International Journal of Development Mathematics Vol 3 Issue 3 Page No. 444 - 481

INTERNATIONAL JOURNAL OF DEVELOPMENT MATHEMATICS

ISSN: 3026-8656 (Print) | 3026-8699 (Online)

journal homepage: https://ijdm.org.ng/index.php/Journals

Mathematical Model and Analysis of Leptospirosis Transmission Dynamics in Human and Dog Populations

Pwaluwo Jonatan[a], Yusuf Bala[b], Abdulfatai A. Momoh[a], Salaudeen Yusuf[c,*], Amos I. Kura[d] and Abubakar Idris[e]

[a]Department of Mathematics, Modibbo Adama University, Yola Adamawa State, Nigeria.
[b]Department of Mathematical Sciences, Federal university of Health Sciences Azare, Bauchi Nigeria.
[c]Department of Epidemiology and Biostatistics, Federal University of Health Sciences Azare, Bauchi State Nigeria.
[d]Department of Mathematics, Federal University, Dutsinma Katsina State Nigeria.
[e]College of Agriculture, Science and Technology, Jalingo Taraba State Nigeria.

ARTICLE INFO

Article history:
Received 03 April 2026
Received in revised form 20 July 2026
Accepted 30 August 2026

Keywords:
Leptospirosis; *Leptospira*; zoonotic disease; transmission dynamics; environmental contamination; One Health; public health; tropical diseases; reservoir hosts; disease control

MSC 2020 Subject classification:
92D30, 34D20, 92D25

ABSTRACT

Leptospirosis is a zoonotic disease caused by Leptospira species and remains a major public health concern in tropical and subtropical regions. It is transmitted primarily through contact with contaminated water or soil. This study develops and analyzes a mathematical model of the transmission dynamics of leptospirosis to enhance understanding of its spread. The basic reproduction number R_0 was derived using the next-generation matrix (NGM) method to quantify the average number of secondary infections. The local and global stability of the system were established using the Routh–Hurwitz criterion and the Castillo–Chavez method, respectively. It was shown that the disease-free equilibrium is both locally and globally asymptotically stable when $R_0 < 1$. Sensitivity analysis was performed using the normalized forward sensitivity index to identify key parameters influencing disease transmission. Parameters with positive sensitivity indices increase the risk of outbreaks, whereas those with negative indices reduce the likelihood of sustained transmission. Numerical simulations were conducted to illustrate the effects of these parameters and to evaluate intervention strategies. The results indicate that effective control of leptospirosis requires a combination of improved environmental sanitation and proper treatment of infections in both humans and animals, providing valuable insights for public health policy in affected communities.

1 Introduction

Leptospirosis is a globally significant zoonotic disease caused by pathogenic *Leptospira* species, with transmission driven by contact with environments contaminated by infected animal urine. This study provides a comprehensive overview of the epidemiology, transmission pathways, clinical manifestations, treatment options, and global burden of the disease, with particular emphasis on tropical and resource-limited settings. The disease persists in a wide range of animal reservoirs, especially rodents and livestock, facilitating environmental contamination and sustained transmission to humans. Clinically, leptospirosis presents a broad spectrum, ranging from mild febrile illness to

* Corresponding author. Tel.: +2348038719847
E-mail address: salaudeeyusuf201@gmail.com (Yusuf, S.)
https://doi.org/10.62054/ijdm/0303.24

