \nu_2)$ represents the probability of infected free range dogs to cause rabies infection to susceptible humans, $\hat{\lambda}_2/(\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + \nu_2)$ signifies the probability of infected free-range dogs causing infection to susceptible domestic dogs, $\hat{\lambda}_3/(\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + \nu_2)$ denotes infected free-range dogs causing infection to free-range dogs, $\nu_2/(\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + \nu_2)$ represents the probability of free-range dogs to shed rabies virus in the environment, $\mu_2/(\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + \nu_2)$ denotes the probability of infected free-range dogs dying naturally, and the term $\sigma_2/(\hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + \nu_2)$ is the probability of infected free-range dogs dying from rabies disease.

If $E_H(0) = 0$, $I_H(0) = 0$, $E_F(0) = 0$, $I_F(0) = 0$, $E_D(0) = 1$, $I_D(0) = 0$, and $M(0) = 0$, then the offspring probability of generating function for E_D is given by

$$f_5(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\gamma_1 u_6 + \gamma_2 + \mu_3}{\gamma_1 + \gamma_2 + \mu_3}, \quad (28)$$

where $\gamma_1/(\gamma_1 + \gamma_2 + \mu_3)$ represents the probability of exposed domestic dogs progressing to infected class, $\gamma_2/(\gamma_1 + \gamma_2 + \mu_3)$ denotes the probability of exposed domestic dogs recovering from exposed class as a result of Rabies Postexposure Prophylaxis (PEP) before progressing to infected class, and $\mu_3/(\gamma_1 + \gamma_2 + \mu_3)$ signifies the probability of infected domestic dogs dying naturally before progressing to infected class.

The offspring probability generating function for I_D , such that $E_H(0) = 0$, $I_H(0) = 0$, $E_F(0) = 0$, $I_F(0) = 0$, $E_D(0) = 0$, $I_D(0) = 1$, and $M(0) = 0$, is given by

$$f_6(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\hat{\lambda}_4 u_5 u_6 + \hat{\lambda}_5 u_1 u_6 + \hat{\lambda}_6 u_3 u_6 + \mu_3 + \sigma_3 + \nu_3 u_6 u_7}{\hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + \nu_3},$$

for $\hat{\lambda}_4 = \frac{\psi_2}{1 + \rho_2} S_D^0$, $\hat{\lambda}_5 = \tau_2 S_H^0$, $\hat{\lambda}_6 = \kappa_2 S_F^0$. (29)

In pgf (29), the term $\hat{\lambda}_4/(\hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + \nu_3)$ represents the probability of infected domestic dogs causing rabies infection to susceptible domestic dogs, $\hat{\lambda}_5/(\hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + \nu_3)$ signifies the probability of domestic dogs causing infection to susceptible humans, $\hat{\lambda}_6/(\hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + \nu_3)$ denotes domestic dogs causing infection to susceptible free-range dogs, $\nu_3/(\hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + \nu_3)$ represents the probability of domestic dogs to shed rabies virus in the environment, $\mu_3/(\hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + \nu_3)$ denotes the probability of infected domestic dogs dying naturally, and the term $\sigma_3/(\hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + \nu_3)$ is the probability of infected domestic dogs dying from rabies disease.

If $E_H(0) = 0$, $I_H(0) = 0$, $E_F(0) = 0$, $I_F(0) = 0$, $E_D(0) = 0$, $I_D(0) = 0$, and $M(0) = 1$, then the offspring probability of generating function for M is given by

$$f_7(u_1, u_2, u_3, u_4, \dots, u_7) = \frac{\hat{\lambda}_7 u_5 u_7 + \hat{\lambda}_8 u_1 u_7 + \hat{\lambda}_9 u_3 u_7 + \mu_4}{\hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4},$$

for $\hat{\lambda}_7 = \frac{\psi_3}{1 + \rho_3} S_D^0$, $\hat{\lambda}_8 = \tau_3 S_H^0$, $\hat{\lambda}_9 = \kappa_3 S_F^0$, (30)

where $\hat{\lambda}_7/(\hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4)$ represents the probability of rabies in environment causing infection to susceptible domestic dogs,

Table 2

Estimated model parameters (Year⁻¹), initial guess for parameters (Year⁻¹) and their respective source.

Parameters	Baseline value	Source	Estimated value	Mean (μ) and std (σ)
θ_1	2000	(Assumed)	1993.382113	$\mathcal{N}(1996.691056 \ 4.4679553)$
τ_1	0.0004	[18]	0.000405	$\mathcal{N}(0.000402 \ 4 \times 10^{-6})$
τ_2	0.0004	[18]	0.000604	$\mathcal{N}(0.000502 \ 1.44 \times 10^{-4})$
τ_3	[0.0003 \ 0.0100]	(Assumed)	0.000303	$\mathcal{N}(0.000302 \ 2 \times 10^{-6})$
β_1	$\frac{1}{6}$	[18,19]	0.165581	$\mathcal{N}(0.166124 \ 7.68 \times 10^{-4})$
β_2	[0.54 \ 1]	[19,20]	0.540487	$\mathcal{N}(0.5402435 \ 3.7815 \times 10^{-4})$
β_3	1	(Assumed)	0.999301	$\mathcal{N}(0.9996505 \ 1.6521 \times 10^{-4})$
μ_1	0.0142	[21]	0.014417	$\mathcal{N}(0.014309 \ 1.53 \times 10^{-4})$
σ_1	1	[19,20]	1.006332	$\mathcal{N}(1.03166 \ 4.47 \times 10^{-3})$
θ_2	1000	(Assumed)	1004.12044	$(1002.060222 \ 2.913594)$
κ_1	0.00006	(Assumed)	0.000020	$\mathcal{N}(0.000040 \ 2.8 \times 10^{-5})$
κ_2	0.00005	(Assumed)	0.000081	$\mathcal{N}(0.000066 \ 2.2 \times 10^{-5})$
κ_3	[0.00001 \ 0.00003]	(Assumed)	0.000040	$\mathcal{N}(0.000025 \ 2.1 \times 10^{-5})$
γ	$\frac{1}{6}$	[18–20]	0.166374	$\mathcal{N}(0.166520 \ 2.07 \times 10^{-4})$
σ_2	0.09	[19,22]	0.089556	$\mathcal{N}(0.089778 \ 3.14 \times 10^{-4})$
μ_2	0.067	(Assumed)	0.066268	$\mathcal{N}(0.066634 \ 1.58 \times 10^{-4})$
θ_3	1200	(Assumed)	1203.844461	$\mathcal{N}(1201.922230 \ 2.718444)$
ψ_1	0.0004	[22,23]	0.000077	$\mathcal{N}(0.000238 \ 2.28 \times 10^{-4})$
ψ_2	0.0004	[4]	0.000066	$\mathcal{N}(0.000233 \ 2.36 \times 10^{-4})$
ψ_3	0.0003	(Assumed)	0.000030	$\mathcal{N}(0.0003 \ 1.91 \times 10^{-4})$
μ_3	0.067	(Assumed)	0.080129	$\mathcal{N}(0.073565 \ 8.056 \times 10^{-3})$
σ_3	0.08	[19]	0.091393	$\mathcal{N}(0.085697 \ 8.056 \times 10^{-3})$
γ_1	$\frac{1}{6}$	[18,19]	0.172489	$\mathcal{N}(0.169578 \ 4.117 \times 10^{-3})$
γ_2	0.09	[19]	0.090308	$\mathcal{N}(0.090154 \ 2.18 \times 10^{-4})$
γ_3	0.05	(Assumed)	0.050128	$\mathcal{N}(0.050128 \ 9.1 \times 10^{-5})$
ν_1	0.001	(Assumed)	0.001958	$\mathcal{N}(0.001479 \ 6.77 \times 10^{-4})$
ν_2	0.006	(Assumed)	0.008971	$\mathcal{N}(0.007485 \ 2.101 \times 10^{-3})$
ν_3	0.001	(Assumed)	0.005735	$\mathcal{N}(0.003367 \ 3.3348 \times 10^{-3})$
μ_4	0.08	(Assumed)	0.080625	$\mathcal{N}(0.080313 \ 4.42 \times 10^{-4})$
ρ_1	10	[5]	9.920733	$\mathcal{N}(9.960366 \ 5.605 \times 10^{-2})$
ρ_2	8	(Assumed)	8.116421	$\mathcal{N}(8.058211 \ 8.2322 \times 10^{-2})$
ρ_3	15	(Assumed)	14.917005	$\mathcal{N}(14.958502 \ 5.8686 \times 10^{-2})$
C	0.003 (PFU)/mL	(Assumed)	0.003011	$\mathcal{N}(0.003005 \ 8.0000 \times 10^{-6})$

$\hat{\lambda}_8/(\hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4)$ signifies the probability of rabies in environment causing infection to susceptible humans, $\hat{\lambda}_9/(\hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4)$ denotes the probability of rabies in environment causing infection to susceptible free-range dogs, and $\mu_4/(\hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4)$ denotes the probability of removal of rabies in environment.

The expectation matrix $\mathbb{M}$ of the branching process is a 7×7 matrix, which is defined by Eq. (31). It is derived from the offspring probability generating functions (pgfs) given in Eqs. (24) to (30), with all variables $(u_1, u_2, u_3, u_4, u_5, u_6, u_7) = (1, 1, 1, 1, 1, 1, 1)$:

$$\mathbb{M} = \begin{pmatrix} 0 & 0 & 0 & \frac{\hat{\lambda}_1}{J_1} & 0 & \frac{\hat{\lambda}_5}{J_2} & \frac{\hat{\lambda}_8}{J_3} \\ \frac{\beta_1}{J_4} & \frac{\nu_1}{J_5} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\hat{\lambda}_3}{J_6} & 0 & \frac{\hat{\lambda}_6}{J_7} & \frac{\hat{\lambda}_9}{J_8} \\ 0 & 0 & \frac{\gamma}{(\gamma + \mu_2)} & \frac{\mathbb{G}_1}{J_1} & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\hat{\lambda}_4}{J_1} & 0 & \frac{\hat{\lambda}_4}{J_2} & \frac{\hat{\lambda}_7}{J_3} \\ 0 & 0 & 0 & 0 & \frac{\gamma_1}{(\gamma_1 + \gamma_2 + \mu_3)} & \frac{\mathbb{G}_2}{J_2} & 0 \\ 0 & \frac{\nu_1}{(\nu_1 + \mu_1 + \sigma_1)} & 0 & \frac{\nu_2}{J_1} & 0 & \frac{\nu_3}{J_2} & \frac{\hat{\lambda}_9}{J_3} \end{pmatrix}, \quad (31)$$

where

$$J_1 = \hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \sigma_2 + \mu_2 + \nu_2, \quad J_2 = \hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \sigma_3 + \mu_3 + \nu_3,$$

$$J_3 = \hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4,$$

$$J_4 = \beta_1 + \beta_2 + \mu_1, \quad J_5 = \nu_1 + \sigma_1 + \mu_1, \quad J_6 = \hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \mu_2 + \sigma_2 + \nu_2,$$

$$J_7 = \hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \mu_3 + \sigma_3 + \nu_3, \quad J_8 = \hat{\lambda}_7 + \hat{\lambda}_8 + \hat{\lambda}_9 + \mu_4,$$

$$\mathbb{G}_1 = \hat{\lambda}_1 + \hat{\lambda}_2 + \hat{\lambda}_3 + \nu_2, \quad \mathbb{G}_2 = \hat{\lambda}_4 + \hat{\lambda}_5 + \hat{\lambda}_6 + \nu_3.$$

The Continuous-Time Markov Chain (CTMC) model identifies a stochastic threshold that determines whether rabies will die out or lead to an outbreak in human and dog populations. This threshold is represented by the spectral radius of the expectation matrix, $\rho(\mathbb{M})$. There is a relationship between $\rho(\mathbb{M})$ in the stochastic model and the basic reproduction number $\mathcal{R}_0$ in the deterministic model. For rabies to be eliminated from both human and dog populations, it is required that $\rho(\mathbb{M}) \leq 1$ or $\mathcal{R}_0 < 1$. Conversely, in deterministic models, rabies persists in humans and dogs if $\mathcal{R}_0 > 1$. The relationship between the deterministic and stochastic thresholds for rabies extinction can be expressed as $\mathcal{R}_0 < 1 \iff \rho(\mathbb{M}) < 1$. In stochastic models, when $\rho(\mathbb{M}) < 1$, there is a possibility of either an outbreak or extinction of the *Rabies lyssavirus*, depending on the initial number of infectives at the onset of the disease outbreak. Conversely, if $\rho(\mathbb{M}) > 1$, a fixed point

$$(f_1, f_2, f_3, f_4, f_5, f_6, f_7) \in (0, 1)^6$$

can be determined using offspring generating functions, which are then used to assess the probability of disease extinction. These generating functions are nonlinear, making analytical computation challenging, and thus numerical methods are typically employed for their calculation.

4. Quantitative analysis: Numerical simulations

Following the analytical assessment of both the deterministic and Continuous-Time Markov Chain (CTMC) stochastic frameworks, numerical simulations were conducted to investigate the qualitative dynamics

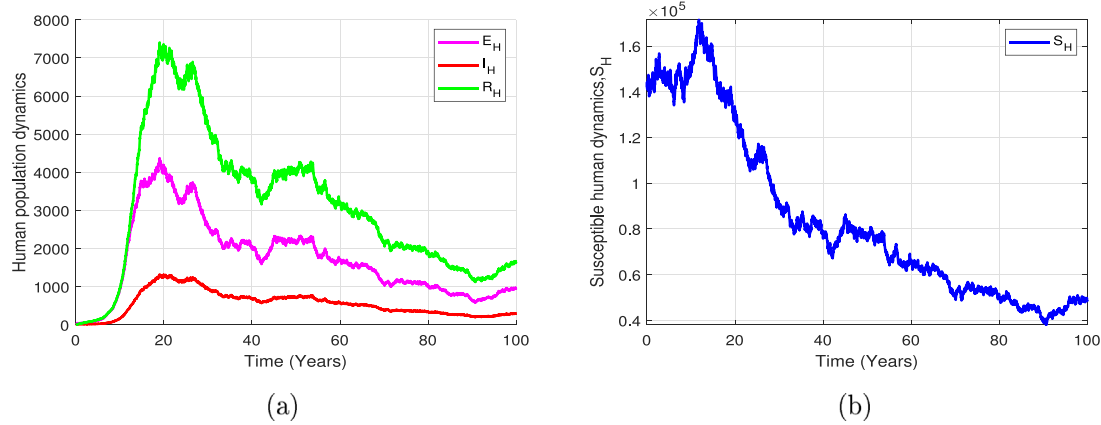


Fig. 2. Stochastic rabies transmission dynamics in humans.

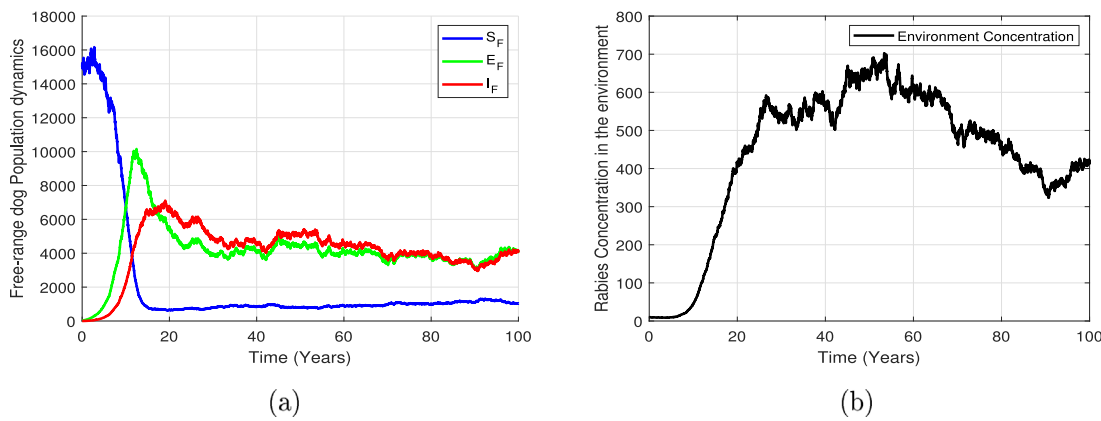


Fig. 3. Stochastic rabies transmission dynamics in (a) Free-range dogs (b) Environment.

of the proposed rabies transmission model. Accurate parameter estimation is essential for generating reliable quantitative forecasts within constrained time frames using real-world epidemiological data [5]. Model parameters in Eq. (1) were estimated using the non-linear least squares method. Synthetic datasets were obtained by numerically integrating equation (1) with a fifth-order Runge–Kutta scheme implemented in MATLAB, employing initial parameter values θ_i from the literature and the initial population conditions:

$$S_H(0) = 142,000, \quad E_H(0) = 40, \quad I_H(0) = 0, \quad R_H(0) = 0,$$

$$S_D(0) = 15,000, \quad E_D(0) = 25, \quad I_D(0) = 0, \quad R_D(0) = 0,$$

$$S_F(0) = 12,500, \quad E_F(0) = 20, \quad I_F(0) = 0, \quad M(0) = 90.$$

The observed data were formalized as a stochastic process:

$$Y_i = RD(t_i, \theta_i) + \eta_i, \quad \eta_i \stackrel{\text{i.i.d.}}{\sim} \mathcal{N}(0, \sigma^2), \quad t_i \in [1, n],$$

where $RD(t_i, \theta_i)$ denotes the model-predicted prevalence and η_i represents Gaussian measurement noise. Parameter estimates were obtained under the assumption that deviations from baseline literature values follow a Gaussian distribution, as reported in Table 2.

The simulation is performed using 10,000 random sample paths, with the results presented graphically alongside the corresponding deterministic numerical solutions for comparative analysis. To conduct the simulations, Euler's method and the Gillespie algorithm are utilized, applying the specified initial conditions $E_H(0) = 10$, $I_H(0) = 5$, $R_H(0) = 0$, $S_H(0) = \frac{\theta_1}{\mu_1} - (E_H(0) + I_H(0) + R_H(0))$, $E_F(0) = 20$, $I_F(0) = 0$, $S_F(0) = \frac{\theta_2}{\mu_2} - (E_F(0) + I_F(0))$, $E_D(0) = 40$, $I_D(0) = 5$, $R_D(0) = 0$, and $S_D(0) = \frac{\theta_3}{\mu_3} - (E_D(0) + I_D(0) + R_D(0))$.

Figs. 2–4 illustrate the stochastic transmission dynamics of rabies between human and dog populations.

Fig. 2(b) illustrates that the number of susceptible humans initially experiences a stochastic decline over the first 40 years, eventually stabilizing at a variable, non-constant level. In contrast, Fig. 2(a) depicts an increase in stochastic fluctuations among the exposed, infected, and recovered human populations during the same period, followed by a decline that approaches a steady, non-zero value. The observed fluctuations in the exposed, infected, and recovered populations likely reflect the effects of control interventions, such as the administration of post-exposure prophylaxis (PEP) to individuals exposed to rabid animals. Fig. 3(a) shows that the number of susceptible free-range dogs initially decreases as the populations of exposed and infected free-range dogs rise stochastically over the first 20 years, eventually stabilizing at a variable, non-constant level. At the same time, Fig. 3(b) indicates increasing stochastic fluctuations in environmental rabies concentration, which subsequently stabilize toward a steady, non-zero level.

Finally, Fig. 4(a) demonstrates that the number of susceptible domestic dogs initially undergoes periodic declines, while the population of exposed, infected, and recovered domestic dogs in Fig. 4(b) increases stochastically over the first 20 years, eventually reaching stability at a variable, non-constant level.

Figs. 5–8 clearly illustrate a stochastic rise in the number of infected individuals among humans, domestic animals, and free-range dogs, while concurrently, there is a decline in the number of susceptible individuals within these populations. Additionally, there is a simultaneous stochastic increase in the concentration of rabies in the environment, driven by variations in contact and shedding rates. These

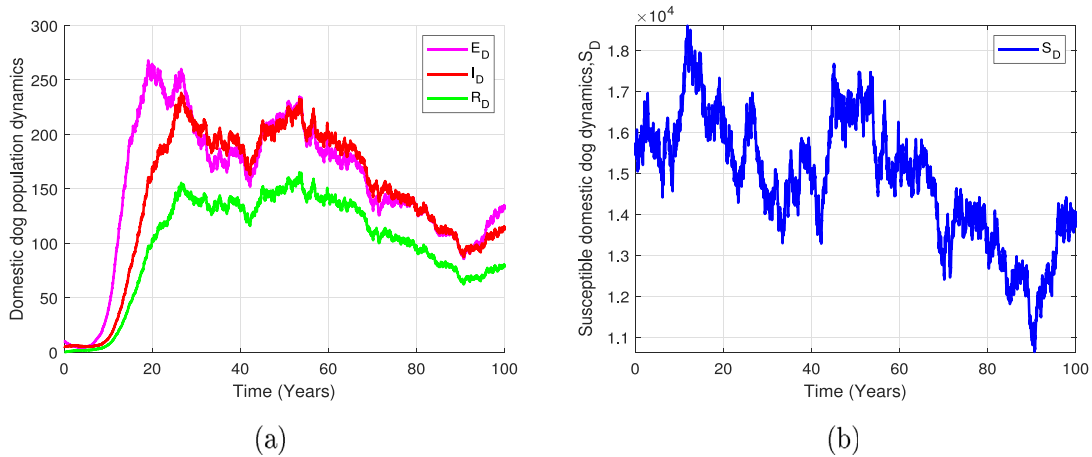


Fig. 4. Stochastic rabies transmission dynamics in Domestic dogs.

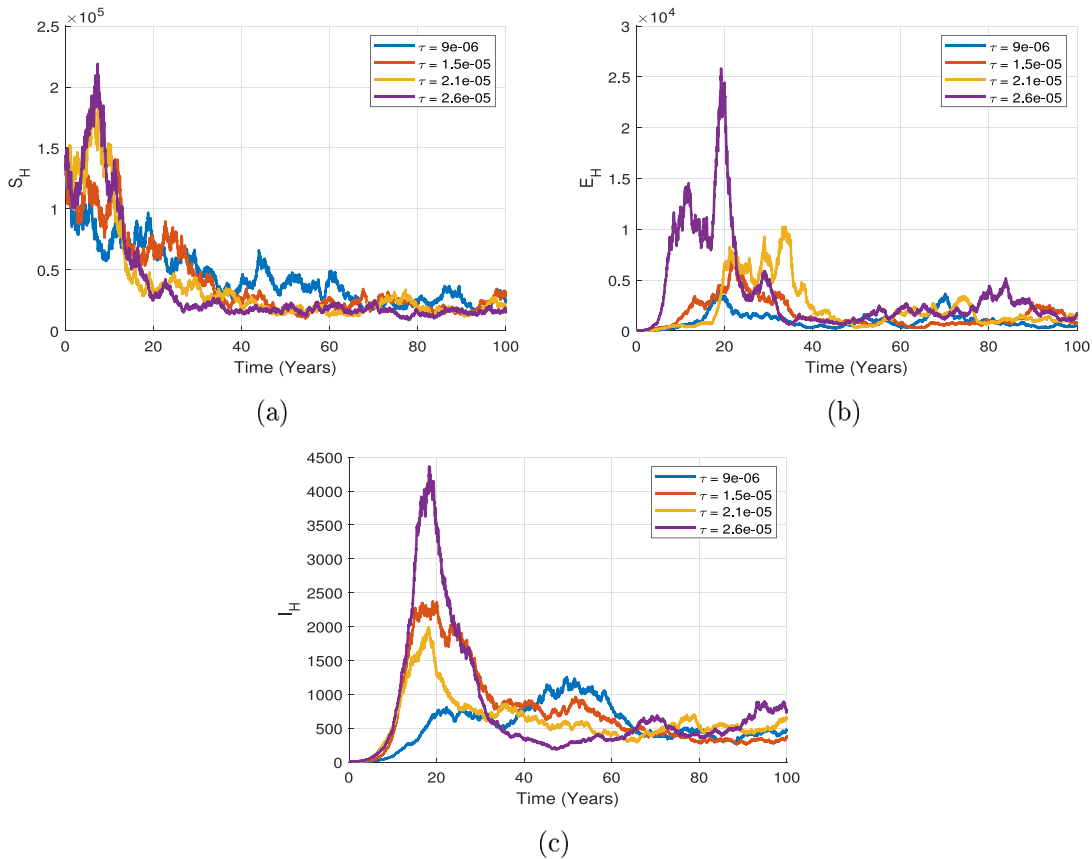


Fig. 5. Stochastic trajectory of human due to impact of contact rate τ .

scenarios provide evidence that the movement of free-roaming dogs could potentially introduce a new rabies infection, suggesting a risk of an outbreak.

Figs. 9–13 present both deterministic and continuous-time Markov chain (CTMC) stochastic results, revealing a comparable trend in rabies transmission dynamics. These figures demonstrate a reduction in susceptible populations following exposure, infection, and recovery events, with stabilization occurring after approximately 20 to 40 years.