severe, life-threatening complications such as Weil's disease and multi-organ failure. Its diagnosis is often challenging due to non-specific symptoms and limited access to reliable diagnostic tools, contributing to widespread underreporting. The global burden remains substantial, with over one million cases reported annually, particularly in regions with poor sanitation, frequent flooding, and high human–animal interaction. In Africa, prevalence rates are notably high, with significant variability across countries and population groups. The study highlights that effective control of leptospirosis requires an integrated One Health approach, combining environmental sanitation, early diagnosis, and coordinated treatment strategies targeting both human and animal populations. Leptospirosis is a globally prevalent zoonotic disease caused by pathogenic *Leptospira* species and remains a major public health concern, particularly in tropical and subtropical regions where poor sanitation, flooding, and close human–animal interaction enhance transmission (Picardeau, 2013). The disease persists in a multi-host system involving rodents, livestock, and domestic animals, which shed the bacteria into the environment through urine, contaminating soil and water (Reis et al., 2008). Human infection occurs through direct contact with infected animal fluids or indirect exposure via contaminated environmental media, highlighting the importance of a One Health framework (Bharti et al., 2003). Clinically, leptospirosis ranges from mild febrile illness to severe disease, including Weil's disease, characterized by jaundice, renal failure, hemorrhage, pulmonary complications, and multi-organ failure. The disease often follows a biphasic pattern and is frequently misdiagnosed due to non-specific symptoms resembling malaria, dengue, and typhoid fever, compounded by limited diagnostic capacity (Haake & Levett, 2015). Globally, leptospirosis accounts for over one million cases and approximately 60,000 deaths annually, with the highest burden in South and Southeast Asia, sub-Saharan Africa, and Latin America (Costa et al., 2015; Guo et al., 2020). In Africa, seroprevalence studies indicate substantial infection rates in both humans and animals, with higher risks among occupational groups and in regions with poor infrastructure. Reported prevalence varies widely, with human infection rates ranging from 2.3% to 19.8% and higher levels observed in livestock and rodent populations (Coutinho et al., 2011). Transmission is strongly influenced by environmental and seasonal factors, particularly rainfall and flooding (Costa et al., 2015). Treatment depends on disease severity, with antibiotics such as doxycycline, amoxicillin, and penicillin proving effective when administered early, while severe cases require hospitalization and supportive care (Haake & Levett, 2015). Preventive strategies, including prophylactic antibiotic use, remain debated. Overall, effective control requires integrated interventions combining environmental sanitation, early diagnosis, and coordinated management of human and animal infections within a One Health framework. Several mathematical models have been developed to understand the complex dynamics of leptospirosis. However, many existing models either oversimplify the role of environmental reservoirs or fail to fully incorporate the interactions between humans, animals, and the environment. This limits their applicability in guiding effective public health interventions, particularly in resource-limited endemic settings. For instance, Ayoade et al. (2024) constructed a toxoplasmosis model including vertical transmission and environmental contamination from infected cats. Their results revealed that vertical transmission, contact rates, and environmental oocyst contribution significantly influence disease persistence, supported by sensitivity and stability analyses. Engida et al. (2022) developed a leptospirosis model

incorporating rodent reservoirs and environmental contamination. Their analysis revealed forward bifurcation and identified key parameters influencing transmission, while simulations showed that reducing transmission rates and controlling rodent populations significantly reduces infection spread. Alalhareth et al. (2023) proposed a fractional-order leptospirosis model with environmental effects, analyzing stability, boundedness, and bifurcation behavior. Their numerical simulations highlighted the role of asymptomatic and symptomatic infections and demonstrated the influence of fractional dynamics on disease spread. Pimpunchat et al. (2013) developed a classical SIR model for leptospirosis and analyzed its stability using nonlinear dynamical system techniques. Their results identified equilibrium points and confirmed consistency between analytical and numerical solutions. Sarkar et al. (2019) formulated a leptospirosis model incorporating exposed individuals, infected vectors, and disease-induced mortality. Although these studies have significantly advanced the mathematical understanding of leptospirosis transmission, several important gaps remain. Most existing models focus on a limited number of transmission pathways, particularly human–rodent or human–environment interactions, and do not comprehensively integrate the coupled transmission dynamics among humans, dogs, and the contaminated environment. Furthermore, many models neglect important epidemiological features such as asymptomatic human infection, hospitalization, waning immunity, and the dynamic accumulation and natural decay of leptospiral bacteria in the environment. These limitations reduce their ability to capture the full complexity of disease transmission and to evaluate integrated intervention strategies within a One Health framework.

To address these limitations, this study develops a novel deterministic compartmental model for leptospirosis that explicitly incorporates the interactions among humans, dogs, and the contaminated environment. Unlike previous studies, the proposed model simultaneously accounts for asymptomatic and symptomatic human infections, hospitalization, recovery with waning immunity, environmental bacterial shedding, and natural pathogen decay, thereby providing a more realistic representation of leptospirosis transmission. The model is analysed by deriving the basic reproduction number using the next-generation matrix approach, investigating the local and global stability of the disease-free and endemic equilibria, and performing both normalized forward sensitivity and Partial Rank Correlation Coefficient (PRCC) analyses to identify the parameters that most strongly influence disease transmission. Furthermore, numerical simulations are used to validate the theoretical findings and to investigate the biological and epidemiological implications of key model parameters. Consequently, this study provides a comprehensive quantitative framework for understanding leptospirosis transmission and offers scientifically informed recommendations for integrated One Health intervention strategies aimed at reducing disease burden in endemic and resource-limited settings.

The remainder of this paper is organized as follows. Section 2 presents the formulation of the proposed leptospirosis transmission model together with the model assumptions and parameter definitions. Section 3 establishes the existence of the equilibrium points, derives the basic reproduction number, and investigates the local and global stability properties of the model. Section 4 presents the normalized forward sensitivity analysis and the Partial Rank Correlation Coefficient (PRCC) analysis to identify the epidemiological parameters that most strongly influence

disease transmission. Numerical simulations, biological interpretations, and discussions of the results are presented in Section 6, while Section 7 concludes the paper with the major findings and recommendations for future research.

2 Leptospiral Model

2.1 Description of the leptospirosis model

The proposed model describes the transmission dynamics of Leptospirosis in a population consisting of humans, dogs (as animal reservoirs), and the environment (contaminated water or soil). The human population is stratified into six compartments: susceptible human $S_H(t)$, latent humans $L_H(t)$,asymptomatic infectious humans $I_{AH}(t)$, symptomatic infectious humans $I_{SH}(t)$,hospitalized humans $H_H(t)$, and recovered humans $R_H(t)$. The dog population is divided into five compartments: susceptible dogs $S_D(t)$, exposed dogs $E_D(t)$, infectious dogs $I_D(t)$, recovered dogs $R_D(t)$, and an environmental compartment $L_E(t)$, which represents the bacterial load in the environment.

Susceptible humans $S_H(t)$ are recruited into the population at a rate Λ_H . They are at risk of infection through direct contact with infectious dogs or indirect contact via the contaminated environment. The force of infection for humans is given by:

$$\lambda_H = \frac{\psi L_E}{1+\alpha L_E} + \frac{\iota I_D}{N_D},$$

where $L_E(t)$ is the bacterial concentration in the environment, E_D is the latent dog population, α is a saturation parameter moderating the environmental infection, ψ is the contact rate from the environment, and ι is the transmission coefficient due to contact with infected dogs. Susceptible humans become exposed at this rate, and natural death occurs at rate μ_H . Recovered individuals may lose immunity and return to the susceptible class at rate σ_H . The equation governing this compartment is:

$$\frac{dS_H}{dt} = \Lambda_H - \lambda_H S_H - \mu_H S_H + \sigma_H R_H.$$

Latently infected humans L_H have contracted the bacteria but are not yet infectious. They progress to the infectious classes at rate ε_H , or die naturally at a rate μ_H :

$$\frac{dL_H}{dt} = \lambda_H S_H - (\mu_H + \varepsilon_H) L_H.$$

Upon leaving the latent state, a fraction q of individuals become asymptomatically infectious I_{AH}, while the remaining fraction $1-q$ develop symptoms and move to the symptomatic class I_{SH}. The asymptomatic class faces natural death, disease-induced death μ_1, or hospitalization at rate δ_1 :

$$\frac{dI_{AH}}{dt} = q\varepsilon_H L_H - (\mu_H + \mu_1 + \delta_1) I_{AH}.$$

Symptomatic human I_{SH} increases at which individuals show symptoms $(1-q)\varepsilon_H$ and are subject to death $(\mu_H + \mu_2)$ or hospitalization at rate δ_2:

$$\frac{dI_{SH}}{dt} = (1-q)\varepsilon_H L_H - (\mu_H + \mu_2 + \delta_2) I_{SH}.$$

Hospitalized humans H_H increases from both infectious classes via δ1 and δ2, respectively. They recover at rate ω_H, die from complications at rate μ_3, or from natural causes:

$$\frac{dH_H}{dt} = \delta_1 I_{AH} + \delta_2 I_{SH} - (\mu_H + \mu_3 + \omega_H) H_H.$$