Likewise, susceptible humans, free-range dogs, and domestic dogs also experience a decline, ultimately reaching a steady state. Both modeling approaches exhibit a similar general pattern; deterministic results indicate an average trend across CTMC sample paths, while stochastic outputs reflect natural fluctuations. The relationship between the susceptible groups and the exposed, infected, and recovered classes is inversely related. Initially, the populations of exposed, infected, and recovered individuals both human and dogs see a rise, peaking around

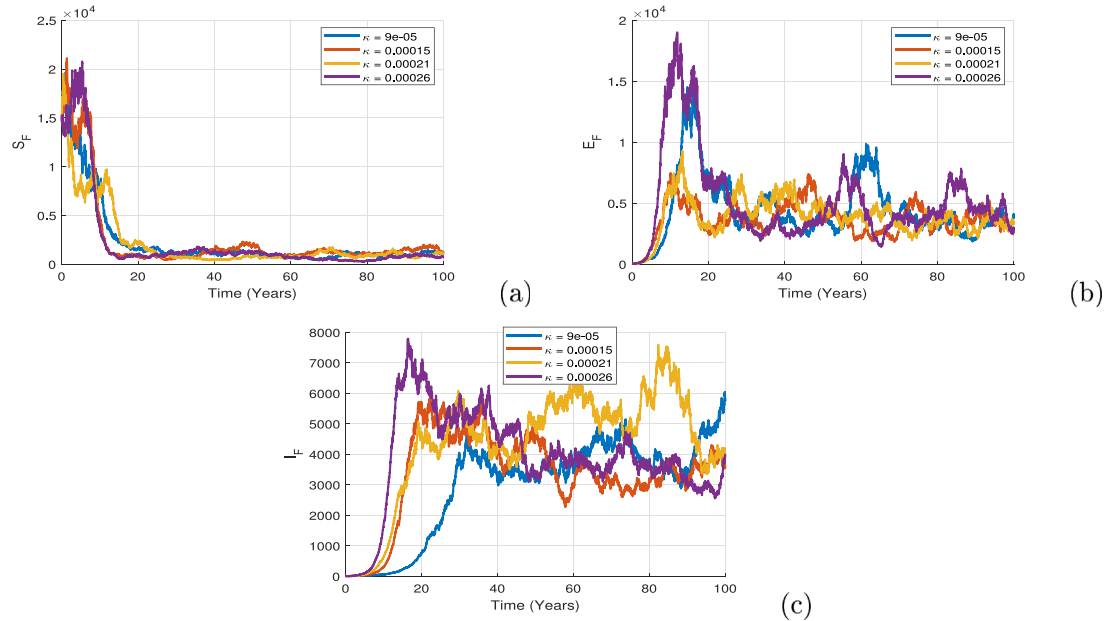


Fig. 6. Stochastic trajectory of free-range dogs due to impact of contact rate κ .

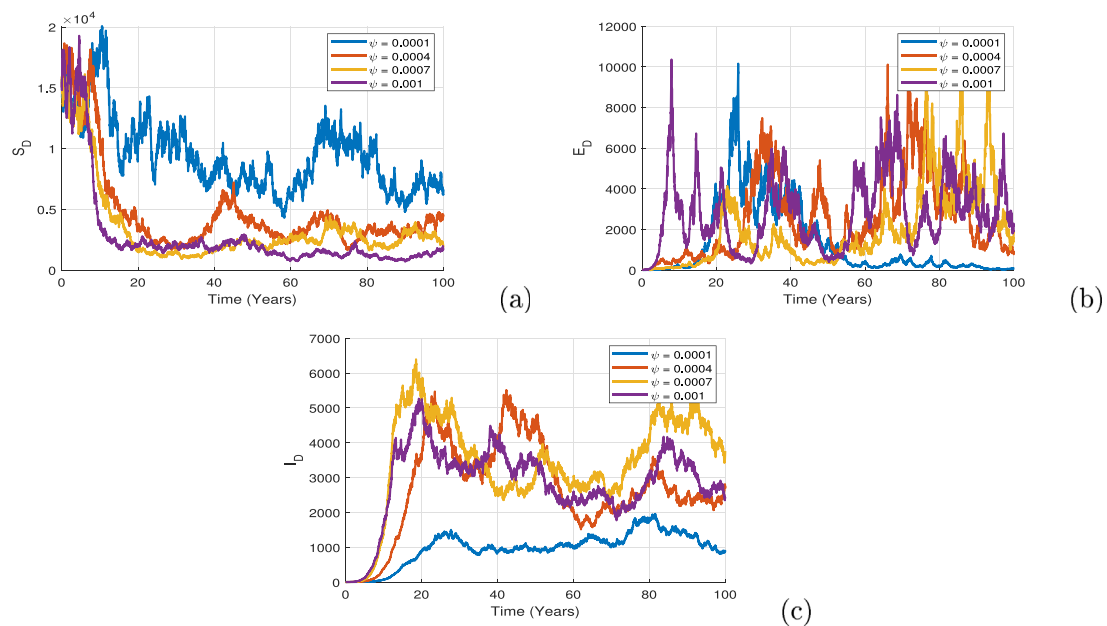


Fig. 7. Stochastic trajectory of domestic dogs due to impact of contact rate ψ .

the first 20 years, followed by a gradual decline that stabilizes by year 30. This pattern suggests that the early increases in infections contribute to herd immunity within populations, ultimately leading to stabilization.

5. Discussion and conclusion

This study presents a comparative analysis of stochastic continuous-time Markov chains (CTMC) and deterministic models to understand rabies persistence in human and dog populations. Using the multitype branching process, the stochastic threshold for rabies persistence is established, offering new insights into how randomness affects disease extinction probabilities. Numerical simulations show that while the stochastic model outcomes closely align with deterministic results,

stochasticity plays a key role in low-infection scenarios. Stochastic models help design flexible control strategies by accounting for uncertainties in disease spread, such as animal behavior or environmental factors (refer to Figs. 2–4). Unlike deterministic models, which assume fixed rates, stochastic models adapt to varying scenarios, like regional differences or population behavior. These strategies include dynamic vaccination programs, real-time monitoring for adjustments, and focusing on high-risk areas for rabies transmission. Long-term planning, informed by continuous data, further refines interventions (refer to Figs. 9–12). Our study provides a policy-driven perspective, advocating for a holistic rabies control approach by considering both predictable trends (deterministic models) and random events (stochastic models) (refer to Figs. 9–13).

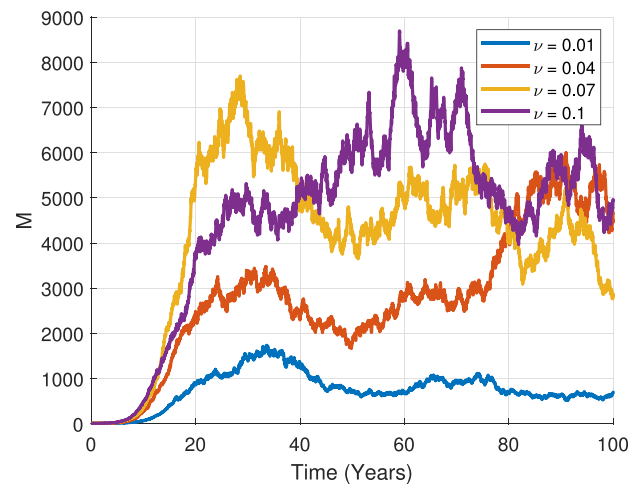


Fig. 8. Stochastic trajectory of Environment due to impact of shedding rate ν .

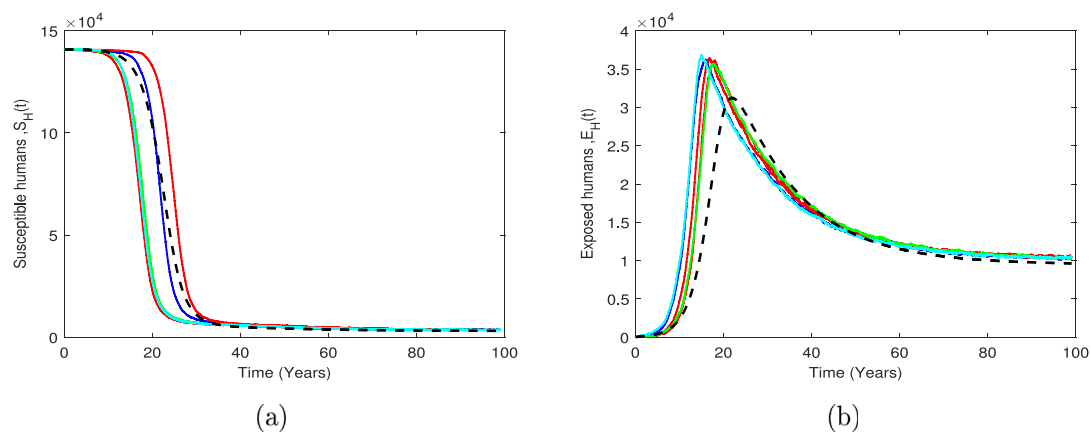


Fig. 9. Comparison of Deterministic (dotted lines) and CTMC Sample Paths for Rabies Transmission Dynamics: (a) Susceptible Humans and (b) Exposed Humans.

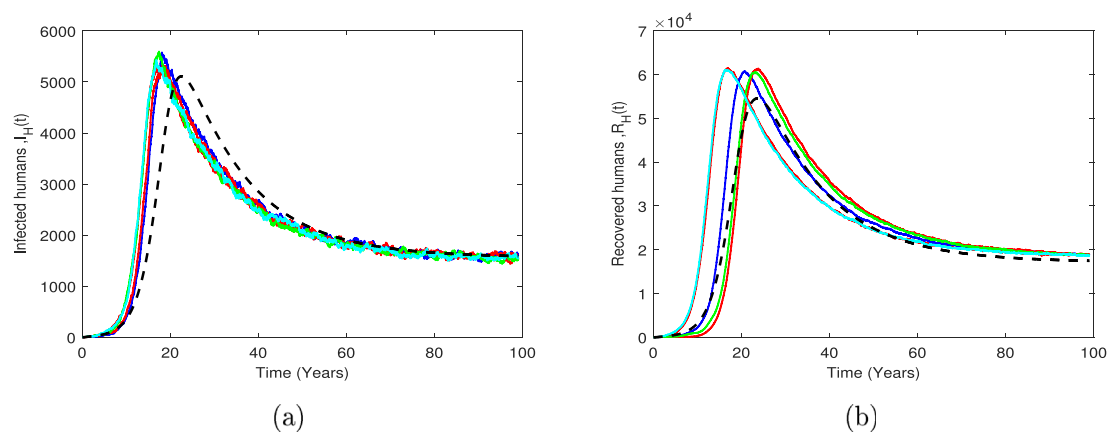


Fig. 10. Comparison of Deterministic (dotted lines) and CTMC Sample Paths for Rabies Transmission Dynamics: (a) Infected Humans and (b) Recovered Humans.

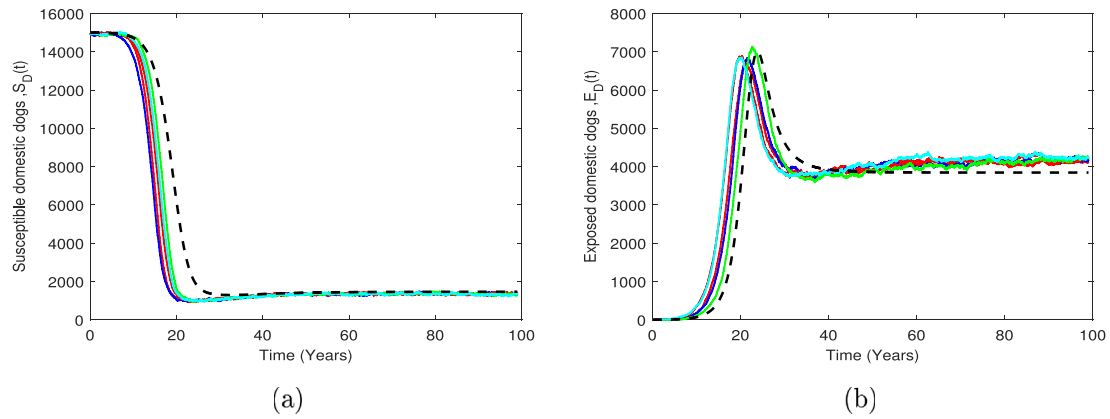


Fig. 11. Comparison of Deterministic (dotted lines) and CTMC Sample Paths for Rabies Transmission Dynamics: (a) Susceptible domestic dogs and (b) Exposed domestic dogs.

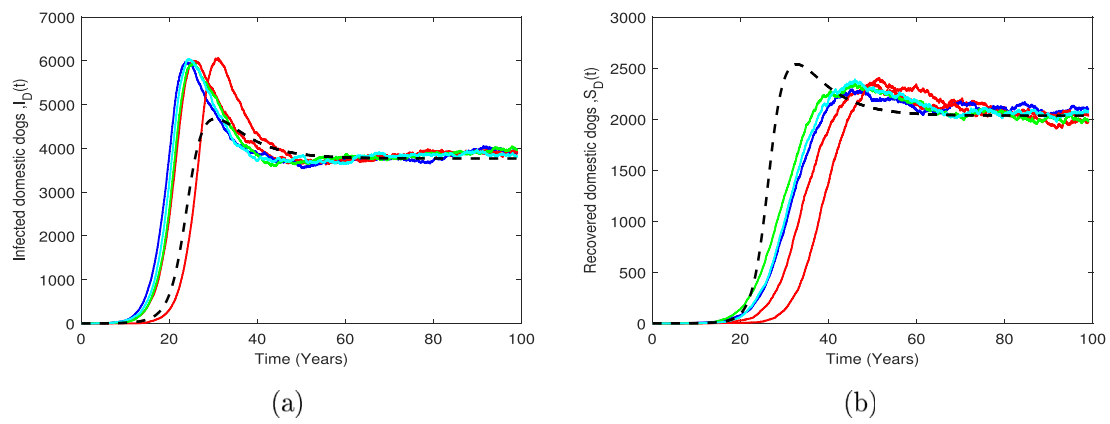


Fig. 12. Comparison of Deterministic (dotted lines) and CTMC Sample Paths for Rabies Transmission Dynamics: (a) Infected domestic dogs and (b) Recovered domestic dogs.

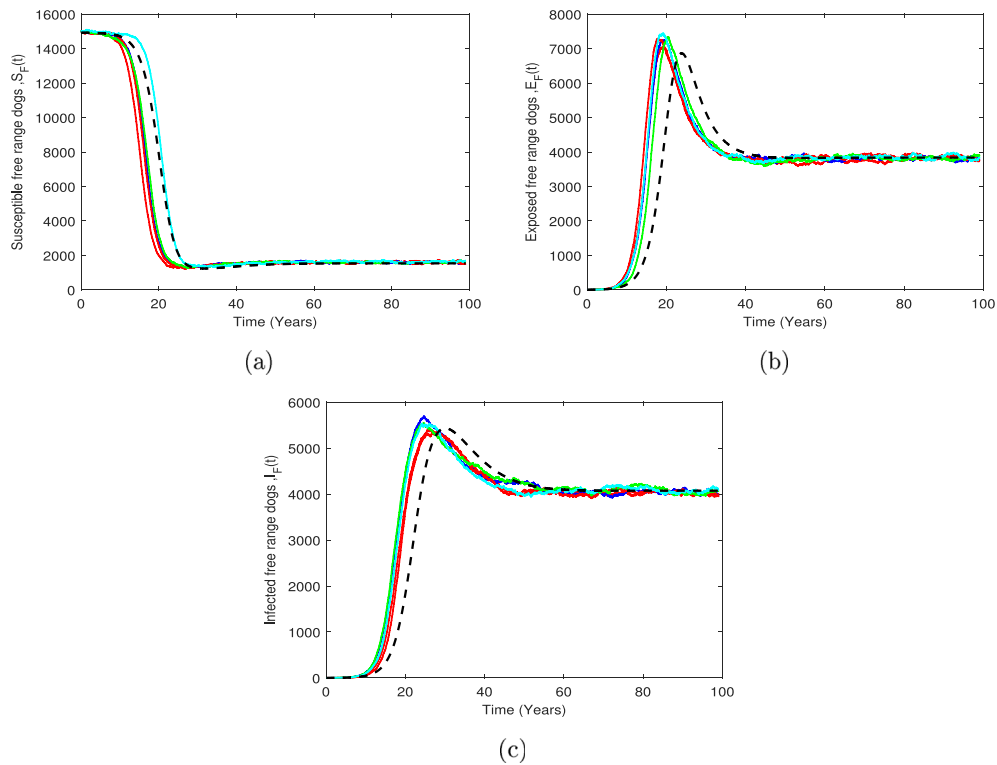


Fig. 13. Comparison of Deterministic (dotted lines) and CTMC Sample Paths for Rabies Transmission Dynamics: (a) Susceptible free-range dogs (b) Exposed free-range dogs and (c) Infected free-range dogs.

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CRediT authorship contribution statement

Mfano Charles: Writing – original draft, Visualization, Software, Resources, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **G.A. Lyakurwa:** Writing – review & editing, Validation, Supervision. **Delfim F.M. Torres:** Writing – review & editing, Visualization, Supervision, Software, Resources, Methodology, Formal analysis, Data curation, Conceptualization. **Verdiana G. Masanja:** Writing – review & editing, Visualization, Supervision, Software, Formal analysis.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix. Global stability of the endemic equilibrium point E^*

Here we prove the result stated at the end of Section 3.2.

Theorem 5. *The endemic equilibrium point E^* of the rabies model (1) is globally asymptotically stable whenever $\mathcal{R}_0 \geq 1$.*

Proof. To prove Theorem 5, we adopt the approach of [5,24] by constructing a Lyapunov function of the form

$$\mathcal{H} = \sum_{i=1}^n D_i \left(U_i - U_i^* + U_i^* \ln \left(\frac{U_i}{U_i^*} \right) \right), D_i > 0 \text{ for } i = 1, 2, 3, \dots, n,$$

where D_i represents a positive constant that needs to be determined, U_i stands for the population variable at compartment i , and U_i^* denotes the equilibrium point of the rabies model at compartment i for $i \in \{1, 2, 3, \dots, 12\}$. Therefore, we define the Lyapunov $\mathcal{H}$ for model system (1) as follows:

$$\mathcal{H} = \begin{cases} D_1 \left(S_H - S_H^* + S_H \ln \left(\frac{S_H}{S_H^*} \right) \right) + D_2 \left(E_H - E_H^* + E_H \ln \left(\frac{E_H}{E_H^*} \right) \right) + D_3 \left(I_H - I_H^* + I_H \ln \left(\frac{I_H}{I_H^*} \right) \right) \\ + D_4 \left(R_H - R_H^* + R_H \ln \left(\frac{R_H}{R_H^*} \right) \right) + D_5 \left(S_F - S_F^* + S_F \ln \left(\frac{S_F}{S_F^*} \right) \right) + D_6 \left(E_F - E_F^* + E_F \ln \left(\frac{E_F}{E_F^*} \right) \right) \\ + D_7 \left(I_F - I_F^* + I_F \ln \left(\frac{I_F}{I_F^*} \right) \right) + D_8 \left(S_D - S_D^* + S_D \ln \left(\frac{S_D}{S_D^*} \right) \right) + D_9 \left(E_D - E_D^* + E_D \ln \left(\frac{E_D}{E_D^*} \right) \right) \\ + D_{10} \left(I_D - I_D^* + I_D \ln \left(\frac{I_D}{I_D^*} \right) \right) + D_{11} \left(R_D - R_D^* + R_D \ln \left(\frac{R_D}{R_D^*} \right) \right) + D_{12} \left(M - M^* + M \ln \left(\frac{M}{M^*} \right) \right). \end{cases} \quad (\text{A.1})$$

Evaluating Eq. (A.1) at the endemic equilibrium point E^* gives

$$\mathcal{H} = \mathbb{E}^* (S_H^*, E_H^*, I_H^*, R_H^*, S_F^*, E_F^*, I_F^*, S_D^*, E_D^*, I_D^*, R_D^*, M^*) = 0.$$

Then, using the time derivative of the Lyapunov function $\mathcal{H}$ in Eq. (A.1) gives

$$\frac{d\mathcal{H}}{dt} = \begin{cases} = D_1 \left(1 - \frac{S_H^*}{S_H} \right) \frac{dS_H}{dt} + D_2 \left(1 - \frac{E_H^*}{E_H} \right) \frac{dE_H}{dt} + D_3 \left(1 - \frac{I_H^*}{I_H} \right) \frac{dI_H}{dt} + D_4 \left(1 - \frac{R_H^*}{R_H} \right) \frac{dR_H}{dt} \\ + D_5 \left(1 - \frac{S_F^*}{S_F} \right) \frac{dS_F}{dt} + D_6 \left(1 - \frac{E_F^*}{E_F} \right) \frac{dE_F}{dt} + D_7 \left(1 - \frac{I_F^*}{I_F} \right) \frac{dI_F}{dt} + D_8 \left(1 - \frac{S_D^*}{S_D} \right) \frac{dS_D}{dt} \\ + D_9 \left(1 - \frac{E_D^*}{E_D} \right) \frac{dE_D}{dt} + D_{10} \left(1 - \frac{I_D^*}{I_D} \right) \frac{dI_D}{dt} + D_{11} \left(1 - \frac{R_D^*}{R_D} \right) \frac{dR_D}{dt} + D_{12} \left(1 - \frac{M^*}{M} \right) \frac{dM}{dt}. \end{cases} \quad (\text{A.2})$$

Consider the endemic equilibrium point E^* of Eq. (1) such that

$$\left\{ \begin{array}{l} \theta_1 = (\tau_1 I_F^* + \tau_2 I_D^* + \tau_3 \lambda(M^*)) S_H^* + \mu_1 S_H^* - \beta_3 R_H^*, \quad \mu_1 + \beta_1 + \beta_2 = \frac{(\tau_1 I_F^* + \tau_2 I_D^* + \tau_3 \lambda(M^*)) S_H^*}{E_H^*}, \\ \sigma_1 + \mu_1 = \frac{\beta_1 E_H^*}{I_H^*}, \quad \beta_3 + \mu_1 = \frac{\beta_2 E_H^*}{R_H^*}, \quad \theta_2 = (\kappa_1 I_F^* + \kappa_2 I_D^* + \kappa_3 \lambda(M^*)) S_F^* + \mu_2 S_F^*, \\ \mu_2 + \gamma = \frac{(\kappa_1 I_F^* + \kappa_2 I_D^* + \tau_3 \lambda(M^*)) S_F^*}{E_F^*}, \quad \sigma_2 + \mu_2 = \frac{\gamma E_F^*}{I_F^*}, \\ \theta_3 = \left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} + \frac{\psi_3 \lambda(M^*)}{1 + \rho_3} \right) S_D^* + \mu_3 S_D^* - \gamma_3 R_D^*, \quad \mu_3 + \gamma_1 + \gamma_2 = \frac{\left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} + \frac{\psi_3 \lambda(M^*)}{1 + \rho_3} \right) S_D^*}{E_D^*}, \\ \sigma_3 + \mu_3 = \frac{\gamma_1 E_D^*}{I_D^*}, \quad \gamma_3 + \mu_3 = \frac{\gamma_2 E_D^*}{R_D^*}, \quad \mu_4 = \frac{(\nu_1 I_H^* + \nu_2 I_F^* + \nu_3 I_D^*)}{M^*}. \end{array} \right. \quad (\text{A.3})$$

Then, by substituting (A.3) into (1), we have

$$\frac{dH}{dt} = \left\{ \begin{array}{l} D_1 \left(1 - \frac{S_H^*}{S_H} \right) (\theta_1 + \beta_3 R_H - \mu_1 S_H - \chi_1) + D_2 \left(1 - \frac{E_H^*}{E_H} \right) (\chi_1 - (\mu_1 + \beta_1 + \beta_2) E_H) \\ + D_3 \left(1 - \frac{I_H^*}{I_H} \right) (\beta_1 E_H - (\sigma_1 + \mu_1) I_H) + D_4 \left(1 - \frac{R_H^*}{R_H} \right) (\beta_2 E_H - (\beta_3 + \mu_1) R_H) \\ + D_5 \left(1 - \frac{S_F^*}{S_F} \right) (\theta_2 - \chi_2 - \mu_2 S_F) + D_6 \left(1 - \frac{E_F^*}{E_F} \right) (\chi_2 - (\mu_2 + \gamma) E_F) \\ + D_7 \left(1 - \frac{I_F^*}{I_F} \right) (\gamma E_F - (\mu_2 + \sigma_2) I_F) + D_8 \left(1 - \frac{S_D^*}{S_D} \right) (\theta_3 - \mu_3 S_D - \chi_3 + \gamma_3 R_D) \\ + D_9 \left(1 - \frac{E_D^*}{E_D} \right) (\chi_3 - (\mu_3 + \gamma_1 + \gamma_2) E_D) + D_{10} \left(1 - \frac{I_D^*}{I_D} \right) (\gamma_1 E_D - (\mu_3 + \delta_3) I_D) \\ + D_{11} \left(1 - \frac{R_D^*}{R_D} \right) (\gamma_2 E_D - (\mu_3 + \gamma_3) R_D) + D_{12} \left(1 - \frac{M^*}{M} \right) ((\nu_1 I_H + \nu_2 I_F + \nu_3 I_D) - \mu_4 M). \end{array} \right. \quad (\text{A.4})$$