Recovered humans R_H are those who have successfully undergone treatment. They may lose immunity and return to the susceptible class or die naturally:

$$\frac{dR_H}{dt} = \omega_H H_H - \mu_H R_H - \sigma_H R_H.$$

The environmental bacterial load L_E increases through contamination by infectious dogs at rate φ and decays at rate ε + τ , where τ represents natural decay and ε environmental cleanup or evaporation:

$$\frac{dL_E}{dt} = \phi I_D - (\varepsilon + \tau) L_E.$$

Dogs are introduced into the population at rate Λ_D . Susceptible dogs S_D are at risk of infection from both the environment and contact with infectious dogs. The force of infection for dogs is:

$$\lambda_D = \frac{\kappa L_E}{1 + \alpha L_E} + \frac{\eta I_D}{N_D},$$

where κ and η are analogous to Ψ and ι for dogs. Susceptible dogs become exposed at this rate or die at μ_D, while recovered dogs rejoin this class via immunity loss at rate σ_D:

$$\frac{dS_D}{dt} = \Lambda_D - \lambda_D S_D - \mu_D S_D + \sigma_D R_D.$$

Exposed dogs E_D are in the latent stage after infection. They progress to the infectious class at rate ε_D or die naturally:

$$\frac{dE_D}{dt} = \lambda_D S_D - (\mu_D + \varepsilon_D) E_D.$$

Infectious dogs I_D actively shed bacteria into the environment, die from the disease at rate μ_4, recover at ω_D, or die naturally:

$$\frac{dI_D}{dt} = \varepsilon_D E_D - (\mu_D + \mu_4 + \omega_D) I_D.$$

Recovered dogs R_D gain temporary immunity and eventually return to the susceptible class at rate σ_D or die:

$$\frac{dR_D}{dt} = \omega_D I_D - \mu_D R_D - \sigma_D R_D.$$

The description of the model is summarized in table 2 and 3 respectively.

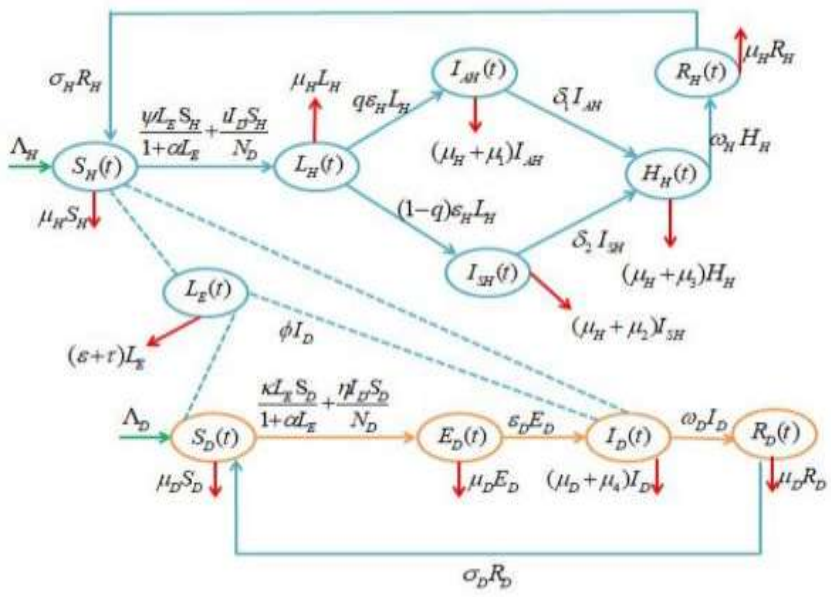

Figure 1: Schematic diagram for the Leptospirosis model with constant control

2.2 Leptospirosis model equations

The Leptospirosis model equation is governed by the following equations:

$$\left.\begin{aligned}
\frac{dS_H(t)}{dt} &= \Lambda_H + \lambda_H S_H - \mu_H S_H + \sigma_H R_H \\
\frac{dL_H(t)}{dt} &= \lambda_H S_H - (\mu_H + \varepsilon_H) L_H \\
\frac{dI_{AH}(t)}{dt} &= q\varepsilon_H L_H - (\delta_1 + \mu_H + \mu_1) I_{AH} \\
\frac{dH_H(t)}{dt} &= \delta_1 I_{AH} + \delta_2 I_{SH} - (\mu_3 + \mu_H + \omega_H) H_H \\
\frac{dI_{SH}(t)}{dt} &= (1-q)\varepsilon_H L_H - (\delta_2 + \mu_H + \mu_2) I_{SH} \\
\frac{dR_H(t)}{dt} &= \omega_H H_H - (\sigma_H + \mu_H) R_H \\
\frac{dL_E(t)}{dt} &= \phi I_D - (\varepsilon + \tau) L_E \\
\frac{dS_D(t)}{dt} &= \Lambda_D - \lambda_D S_D - \mu_D S_D + \sigma_H R_D \\
\frac{dE_D(t)}{dt} &= \lambda_D S_D - (\mu_D + \varepsilon_D) E_D \\
\frac{dI_D(t)}{dt} &= \varepsilon_D E_D - (\mu_D + \mu_4 + \omega_D) I_D \\
\frac{dR_D(t)}{dt} &= \omega_D I_D - (\mu_D + \sigma_D) R_D
\end{aligned}\right\} \quad (1)$$

with initial conditions:

$$\left.\begin{aligned}
&S_H(0) = S_{H0},\ L_H(0) = L_{H0},\ I_{AH}(0) = I_{AH0},\ I_{SH}(0) = I_{SHo},\ H_H(0) = H_{H0},\ R_H(0) = R_{H0}, \\
&L_E(0) = L_{E0},\ S_D(0) = S_{D0},\ E_D(0) = E_{D0},\ I_D(0) = I_{D0},\ R_D(0) = R_{D0}
\end{aligned}\right\}$$

Table 1: Parametric values for the simulation of Leptospirosis infection model.

Parameters	Description	Values	Source
Λ_H	Recruitment rate of susceptible humans.	1.6	Adler, (2015)
Λ_D	Recruitment rate of susceptible dogs.	1	Adler, (2015)
μ_H	Natural death rate of humans.	0.034	Adler, (2015)
μ_D	Natural death rate of dogs.	0.6	Phineas & Isaac, (2022)
ψ	Contact rate between the environment and	0.009	Hiver, Phineas & Isaac,

	humans.		(2022)
η	Contact rate between infected dogs and susceptible dogs.	0.0078	Adler, (2015)
ι	Contact rate between infected dogs and susceptible humans.	0.1	Phineas & Isaac, (2022)
α	Saturation parameter for infection.	0.05	Alalhareth teal., (2022)
q	Proportion of exposed humans who become asymptotically infected.	0.7	Assumed
ε_H	Rate at which exposed humans progresses to infection.	0.0098	Adler, (2015)
ε_D	Rate at which exposed dogs become infectious.	0.0018	Hiver, Phineas & Isaac, (2022)
δ_1	Rate at which asymptomatically infected humans are hospitalized.	0.1	Assumed
δ_2	Rate at which symptomatically infected humans are hospitalized.	0.3	Assumed
μ_1	Disease-induced death rate for asymptomatic humans.	0.000016	Assumed
μ_2	Disease-induced death rate for symptomatic humans.	0.000046	Assumed
μ_3	Disease-induced death rate for hospitalized humans.	0.000056	Assumed
μ_4	Disease-induced death rate for infected dogs.	0.0018	Adler, (2015)
ω_H	Recovery rate for hospitalized humans.	0.007	Adler, (2015)
ω_D	Recovery rate for hospitalized dogs.	0.009	Assumed
σ_H	Loss of immunity in recovered humans.	0.0027	Assumed
σ_D	Loss of immunity in recovered dogs.	0.00548	Assumed
ϕ	Bacteria shedding rate from infected dogs into the environment.	0.00357	Hiver, Phineas & Isaac,(2022)
ε	Natural decay rate of leptospiral in the environment.	0.011	Hiver, Phineas & Isaac,(2022)

τ	Environmental cleaning/intervention rate	0.0055	Hiver, Phineas & Isaac,(2022)
κ	Contact rate of susceptible dogs with contaminated environment	0.0018	Hiver, Phineas & Isaac,(2022)

3 Results

3.1 Basic Properties of Leptospirosis model

The invariant region and positivity of solution of dynamical system (1) is given, coupled with the fact that, the model describes human population. Hence, the study claims the following results.