Using the endemic equilibrium point E^0 described in Eq. (A.3), we simplify Eq. (A.4) as

$$\frac{dH}{dt} = \left\{ \begin{array}{l} D_1 \left(1 - \frac{S_H^*}{S_H} \right) \left((\tau_1 I_F^* + \tau_2 I_D^* + \tau_3 \lambda(M^*)) S_H^* + \mu_1 S_H^* - \beta_3 R_H^* - \mu_1 S_H \right. \\ \quad \left. - (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H + \beta_3 R_H \right) \\ + D_2 \left(1 - \frac{E_H^*}{E_H} \right) \left((\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H - \frac{(\tau_1 I_F^* + \tau_2 I_D^* + \tau_3 \lambda(M^*)) S_H^* E_H}{E_H^*} \right) \\ + D_3 \left(1 - \frac{I_H^*}{I_H} \right) \left(\beta_1 E_H - \frac{\beta_1 E_H^* I_H}{I_H^*} \right) + D_4 \left(1 - \frac{R_H^*}{R_H} \right) \left(\beta_2 E_H - \frac{\beta_2 E_H^* R_H}{R_H^*} \right) \\ + D_5 \left(1 - \frac{S_F^*}{S_F} \right) \left((\kappa_1 I_F^* + \kappa_2 I_D^* + \kappa_3 \lambda(M^*)) S_F^* + \mu_2 S_F^* - \mu_2 S_F \right. \\ \quad \left. - (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F \right) \\ + D_6 \left(1 - \frac{E_F^*}{E_F} \right) \left((\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F - \frac{(\tau_1 I_F^* + \kappa_2 I_D^* + \kappa_3 \lambda(M^*)) S_F^* E_F}{E_F^*} \right) \\ + D_7 \left(1 - \frac{I_F^*}{I_F} \right) \left(\gamma E_F - \frac{\gamma E_F^* I_F}{I_F^*} \right) \\ + D_8 \left(1 - \frac{S_D^*}{S_D} \right) \left(\left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} + \frac{\psi_3 \lambda(M^*)}{1 + \rho_3} \right) S_D^* + \mu_3 S_D^* - \gamma_3 R_D^* \right. \\ \quad \left. - \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3 \lambda(M)}{1 + \rho_3} \right) S_D - \mu_3 S_D + \gamma_3 R_D \right) \\ + D_9 \left(1 - \frac{E_D^*}{E_D} \right) \left(\left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3 \lambda(M)}{1 + \rho_3} \right) S_D - \frac{\left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} + \frac{\psi_3 \lambda(M^*)}{1 + \rho_3} \right) S_D^* E_D}{E_D^*} \right) \\ + D_{10} \left(1 - \frac{I_D^*}{I_D} \right) \left(\gamma_1 E_D - \frac{\gamma_1 E_D^* I_D}{I_D^*} \right) + D_{11} \left(1 - \frac{R_D^*}{R_D} \right) \left(\gamma_2 E_D - \frac{\gamma_2 E_D^* R_D}{R_D^*} \right) \\ + D_{12} \left(1 - \frac{M^*}{M} \right) \left(\nu_1 I_H + \nu_2 I_F + \nu_3 I_D - \frac{(\nu_1 I_H^* + \nu_2 I_F^* + \nu_3 I_D^*) M}{M^*} \right). \end{array} \right. \quad (\text{A.5})$$

Then, Eq. (A.5) can be expressed as follows:

$$\frac{dH}{dt} = \left[\begin{aligned} & -D_1\mu_1 S_H \left(1 - \frac{S_H^*}{S_H}\right)^2 + D_1\tau_1 S_H I_F \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{I_F^* S_H^*}{I_F S_H} - 1\right) + D_1\tau_2 S_H I_D \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{I_D^* S_H^*}{I_D S_H} - 1\right) \\ & + D_1\tau_3 S_H \lambda(M) \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{\lambda(M^*) S_H^*}{\lambda(M) S_H} - 1\right) + D_1\beta_3 R_H \left(1 - \frac{S_H^*}{S_H}\right) \left(1 - \frac{R_H^*}{R_H}\right) \\ & + D_2\tau_1 S_H I_F \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{I_F^* S_H^* E_H}{I_F S_H E_H^*}\right) + D_2\tau_2 S_H I_D \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{I_D^* S_H^* E_H}{I_D S_H E_H^*}\right) \\ & + D_2\tau_3 S_H \lambda(M) \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{\lambda(M^*) S_H^* E_H}{\lambda(M) S_H E_H^*}\right) \\ & + D_3\beta_1 E_H \left(1 - \frac{I_H^*}{I_H}\right) \left(1 - \frac{E_H^* I_H}{E_H I_H^*}\right) + D_4\beta_2 E_H \left(1 - \frac{R_H^*}{R_H}\right) \left(-\frac{E_H^* R_H}{E_H R_H^*}\right) \\ & - D_5\mu_2 S_F \left(1 - \frac{S_F^*}{S_F}\right)^2 + D_5\kappa_1 S_F I_F \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{I_F^* S_F^*}{I_F S_F} - 1\right) + D_5\kappa_2 S_F I_D \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{I_D^* S_F^*}{I_D S_F} - 1\right) \\ & + D_5\kappa_3 S_F \lambda(M) \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{\lambda(M^*) S_F^*}{\lambda(M) S_F} - 1\right) + D_6\kappa_1 S_F I_F \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{I_F^* S_F^* E_F}{I_F S_F E_F^*}\right) \\ & + D_6\kappa_2 S_F I_D \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{I_D^* S_F^* E_F}{I_D S_F E_F^*}\right) \\ & + D_6\kappa_3 S_F \lambda(M) \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{\lambda(M^*) S_F^* E_F}{\lambda(M) S_F E_F^*}\right) + D_7\gamma E_F \left(1 - \frac{I_F^*}{I_F}\right) \left(1 - \frac{E_F^* I_F}{E_F I_F^*}\right) \\ & - D_8\mu_3 S_D \left(1 - \frac{S_D^*}{S_D}\right)^2 + \frac{\psi_1 S_D I_F D_8}{(1 + \rho_1)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{I_F^* S_D^*}{I_F S_D} - 1\right) + \frac{\psi_2 S_D I_F D_8}{(1 + \rho_2)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{I_D^* S_D^*}{I_D S_D} - 1\right) \\ & + \frac{\psi_3 S_D \lambda(M) D_8}{(1 + \rho_3)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{\lambda(M^*) S_D^*}{\lambda(M) S_D} - 1\right) + D_8\gamma_3 R_D \left(1 - \frac{S_D^*}{S_D}\right) \left(1 - \frac{R_D^*}{R_D}\right) \\ & + \frac{\psi_1 S_D I_F D_9}{(1 + \rho_1)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{I_F^* S_D^* E_D}{I_F S_D E_D^*}\right) + \frac{\psi_2 S_D I_F D_9}{(1 + \rho_2)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{I_D^* S_D^* E_D}{I_D S_D E_D^*}\right) \\ & + \frac{\psi_3 S_D I_F D_9}{(1 + \rho_3)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{\lambda(M^*) S_D^* E_D}{\lambda(M) S_D E_D^*}\right) \\ & + D_{10}\gamma_1 E_D \left(1 - \frac{I_D^*}{I_D}\right) \left(1 - \frac{E_D^* I_D}{E_D I_D^*}\right) + D_{11}\gamma_2 E_D \left(1 - \frac{R_D^*}{R_D}\right) \left(-\frac{E_D^* R_D}{E_D R_D^*}\right) \\ & + D_{12}\nu_1 I_H \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_H^* M}{I_H M^*}\right) + D_{12}\nu_2 I_F \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_F^* M}{I_F M^*}\right) + D_{12}\nu_3 I_D \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_D^* M}{I_D M^*}\right). \end{aligned} \right] \quad (A.6)$$

Eq. (A.6) can be written as

$$\frac{dH}{dt} = \mathcal{G} + \mathcal{P},$$

where

$$\begin{aligned} \mathcal{P} = & -D_1\mu_1 S_H \left(1 - \frac{S_H^*}{S_H}\right)^2 - D_5\mu_2 S_F \left(1 - \frac{S_F^*}{S_F}\right)^2 \\ & - D_8\mu_3 S_D \left(1 - \frac{S_D^*}{S_D}\right)^2 \end{aligned}$$

and

$$\mathcal{G} = \left\{ \begin{aligned} & D_1 \tau_1 S_H I_F \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{I_F^* S_H^*}{I_F S_H} - 1\right) + D_1 \tau_2 S_H I_D \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{I_D^* S_H^*}{I_D S_H} - 1\right) \\ & + D_1 \tau_3 S_H \lambda(M) \left(1 - \frac{S_H^*}{S_H}\right) \left(\frac{\lambda(M^*) S_H^*}{\lambda(M) S_H} - 1\right) + D_1 \beta_3 R_H \left(1 - \frac{S_H^*}{S_H}\right) \left(1 - \frac{R_H^*}{R_H}\right) \\ & + D_2 \tau_1 S_H I_F \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{I_F^* S_H^* E_H^*}{I_F S_H E_H^*}\right) + D_2 \tau_2 S_H I_D \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{I_D^* S_H^* E_H^*}{I_D S_H E_H^*}\right) \\ & + D_2 \tau_3 S_H \lambda(M) \left(1 - \frac{E_H^*}{E_H}\right) \left(1 - \frac{\lambda(M^*) S_H^* E_H^*}{\lambda(M) S_H E_H^*}\right) \\ & + D_3 \beta_1 E_H \left(1 - \frac{I_H^*}{I_H}\right) \left(1 - \frac{E_H^* I_H^*}{E_H I_H^*}\right) + D_4 \beta_2 E_H \left(1 - \frac{R_H^*}{R_H}\right) \left(-\frac{E_H^* R_H^*}{E_H R_H^*}\right) \\ & + D_5 \kappa_1 S_F I_F \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{I_F^* S_F^*}{I_F S_F} - 1\right) + D_5 \kappa_2 S_F I_D \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{I_D^* S_F^*}{I_D S_F} - 1\right) \\ & + D_5 \kappa_3 S_F \lambda(M) \left(1 - \frac{S_F^*}{S_F}\right) \left(\frac{\lambda(M^*) S_F^*}{\lambda(M) S_F} - 1\right) + D_6 \kappa_1 S_F I_F \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{I_F^* S_F^* E_F^*}{I_F S_F E_F^*}\right) \\ & + D_6 \kappa_2 S_F I_D \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{I_D^* S_F^* E_F^*}{I_D S_F E_F^*}\right) \\ & + D_6 \kappa_3 S_F \lambda(M) \left(1 - \frac{E_F^*}{E_F}\right) \left(1 - \frac{\lambda(M^*) S_F^* E_F^*}{\lambda(M) S_F E_F^*}\right) + D_7 \gamma E_F \left(1 - \frac{I_F^*}{I_F}\right) \left(1 - \frac{E_F^* I_F^*}{E_F I_F^*}\right) \\ & + \frac{\psi_1 S_D I_F D_8}{(1 + \rho_1)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{I_F^* S_D^*}{I_F S_D} - 1\right) + \frac{\psi_2 S_D I_F D_8}{(1 + \rho_2)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{I_D^* S_D^*}{I_D S_D} - 1\right) \\ & + \frac{\psi_3 S_D \lambda(M) D_8}{(1 + \rho_3)} \left(1 - \frac{S_D^*}{S_D}\right) \left(\frac{\lambda(M^*) S_D^*}{\lambda(M) S_D} - 1\right) + D_8 \gamma_3 R_D \left(1 - \frac{S_D^*}{S_D}\right) \left(1 - \frac{R_D^*}{R_D}\right) \\ & + \frac{\psi_1 S_D I_F D_9}{(1 + \rho_1)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{I_F^* S_D^* E_D^*}{I_F S_D E_D^*}\right) + \frac{\psi_2 S_D I_F D_9}{(1 + \rho_2)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{I_D^* S_D^* E_D^*}{I_D S_D E_D^*}\right) \\ & + \frac{\psi_3 S_D I_F D_9}{(1 + \rho_3)} \left(1 - \frac{E_D^*}{E_D}\right) \left(1 - \frac{\lambda(M^*) S_D^* E_D^*}{\lambda(M) S_D E_D^*}\right) \\ & + D_{10} \gamma_1 E_D \left(1 - \frac{I_D^*}{I_D}\right) \left(1 - \frac{E_D^* I_D^*}{E_D I_D^*}\right) + D_{11} \gamma_2 E_D \left(1 - \frac{R_D^*}{R_D}\right) \left(-\frac{E_D^* R_D^*}{E_D R_D^*}\right) \\ & + D_{12} \nu_1 I_H \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_H^* M^*}{I_H M^*}\right) + D_{12} \nu_2 I_F \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_F^* M^*}{I_F M^*}\right) + D_{12} \nu_3 I_D \left(1 - \frac{M^*}{M}\right) \left(1 - \frac{I_D^* M^*}{I_D M^*}\right). \end{aligned} \right. \quad (\text{A.7})$$

To simplify (A.7), let

$$\begin{aligned} a &= \frac{S_H}{S_H^*}, \quad b = \frac{E_H}{E_H^*}, \quad c = \frac{I_H}{I_H^*}, \quad d = \frac{R_H}{R_H^*}, \quad e = \frac{S_F}{S_F^*}, \quad f = \frac{E_F}{E_F^*}, \quad g = \frac{I_F}{I_F^*}, \\ h &= \frac{S_D}{S_D^*}, \quad r = \frac{E_D}{E_D^*}, \quad n = \frac{I_D}{I_D^*}, \quad m = \frac{\lambda(M)}{\lambda(M^*)}, \quad l = \frac{R_D}{R_D^*}, \quad \text{and } k = \frac{M}{M^*}. \end{aligned}$$

We express Eq. (A.7) as

$$\mathcal{Q} = \left\{ \begin{aligned} & \tau_1 S_H I_F \left(1 - \frac{b}{ac} + \frac{1}{ac} - \frac{1}{b}\right) + \tau_2 S_H I_D \left(\frac{1}{an} - 1 + \frac{1}{a^2 n} + \frac{1}{a}\right) + \tau_3 S_H \lambda(M) \left(\frac{1}{am} - 1 + \frac{1}{a^2 m} + \frac{1}{a}\right) \\ & + \beta_3 R_H \left(1 - \frac{1}{d} - \frac{1}{a} + \frac{1}{ad}\right) + \tau_1 S_H I_F \left(1 - \frac{b}{af} - \frac{1}{b} + \frac{1}{af}\right) + \tau_2 S_H I_D \left(1 - \frac{b}{an} - \frac{1}{b} + \frac{1}{an}\right) \\ & + \tau_3 S_H \lambda(M) \left(1 - \frac{b}{af} - \frac{1}{b} + \frac{1}{af}\right) + \tau_2 S_H I_D \left(1 - \frac{b}{am} - \frac{1}{b} + \frac{1}{am}\right) + \beta_1 E_H \left(1 - \frac{b}{c} - \frac{1}{c} + \frac{b}{c^2}\right) \\ & + \beta_2 E_H \left(1 - \frac{b}{d} - \frac{1}{d} + \frac{b}{d^2}\right) + \kappa_1 S_F I_F \left(\frac{1}{ef} - 1 - \frac{1}{e^2 f} + \frac{1}{e}\right) + \kappa_2 S_F I_D \left(\frac{1}{en} - 1 - \frac{1}{e^2 n} + \frac{1}{e}\right) \\ & + \kappa_3 S_F \lambda(M) \left(\frac{1}{em} - 1 - \frac{1}{e^2 m} + \frac{1}{e}\right) + \kappa_1 S_F I_F \left(1 - \frac{f}{en} - \frac{1}{f} + \frac{1}{en}\right) + \kappa_2 S_F I_D \left(1 - \frac{f}{en} - \frac{1}{f} + \frac{1}{en}\right) \\ & + \kappa_3 S_F \lambda(M) \left(1 - \frac{f}{me} - \frac{1}{f} + \frac{1}{me}\right) + \gamma E_F \left(1 - \frac{g}{f} - \frac{1}{f} + \frac{g}{f^2}\right) + \frac{\psi_1 S_D I_F}{(1 + \rho_1)} \left(1 - \frac{1}{h} - \frac{1}{h^2 g} + \frac{1}{h}\right) \\ & + \frac{\psi_2 S_D I_F}{(1 + \rho_2)} \left(1 - \frac{1}{h} - \frac{1}{h^2 g} + \frac{1}{h}\right) + \frac{\psi_3 S_D \lambda(M)}{(1 + \rho_3)} \left(\frac{1}{mh} - 1 - \frac{1}{h^2 m} + \frac{1}{h}\right) + \gamma_3 R_D \left(1 - \frac{1}{l} - \frac{1}{h} + \frac{1}{hl}\right) \\ & + \frac{\psi_1 S_D I_F}{(1 + \rho_1)} \left(1 - \frac{r}{hg} - \frac{1}{r} + \frac{1}{hg}\right) + \frac{\psi_2 S_D I_F}{(1 + \rho_2)} \left(1 - \frac{r}{hn} - \frac{1}{r} + \frac{1}{hn}\right) + \frac{\psi_3 S_D I_F}{(1 + \rho_3)} \left(1 - \frac{r}{hm} - \frac{1}{r} + \frac{1}{hm}\right) \\ & + \gamma_1 E_D \left(1 - \frac{l}{r} - \frac{1}{l} + \frac{1}{r}\right) + \gamma_2 E_D \left(1 - \frac{r}{hg} - \frac{1}{r} + \frac{1}{hg}\right) + \nu_1 I_H \left(1 - \frac{k}{c} - \frac{1}{k} + \frac{1}{c}\right) + \nu_2 I_F \left(1 - \frac{k}{g} - \frac{1}{k} + \frac{1}{g}\right) \\ & + \nu_3 I_D \left(1 - \frac{k}{n} - \frac{1}{k} + \frac{1}{n}\right). \end{aligned} \right. \quad (\text{A.8})$$

From Eq. (A.8), we have

$$1 - \frac{1}{d} - \frac{1}{a} + \frac{1}{ad} = \left(1 - \frac{1}{d}\right) + \left(1 - \frac{1}{a}\right) - \left(1 - \frac{1}{ad}\right). \quad (\text{A.9})$$

To proceed, we make use of the following basic inequality: if $\epsilon(y) = 1 - y + \ln y$, then $\epsilon(y) \leq 0$ such that $1 - y \leq -\ln y$ if, and only if, $y > 0$ and, from the concept of geometric mean, Eq. (A.9) is written as

$$\begin{aligned} \left(1 - \frac{1}{d}\right) + \left(1 - \frac{1}{a}\right) - \left(1 - \frac{1}{ad}\right) &\leq -\ln\left(\frac{1}{d}\right) - \ln\left(\frac{1}{a}\right) + \ln\left(\frac{1}{ad}\right) \\ &\leq \ln\left(a \times d \times \frac{1}{ad}\right) = \ln(1) = 0. \end{aligned} \quad (\text{A.10})$$

Following similar procedures as in (A.10), we get

$$1 - \frac{c}{b} - \frac{1}{c} + \frac{1}{b} \leq 0, \quad 1 - \frac{p}{m} - \frac{1}{p} + \frac{1}{m} \leq 0, \quad 1 - \frac{d}{b} - \frac{1}{d} + \frac{1}{b} \leq 0.$$

From Eq. (A.6), the global stability holds only if $\frac{dH}{dt} \leq 0$. Now, if $\mathcal{P} < \mathcal{G}$, then $\frac{dH}{dt}$ will be negative definite, which implies that $\frac{dH}{dt} < 0$ and $\frac{dH}{dt} = 0$ only at the endemic equilibrium point E^* . Hence, by LaSalle's invariance principle [25], any solution to the rabies model (1) which intersects the interior $\mathbb{R}_+^{12}$ limits to E^* is globally asymptotically stable whatever $\mathcal{R}_0 > 1$. $\square$

Data availability

The data used in this study are included in the manuscript.

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Research Article

A Mathematical and Optimal Control Model for Rabies Transmission Dynamics Among Humans and Dogs With Environmental Effects

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This study presents a deterministic model to investigate rabies transmission dynamics, incorporating environmental effects and control strategies using optimal control theory. Qualitative and quantitative analyses reveal that the disease-free equilibrium is stable when the effective reproduction number $\mathcal{R}_e < 1$ and unstable when $\mathcal{R}_e > 1$. Mesh and contour plots illustrate an inverse relationship between $\mathcal{R}_e$ and control strategies, including dog vaccination, health promotion, and postexposure treatment. Increased intervention reduces transmission, while higher contact rates among dogs raise $\mathcal{R}_e$. Numerical simulations with optimal control confirm the effectiveness of integrated strategies. Vaccination and treatment are identified as key interventions for achieving rabies elimination within 5 years.

Keywords: contact rate; deterrence factors; environment; optimal control; rabies

1. Introduction

Rabies is a zoonotic viral disease characterized by nearly 100% mortality in humans once clinical symptoms manifest, rendering it a significant public health threat, particularly in resource-limited settings [1, 2]. Although rabies has been successfully eliminated in parts of the Western Hemisphere through comprehensive control programs, legislative action, and sustained vaccination campaigns, the disease remains endemic in many parts of the world, particularly in Africa and Asia where the burden is disproportionately high [3–5]. In Tanzania, as in many other countries of the Global South, rabies continues to pose a serious public health threat. An estimated 1500 human deaths occur annually, with over 98% resulting from bites by rabid domestic dogs [6]. Surveil-

lance data from 2022 documented more than 26,000 reported bite incidents, with notable clusters in high transmission areas such as Songea, Mkuranga, and Kigoma. These persistent hotspots emphasize the need for intensified and sustained control interventions [7].

One such intervention, a coordinated *One Health* mass dog vaccination campaign conducted in Moshi District in 2018, achieved 74.5% coverage, reaching nearly 30,000 dogs at an estimated cost of just \$1.61 per animal (equivalent to \$0.067 per person protected). This initiative was associated with the complete elimination of reported human and animal rabies cases in the district by April 2019. Comparable modeling studies in regions such as Ngorongoro and Serengeti corroborate that achieving annual vaccination coverage of at least 70% is both cost-

effective and critical for interrupting transmission. Nevertheless, postexposure prophylaxis (PEP) access remains a significant challenge. In Moshi, only 65% of bite victims sought PEP, while studies across northern and southern Tanzania indicate that 20%–30% of exposed individuals failed to initiate treatment. Barriers such as the high cost of treatment (approximately \$100 per full regimen), inconsistent availability, and logistical delays hinder effective response.

The global health community has set the 2030 ambitious plan to eradicate dog-mediated rabies [4, 8]. However, achieving this objective necessitates addressing significant challenges, including limited awareness, vaccine availability, and dog population management [9]. Notably, free-roaming or stray dogs, which are difficult to vaccinate, can perpetuate transmission cycles even in communities where owned dogs are vaccinated [10]. Inadequate healthcare services and underreporting exacerbate the issue. Effectively addressing these challenges requires moving beyond vaccination and comprehending the interaction between humans, dogs, and their environment by integrating strategies necessary to disrupt the transmission cycle between dogs and humans. A pivotal strategy is mass dog vaccination, aiming to reach the threshold of 70% vaccination coverage within the dog population in areas where rabies is prevalent [11, 12]. Public awareness campaigns are also essential for educating communities about rabies prevention and responsible pet ownership, thereby enhancing participation in vaccination programs. Enhancing healthcare provision, particularly preventive treatment after exposure, is vital for preventing rabies in humans following a bite. Additionally, effective dog population management, improved surveillance and reporting systems, increased funding, and political commitment are critical for sustaining rabies control initiatives and advancing the elimination endeavor [13]. For comprehending the dynamics of infectious diseases and its application in rabies research, mathematical models have been instrumental in revealing insights into transmission patterns and the impact of control measures. They also emphasize the need for targeted human–dog contact rates and environmental factors such as urban versus rural settings. A nonlinear mathematical model proposed by [14] to study the dynamics of rabies infection among dogs and in a human population that is exposed to dog bites revealed that it is possible to control rabies disease infection through reducing contacts of humans to infected dogs and dogs to infected dogs, increasing immunization of dogs, and culling of infected dogs. In [15], the most effective rabies vaccination strategies for domestic dog populations with heterogeneous roaming patterns are investigated; in the study, a rabies-spread model was used to simulate a potential outbreak and evaluate various disease control strategies where 27 vaccination strategies were employed based on a complete block design of 50%, 70%, and 90% in four sampled population structures. Simulation scenarios provided some theoretical evidence that targeting subpopulations of dogs for vaccination, based on the roaming behavior of dogs, is more effective and efficient than recommending 70% vaccination campaigns. In [16], an analysis of a time-

delayed rabies model in human and dog populations with controls reveals that time delay influences the endemicity of rabies; it was also observed that in dog populations, vaccination and the birth of puppies are equally effective measures for rabies control. The work [17] uses the SEIR (susceptible, exposed, infection, and recovered) model to study the effect of vaccination and culling on the dynamics of rabies transmission from stray dogs to domestic dogs. The model was constructed by dividing the dog population into two categories: stray dogs and domestic dogs. The results of analytical and numerical studies demonstrated that the annual dog birth rate is a critical factor in influencing the occurrence of rabies.