3.2 Invariant region of Leptospirosis model

In this section, we get a region in which the solution of equations (1) is bounded. To obtain this, first we considered the total population of human N_H and dog N_D, where

$$\left.\begin{aligned} N_H &= S_H(t) + L_H(t) + I_{AH}(t) + I_{SH}(t) + H_H(t) + R_H(t) \\ N_D(t) &= S_D(t) + E_D(t) + I_D(t) + R_D(t) \end{aligned}\right\} \tag{2}$$

Then, differentiating both sides of equation (2) with respect to and simplifying leads to

$$\begin{aligned} \frac{dN_H}{dt} &= \Lambda_H - \mu N_H - \mu_1 I_{AH} - \mu_2 I_{SH} - \mu_3 H_H \\ \frac{dN_D}{dt} &= \Lambda_D - \mu N_D - \mu_4 I_D \end{aligned} \tag{3}$$

And the environmental reservoir is given by

$$L_E \le \frac{\phi \Lambda_D}{\mu_D} - (\varepsilon + \tau) L_E$$

Theorem 1: *The closet*

$$\Omega = \left(S_H, L_H, I_{AH}, I_{SH}, H_H, R_H, L_E, S_D, E_D, I_D, R_D) \in \mathbb{R}_+^{11} : N_H \le \frac{\Lambda_H}{\mu_H}, N_D \le \frac{\Lambda_D}{\mu_D}, L_E \le \frac{\phi \Lambda_D}{\mu_D} - (\varepsilon + \tau) L_E \right) \text{ is}$$

positively invariant and attracting with respect to the model (1).

Proof:

In the absence of disease-induced death, (i.e. $\mu_1 = \mu_2 = \mu_3 = \mu_4 = 0$), and by comparison theorem, the first equation of equation (3) becomes

$$\frac{dN_H}{dt} \le \Lambda_H - \mu_H N_H \tag{4}$$

Solving equation (4) by method of integrating factor and applying initial condition resulted into

$$N_H(t) \le \frac{\Lambda_H}{\mu_H} + \left(N_H(0) - \frac{\Lambda_H}{\mu_H} \right) e^{-\mu_H t} \tag{5}$$

Further simplification of equation (5) resulted into

$$N_H(t) \le N_H(0) e^{-\mu t} + \frac{\Lambda_H}{\mu_H} \left(1 - e^{-\mu_H t} \right) \tag{6}$$

From equation (6), it can be observed that $N_H(t) \to \left(\frac{\Lambda_H}{\mu_H} \right)$ as $t \to \infty$. That is, the total population size $N_H(t)$ takes off from the value $N_H(0)$ at the initial time $t = 0$ and ends up with the bounded value $\frac{\Lambda_H}{\mu_H}$ as the time t grows to infinity. Thus, it can be concluded that $N_H(t)$ is bounded within $0 \le N_H(t) \le \frac{\Lambda_H}{\mu_H}$.

Solving for the Dog population also resulted into

$$N_D(t) \le N_D(0) e^{-\mu_D t} + \frac{\Lambda_D}{\mu_D} \left(1 - e^{-\mu_D t} \right) \tag{7}$$

From equation (7), it can be observed that $N_D(t) \to \left(\frac{\Lambda_D}{\mu_D} \right)$ as $t \to \infty$. That is, the total population size $N_D(t)$ takes off from the value $N_D(0)$ at the initial time $t = 0$ and ends up with the bounded value $\frac{\Lambda_D}{\mu_D}$ as the time t grows to infinity. Thus, it can be concluded that $N_D(t)$ is bounded within $0 \le N_D(t) \le \frac{\Lambda_D}{\mu_D}$.

Thus, the feasible solution set of the system (1) of the model enters and remains in the region: $\Omega = \left(S_H, L_H, I_{AH}, I_{SH}, H_H, R_H, L_E, S_D, E_D, I_D, R_D \right) \in \mathbb{R}_+^{11} : N_H \le \frac{\Lambda_H}{\mu_H}, N_D \le \frac{\Lambda_D}{\mu_D}, L_E \le \frac{\phi \Lambda_D}{\mu_D} - (\varepsilon + \tau) L_E \Bigg)$,

Hence, our Leptospirosis model is well posed and biologically meaningful.

3.3 Positivity of the solution for the Leptospirosis model

In this section, it was shown that all the solutions of the model equation (1) remain positive for future time if their respective initial values are positive.

Theorem 2*:* Let the initial data be

$\{(S_H(0), L_H(0), I_{AH}(0), I_{SH}(0), H_H(0), R_H(0), L_E(0), S_D(0), E_D(0), I_D(0), R_D(0)) \geq 0\} \in \Omega$, *then all the solution sets;* $S_H(t), L_H(t), I_{AH}(t), I_{SH}(t), H_H(t), R_H(t), L_E(t), S_D(t), E_D(t), I_D(t), R_D(t)$ of the model equations (1) is non-negative for all time, $t > 0$.