Despite the existing body of study, critical factors such as the presence of multiple rabies hosts, including free-roaming dogs, domestic dogs, and environmental contributors, have often been overlooked. This gap highlights the urgent need for more comprehensive approaches to rabies control. Motivated by the pressing demand to enhance strategies for managing both emerging and re-emerging rabies outbreaks, this study employs mathematical modeling to optimize intervention efforts, particularly in high-risk areas. It emphasizes the integration of environmental and deterrent factors, which are frequently underrepresented in traditional control theory frameworks.

2. Model Formulation

A mathematical modeling approach to examine rabies transmission among humans, free-ranging, and domestic dogs was put forward by [3]. Here, we administer time-dependent control variables to that model to assess their efficacy in mitigating the transmission of rabies disease in both humans and dogs. The selection of these variables is guided by strategic interventions aimed at decreasing the likelihood of rabies infections in host population, as well as minimizing the shedding rate of rabies virus by dogs within the environment, including both domestic and free-range populations. To achieve optimal control over rabies transmission in humans, domesticated, and free-roaming dogs, we integrate four distinct time-dependent control variables that are meticulously tailored to address the intricacies of this complex epidemiological scenario: $u_1(t)$ measures the effect of promoting good health practices and management (surveillance and monitoring) in reducing the possibility of human and dog infections with rabies, $u_2(t)$ represents the control effort due to vaccination of domestic dogs, $u_3(t)$ measures the impact of educating communities about rabies-related risks and preventive measures against exposure (public awareness and education), and $u_4(t)$ represents treatment efforts for individuals exposed to suspected rabid animals through bites or scratches (PEP). The model of the transmission dynamics of rabies disease in human, free-range dogs, domestic dogs, and environment is developed based on the following assumptions:

- The transmission is due to adequate contact between the susceptible individual and the infectious agent.

- All infectious individuals are subject to both natural and disease-induced mortality.
- All noninfectious individuals are subject to natural death.
- There is a reduced chance for a domestic dog to get the disease due to protection from human beings.
- The recruitment rate for each population is greater than natural mortality.
- Each population is homogeneously mixed; that is, each individual (human or dogs) has an equal chance of getting the disease.
- No PEP or pre-exposure prophylaxis (PREP) is applied for free-range dogs.
- Environment is any material or object that is contaminated by the viruses that cause rabies disease.
- All individuals are recruited at a constant rate.
- Upon recovery, human beings and domestic dogs attain temporary immunity.
- Upon exposure, humans and domestic dogs receive PEP.

Based on the model formulation and underlying assumptions, the flow diagram presented in Figure 1 delineates the transmission dynamics of rabies by integrating the model's structural assumptions, definitions of state variables in Table 1, and parameter specifications.

The dynamics of rabies are summarized by the following system of ordinary differential equations (ODEs):

$$\begin{cases} \dot{S}_H = \theta_1 + \beta_3 R_H - \mu_1 S_H - (1 - (u_1 + u_3))(\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H, \\ \dot{E}_H = (1 - (u_1 + u_3))(\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H - (\mu_1 + \beta_1 + \beta_2 + u_4) E_H, \\ \dot{I}_H = \beta_1 E_H - (\sigma_1 + \mu_1) I_H, \\ \dot{R}_H = (\beta_2 + u_4) E_H - (\beta_3 + \mu_1) R_H, \\ \dot{S}_F = \theta_2 - (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F - \mu_2 S_F, \\ \dot{E}_F = (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F - (\mu_2 + \gamma) E_F, \\ \dot{I}_F = \gamma E_F - (\mu_2 + \sigma_2) I_F, \\ \dot{S}_D = \theta_3 - \mu_3 S_D - (1 - (u_1 + u_2)) \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right) S_D + \gamma_3 R_D, \\ \dot{E}_D = (1 - (u_1 + u_2)) \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right) S_D - (\mu_3 + \gamma_1 + \gamma_2 + u_4) E_D, \\ \dot{I}_D = \gamma_1 E_D - (\mu_3 + \sigma_3) I_D, \\ \dot{R}_D = (\gamma_2 + u_4) E_D - (\mu_3 + \gamma_3) R_D, \\ \dot{M} = (\nu_1 I_H + \nu_2 I_F + \nu_3 I_D) - \mu_4 M, \end{cases} \quad (1)$$

where

$$\lambda(M) = \frac{M}{M + C},$$

subject to initial nonnegative conditions

$$\begin{aligned} S_H(0) &> 0, E_H(0) \geq 0, I_H(0) \geq 0, R_H(0) \geq 0, \\ S_F(0) &> 0, E_F(0) \geq 0, I_F(0) \geq 0, \\ S_D(0) &\geq 0, E_D(0) \geq 0, I_D(0) \geq 0, R_D(0) \geq 0. \end{aligned} \quad (2)$$

3. Qualitative Analysis

In this section, we analyze the rabies model (1) by taking into consideration the model's boundedness as well as the positivity of the solutions. Stability and the equilibrium point are also discussed.

3.1. Positivity of the Solutions and Boundedness of the System. We begin by proving that the model is well-posed.

Lemma 1. All solutions of the system (1) that start in $\Omega \subset \mathbb{R}_+^{12}$ remain positive for all the time.

Proof 1. To establish the existence of a solution for the model given in (1), we begin by considering the initial conditions in (2). Applying the integral operator $\int_0^t (\cdot) ds$ to each equation in the model (1), we derive

$$\begin{cases} S_H(t) - S_H(0) = \int_0^t (\theta_1 + \beta_3 R_H - \mu_1 S_H - \chi_1 S_H) ds, \\ E_H(t) - E_H(0) = \int_0^t (\chi_1 S_H - (\mu_1 + \beta_1 + \beta_2 + u_4) E_H) ds, \\ I_H(t) - I_H(0) = \int_0^t (\beta_1 E_H - (\sigma_1 + \mu_1) I_H) ds, \\ R_H(t) - R_H(0) = \int_0^t ((\beta_2 + u_4) E_H - (\beta_3 + \mu_1) R_H) ds, \\ S_F(t) - S_F(0) = \int_0^t (\theta_2 - \chi_2 S_F - \mu_2 S_F) ds, \\ E_F(t) - E_F(0) = \int_0^t (\chi_2 S_F - (\mu_2 + \gamma) E_F) ds, \\ I_F(t) - I_F(0) = \int_0^t (\gamma E_F - (\mu_2 + \sigma_2) I_F) ds, \\ S_D(t) - S_D(0) = \int_0^t (\theta_3 - \mu_3 S_D - \chi_3 S_D + \gamma_3 R_D) ds, \\ E_D(t) - E_D(0) = \int_0^t (\chi_3 S_D - (\mu_3 + \gamma_1 + \gamma_2 + u_4) E_D) ds, \\ I_D(t) - I_D(0) = \int_0^t (\gamma_1 E_D - (\mu_3 + \sigma_3) I_D) ds, \\ R_D(t) - R_D(0) = \int_0^t ((\gamma_2 + u_4) E_D - (\mu_3 + \gamma_3) R_D) ds, \\ M(t) - M(0) = \int_0^t ((\nu_1 I_H + \nu_2 I_F + \nu_3 I_D) - \mu_4 M) ds, \end{cases} \quad (3)$$

where

$$\begin{aligned} \chi_1 &= (1 - (u_1 + u_3))(\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)), \\ \chi_2 &= (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)), \\ \chi_3 &= (1 - (u_1 + u_2)) \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right). \end{aligned}$$

For convenience, from Equation (3), we define the following functions:

$$\begin{cases} f_1(t, S_H) = \theta_1 + \beta_3 R_H - \mu_1 S_H - \chi_1 S_H, \\ f_2(t, E_H) = \chi_1 S_H - (\mu_1 + \beta_1 + \beta_2 + u_4) E_H, \\ f_3(t, E_H) = \beta_1 E_H - (\sigma_1 + \mu_1) I_H, \\ f_4(t, R_H) = (\beta_2 + u_4) E_H - (\beta_3 + \mu_1) R_H, \\ f_5(t, S_F) = \theta_2 - \chi_2 S_F - \mu_2 S_F, \\ f_6(t, E_F) = \chi_2 S_F - (\mu_2 + \gamma) E_F, \\ f_7(t, I_F) = \gamma E_F - (\mu_2 + \sigma_2) I_F, \\ f_8(t, S_D) = \theta_3 - \mu_3 S_D - \chi_3 S_D + \gamma_3 R_D, \\ f_9(t, E_D) = \chi_3 S_D - (\mu_3 + \gamma_1 + \gamma_2 + u_4) E_D, \\ f_{10}(t, I_D) = \gamma_1 E_D - (\mu_3 + \sigma_3) I_D, \\ f_{11}(t, R_D) = (\gamma_2 + u_4) E_D - (\mu_3 + \gamma_3) R_D, \\ f_{12}(t, M) = (\nu_1 I_H + \nu_2 I_F + \nu_3 I_D) - \mu_4 M. \end{cases} \quad (4)$$

Since $\{S_H, E_H, I_H, R_H, S_F, E_F, I_F, S_D, E_D, I_D, R_D, M\} \in \Omega \subset \mathbb{R}_+^{12}$ and $0 \leq \{S_H, E_H, I_H, R_H, S_F, E_F, I_F, S_D, E_D, I_D, R_D, M\} \leq \mathbb{L}_i, i = 1, 2, \dots, 12$, such that

$$\begin{aligned} & \|f_1(t, S_H(t)) - f_1(t, S_H(t)_1)\| \\ & \leq \|(\theta_1 + \beta_3 R_H(t) - \mu_1 S_H(t) - \chi_1 S_H(t)) \\ & \quad - (\theta_1 + \beta_3 R_H(t)_1 - \mu_1 S_H(t)_1 - \chi_1 S_H(t)_1)\| \\ & \leq \mu_1 \|S_H(t)_1 - S_H(t)\| + \chi_1 \|S_H(t)_1 - S_H(t)\| \\ & \leq (\mu_1 + \chi_1) \|S_H(t)_1 - S_H(t)\| \leq \varepsilon_1 \|S_H(t)_1 - S_H(t)\|. \end{aligned} \quad (5)$$

Using a similar approach as in Equation (5) for functions $f_i, i = 2, 3, \dots, 12$, we obtain that

$$\begin{cases} \|f_2(t, E_H(t)) - f_2(t, E_H(t)_1)\| \leq \varepsilon_2 \|E_H(t)_1 - E_H(t)\|, \\ \|f_3(t, I_H(t)) - f_3(t, I_H(t)_1)\| \leq \varepsilon_3 \|I_H(t)_1 - I_H(t)\|, \\ \|f_4(t, R_H(t)) - f_4(t, R_H(t)_1)\| \leq \varepsilon_4 \|R_H(t)_1 - R_H(t)\|, \\ \|f_5(t, S_F(t)) - f_5(t, S_F(t)_1)\| \leq \varepsilon_5 \|S_F(t)_1 - S_F(t)\|, \\ \|f_6(t, E_F(t)) - f_6(t, E_F(t)_1)\| \leq \varepsilon_6 \|E_F(t)_1 - E_F(t)\|, \\ \|f_7(t, I_F(t)) - f_7(t, I_F(t)_1)\| \leq \varepsilon_7 \|I_F(t)_1 - I_F(t)\|, \\ \|f_8(t, S_D(t)) - f_8(t, S_D(t)_1)\| \leq \varepsilon_8 \|S_D(t)_1 - S_D(t)\|, \\ \|f_9(t, E_D(t)) - f_9(t, E_D(t)_1)\| \leq \varepsilon_9 \|E_D(t)_1 - E_D(t)\|, \\ \|f_{10}(t, I_D(t)) - f_{10}(t, I_D(t)_1)\| \leq \varepsilon_{10} \|I_D(t)_1 - I_D(t)\|, \\ \|f_{11}(t, R_D(t)) - f_{11}(t, R_D(t)_1)\| \leq \varepsilon_{11} \|R_D(t)_1 - R_D(t)\|, \\ \|f_{12}(t, M(t)) - f_{12}(t, M(t)_1)\| \leq \varepsilon_{12} \|M(t)_1 - M(t)\|, \end{cases} \quad (6)$$

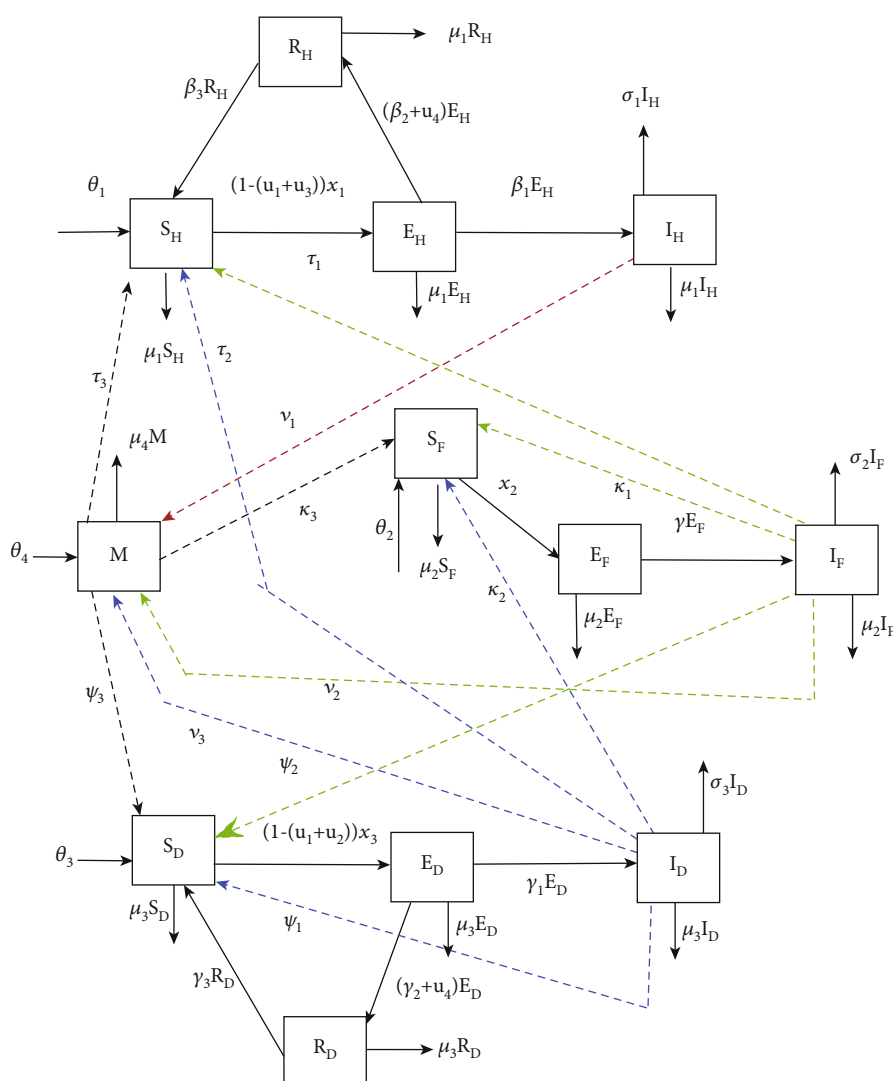


FIGURE 1: Compartmental diagram for rabies transmission with control.

TABLE 1: Model state variables and their descriptions.

Variable	Description
$S_H(t)$	Susceptible human at time t
$E_H(t)$	Exposed human at time t
$I_H(t)$	Infected human at time t
$R_H(t)$	Recovered human at time t
$S_F(t)$	Susceptible free-range dog at time t
$E_F(t)$	Exposed free-range dog at time t
$I_F(t)$	Infected free-range dog at time t
$S_D(t)$	Susceptible domestic dog at time t
$E_D(t)$	Exposed domestic dog at time t
$I_D(t)$	Infected domestic dog at time t
$R_D(t)$	Recovered domestic dog at time t
$M(t)$	Number of rabies virus per unit volume in the environment at time t

where $\varepsilon_1 = \mu_1 + \chi_1$, $\varepsilon_2 = \mu_1 + \beta_1 + \beta_2 + u_4$, $\varepsilon_3 = \sigma_1 + \mu_1$, $\varepsilon_4 = \beta_3 + \mu_1$, $\varepsilon_5 = \mu_2 + \chi_2$, $\varepsilon_6 = \mu_2 + \gamma$, $\varepsilon_7 = \mu_2 + \sigma_1$, $\varepsilon_8 = \mu_3 + \chi_3$, $\varepsilon_9 = \mu_3 + \gamma_1 + \gamma_2 + u_4$, $\varepsilon_{10} = \mu_3 + \sigma_3$, $\varepsilon_{11} = \mu_3 + \gamma_3$, and $\varepsilon_{12} = \mu_4$. This satisfy Lipschitz condition which guarantees the existence of a solution for the model in (1). $\square$

Theorem 1. *All solutions of the system (1) starting in $\mathbb{R}^{12+}$ are uniformly bounded.*

Proof 2. The model system (1) can be divided into subsections as human population, free-range dogs, and domestic dogs as follows:

$$\begin{aligned} & \frac{d(S_H + E_H + I_H + R_H)}{dt} \\ &= \theta_1 + \beta_3 R_H - \mu_1 S_H - (1 - (u_1 + u_3))\chi_1 \\ & \quad + (1 - (u_1 + u_3))\chi_1 - (\mu_1 + \beta_1 + \beta_2 + u_4)E_H \\ & \quad + \beta_1 E_H - (\sigma_1 + \mu_1)I_H + (\beta_2 + u_4)E_H - (\beta_3 + \mu_1)R_H. \end{aligned} \quad (7)$$

Then, Equation (7) becomes

$$\frac{dN_H}{dt} = \theta_1 - (S_H + E_H + I_H + R_H)\mu_1 - \sigma_1 I_H, \quad (8)$$

$$\frac{dN_H}{dt} = \theta_1 - (N_H)\mu_1 - \sigma_1 I_H. \quad (9)$$

We define the integrating factor as

$$N_H(t) = e^{\int_0^t \mu_1 dt} = e^{\mu_1 t}, \quad (10)$$

for $t \rightarrow 0$. Then, Equation (10) is simplified as

$$N_H(0) \leq \frac{\theta_1}{\mu_1} + Ce^{0} N_H(0) - \frac{\theta_1}{\mu_1} \leq C. \quad (11)$$

A simple manipulation of Equation (11) allows us to write that

$$\Omega_H = \left\{ (S_H, E_H, I_H, R_H) \in \mathbb{R}_+^4 : 0 \leq S_H + E_H + I_H + R_H \leq \frac{\theta_1}{\mu_1} \right\}. \quad (12)$$

So, using the same procedure, it can be concluded that

$$\Omega_F = \left\{ (S_F, E_F, I_F) \in \mathbb{R}_+^3 : 0 \leq S_F + E_F + I_F \leq \frac{\theta_2}{\mu_2} \right\},$$

$$\Omega_D = \left\{ (S_D, E_D, I_D, R_D) \in \mathbb{R}_+^4 : 0 \leq S_D + E_D + I_D + R_D \leq \frac{\theta_3}{\mu_3} \right\},$$

$$M(t) \leq \Omega_M = \max \left\{ \frac{\theta_1 v_1}{\mu_1 \mu_4} + \frac{\theta_2 v_2}{\mu_2 \mu_4} + \frac{\theta_3 v_3}{\mu_3 \mu_4}, M(0) \right\},$$

and the solution is biologically and mathematically meaningful. $\square$

3.2. Rabies-Free Equilibrium Point E^0 and the Effective Reproduction Rabies Number $\mathcal{R}_e$. To obtain the rabies

disease-free equilibrium point E^0 , the left-hand side of equation in the model system (1) is set to zero, such that

$$E^0 = \left(\frac{\theta_1}{\mu_1}, 0, 0, 0, \frac{\theta_2}{\mu_2}, 0, 0, \frac{\theta_3}{\mu_3}, 0, 0, 0, 0 \right).$$

The $\mathcal{R}_e$ is obtained by

$$FV^{-1} = \left[\frac{\partial \mathcal{F}_i(E^0)}{\partial t} \right] \left[\frac{\partial \mathcal{V}_i(E^0)}{\partial t} \right]^{-1}, \quad (13)$$

where

$$\mathcal{F}_i = \begin{pmatrix} (1 - (u_1 + u_3))(\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H \\ 0 \\ (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F \\ 0 \\ (1 - (u_1 + u_2)) \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right) S_D \\ 0 \\ 0 \end{pmatrix},$$

$$\mathcal{V}_i = \begin{pmatrix} (\mu_1 + \beta_1 + \beta_2 + u_4) E_H \\ (\sigma_1 + \mu_1) I_H - \beta_1 E_H \\ (\mu_2 + \gamma) E_F \\ (\mu_2 + \sigma_2) I_F - \gamma E_F \\ (\mu_3 + \gamma_1 + \gamma_2 + u_4) E_D \\ (\mu_3 + \delta_3) I_D - \gamma_1 E_D \\ \mu_4 M - (v_1 I_H + v_2 I_F + v_3 I_D) \end{pmatrix}.$$

The Jacobian matrices of $\mathcal{F}_i$ and $\mathcal{V}_i$ at E^0 provide F and V as follows:

$$\frac{\partial \mathcal{F}_i}{\partial y_j} \Big|_{E^0} = F = \begin{pmatrix} 0 & 0 & 0 & \frac{(1 - u_1 - u_3)\tau_1\theta_1}{\mu_1} & 0 & \frac{(1 - u_1 - u_3)\tau_2\theta_1}{\mu_1} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\kappa_2\theta_2}{\mu_2} & 0 & \frac{\kappa_2\theta_2}{\mu_2} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{(1 - u_1 - u_2)\psi_2\theta_3}{(1 + \rho_2)\mu_3} & 0 & \frac{(1 - u_1 - u_2)\psi_1\theta_3}{(1 + \rho_1)\mu_3} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \quad (14)$$

$$\frac{\partial \mathcal{V}_i}{\partial y_j} \Big|_{E^0} = V = \begin{pmatrix} \mu_1 + \beta_1 + \beta_2 + u_4 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\beta_1 & \sigma_1 + \mu_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \mu_2 + \gamma & 0 & 0 & 0 & 0 \\ 0 & 0 & -\gamma & \mu_2 + \sigma_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \mu_3 + \gamma_1 + \gamma_2 + u_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\gamma_1 & \mu_3 + \sigma_3 & 0 \\ 0 & -v_1 & 0 & -v_2 & 0 & -v_3 & \mu_4 \end{pmatrix}. \quad (15)$$

Thus, the maximum of the eigenvalues of (1) gives the effective rabies production number as

$$\mathcal{R}_e = \rho(FV^{-1}) = \frac{R_{33} + R_{21} + \sqrt{R_{21}^2 - 2R_{33}R_{21} + 4R_{31}R_{23} + R_{33}^2}}{2} \quad (16)$$

with

$$\begin{aligned} R_{21} &= \frac{\kappa_1 \theta_2 \gamma}{\mu_2 (\mu_2 + \gamma) (\sigma_2 + \mu_2)}, R_{23} = \frac{\kappa_2 \theta_2 a_3}{\mu_2}, \\ a_3 &= \frac{\gamma}{(\mu_3 + \gamma_1 + \gamma_2 + u_4) (\sigma_3 + \mu_3)}, \\ R_{31} &= \frac{(1 - u_1 - u_2) \psi_1 \theta_3 \gamma}{(1 + \rho_1) \mu_3 (\mu_2 + \gamma) (\sigma_2 + \mu_2)}, \\ R_{33} &= \frac{(1 - u_1 - u_2) \psi_2 \theta_3 a_3}{(1 + \rho_2) \mu_3}. \end{aligned}$$

3.3. Global Stability of Rabies E^0 . The equilibrium behavior of the model at E^0 , as defined in Equation (1), is analyzed using the Metzler matrix framework, following the approaches in [3, 18].

Theorem 2. *The rabies E^0 is globally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable otherwise.*

Proof 3. Let U_s denote the compartments that do not contribute to rabies transmission, while U_i corresponds to those that do. If G_2 is identified as a Metzler matrix (i.e., all off-diagonal elements are nonnegative) and G_0 possesses only

negative real eigenvalues, the rabies-free equilibrium is shown to be globally asymptotically stable. Consequently, the model represented by Equation (1) is reformulated as follows:

$$\begin{cases} \frac{dU_s}{dt} = G_0(U_s - U(E^0)) + G_1 U_i, \\ \frac{dU_i}{dt} = G_2 U_i, \end{cases} \quad (17)$$

where

$$\begin{aligned} G_s - U(E^0) &= \begin{pmatrix} S_H - \frac{\theta_1}{\mu_1} \\ R_H \\ S_F - \frac{\theta_2}{\mu_2} \\ S_D - \frac{\theta_3}{\mu_3} \\ R_D \end{pmatrix}, G_0 = \begin{pmatrix} -\mu & \beta_3 & 0 & 0 & 0 \\ 0 & -(\beta_3 + \mu_1) & 0 & 0 & 0 \\ 0 & 0 & -\mu_2 & 0 & 0 \\ 0 & 0 & 0 & -\mu_3 & \gamma_3 \\ 0 & 0 & 0 & 0 & -(\mu_3 + \gamma_3) \end{pmatrix}, \\ G_1 &= \begin{pmatrix} 0 & 0 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ \beta_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} & 0 \\ 0 & 0 & 0 & \frac{\psi_1 \theta_3}{\mu_3(1 + \rho_1)} & 0 & \frac{\psi_2 \theta_3}{\mu_3(1 + \rho_2)} & 0 \\ 0 & 0 & 0 & 0 & \gamma_2 & 0 & 0 \end{pmatrix}, \end{aligned}$$

and

$$G_2 = \begin{pmatrix} -\mu_1 - \beta_1 - \beta_2 & 0 & 0 & \frac{\tau_1 \theta_1}{\mu_1} & 0 & \frac{\tau_2 \theta_1}{\mu_1} & 0 \\ \beta_1 & -\sigma_1 - \mu_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\mu_2 - \gamma & \frac{\kappa_1 \theta_2}{\mu_2} & 0 & \frac{\kappa_2 \theta_2}{\mu_2} & 0 \\ 0 & 0 & \gamma & -\mu_2 - \sigma_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\psi_1 \theta_3}{\mu_3(1 + \rho_1)} & -\mu_3 - \gamma_1 - \gamma_2 & \frac{\psi_2 \theta_3}{\mu_3(1 + \rho_1)} & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\mu_3 - \sigma_3 & 0 \\ 0 & \nu_1 & 0 & \nu_2 & 0 & \nu_3 & -\mu_4 \end{pmatrix}.$$

Given that the eigenvalues of the matrix E_0 are negative and the off-diagonal entries of the Metzler matrix G_2 are nonnegative, it follows that the rabies equilibrium point E^0 is globally asymptotically stable. $\square$

3.4. Rabies Persistent Equilibrium Point E^* . The point $E^* = (S_H^*, E_H^*, I_H^*, R_H^*, S_F^*, E_F^*, I_F^*, S_D^*, E_D^*, I_D^*, R_D^*, M^*)$ is obtained

by equating Equation (1) to zero:

$$\begin{aligned} \frac{dS_H}{dt} = \frac{dE_H}{dt} = \frac{dI_H}{dt} = \frac{dR_H}{dt} = \frac{dS_F}{dt} = \frac{dE_F}{dt} = \frac{dI_F}{dt} \\ = \frac{dS_D}{dt} = \frac{dE_D}{dt} = \frac{dI_D}{dt} = \frac{dR_D}{dt} = 0. \end{aligned}$$

Upon solving and considering the force infection as

$$\lambda_H^* = \tau_2 I_D^* + \tau_1 I_F^* + \frac{\tau_3 M^*}{M^* + C}, \lambda_F^* = \kappa_1 I_F^* + \kappa_2 I_D^* + \frac{\kappa_3 M^*}{M^* + C},$$

$$\lambda_D^* = (1 - u_1 - u_2) \left(\frac{\psi_1 I_F^*}{1 + \rho_1} + \frac{\psi_2 I_D^*}{1 + \rho_2} + \frac{M^* \psi_3}{(1 + \rho_3)(M^* + C)} \right),$$

the resulting system is

$$\left. \begin{aligned} S_H^* &= \frac{\beta_3(\beta_2(\beta_2 + u_4)E_H^*) - \theta_1(\beta_3 + \mu_1)}{\lambda_H^*(u_1 + u_3 - 1)(\beta_3 + \mu_1)}, I_H^* = \frac{\beta_1 E_H^*}{\sigma_1 + \mu_1}, R_H^* = \frac{(\beta_2 + u_4)E_H^*}{\beta_3 + \mu_1}, \\ E_H^* &= \frac{\theta_1(\beta_3 + \mu_1)}{\mu_1^2 + (u_4 + \beta_1 + \beta_2 + \beta_3)\mu_1 + (u_4 + \beta_1 + \beta_2)\beta_3 - \beta_2(\beta_2 + u_4)}, \end{aligned} \right\} \text{for humans} \quad (18)$$

$$\left. \begin{aligned} S_F^* &= \frac{(-\gamma - \mu_2)E_F^* + \theta_2}{\mu_2}, \lambda_F^* = -\frac{E_F^* \mu_2 (\mu_2 + \gamma)}{(\mu_2 + \gamma)E_F^* - \theta_2}, I_F^* = \frac{\gamma E_F^*}{\mu_2 + \sigma_2}, \end{aligned} \right\} \text{free-range dogs} \quad (19)$$

$$\left. \begin{aligned} S_D^* &= \frac{(-u_4 - \mu_3 - \gamma_1 - \gamma_2)E_D^* + \theta_3}{\mu_3}, \lambda_D^* = -\frac{E_D^* \mu_3 (\mu_3 + \gamma_1 + \gamma_2 + u_4)}{(\mu_3 + \gamma_1 + \gamma_2 + u_4)E_D^* - \theta_3}, \\ I_D^* &= \frac{E_D^* \gamma_1}{\mu_3 + \sigma_3}, R_D^* = \frac{E_D^* (\gamma_2 + u_4)}{\mu_3 + \gamma_3}, M^* = \frac{\gamma_1 E_D^* \nu_3}{\mu_4 (\mu_3 + \sigma_3)} + \frac{\beta_1 E_H^* \nu_1}{\mu_4 (\sigma_1 + \mu_1)} + \frac{\gamma E_F^* \nu_2}{\mu_4 (\mu_2 + \sigma_2)}. \end{aligned} \right\} \text{domestic dogs} \quad (20)$$

Thus, E^* persists if $E_H, E_F, E_D > 0$ and $R_e \geq 1$, as stated in Theorem 3.

Theorem 3. The system model (1) has a unique endemic equilibrium E^* if $\mathcal{R}_e \geq 1$ and $E_H, E_F, E_D > 0$.

The endemic equilibrium point E^* is globally stable. This is proved in Appendix A (see Theorem 7).

3.5. Global Sensitivity Analysis. In this study, we employed Latin hypercube sampling (LHS) and the partial rank correlation coefficient (PRCC) methods to conduct a comprehensive global sensitivity analysis. The LHS technique was used to generate 1000 model parameter runs, incorporating uncertainty by treating these parameters as probabilistic variables uniformly distributed over predefined ranges. Following this, the PRCC approach was utilized to quantify the degree of monotonic relationship between each model input (parameter) and output (state variable), providing insights into how each parameter influences the model dynamics. Let X_i represent the sampled input parameter and Z_i the corresponding model output, with respective means $\bar{X}$ and $\bar{Z}$. The PRCC value r , which denotes the correlation between X_i and Z_i , is computed as follows:

$$r = \frac{\text{Cov}(X_i, Z_i)}{\sqrt{\text{Var}(X_i)\text{Var}(Z_i)}} = \frac{\sum_{i=1}^N (X_i - \bar{X})(Z_i - \bar{Z})}{\sqrt{\sum_{i=1}^N (X_i - \bar{X})^2 \sum_{i=1}^N (Z_i - \bar{Z})^2}}. \quad (21)$$

The PRCC values range within the closed interval $[-1, +1]$, where values closer to either bound indicate a stronger monotonic relationship between input parameters and model outputs. A positive PRCC value ($r > 0$) reflects a direct (increasing) monotonic relationship, suggesting that

an increase in the input parameter leads to a corresponding increase in the output variable. In contrast, a negative PRCC value ($r < 0$) denotes an inverse (decreasing) monotonic relationship, whereby increasing the input parameter results in a decline in the associated output. The results of the sensitivity analysis are visually summarized through histograms presented in Figure 2a,b, and Figure 3a,b, illustrating the evolution of parameter influence over time. Specifically, Figure 2a reveals that the parameters $\theta_1, \tau_1, \tau_2, \kappa_1$, and κ_2 exhibit strong positive correlations with the number of infected humans (I_H), indicating that higher values of these parameters lead to an increased burden of infection in human populations. Moreover, parameters associated with environmental contamination, such as the shedding rates ν_1, ν_2 , and ν_3 , also display positive PRCC values, implying their significant role in enhancing indirect rabies transmission through environmental exposure. On the other hand, parameters such as β_2, ρ_3, ρ_1 , and γ_2 exhibit negative PRCC values with respect to I_H , suggesting that increasing these parameter values contributes to a reduction in the number of infections.

Figure 2b of infected free-range dogs I_F identifies $\kappa_1, \kappa_2, \kappa_3, \theta_2, \theta_3, \nu_1, \nu_2, \nu_3$, and C as significant influences of I_F throughout the epidemic.

Figure 3a,b illustrates the sensitivity of the model outputs to various input parameters. Specifically, Figure 3a shows that the parameters $\kappa_1, \kappa_2, \theta_3, \psi_1, \psi_2, \psi_3, \beta_2$, and θ_2 exhibit positive PRCC values with respect to the number of infected domestic dogs (I_D), indicating that increases in these parameters contribute to a rise in infection levels within the domestic dog population. Conversely, the parameters ρ_1, ρ_2 , and ρ_3 demonstrate negative PRCC values, suggesting that enhancing these parameters may contribute to a reduction in I_D .

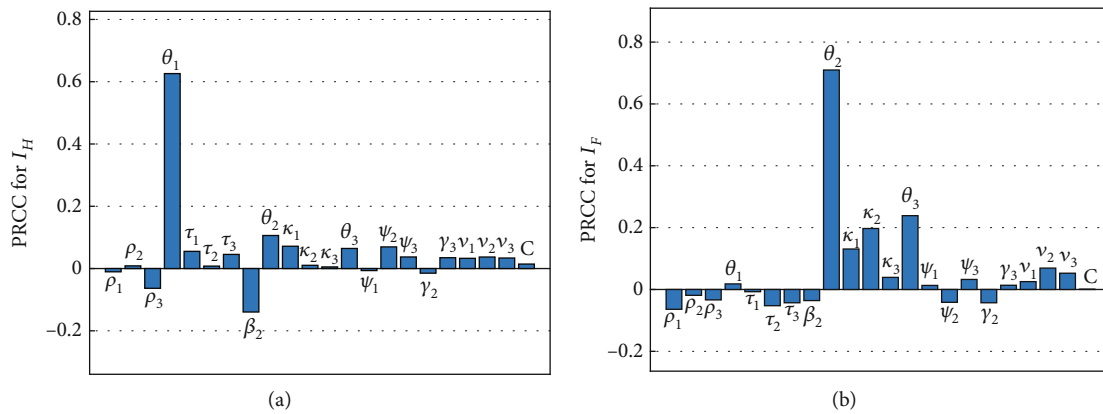


FIGURE 2: The sensitivity analysis of the model outputs: (a) populations of infected humans and (b) infected free-range dogs.

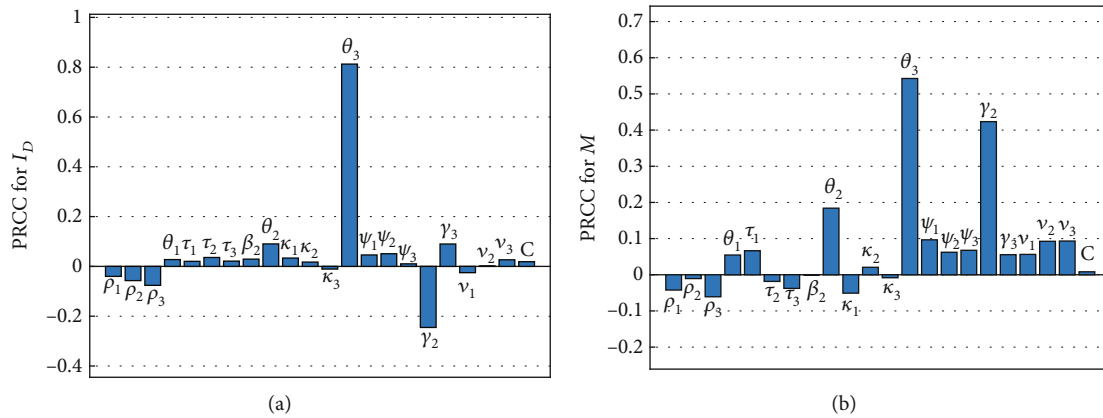


FIGURE 3: The sensitivity analysis of the model outputs: (a) populations of infected domestic dogs and (b) rabies virus in the environment.

Furthermore, Figure 3b highlights the significant influence of the environmental shedding rates v_1 , v_2 , and v_3 on the persistence and amplification of rabies in the environment. These findings underscore the critical role of environmental contamination in the indirect transmission of the disease. Consequently, effective control strategies should prioritize sustained reductions in these key parameters throughout the course of the outbreak to mitigate environmental transmission and disease burden.

3.6. Formulation of the Optimal Control Problem. We aim to reduce the infected human and dog populations while also optimizing the costs linked to the implementation of control strategies. The objective function that reduces the I_H and I_D is given by

$$J(u_j)_{1 \leq j \leq 4} = \min_{(u_1, u_2, u_3, u_4)} \int_{t_0}^{t_f} \left(K_1 M + K_2 E_H + K_3 I_H + K_4 E_D + K_5 I_D - K_6 S_D + \frac{1}{2} \sum_{j=1}^4 A_j u_j^2 \right) dt \quad (22)$$

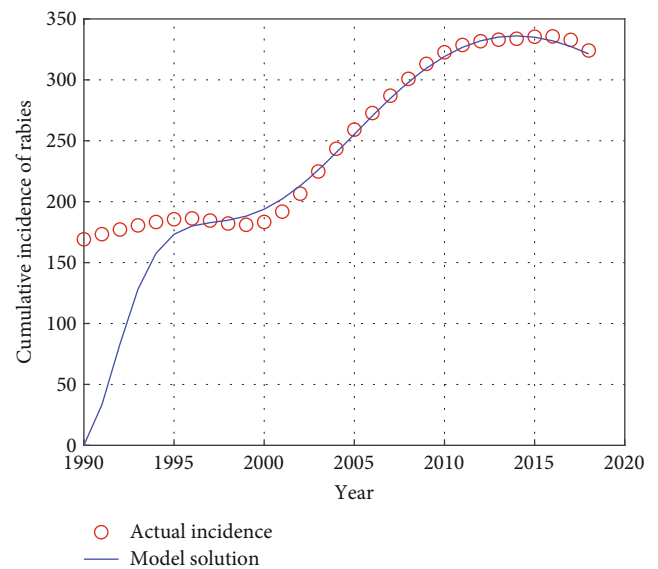


FIGURE 4: The comparison between the reported human rabies incidence in Tanzania from 1990 to 2018 and the simulation of $I_H(t)$ from our model (1).

TABLE 2: Epidemiological parameter data (year⁻¹) for Model 1.

Parameters	Baseline value	Source	Estimated value	Mean(μ) and std(σ)
θ_1	2000	(Estimated)	1993.382113	$\mathcal{N}(1996.691056 \quad 4.4679553)$
τ_1	0.0004	[26]	0.000405	$\mathcal{N}(0.000402 \quad 4 \times 10^{-6})$
τ_2	0.0004	[26]	0.000604	$\mathcal{N}(0.000502 \quad 1.44 \times 10^{-4})$
τ_3	[0.0003 0.0100]	(Estimated)	0.000303	$\mathcal{N}(0.000302 \quad 2 \times 10^{-6})$
β_1	1/6	[26, 27]	0.165581	$\mathcal{N}(0.166124 \quad 7.68 \times 10^{-4})$
ν_3	0.001	(Estimated)	0.005735	$\mathcal{N}(0.003367 \quad 3.3348 \times 10^{-3})$
β_2	[0.54 1]	[26, 27]	0.540487	$\mathcal{N}(0.5402435 \quad 3.7815 \times 10^{-4})$
β_3	1	(Estimated)	0.999301	$\mathcal{N}(0.9996505 \quad 1.6521 \times 10^{-4})$
μ_1	0.0142	[28, 29]	0.014417	$\mathcal{N}(0.014309 \quad 1.53 \times 10^{-4})$
σ_1	1	[16, 27]	1.006332	$\mathcal{N}(1.03166 \quad 4.47 \times 10^{-3})$
θ_2	1000	(Estimated)	1004.12044	(1002.060222 2.913594)
κ_1	0.00006	(Estimated)	0.000020	$\mathcal{N}(0.000040 \quad 2.8 \times 10^{-5})$
κ_2	0.00005	(Estimated)	0.000081	$\mathcal{N}(0.000066 \quad 2.2 \times 10^{-5})$
κ_3	[0.00001 0.00003]	(Estimated)	0.000040	$\mathcal{N}(0.000025 \quad 2.1 \times 10^{-5})$
γ	1/6	[16, 26, 27]	0.166374	$\mathcal{N}(0.166520 \quad 2.07 \times 10^{-4})$
ν_1	0.001	(Estimated)	0.001958	$\mathcal{N}(0.001479 \quad 6.77 \times 10^{-4})$
σ_2	0.09	[27, 30]	0.089556	$\mathcal{N}(0.089778 \quad 3.14 \times 10^{-4})$
μ_4	0.08	(Estimated)	0.080625	$\mathcal{N}(0.080313 \quad 4.42 \times 10^{-4})$
μ_2	0.067	(Estimated)	0.066268	$\mathcal{N}(0.066634 \quad 1.58 \times 10^{-4})$
θ_3	1200	(Estimated)	1203.844461	$\mathcal{N}(1201.922230 \quad 2.718444)$
ψ_1	0.0004	[30, 31]	0.000077	$\mathcal{N}(0.000238 \quad 2.28 \times 10^{-4})$
ψ_2	0.0004	[17]	0.000066	$\mathcal{N}(0.000233 \quad 2.36 \times 10^{-4})$
ψ_3	0.0003	(Estimated)	0.000030	$\mathcal{N}(0.0003 \quad 1.91 \times 10^{-4})$
μ_3	0.067	(Estimated)	0.080129	$\mathcal{N}(0.073565 \quad 8.056 \times 10^{-3})$
σ_3	0.08	[27]	0.091393	$\mathcal{N}(0.085697 \quad 8.056 \times 10^{-3})$
γ_1	1/6	[26, 27]	0.172489	$\mathcal{N}(0.169578 \quad 4.117 \times 10^{-3})$
γ_2	0.09	[27]	0.090308	$\mathcal{N}(0.090154 \quad 2.18 \times 10^{-4})$
γ_3	0.05	(Estimated)	0.050128	$\mathcal{N}(0.050128 \quad 9.1 \times 10^{-5})$
ν_2	0.006	(Estimated)	0.008971	$\mathcal{N}(0.007485 \quad 2.101 \times 10^{-3})$
ρ_1	10	[32]	9.920733	$\mathcal{N}(9.960366 \quad 5.605 \times 10^{-2})$
ρ_2	8	(Estimated)	8.116421	$\mathcal{N}(8.058211 \quad 8.2322 \times 10^{-2})$
ρ_3	15	(Estimated)	14.917005	$\mathcal{N}(14.958502 \quad 5.8686 \times 10^{-2})$
C	0.003 (PFU)/mL	(Estimated)	0.003011	$\mathcal{N}(0.003005 \quad 8.0000 \times 10^{-6})$

subject to the control system (1), where K_1 represents the constant weight for the environment, while K_2 and K_4 are the constant weights for the exposed classes of humans and dogs, respectively. Likewise, K_3 and K_5 are the constant weights for the infectious classes of humans and dogs, respectively, and K_6 is the constant weight for vaccinated domestic dogs. The coef-

ficients A_1, A_2, A_3 , and A_4 are relative cost weights allocated to each specific control measure to transform the integral into the cost occurred over a time frame of t_f years. This duration represents the time period for implementing the controls. In essence, the cost associated with each control scenario is considered to follow a nonlinear quadratic pattern, denoted as

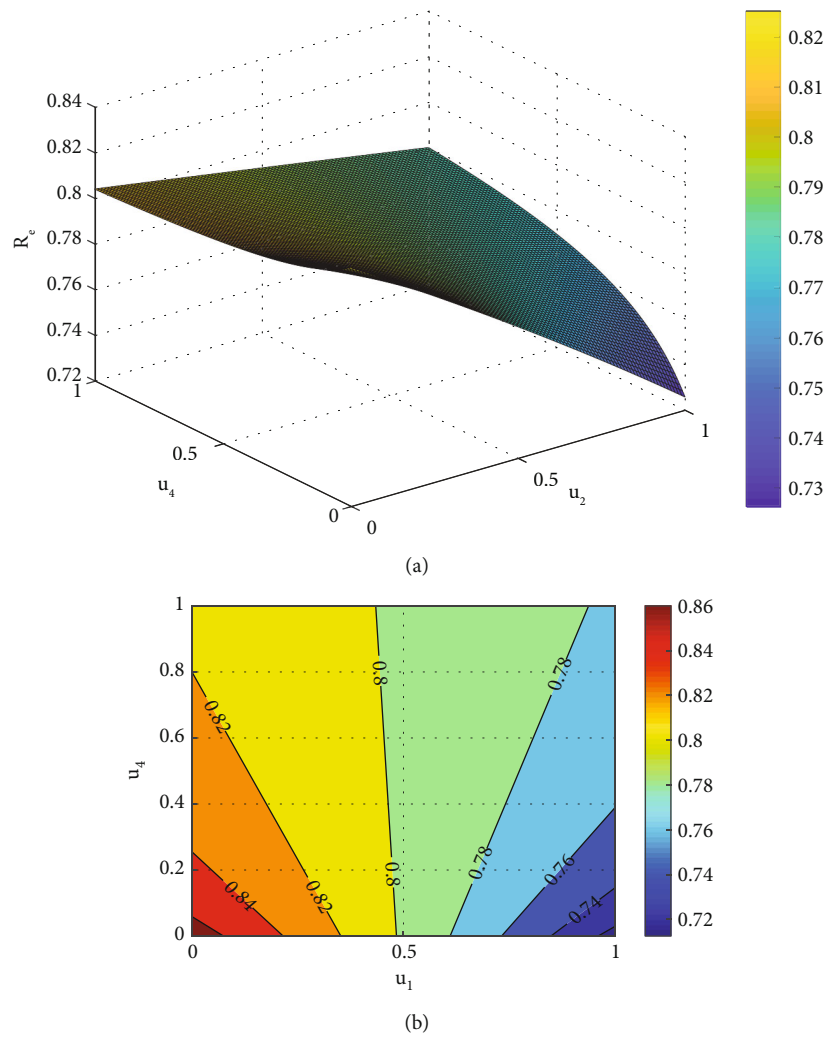


FIGURE 5: Mesh and contour plots showing the combined influence of parameters on $\mathcal{R}_e$. (a) $\mathcal{R}_e$ in terms of u_2 and u_4 . (b) $\mathcal{R}_e$ in terms of u_1 and u_4 .

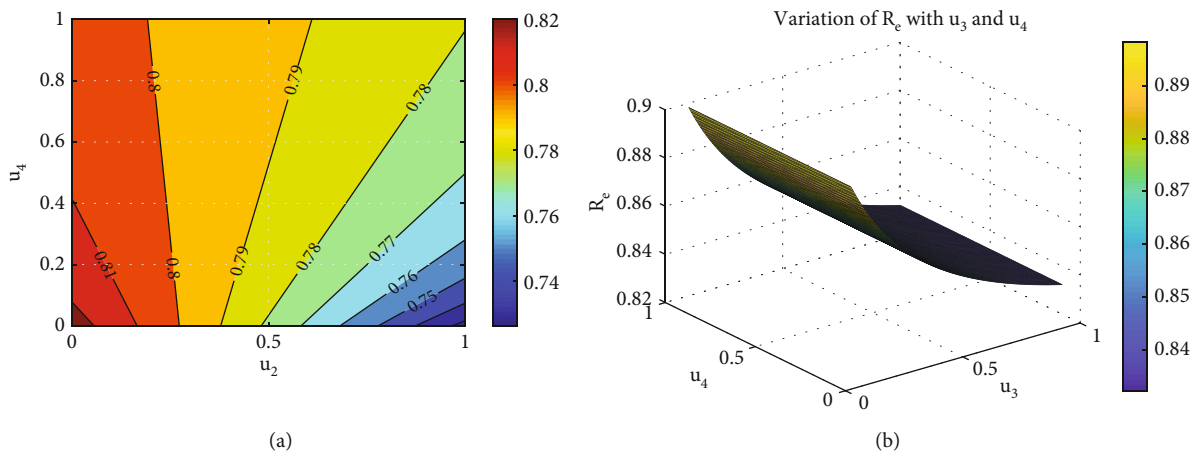


FIGURE 6: Mesh and contour plots showing the combined influence of parameters on $\mathcal{R}_e$. (a) $\mathcal{R}_e$ in terms of u_2 and u_4 . (b) $\mathcal{R}_e$ in terms of u_3 and u_4 .

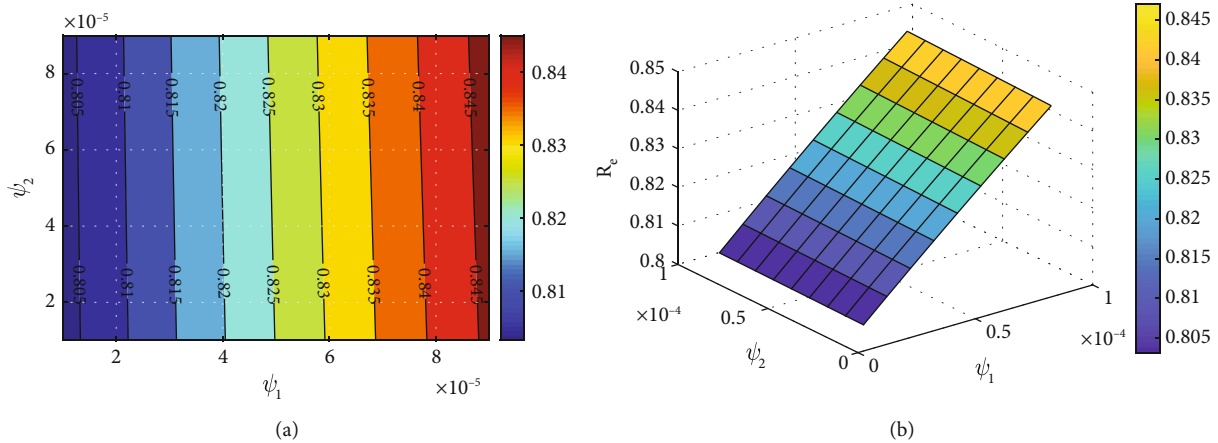


FIGURE 7: Variations in $\mathcal{R}_e$ with respect to changes on (a) the rate that S_D gets infection from I_F and ψ_1 and (b) the rate that S_D gets infection from I_D and ψ_1 .

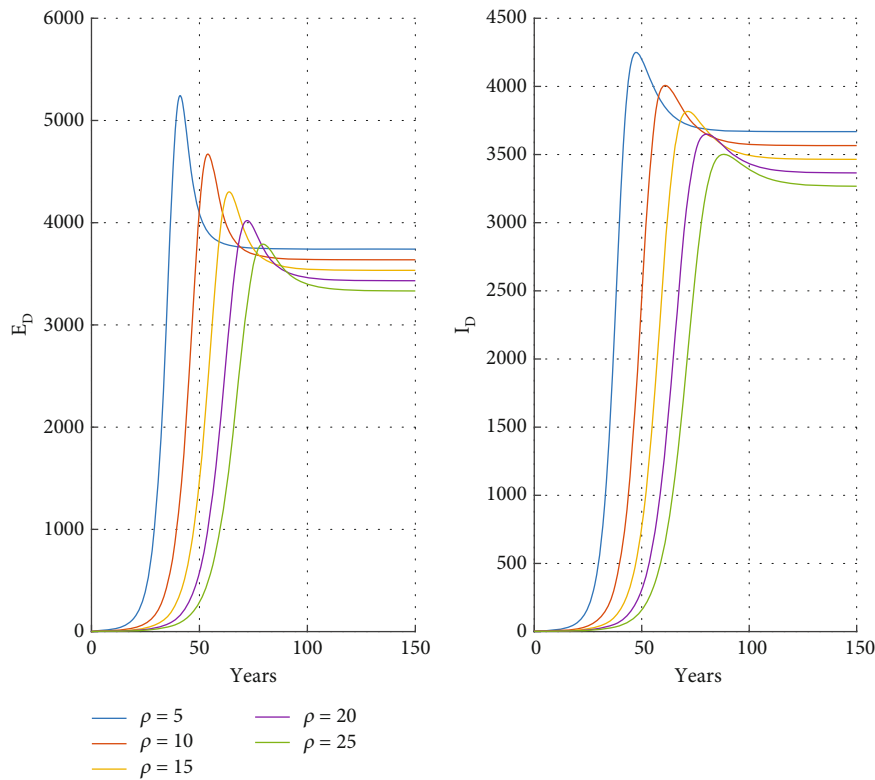


FIGURE 8: Impact of the deterrent factor ρ on the transmission dynamics of rabies in domestic dogs.

$A_1 u_1^2/2$. This cost reflects the expenses incurred in implementing control strategies aimed at promoting environmentally sustainable health practices and management, specifically surveillance and monitoring. Similarly, $A_2 u_2^2/2$ represents the cost of vaccinating domestic dogs, while $A_3 u_3^2/2$ is linked to community education about the risks of rabies exposure and prevention (public awareness and education). Meanwhile, $A_4 u_4^2/2$ pertains to the gradual treatment of infected individuals by potentially rabid animals (PEP). Each

of these costs contributes to the overall expenditure required to address and mitigate the risks associated with rabies.

Considering $J(u_1, u_2, u_3, u_4)$, the primary goal is to minimize the number of exposed and infected for both human and domestic dogs while simultaneously maximizing the number of recovered humans and domestic dogs. Consequently, our aim is to determine u_1^* , u_2^* , u_3^* , and u_4^* that satisfy the condition $J(u_1^*, u_2^*, u_3^*, u_4^*) = \min J(u_1, u_2, u_3, u_4)$, where $U = \{u : u \text{ is measurable and } 0 \leq u_j(t) \leq 1, j = 1, \dots, 4,$

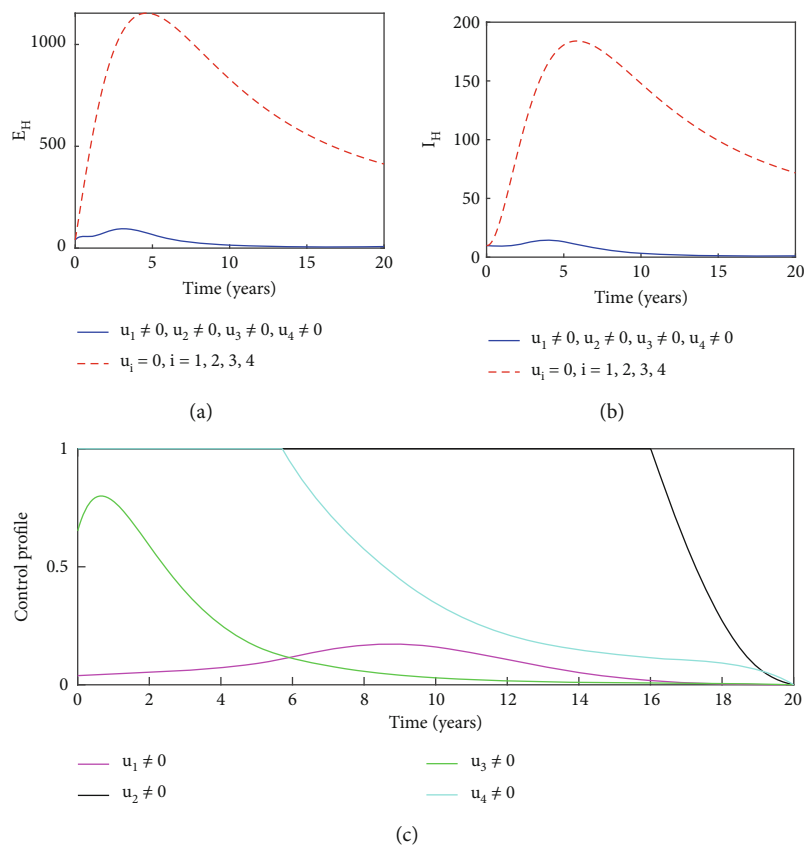


FIGURE 9: (a–c) Impact of Strategy A on human population.

for $t \in [t_0, t_f]$ is the control set, with the state variables $S_H(t)$, $E_H(t)$, $I_H(t)$, $R_H(t)$, $S_F(t)$, $E_F(t)$, $I_F(t)$, $S_D(t)$, $E_D(t)$, $I_D(t)$, $R_D(t)$, and $M(t)$ satisfying the control system (1) and given initial conditions (2).

3.7. Analysis of the Optimal Control Problem. This can be proved by characterizing the optimal control via Pontryagin's maximum principle (PMP), elucidating its essential features and providing a comprehensive understanding of its structure and dynamics.

3.7.1. Existence of a Unique Optimal Control. The solution to our optimal control problem is ensured by applying the results found in [19, 20].

Theorem 4. *The optimal control problem formulated in Section 3.6 admits a solution.*

Proof 4. We want to show that there exists a control $\bar{u} = (\bar{u}_1, \bar{u}_2, \bar{u}_3, \bar{u}_4) \in U$ such that $J(\bar{u}) \leq J(u)$ for all $u = (u_1, u_2, u_3, u_4) \in U$ subject to (1) and (2). This follows from [19, 20] noting that (i) the control system (1) is linear with respect to the control variables; (ii) the control set U is bounded, convex, and closed; and (iii) the integrand of the objective functional (22) is convex in U . $\square$

Theorem 5. *The solution to the optimal control problem formulated in Section 3.6 is unique.*

Proof 5. Assume that there exists optimal controls $\bar{u}_1, \bar{u}_2, \bar{u}_3, \bar{u}_4$ and $\tilde{u}_1, \tilde{u}_2, \tilde{u}_3, \tilde{u}_4$ for the problem of Section 3.6, both achieving the minimum value of the objective functional:

$$J(\bar{u}_1, \bar{u}_2, \bar{u}_3, \bar{u}_4) = J(\tilde{u}_1, \tilde{u}_2, \tilde{u}_3, \tilde{u}_4) = \min_{u_1, u_2, u_3, u_4} J(u_1, u_2, u_3, u_4). \quad (23)$$

Consider the difference between the two optimal controls such that

$$\delta u_j = \bar{u}_j - \tilde{u}_j \text{ for } j = 1, 2, 3, 4.$$

Since both controls are optimal, we have

$$J(\tilde{u}_1, \tilde{u}_2, \tilde{u}_3, \tilde{u}_4) \leq J(\tilde{u}_1 + \delta u_1, \tilde{u}_2 + \delta u_2, \tilde{u}_3 + \delta u_3, \tilde{u}_4 + \delta u_4). \quad (24)$$

Expanding the expression (24), and subtracting $J(\tilde{u}_1, \tilde{u}_2, \tilde{u}_3, \tilde{u}_4)$ from both sides, we get

$$\begin{aligned} & J(\tilde{u}_1 + \delta u_1, \tilde{u}_2 + \delta u_2, \tilde{u}_3 + \delta u_3, \tilde{u}_4 + \delta u_4) - J(\tilde{u}_1, \tilde{u}_2, \tilde{u}_3, \tilde{u}_4) \\ &= \int_{t_0}^{t_f} \left(\sum_{j=1}^4 2A_j \tilde{u}_j \delta u_j + \sum_{j=1}^4 A_j \delta u_j^2 \right) dt. \end{aligned} \quad (25)$$

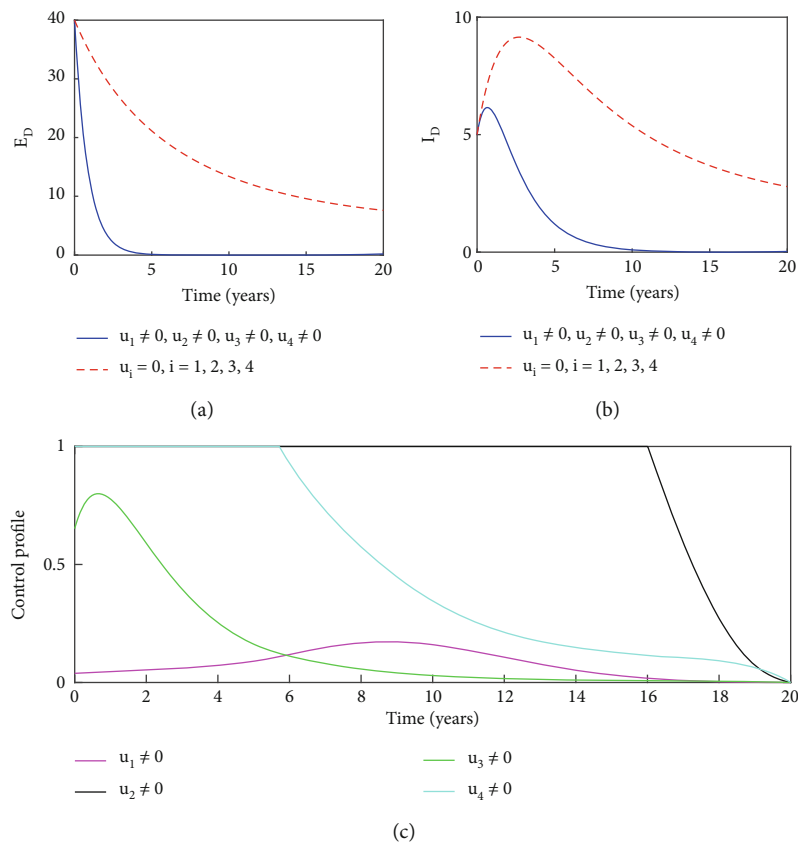


FIGURE 10: (a-c) Impact of Strategy A on domestic dog population.

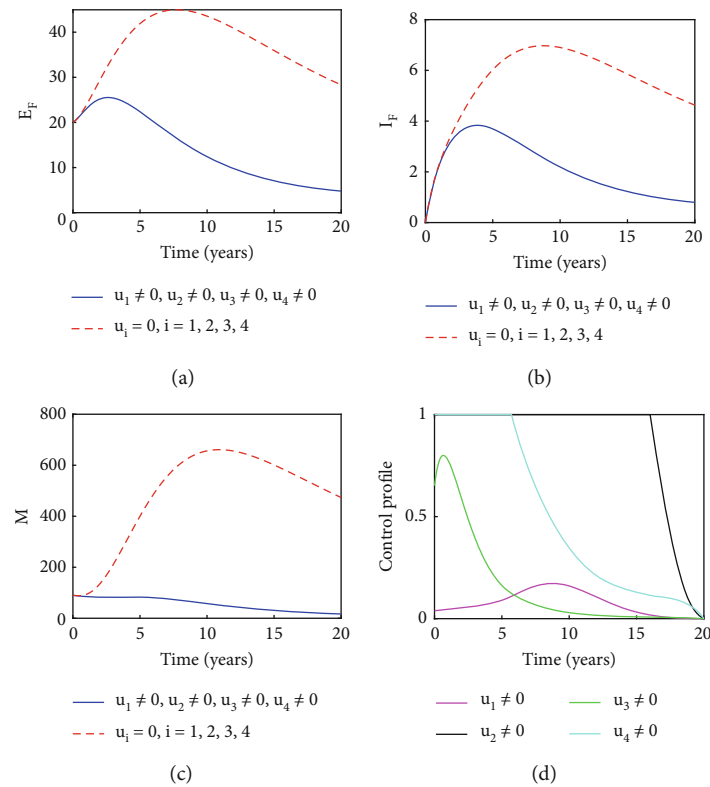


FIGURE 11: (a-d) Impact of Strategy A on free-range dogs and rabies virus.

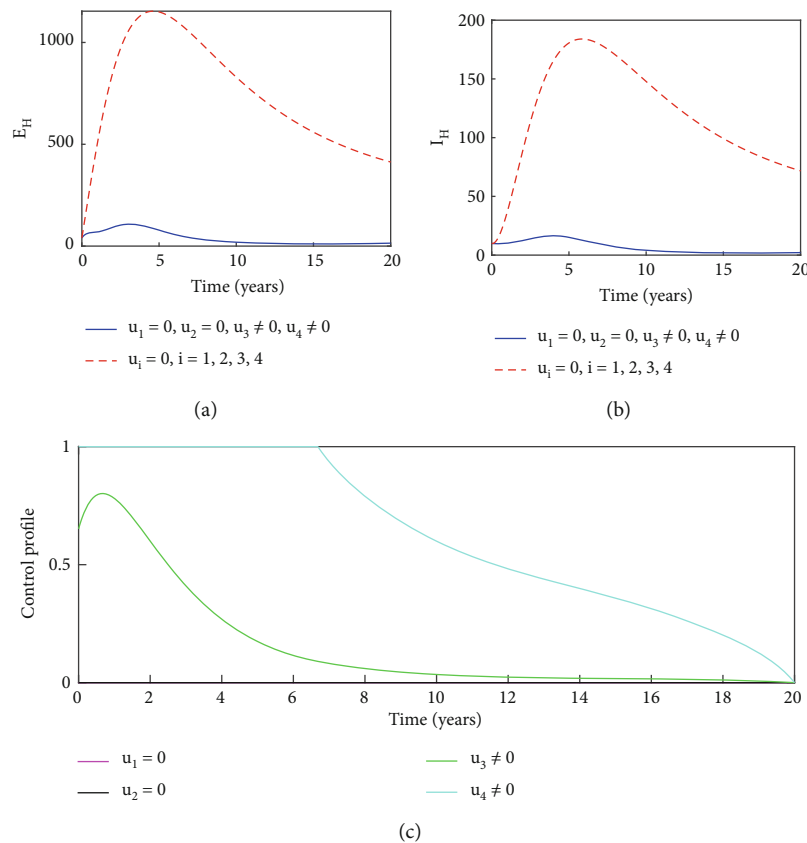


FIGURE 12: (a–c) Impact of Strategy B on human population.

Since all terms in expression (25) have nonnegative integrands,

$$0 \leq \int_{t_0}^{t_f} \left(\sum_{j=1}^4 2A_j \tilde{u}_j \delta u_j + \sum_{j=1}^4 A_j \delta u_j^2 \right) dt. \quad (26)$$

Now, since $0 \leq \tilde{u}_j(t) \leq 1$ and $0 \leq \delta u_j(t) \leq 1$ for all $t \in [t_0, t_f]$ and $j = 1, 2, 3, 4$, we have

$$0 \leq 2A_j \tilde{u}_j \delta u_j \leq 2A_j |\delta u_j|,$$

$$0 \leq A_j \delta u_j^2 \leq A_j |\delta u_j|,$$

where $|\delta u_j|$ represents the absolute value of δu_j . Therefore,

$$\begin{aligned} 0 &\leq \int_{t_0}^{t_f} \left(\sum_{j=1}^4 2A_j \tilde{u}_j \delta u_j + \sum_{j=1}^4 A_j \delta u_j^2 \right) dt \\ &\leq \int_{t_0}^{t_f} \left(\sum_{j=1}^4 2A_j |\delta u_j| + \sum_{j=1}^4 A_j |\delta u_j| \right) dt = 0. \end{aligned} \quad (27)$$

The inequality (27) holds if $\delta u_j(t) = 0$ for all $t \in [t_0, t_f]$ and $j = 1, 2, 3, 4$. This implies that $\tilde{u}_j(t) = \tilde{u}_j(t)$ for all $t \in$

$[t_0, t_f]$ and $j = 1, 2, 3, 4$, which means the two optimal controls are the same. $\square$

3.7.2. Characterization of the Optimal Control. PMP, as applied in [21–23], is now employed to characterize the solution in Section 3.6. The conditions outlined by the PMP transform the optimal control problem into the task of minimizing a pointwise function.

We introduce the Hamiltonian H given by

$$\begin{aligned} H = & K_1 M + K_2 E_H + K_3 I_H + K_4 E_D + K_5 I_D \\ & - K_6 S_D + \frac{1}{2} u_1^2 A_1 + \frac{1}{2} u_2^2 A_2 + \frac{1}{2} u_3^2 A_3 + \frac{1}{2} u_4^2 A_4 \\ & + \lambda_1 (\theta_1 + \beta_3 R_H - \mu_1 S_H - (1 - (u_1 + u_3)) \chi_1) \\ & + \lambda_2 ((1 - (u_1 + u_3)) \chi_1 - (\mu_1 + \beta_1 + \beta_2 + u_4) E_H) \\ & + \lambda_3 (\beta_1 E_H - (\sigma_1 + \mu_1) I_H) + \lambda_4 ((\beta_2 + u_4) E_H \\ & - (\beta_3 + \mu_1) R_H) + \lambda_5 (\theta_2 - \chi_2 - \mu_2 S_F) \\ & + \lambda_6 (\chi_2 - (\mu_2 + \gamma) E_F) + \lambda_7 (\gamma E_F - (\mu_2 + \sigma_2) I_F) \\ & + \lambda_8 (\theta_3 - \mu_3 S_D - (1 - (u_1 + u_2)) \chi_3 + \gamma_3 R_D) \\ & + \lambda_9 ((1 - (u_1 + u_2)) \chi_3 - (\mu_3 + \gamma_1 + \gamma_2 + u_4) E_D) \\ & + \lambda_{10} (\gamma_1 E_D - (\mu_3 + \sigma_3) I_D) + \lambda_{11} ((\gamma_2 + u_4) E_D \\ & - (\mu_3 + \gamma_3) R_D) + \lambda_{12} ((v_1 I_H + v_2 I_F + v_3 I_D) - \mu_4 M), \end{aligned} \quad (28)$$

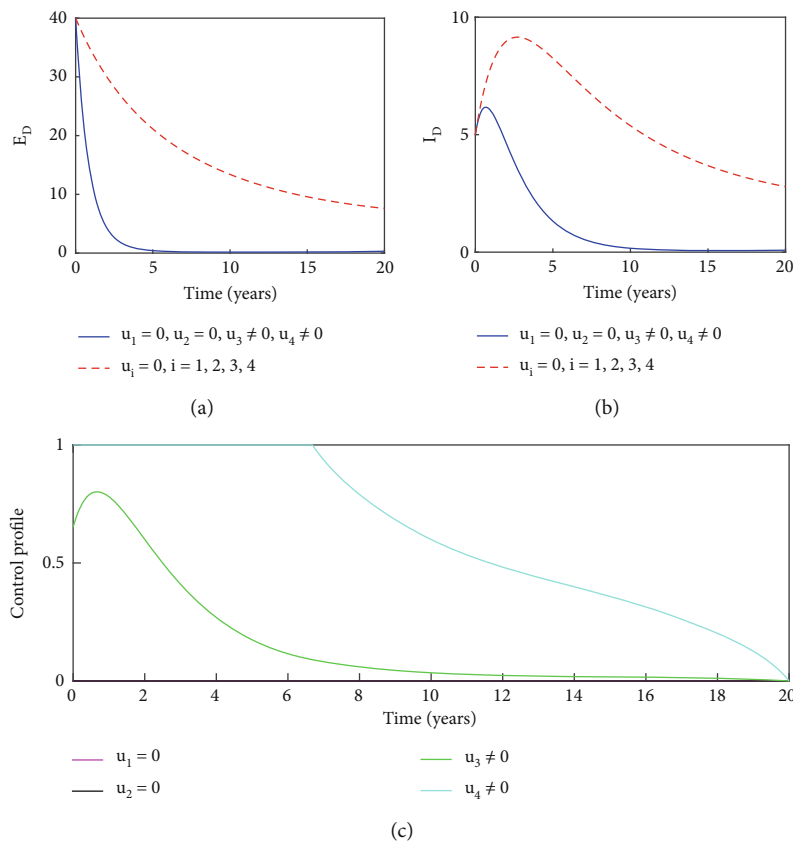


FIGURE 13: (a–c) Impact of Strategy B on domestic dog population.

where

$$\begin{aligned}\chi_1 &= (\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)) S_H, \\ \chi_2 &= (\kappa_1 I_F + \kappa_2 I_D + \kappa_3 \lambda(M)) S_F, \\ \chi_3 &= \left(\frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3} \lambda(M) \right) S_D, \quad \lambda(M) = \frac{M}{M + C},\end{aligned}$$

and $\lambda_1, \dots, \lambda_{12}$ represent the adjoint variables, and $y = (M, S_H, E_H, I_H, R_H, S_F, E_F, I_F, S_D, E_D, I_D, R_D)$ are the state variables. The following result is a direct consequence of PMP [24] and our results of Section 3.7.1.

Theorem 6. *If the optimal control to the problem formulated in Section 3.6 is $u^* = (u_1^*, u_2^*, u_3^*, u_4^*)$, then there exist corresponding adjoint variables $\lambda_1, \dots, \lambda_{12}$ that satisfy the adjoint system*

$$\lambda'_j(t) = -\frac{\partial H}{\partial y_j}$$

subject to the transversality conditions

$$\lambda_j(t_f) = 0,$$

for $1 \leq j \leq 12$. Furthermore, the optimal controls $u_i^*, i = 1, \dots, 4$, are characterized by the maximality condition as follows:

$$\begin{cases} u_1^* = \max \left\{ 0, \min \left(1, \frac{(\lambda_2 - \lambda_1)(\tau_2 I_D + \tau_1 I_F + (M\tau_3/(M+C)))S_H + (\lambda_9 - \lambda_8)(G)S_D}{A_1} \right) \right\}, \\ u_2^* = \max \left\{ 0, \min \left(1, \frac{(\lambda_9 - \lambda_8)((\psi_1 I_F/(1 + \rho_1)) + (\psi_2 I_D/(1 + \rho_2)) + \psi_3 M/((M+C)(1 + \rho_3)))S_D}{A_2} \right) \right\}, \\ u_3^* = \max \left\{ 0, \min \left(1, \frac{(\lambda_2 - \lambda_1)(\tau_1 I_F + \tau_2 I_D + (\tau_3 M/(M+C)))S_H}{A_3} \right) \right\}, \\ u_4^* = \max \left\{ 0, \min \left(1, \frac{E_H(\lambda_2 - \lambda_4) + E_D(\lambda_9 - \lambda_{11})}{A_4} \right) \right\}, \end{cases} \quad (29)$$

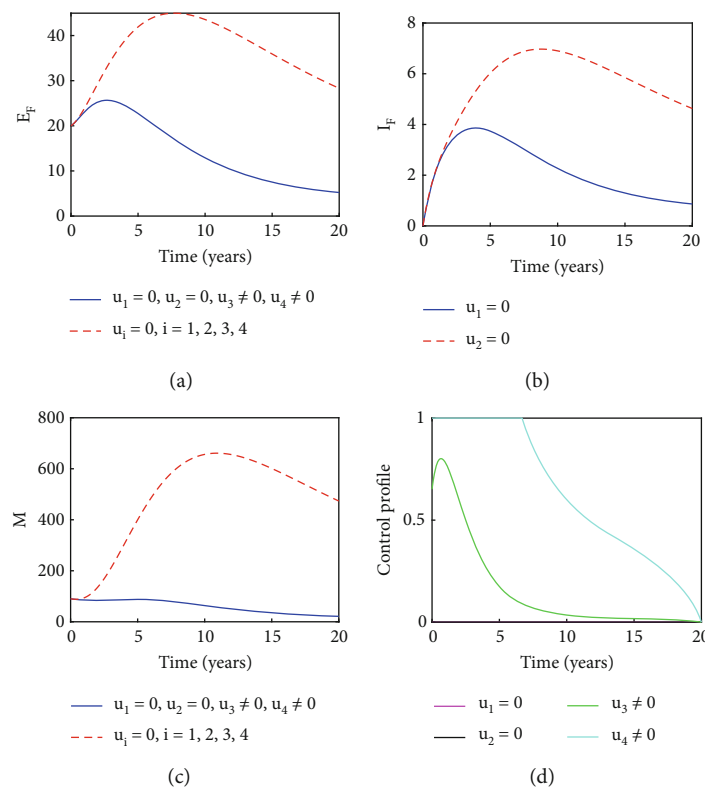


FIGURE 14: (a-d) Impact of Strategy B on free-range dogs and rabies virus.

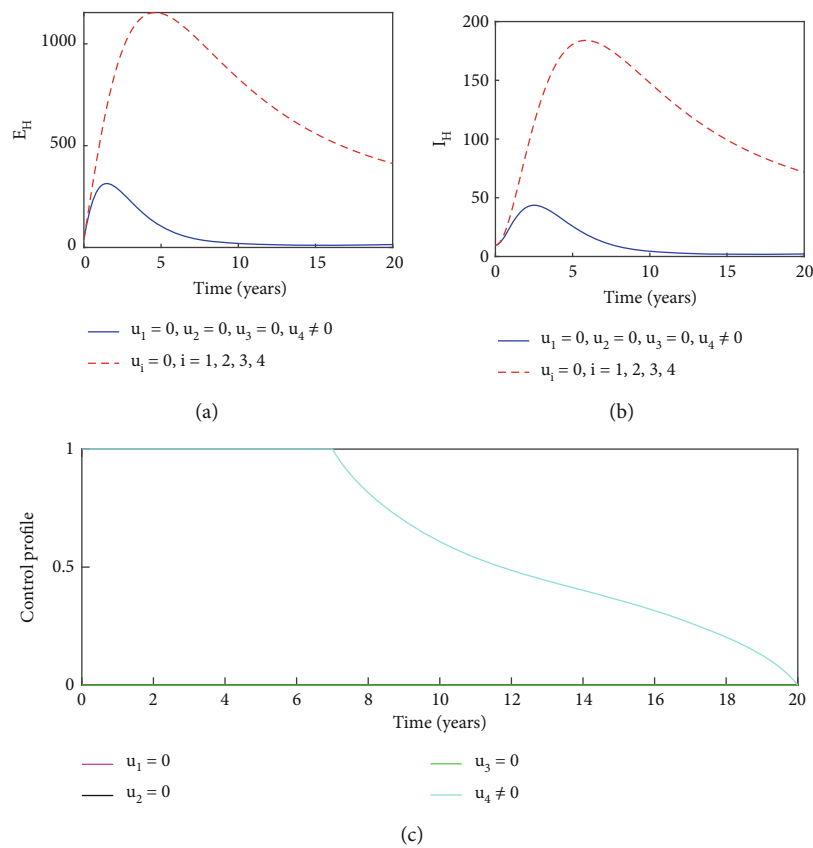


FIGURE 15: (a-c) Impact of Strategy C on human population.

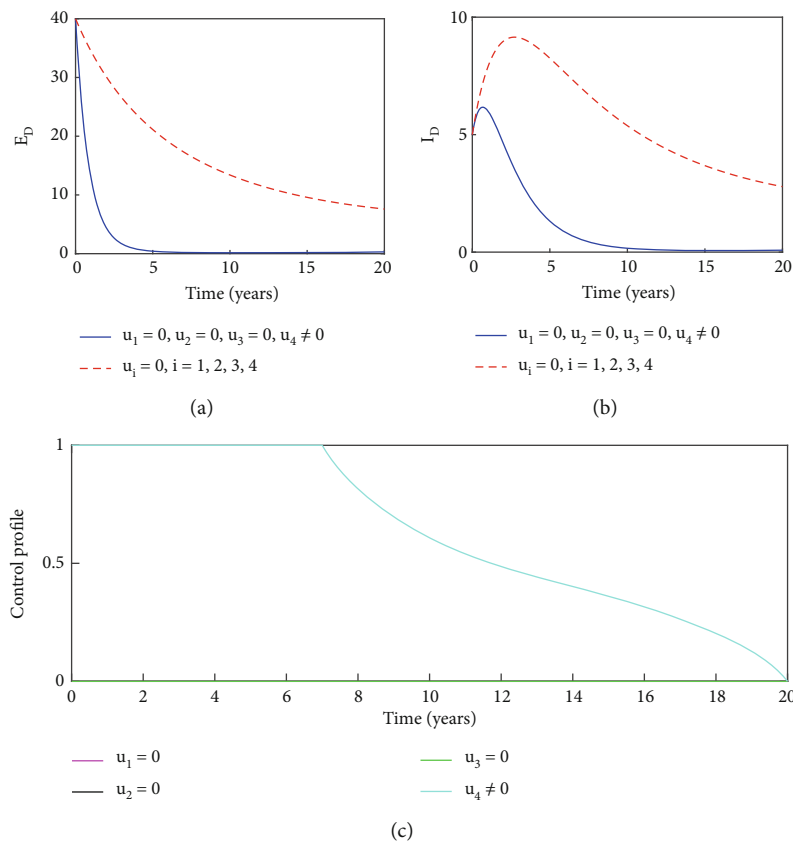


FIGURE 16: (a–c) Impact of Strategy C on domestic dog population.

where

$$G = \frac{\psi_1 I_F}{1 + \rho_1} + \frac{\psi_2 I_D}{1 + \rho_2} + \frac{\psi_3 M}{(M + C)(1 + \rho_3)}.$$

In Section 4.4, we follow [25] to implement Theorem 6 in MATLAB in an algorithmic way.

4. Quantitative Analysis

Before numerical simulations, the parameters of the mathematical model (1) are estimated to understand the transmission dynamics of the system with data from Tanzania.

4.1. Model Fitting and Parameter Estimation. To estimate parameters Θ_i , the model is fitted to real-world data using optimization techniques. The Nelder–Mead algorithm was employed to minimize the difference between the model's predictions $\hat{Y}(\Theta_i)$ and the observed data Y . The estimated parameters were obtained using the least square fitting of $I_H(t_i)$ through discretization of the system of ODEs, that is, (1) without controls, as follows:

$$I_H(t_i + \Delta t) = I_H(t_i) + (\beta_1 \cdot E_H(t_i) - (\sigma_1 + \mu_1) \cdot I_H(t_i)) \cdot \Delta t. \quad (30)$$

The least square fitting is to minimize the objective function given as

$$\min_{\Theta_i} \text{MSE}(\Theta_i) = \frac{1}{n} \sum_{i=1}^n (Y_i - \hat{Y}_i(\Theta_i))^2, \quad (31)$$

where $\text{MSE}(\Theta_i)$ represents the mean squared error, n is the number of data points, while Y_i and $\hat{Y}_i(\Theta_i)$ are the observed and predicted values, respectively, at time i .

We have used data from Tanzania from 1990 to 2018 (see Figure 4). The estimated model parameters that we obtained are shown in Table 2, together with some other values taken from the literature.

4.2. Impact of Contact Rate, Optimal Control Strategy, and $\mathcal{R}_e$. Mesh and contour plots are shown in Figures 5 and 6. They illustrate the relationship between $\mathcal{R}_e$ and $u_1(t)$, which measures the impact of promoting good health practice and management; $u_2(t)$, which represents control effort due to vaccination of domestic dogs; and $u_4(t)$, which measures the treatment effort given to people bitten or scratched by a potentially rabid animal (PREP and PEP), along with the recovery rate as a result of treating infected individuals. It is observed that the magnitude of $\mathcal{R}_e$ decreases with an increase in $u_1(t)$, $u_2(t)$, and $u_4(t)$. This observation highlights the effectiveness

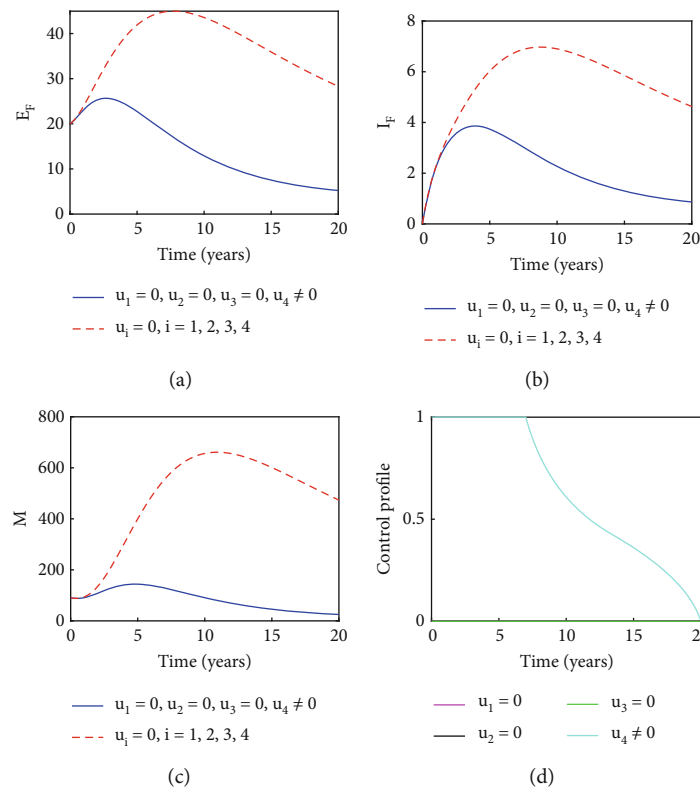


FIGURE 17: (a-d) Impact of Strategy C on free-range dogs and rabies virus.

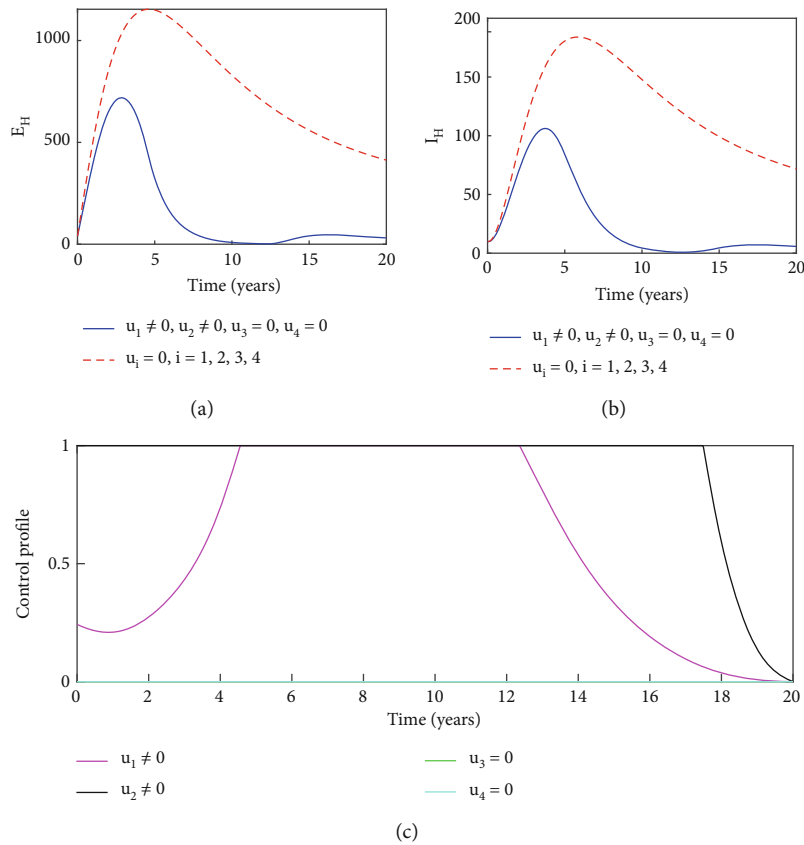


FIGURE 18: (a-c) Impact of Strategy D on human population.

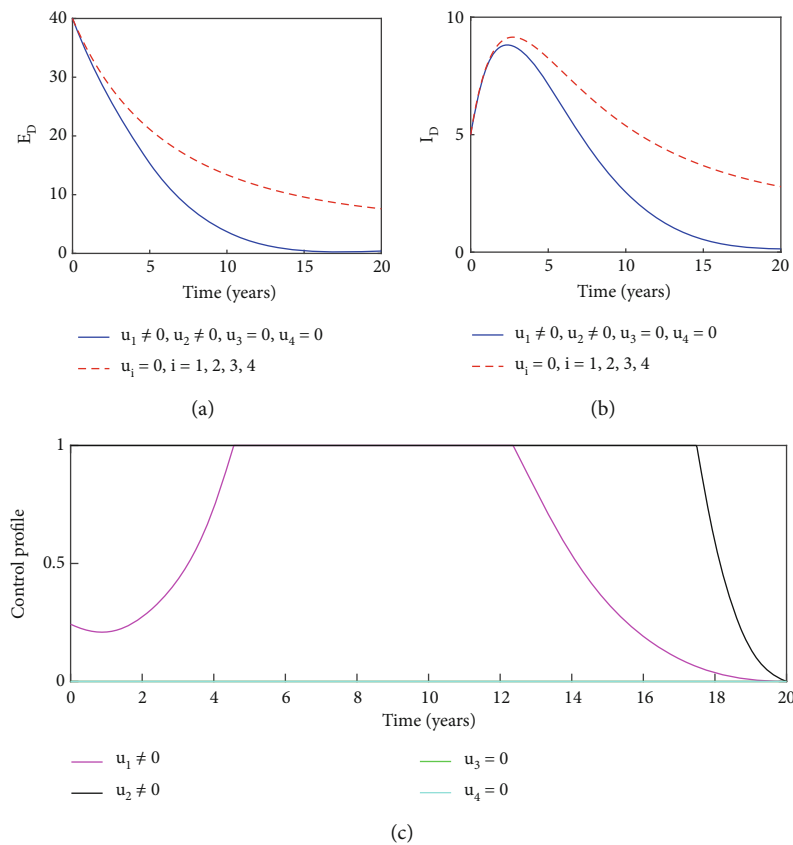


FIGURE 19: (a–c) Impact of Strategy D on domestic dog population.

of $u_1(t)$, $u_2(t)$, and $u_4(t)$ interventions in reducing the transmission of rabies across the community.

The mesh and contour plot representing $\mathcal{R}_e$ as a function of ψ_1 and ψ_2 , which influence the effective reproduction number in the dynamics of rabies transmission, are shown in Figure 7. The results demonstrate that the effectiveness increases along with the values of ψ_1 and ψ_2 . This indicates that increased rates of contact have a positive effect on the dynamics of rabies transmission in domestic dogs.

4.3. Impact of Deterrence Factors on the Transmission of Rabies in Domestic Dogs. Figure 8 demonstrates that an increase in deterrence factors leads to a reduction in the number of exposed and infected domestic dogs. This suggests that deterrence factors exert a significant influence on the transmission dynamics of rabies in domestic dogs.

4.4. Numerical Simulations. Now, we conduct numerical simulations involving an optimal control model to analyze the dynamics and management of rabies in humans, as well as in free-range and domestic dog populations. The optimal control solution is obtained by solving the optimality system, which consists of the adjoint system and the state system, according with Theorem 6. To solve such system, the fourth-order Runge–Kutta iterative scheme method is employed, initializing controls over the simulated time. For solving the adjoint equations, the backward fourth-order Runge–Kutta scheme is employed, leveraging existing solu-

tions of the state equations in accordance with the transversality conditions [33]. Furthermore, updates to the controls (u_1, u_2, u_3, u_4) are made using a convex combination of the prior controls and values from the characterizations [33]. This iterative process continues until the values of unknowns in subsequent iterations closely approximate those in the current iteration [20]. Our MATLAB implementation follows the one discussed in [25]. The values used for numerical simulation are shown in Table 2.

We incorporate four time-dependent control variables that are as follows: $u_1(t)$, which measures the effect of promoting good health practice and management (surveillance and monitoring) in reducing the possibility of human and dogs infection with rabies; $u_2(t)$, which represents the control effort due to vaccination of domestic dogs; $u_3(t)$, which measures the impact of educating communities about rabies transmission risks and protective measures to avoid exposure (public awareness and education); and $u_4(t)$, which represents the treatment effort to individuals exposed to suspected rabid animals through bites or scratches (PEP). Strategies for eliminating rabies in humans and dog populations have been ranked based on their effectiveness, starting from the least effective and advancing to the most effective as follows.

4.4.1. Strategy A: Optimal Control With all the Controls u_i , $i = 1, 2, 3, 4$. Here, we measure the impact of promoting good health practices and management, vaccinating domestic

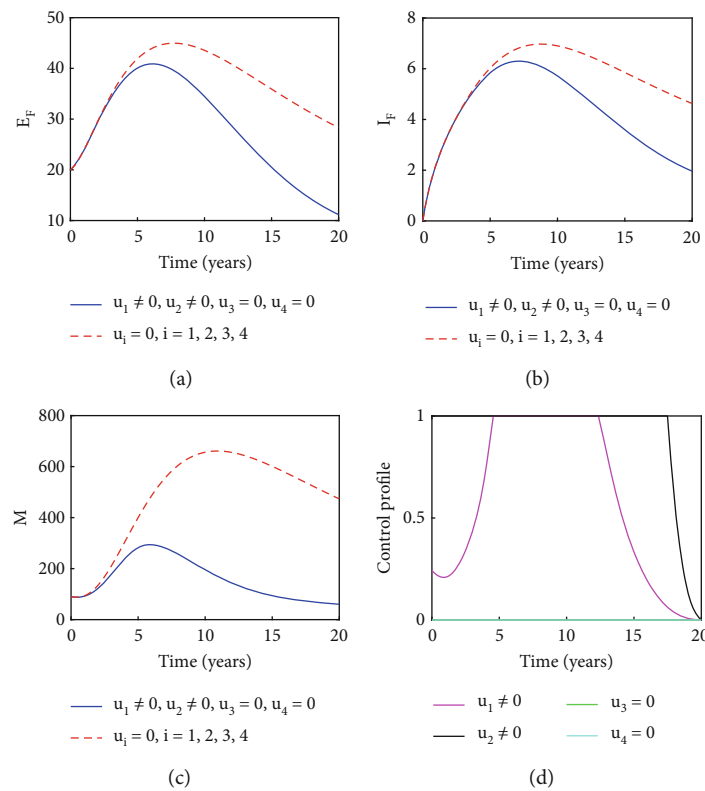


FIGURE 20: (a–d) Impact of Strategy D on free-range dogs and rabies virus.

dogs, educating communities, and lastly, providing treatment for those who have been in contact with a potentially rabid animal through bites or scratches (PEP). This strategy is aimed at eradicating rabies within the community by focusing on four key interventions: promoting good health practices and management, denoted by $u_1(t)$; vaccinating domestic dogs, represented by $u_2(t)$; educating communities, captured by $u_3(t)$; and administering treatment to individuals exposed to potentially rabid animals through PEP, modeled by $u_4(t)$. The results demonstrate that the strategy effectively eliminates rabies within 5 years (see Figures 9, 10, and 11), with optimal utilization of $u_2(t)$ and $u_4(t)$ at 100% during the first 16 and 6 years, respectively, followed by a gradual reduction to nonzero. Meanwhile, $u_1(t)$ and $u_3(t)$ declined over time, reflecting the fact that vaccination does not confer permanent immunity, as illustrated in the control profile.

4.4.2. Strategy B: Optimal Control With Controls u_3 and u_4 . We assess the outcomes of public education and awareness campaigns and the availability of treatment for people exposed to potential rabies via animal bites or scratches (PEP). The results are given in Figures 12, 13, and 14.

4.4.3. Strategy C: Optimal Control Using Only Control u_4 . Now, we concentrate on the impact of providing treatment for bitten or scratched individuals by an animal suspected to be rabid (PEP). Figures 15, 16, and 17 illustrate that the timely administration of PEP at 100% effectiveness plays a

pivotal role in reducing rabies mortality and curbing disease transmission within the first 5 years. By treating individuals immediately after exposure, PEP significantly reduces the risk of death and halts secondary transmission, particularly in high-risk areas. The results also reveal that PEP complements long-term rabies control measures, such as vaccination and health education. While these strategies are vital, PEP offers immediate protection, ensuring effective containment of the disease even after exposure.

4.4.4. Strategy D: Optimal Control With Controls u_1 and u_2 . Finally, we consider the impact of promoting good health practices and management (surveillance and monitoring) and vaccinating on domestic dogs. This strategy focuses on the combined impact of promoting good health practices and management, including surveillance and monitoring, represented by $u_1(t)$, along with the vaccination of domestic dogs, denoted by $u_2(t)$. The results indicate that the strategy is most effective when both control measures are applied at 100%, as illustrated in Figures 18, 19, and 20. The effectiveness of the strategy was observed between the 4th and 14th years, followed by a decline after the reduction in control application. These findings suggest that promoting good health practices and vaccination can significantly reduce periodic rabies transmission. However, the data also highlights that vaccinating domestic dogs alone is not sufficient to fully prevent cyclical rabies transmission, primarily due to the movement of free-roaming dogs that may travel to rabies-free areas and initiate new outbreaks.

5. Conclusion

In this work, a new deterministic mathematical model for rabies disease, incorporating time-dependent control measures, was formulated and rigorously analyzed. The comparison between our effective reproduction rabies number $\mathcal{R}_e$ and the basic reproduction number $\mathcal{R}_0$ of [3] underscores the importance of implementing robust control measures to reduce the effective reproduction number and control rabies transmission. The findings highlight the effectiveness of promoting good health practices, vaccinating domestic dogs, and providing treatment to exposed individuals. Among the control strategies, vaccination and PEP treatment stand out as the most impactful measures for reducing the rabies spread. However, it is clear that relying on a single intervention may not be sufficient to fully eliminate rabies transmission. A combination of interventions, including vaccination, health education, and treatment, proves to be the most effective approach. Additionally, controlling the movement of free-roaming dogs and implementing deterrence measures further enhance the success of rabies elimination strategies. Ultimately, sustained control efforts, especially in regions with high rates of dog-to-dog and dog-to-human contact, are necessary to achieve long-term rabies eradication. A holistic approach that incorporates vaccination, education, treatment, and deterrence can significantly reduce the transmission dynamics and bring the effective reproduction number below the critical threshold needed to eliminate rabies.

Appendix A. Global Stability of Endemic Equilibrium Point E^*

Follows the proof of the result asserted in Section 3.4.

Theorem 7. *The rabies disease endemic equilibrium point of the model (1) is globally asymptotically stable if $\mathcal{R}_e > 1$.*

Proof 6. We use the Lyapunov function of the model system (1) as described by [3, 34, 35]. The Lyapunov function $\mathcal{H}$ is defined by

$$\mathcal{H} = \sum_{i=1}^{12} Q_i \left(y_i - y_i^* - y_i^* \ln \left(\frac{y_i}{y_i^*} \right) \right),$$

where Q_i denotes an undetermined positive constant, y_i represents the population of the i^{th} compartment, and y_i^* indicates the endemic equilibrium point of the model (1). It is apparent that the function $\mathcal{H}$ satisfies all the criteria for being a Lyapunov function:

- i. At the equilibrium point, $\mathcal{H} = 0$, thus $E^*(S_H^*, E_H^*, I_H^*, R_H^*, S_F^*, E_F^*, I_F^*, S_D^*, E_D^*, I_D^*, R_D^*, M)$;
- ii. $\mathcal{H}$ is positive for all other values of $S_H, E_H, I_H, R_H, S_F, E_F, I_F, S_D, E_D, I_D, R_D$ and M .

A Lyapunov function $\mathcal{H}$ of the model system (1) is then defined as

$$\begin{aligned} \mathcal{H} = & Q_1 \left(S_H - S_H^* - S_H^* \ln \left(\frac{S_H}{S_H^*} \right) \right) + Q_2 \left(E_H - E_H^* \right. \\ & \left. - E_H^* \ln \left(\frac{E_H}{E_H^*} \right) \right) + Q_3 \left(I_H - I_H^* - I_H^* \ln \left(\frac{I_H}{I_H^*} \right) \right) \\ & + Q_4 \left(R_H - R_H^* - R_H^* \ln \left(\frac{R_H}{R_H^*} \right) \right) + Q_5 \left(S_F - S_F^* \right. \\ & \left. - S_F^* \ln \left(\frac{S_F}{S_F^*} \right) \right) + Q_6 \left(E_F - E_F^* - E_F^* \ln \left(\frac{E_F}{E_F^*} \right) \right) \\ & + Q_7 \left(I_F - I_F^* - I_F^* \ln \left(\frac{I_F}{I_F^*} \right) \right) + Q_8 \left(S_D - S_D^* \right. \\ & \left. - S_D^* \ln \left(\frac{S_D}{S_D^*} \right) \right) + Q_9 \left(E_D - E_D^* - E_D^* \ln \left(\frac{E_D}{E_D^*} \right) \right) \\ & + Q_{10} \left(I_D - I_D^* - I_D^* \ln \left(\frac{I_D}{I_D^*} \right) \right) + Q_{11} \left(R_D - S_D^* \right. \\ & \left. - R_D^* \ln \left(\frac{R_D}{R_D^*} \right) \right) + Q_{12} \left(M - M^* - M^* \ln \left(\frac{M}{M^*} \right) \right). \end{aligned} \quad (A.1)$$

The time derivative of Equation (A.1) leads to

$$\begin{aligned} \frac{d\mathcal{H}}{dt} = & Q_1 \left(1 - \frac{S_H^*}{S_H} \right) \frac{dS_H}{dt} + Q_2 \left(1 - \frac{E_H^*}{E_H} \right) \frac{dE_H}{dt} \\ & + Q_3 \left(1 - \frac{I_H^*}{I_H} \right) \frac{dI_H}{dt} + Q_4 \left(1 - \frac{R_H^*}{R_H} \right) \frac{dR_H}{dt} \\ & + Q_5 \left(1 - \frac{S_F^*}{S_F} \right) \frac{dS_F}{dt} + Q_6 \left(1 - \frac{E_F^*}{E_F} \right) \frac{dE_F}{dt} \\ & + Q_7 \left(1 - \frac{I_F^*}{I_F} \right) \frac{dI_F}{dt} + Q_8 \left(1 - \frac{S_D^*}{S_D} \right) \frac{dS_D}{dt} \\ & + Q_9 \left(1 - \frac{E_D^*}{E_D} \right) \frac{dE_D}{dt} + Q_{10} \left(1 - \frac{I_D^*}{I_D} \right) \frac{dI_D}{dt} \\ & + Q_{11} \left(1 - \frac{R_D^*}{R_D} \right) \frac{dR_D}{dt} + Q_{12} \left(1 - \frac{M^*}{M} \right) \frac{dM}{dt}. \end{aligned} \quad (A.2)$$

Substituting Equation (1) into Equation (A.2) yields

$$\begin{aligned} \frac{d\mathcal{H}}{dt} = & Q_1 \left(1 - \frac{S_H^*}{S_H} \right) [\theta_1 + \beta_3 R_H - \mu_1 S_H - (1 - \eta_1) f_1 S_H] \\ & + Q_2 \left(1 - \frac{E_H^*}{E_H} \right) [(1 - \eta_1) f_1 S_H - \eta_{12} E_H] \\ & + Q_3 \left(1 - \frac{I_H^*}{I_H} \right) [\beta_1 E_H - \eta_2 I_H] + Q_4 \left(1 - \frac{R_H^*}{R_H} \right) \\ & \cdot [\eta_3 E_H - \eta_4 R_H] + Q_5 \left(1 - \frac{S_F^*}{S_F} \right) [\theta_2 - f_2 S_F - \mu_2 S_F] \\ & + Q_6 \left(1 - \frac{E_F^*}{E_F} \right) [f_2 S_F - \eta_5 E_F] + Q_7 \left(1 - \frac{I_F^*}{I_F} \right) \\ & \cdot [\gamma E_F - \eta_6 I_F] + Q_8 \left(1 - \frac{S_D^*}{S_D} \right) [\theta_3 - \mu_3 S_D] \end{aligned}$$

$$\begin{aligned}
& - (1 - \eta_7)f_3S_D + \gamma_3R_D] + Q_9\left(1 - \frac{E_D^*}{E_D}\right) \\
& \cdot [(1 - \eta_7)f_3S_D - \eta_8E_D] + Q_{10}\left(1 - \frac{I_D^*}{I_D}\right) \\
& \cdot [\gamma_1E_D - \eta_9I_D] + Q_{11}\left(1 - \frac{R_D^*}{R_D}\right)[\eta_{10}E_D - \eta_{11}R_D] \\
& + Q_{12}\left(1 - \frac{M^*}{M}\right)[f_4 - \mu_4M],
\end{aligned} \tag{A.3}$$

where

$$\begin{aligned}
\eta_1 &= u_1 + u_3, \eta_2 = \sigma_1 + \mu_1, f_1 = \tau_1I_F + \tau_2I_D + \tau_3\lambda(M), \\
\eta_3 &= \beta_2 + u_4, \eta_4 = \beta_3 + \mu_1, f_2 = \kappa_1I_F + \kappa_2I_D + \kappa_3\lambda(M), \\
\eta_5 &= \mu_2 + \gamma, \eta_6 = \mu_2 + \sigma_2, \\
f_3 &= \left(\frac{\psi_1I_F}{1 + \rho_1} + \frac{\psi_2I_D}{1 + \rho_2} + \frac{\psi_3}{1 + \rho_3}\lambda(M)\right), \\
\eta_7 &= u_1 + u_2, \eta_8 = \mu_3 + \gamma_1 + \gamma_2 + u_4, \\
\eta_9 &= \mu_3 + \sigma_3, \eta_{10} = \gamma_2 + u_4 \\
\eta_{11} &= \mu_3 + \gamma_3, f_4 = \nu_1I_H + \nu_2I_F + \nu_3I_D, \lambda(M) = \frac{M}{M + C}, \\
\eta_{12} &= \mu_1 + \beta_1 + \beta_2 + u_4.
\end{aligned}$$

At the rabies disease endemic equilibrium point E^* , Equation (A.3) gives

$$\begin{aligned}
\frac{d\mathcal{H}}{dt} &= Q_1\left(1 - \frac{S_H^*}{S_H}\right)[(1 - \eta_1)f_1^*S_H^* + \mu_1S_H^* - \beta_3R_H^* \\
& + \beta_3R_H - \mu_1S_H - (1 - \eta_1)f_1S_H] \\
& + Q_2\left(1 - \frac{E_H^*}{E_H}\right)\left[(1 - \eta_1)f_1S_H - \frac{(1 - \eta_1)f_1^*S_H^*E_H}{E_H^*}\right] \\
& + Q_3\left(1 - \frac{I_H^*}{I_H}\right)\left[\beta_1E_H - \frac{\beta_1E_H^*I_H}{I_H^*}\right] \\
& + Q_4\left(1 - \frac{R_H^*}{R_H}\right)\left[\eta_3E_H - \frac{\eta_3E_H^*R_H}{R_H^*}\right] \\
& + Q_5\left(1 - \frac{S_F^*}{S_F}\right)[f_2^*S_F^* + \mu_2S_F^* - f_2S_F - \mu_2S_F] \\
& + Q_6\left(1 - \frac{E_F^*}{E_F}\right)\left[f_2S_F - \frac{f_2^*S_F^*E_F}{E_F^*}\right] \\
& + Q_7\left(1 - \frac{I_F^*}{I_F}\right)\left[\gamma E_F - \frac{\gamma E_F^*I_F}{I_F^*}\right] \\
& + Q_8\left(1 - \frac{S_D^*}{S_D}\right)[(1 - \eta_7)f_3^*S_D^* + \mu_3S_D^* - \gamma_3R_D^* \\
& - (1 - \eta_7)f_3S_D - \mu_3S_D + \gamma_3R_D] \\
& + Q_9\left(1 - \frac{E_D^*}{E_D}\right)\left[(1 - \eta_7)f_3S_D - \frac{(1 - \eta_7)f_3^*S_D^*E_D}{E_D^*}\right]
\end{aligned}$$

$$\begin{aligned}
& + Q_{10}\left(1 - \frac{I_D^*}{I_D}\right)\left[\gamma_1E_D - \frac{\gamma_1E_D^*I_D}{I_D^*}\right] \\
& + Q_{11}\left(1 - \frac{R_D^*}{R_D}\right)\left[\eta_{10}E_D - \frac{\eta_{10}E_D^*R_D}{R_D^*}\right] \\
& + Q_{12}\left(1 - \frac{M^*}{M}\right)\left[f_4 - \frac{f_4^*M}{M^*}\right].
\end{aligned} \tag{A.4}$$

Expanding (A.4), we obtain that

$$\begin{aligned}
\frac{d\mathcal{H}}{dt} &= -Q_1\mu_1S_H\left(1 - \frac{S_H^*}{S_H}\right)^2 - Q_5\mu_2S_F\left(1 - \frac{S_F^*}{S_F}\right)^2 \\
& - Q_8\mu_3S_D\left(1 - \frac{S_D^*}{S_D}\right)^2 + (1 - \eta_1)Q_1f_1S_H\left(1 - \frac{S_H^*}{S_H}\right) \\
& \cdot \left(1 - \frac{f_1^*S_H^*}{f_1S_H}\right) + Q_1\beta_1R_H\left(1 - \frac{S_H^*}{S_H}\right)\left(1 - \frac{R_H^*}{R_H}\right) \\
& + Q_2(1 - \eta_1)f_1S_H\left(1 - \frac{E_H^*}{E_H}\right)\left(1 - \frac{f_1^*S_H^*E_H}{f_1S_HE_H^*}\right) \\
& + Q_3\beta_1E_H\left(1 - \frac{I_H^*}{I_H}\right)\left(1 - \frac{E_H^*I_H}{I_H^*E_H}\right) \\
& + Q_4\eta_3E_H\left(1 - \frac{R_H^*}{R_H}\right)\left(1 - \frac{E_H^*R_H}{R_H^*E_H}\right) \\
& + Q_5f_2S_F\left(1 - \frac{S_F^*}{S_F}\right)\left(1 - \frac{f_2^*S_F^*}{f_2S_F}\right) \\
& + Q_6f_2S_F\left(1 - \frac{E_F^*}{E_F}\right)\left(1 - \frac{f_2^*S_F^*E_F}{E_F^*S_Ff_2}\right) \\
& + Q_7\gamma E_F\left(1 - \frac{I_F^*}{I_F}\right)\left(1 - \frac{E_F^*I_F}{I_F^*E_F}\right) \\
& + (1 - \eta_7)Q_8f_3S_D\left(1 - \frac{S_D^*}{S_D}\right)\left(1 - \frac{f_3^*S_D^*}{f_3S_D}\right) \\
& + Q_8\gamma_3R_D\left(1 - \frac{S_D^*}{S_D}\right)\left(1 - \frac{R_D^*}{R_D}\right) \\
& + Q_9(1 - \eta_7)f_3S_D\left(1 - \frac{E_D^*}{E_D}\right)\left(1 - \frac{f_3^*S_D^*E_D}{E_D^*f_3S_D}\right) \\
& + Q_{10}\gamma_1E_D\left(1 - \frac{I_D^*}{I_D}\right)\left(1 - \frac{E_D^*I_D}{I_D^*E_D}\right) \\
& + Q_{11}\eta_{10}E_D\left(1 - \frac{R_D^*}{R_D}\right)\left(1 - \frac{E_D^*R_D}{R_D^*E_D}\right) \\
& + Q_{12}f_4\left(1 - \frac{M^*}{M}\right)\left(1 - \frac{f_4^*M}{M^*f_4}\right).
\end{aligned} \tag{A.5}$$

To simplify Equation (A.5), let

$$\begin{aligned}
a &= \frac{S_H}{S_H^*}, b = \frac{E_H}{E_H^*}, c = \frac{I_H}{I_H^*}, d = \frac{R_H}{R_H^*}, \\
e &= \frac{S_F}{S_F^*}, f = \frac{E_F}{E_F^*}, g = \frac{I_F}{I_F^*}, h = \frac{S_D}{S_D^*}, \\
m &= \frac{E_D}{E_D^*}, n = \frac{I_D}{I_D^*}, p = \frac{R_D}{R_D^*}, q = \frac{M}{M^*},
\end{aligned}$$

$$k = \frac{f_1}{f_1^*}, l = \frac{f_2}{f_2^*}, t = \frac{f_3}{f_3^*}, \quad \text{and} \quad s = \frac{f_4}{f_4^*}.$$

Upon solving (A.5), the implication equation yields to

$$\begin{aligned} \frac{d\mathcal{H}}{dt} = & -Q_1\mu_1S_H\left(1-\frac{1}{a}\right)^2 - Q_5\mu_2S_F\left(1-\frac{1}{e}\right)^2 \\ & - Q_8\mu_3S_D\left(1-\frac{1}{h}\right)^2 + (1-\eta_1)Q_1f_1S_H\left(1-\frac{1}{a}\right) \\ & \cdot \left(1-\frac{1}{ak}\right) + Q_1\beta_1R_H\left(1-\frac{1}{a}\right)\left(1-\frac{1}{d}\right) \\ & + Q_2(1-\eta_1)f_1S_H\left(1-\frac{1}{b}\right)\left(1-\frac{b}{ka}\right) \\ & + Q_3\beta_1E_H\left(1-\frac{1}{c}\right)\left(1-\frac{c}{b}\right) + Q_4\eta_3E_H\left(1-\frac{1}{d}\right) \\ & \cdot \left(1-\frac{d}{b}\right) + Q_5f_2S_F\left(1-\frac{1}{e}\right)\left(1-\frac{1}{el}\right) \\ & + Q_6f_2S_F\left(1-\frac{1}{f}\right)\left(1-\frac{f}{el}\right) + Q_7\gamma E_F\left(1-\frac{1}{g}\right) \\ & \cdot \left(1-\frac{g}{f}\right) + (1-\eta_7)Q_8f_3S_D\left(1-\frac{1}{h}\right) \\ & \cdot \left(1-\frac{1}{ht}\right) + Q_8\gamma_3R_D\left(1-\frac{1}{h}\right)\left(1-\frac{1}{p}\right) \\ & + Q_9(1-\eta_7)f_3S_D\left(1-\frac{1}{m}\right)\left(1-\frac{m}{ht}\right) \\ & + Q_{10}\gamma_1E_D\left(1-\frac{1}{n}\right)\left(1-\frac{n}{m}\right) + Q_{11}\eta_{10}E_D\left(1-\frac{1}{p}\right) \\ & \cdot \left(1-\frac{p}{m}\right) + Q_{12}f_4\left(1-\frac{1}{q}\right)\left(1-\frac{q}{s}\right). \end{aligned} \quad (\text{A.6})$$

If $Q_i = 1$ for $i = 1 \leq i \leq 12$, whereas the coefficients of ak , el , and ht are set to zero, then Equation (A.6) becomes

$$\begin{aligned} \frac{d\mathcal{H}}{dt} = & -\mu_1S_H\left(1-\frac{1}{a}\right)^2 - \mu_2S_F\left(1-\frac{1}{e}\right)^2 - \mu_3S_D\left(1-\frac{1}{h}\right)^2 \\ & + \beta_1R_H\left(1-\frac{1}{d}-\frac{1}{a}+\frac{1}{ad}\right) + \beta_1E_H\left(1-\frac{c}{b}-\frac{1}{c}+\frac{1}{b}\right) \\ & + \eta_3E_H\left(1-\frac{d}{b}-\frac{1}{d}+\frac{1}{b}\right) + \gamma E_F\left(1-\frac{g}{f}-\frac{1}{g}+\frac{1}{f}\right) \\ & + \gamma_3R_D\left(1-\frac{1}{p}-\frac{1}{h}+\frac{1}{ph}\right) + \gamma_1E_D\left(1-\frac{n}{m}-\frac{1}{n}+\frac{1}{m}\right) \\ & + \eta_{10}E_D\left(1-\frac{p}{m}-\frac{1}{p}+\frac{1}{m}\right) + f_4\left(1-\frac{q}{s}-\frac{1}{q}+\frac{1}{s}\right). \end{aligned} \quad (\text{A.7})$$

From Equation (A.7), we have

$$H_1 = -\mu_1S_H\left(1-\frac{1}{a}\right)^2 - \mu_2S_F\left(1-\frac{1}{e}\right)^2 - \mu_3S_D\left(1-\frac{1}{h}\right)^2, \quad (\text{A.8})$$

$$\begin{aligned} H_2 = & \beta_1R_H\left(1-\frac{1}{d}-\frac{1}{a}+\frac{1}{ad}\right) + \beta_1E_H\left(1-\frac{c}{b}-\frac{1}{c}+\frac{1}{b}\right) \\ & + \eta_3E_H\left(1-\frac{d}{b}-\frac{1}{d}+\frac{1}{b}\right) + \gamma E_F\left(1-\frac{g}{f}-\frac{1}{g}+\frac{1}{f}\right) \\ & + \gamma_3R_D\left(1-\frac{1}{p}-\frac{1}{h}+\frac{1}{ph}\right) + \gamma_1E_D\left(1-\frac{n}{m}-\frac{1}{n}+\frac{1}{m}\right) \\ & + \eta_{10}E_D\left(1-\frac{p}{m}-\frac{1}{p}+\frac{1}{m}\right) + f_4\left(1-\frac{q}{s}-\frac{1}{q}+\frac{1}{s}\right). \end{aligned} \quad (\text{A.9})$$

Lemma 2. If $\delta(y) = 1 - y + \ln y$, then $\delta(y) \leq 0$ where $1 - y \leq -\ln y$ if and only if $y > 0$ for all $y \in \mathbb{R}^{12+}$.

From Equation (A.9), we have

$$1 - \frac{1}{d} - \frac{1}{a} + \frac{1}{ad} = \left(1 - \frac{1}{d}\right) + \left(1 - \frac{1}{a}\right) - \left(1 - \frac{1}{ad}\right). \quad (\text{A.10})$$

Equation (A.10) is then written using Lemma 2, and the concept of geometric mean is as follows:

$$\begin{aligned} & \left(1 - \frac{1}{d}\right) + \left(1 - \frac{1}{a}\right) - \left(1 - \frac{1}{ad}\right) \\ & \leq -\ln\left(\frac{1}{d}\right) - \ln\left(\frac{1}{a}\right) + \ln\left(\frac{1}{ad}\right) \\ & \leq \ln\left(a \times d \times \frac{1}{ad}\right) = \ln(1) = 0. \end{aligned} \quad (\text{A.11})$$

Following similar procedures in (A.11), we get

$$1 - \frac{c}{b} - \frac{1}{c} + \frac{1}{b} \leq 0, 1 - \frac{p}{m} - \frac{1}{p} + \frac{1}{m} \leq 0, 1 - \frac{d}{b} - \frac{1}{d} + \frac{1}{b} \leq 0. \quad (\text{A.12})$$

Therefore, it follows that $d\mathcal{H}/dt \leq 0$ and $d\mathcal{H}/dt = 0$ only at the endemic equilibrium point (E^*). Hence, using Lasalle's extension to Lyapunov's method, the limit set of each solution is obtained in the largest invariant set for which $S_H^* = S_H$, $E_H^* = E_H$, $I_H^* = I_H$, $R_H^* = R_H$, $S_F^* = S_F$, $E_F^* = E_F$, $S_D^* = S_D$, $E_D^* = E_D$, $I_D^* = I_D$, $R_D^* = R_D$, and $M^* = M$, which is the singleton $\{E^*\}$. Hence, the rabies disease endemic equilibrium point (E^*) of the model system (1) is globally asymptotically stable on a given set when $\mathcal{R}_e > 1$.

Data Availability Statement

The data supporting this study were sourced from existing literature.

Conflicts of Interest

The authors declare no conflicts of interest.

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POSTER PRESENTATION

Mathematical Modeling and Analysis of Emerging and Re-emerging of Rabies Transmission Dynamics: Deterministic and Stochastic Approaches with Control Strategies

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Abstract

Background: Rabies is a fatal zoonotic disease causing approximately 70,000 deaths annually worldwide, with 99% of human cases resulting from dog bites (Fatima et al., 2023). Despite being preventable through vaccination, rabies remains endemic in many developing countries due to limited access to post-exposure prophylaxis and inadequate dog vaccination coverage (Alemayehu et al., 2024).

Methods: We developed and analyzed integrated deterministic and stochastic mathematical models to understand rabies transmission dynamics among humans, domestic dogs, and free-roaming dogs. The models incorporate environmental contamination and evaluate multiple control strategies including vaccination, public education, surveillance, and post-exposure prophylaxis.

Results: The basic reproduction number $\mathcal{R}_0$ was derived and analyzed. Stochastic modeling revealed that even when $\mathcal{R}_0 > 1$, disease extinction can occur due to random effects. Optimal control analysis identified that combined vaccination and public education strategies are most effective for rabies elimination.

Conclusion: The study provides evidence-based recommendations for rabies control policies, supporting the global goal of "Zero dog-mediated human rabies by 2030."

Introduction

Rabies is a zoonotic viral disease caused by *Rabies lyssavirus*, primarily affecting the central nervous system of humans, domestic animals, and livestock (Fatima et al., 2023). **Transmission** occurs mainly through dog bites or scratches, with 99% of human cases linked to dog-mediated exposure (Turner, 2022). Once infected, the virus causes **severe neuroinflammation** and progressive neurological decline (Kumar et al., 2023). Despite being **preventable by vaccination**, rabies remains a major public health threat, especially in LMICs, accounting for 59.6% of cases in Asia and 36.4% in Africa, with 70,000 deaths annually (Alemayehu et al., 2024).

Research Objectives:

The general objective of the study is to develop and analyze mathematical models of dog bites and estimate probability of developing rabies;

- To formulate and analyze a deterministic model that captures the transmission dynamics of rabies in human and dog populations.
- To conduct parameter estimation and sensitivity analysis for a deterministic model of rabies transmission in human and dog populations.
- To quantitatively assess the performance of the deterministic model in predicting rabies transmission.
- Develop and analyze a CTMC stochastic model to estimate the probability of rabies infection.
- Assess the impact of intervention strategies using optimal control theory.

Deterministic SEIR Model Formulation

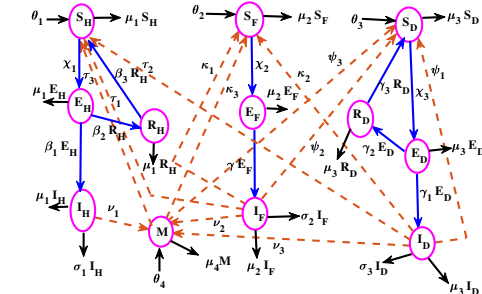


Figure 1: Model Schematic

Results for Deterministic SEIR Model

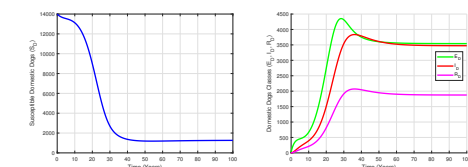


Figure 2: Population dynamics for (a) S_D , and (b) E_D , I_D and R_D .

Parameter Estimation & Sensitivity

Parameter Estimation Method:

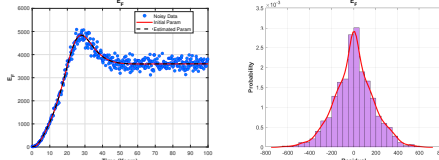


Figure 4: (a) Model fitting, and (b) Normal Distribution at $\sigma = 0.05$ and 95%

Global Sensitivity Analysis:

- Latin Hypercube Sampling (LHS) with 1000 simulations.
- Partial Rank Correlation Coefficients (PRCC).

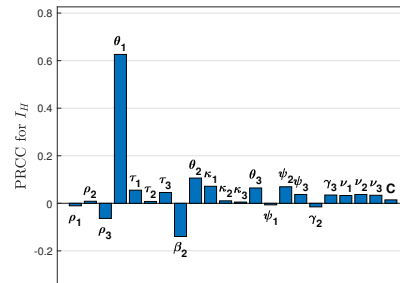


Figure 5: PRCC Analysis

Model Performance:

Variable	MAE	MRE (%)	R ²
S_H	753.83	4.25	0.99656
I_H	89.54	4.00	0.98543
S_D	168.23	4.20	0.99544
I_D	104.33	4.00	0.98516

Stochastic CTMC Model

Extinction Probability:

$$\mathbb{P}_0 = q_1^i q_2^b \dots q_7^i.$$

Outbreak Probability:

$$\mathbb{P}_{\text{outbreak}} = 1 - \mathbb{P}_0.$$

Key Findings:

Initial Infection	Ext. Prob	Outb. Prob
Multiple populations	0.12	0.87
Only free-range dogs	0.27	0.72
Single domestic dog	1.00	0.00

- High outbreak probability when multiple populations infected.
- Free-range and domestic dogs are main transmission sources.

Optimal Control Strategies

Control Variables:

- $u_1(t)$: Surveillance and monitoring.
- $u_2(t)$: Dog vaccination.
- $u_3(t)$: Public education.
- $u_4(t)$: Post-exposure prophylaxis.

Most Effective Strategies:

- Strategy A:** All controls (near eradication).

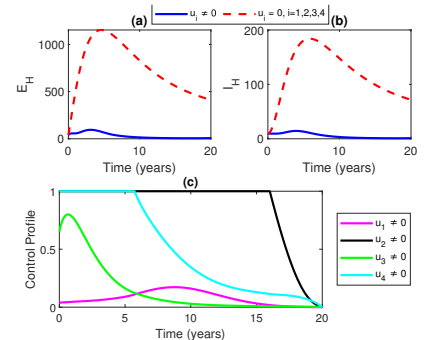


Figure 6: Strategy A Results

Conclusions & Recommendations

Key Conclusions:

- Dog populations are primary transmission drivers.
- Combined vaccination, PEP, and education most effective.

Policy Recommendations:

- Prioritize mass dog vaccination campaigns.
- Enhance public education on rabies prevention.
- Implement real-time surveillance systems.
- Enforce dog movement control policies.
- Integrate environmental hygiene measures.

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