Proof: Let $T_1 = \sup\left\{\begin{matrix} T_1 > 0 : S_H > 0,\ L_H > 0,\ I_{AH} > 0,\ I_{SH} > 0, H_H > 0, \\ R_H > 0,\ L_E > 0,\ S_D > 0,\ E_D > 0,\ I_D > 0, R_D > 0 \in [0,\ t] \end{matrix}\right\}$,thus, $T_1 > 0$. Then it follows from the first equations of (1) after eliminating the positive term that

$$\frac{dS_H}{dt} = -(\lambda_H + \mu_H)S_H \tag{7}$$

Applying method of separable variable on equation (7), we have

$$\frac{dS_H}{S_H} \geq -(\lambda_H(u)du + \mu_H)dt > 0 \tag{8}$$

Integrating equation (8), we get

$$\ln S_H(t) \geq -\int_0^t \lambda_H(u)du - \mu_H \int dt \tag{9}$$

Taking exponential of both sides on equation (9)

$$S_H(t) \geq e^{-\int_0^t \lambda_H(u)du - \mu_H t + c} \tag{10}$$

where $e^c = C_1$

Simplifying equation (10), we have

$$S_H(t) \geq C_1 e^{-\int_0^t \lambda_H(u)du - \mu_H t} \tag{11}$$

Applying the initial condition at $t = 0$ on equation (11), we have

$$S(0) = C_1 \tag{12}$$

Substitute (12) back into (11) to have

$$S_H(t) \geq S_H(0) e^{-\int_0^t \lambda_H(u)du - \mu_H t} > 0 \tag{13}$$

Applying the same method above to other state variables $L_H(t),\ I_{AH}(t),\ I_{SH}(t),\ H_H(t),\ R_H(t),\ L_E(t),\ S_D(t),\ E_D(t),\ I_D(t),\ R_D(t)$,it can be shown that the solutions are positive for all time $t > 0$.

4 Existence of Equilibrium

4.1 Leptospirosis-free equilibrium point

The Leptospirosis-free equilibrium point is steady-state solution where there is no leptospirosis infection in the population. Thus, in the absence of Leptospirosis infection , we set infectious states to zero, $L_H = E_D = I_{AH} = I_{SH} = I_D = 0$. Equations of the system (1) are solved simultaneously and therefore, the Leptospirosis-free equilibrium point is obtained, Hence,

$$M_0 = \left\{ \frac{\Lambda_H}{\mu_H}, 0, 0, 0, 0, 0, \frac{\Lambda_D}{\mu_D}, 0, 0, 0, 0 \right\} \tag{15}$$

4.2 Endemic equilibrium points of the leptospirosis model

The Leptospirosis endemic equilibrium points are steady-state solution where leptospiral infection persists in the population. At endemic equilibrium, the infectious states are not equal to zero, $L_H \neq 0, E_D \neq 0,\ I_{AH} \neq 0,\ I_{SH} \neq 0, I_D \neq 0$. To establish the existence of endemic equilibria of our Leptospirosis model, we solve equations of the system (1) at steady state simultaneously.

where the force of infection is,

$$\lambda_v^* = \frac{\psi L_E^*}{L_E^*} + \frac{\iota I_D^*}{N_D^*}, \lambda_D^* = \frac{\kappa L_E^*}{L_E^*} + \frac{\eta I_D^*}{N_D^*} \tag{16}$$

And the endemic equilibrium point is obtained as:

$$M_1 = \begin{pmatrix} S_H^* \\ L_H^* \\ I_{AH}^* \\ I_{SH}^* \\ H_H^* \\ R_H^* \\ L_E^* \\ S_D^* \\ E_D^* \\ I_D^* \\ R_D^* \end{pmatrix} = \begin{pmatrix} \frac{\Lambda_H + \sigma_H R_H^*}{g_1} \\ \frac{\lambda_H^*(\Lambda_H + \sigma_H R_H^*)}{k_1 g_1} \\ \frac{\lambda_H^* q \varepsilon_H (\Lambda_H + \sigma_H R_H^*)}{k_1 k_2 g_1} \\ \frac{\lambda_H^*(1-q)\varepsilon_H(\Lambda_H + \sigma_H R_H^*)}{k_3 g_1} \\ \frac{\lambda_H^*(\Lambda_H + \sigma_H R_H^*)(k_3\lambda_H^* q\varepsilon_H\delta_1 + k_2 k_1(1-q)\varepsilon_H\delta_2)}{k_2 k_3 k_4 k_1 g_1} \\ \frac{\lambda_H^*\omega_H(\Lambda_H + \sigma_H R_H^*)(k_3\lambda_H^* q\varepsilon_H\delta_1 + k_2 k_1(1-q)\varepsilon_H\delta_2)}{k_5 k_2 k_3 k_4 k_1 g_1} \\ \frac{\Lambda_D + \sigma_D R_D^*}{g_2} \\ \frac{\lambda_D(\Lambda_D + \sigma_D R_D^*)}{k_7 g_2} \\ \frac{\lambda_D^* \varepsilon_D(\Lambda_D + \sigma_D R_D^*)}{k_8 k_7 g_2} \\ \frac{\lambda_{DD}^* \phi(\Lambda_D + \sigma_D R_D^*)}{k_6 k_8 k_7 g_2} \\ \frac{\omega_D\left\{\lambda_D^* \varepsilon_D(\Lambda_D + \sigma_D R_D^*)\right\}}{k_9 k_6 k_8 k_7 g_2} \end{pmatrix} \tag{17}$$

4.3 Basic Reproduction Number of Leptospirosis Model

Calculation of effective reproduction number for the system model (1) This section studies the effective reproduction number, which is a threshold parameter that controls the spread of a disease. To obtain the effective reproduction number, we apply the next generation approach described in (Driessche & Watmough, 2002). The associated incidence term F and the transfer term V for the Leptospirosis model for the unvaccinated and vaccinated proportions, are respectively obtained as:

$$F=\begin{pmatrix} E_H \\ I_{AH} \\ I_{SH} \\ L_E \\ E_D \\ I_D \end{pmatrix}=\begin{pmatrix} \left(\frac{\psi L_E}{1+\alpha L_E}+\frac{\iota I_D}{N_D}\right)S_H \\ 0 \\ 0 \\ 0 \\ \left(\frac{\kappa L_E}{1+\alpha L_E}+\frac{\iota I_D}{N_D}\right)S_D \\ 0 \end{pmatrix}, V=\begin{pmatrix} (\varepsilon_H+\mu_H)E_H \\ -q\varepsilon_H E_H+(\mu_H+\mu_1+\delta_1)I_{AH} \\ -(1-q)\varepsilon_H E_H+(\mu_H+\mu_2+\delta_2)I_{SH} \\ -\varphi I_D+(\tau+\varepsilon)L_E \\ (\mu_D+\varepsilon_D)E_D \\ -\varepsilon_D E_D+(\mu_D+\mu_4+\omega_D)I_D \end{pmatrix} \tag{18}$$

Thus, the effective reproductive number of system model (1) is calculated from the spectral radius $\rho(FV^{-1})$, Therefore, the Leptospirosis reproduction number is

$$R_0=\frac{\kappa\Lambda_D\phi\varepsilon_D}{\mu_D(\tau+\varepsilon)(\mu_D+\mu_4+\omega_D)(\varepsilon_D+\mu_D)}+\frac{\eta\varepsilon_D}{(\mu_D+\mu_4+\omega_D)(\varepsilon_D+\mu_D)} \tag{19}$$

Local stability of modified Leptospirosis-free equilibrium point

Theorem 3: The leptospirosis-free equilibrium M_0 point of the model is locally asymptotically stable (LAS) if $R_0<1$ and unstable if $R_0>1$

Proof

To prove the system's local stability, the Jacobian of the proposed model system (1) is investigated at DFE, which is then given by:

$$J_{LFE}=\begin{pmatrix} -\mu_H & 0 & 0 & 0 & 0 & \sigma_H & -\psi S_H^0 & 0 & 0 & -\frac{\iota}{N_D^0} & 0 \\ 0 & -k_1 & 0 & 0 & 0 & 0 & \psi S_H^0 & 0 & 0 & \frac{\iota}{N_D^0} & 0 \\ 0 & q\in_H & -k_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & (1-q)\in_H & 0 & -k_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \delta_1 & \delta_2 & -k_4 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \omega_H & -k_5 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau_2 e & -k_6 & 0 & 0 & \phi & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{(1-\psi)\beta\theta V_0}{N^0} & -\kappa S_D^0 & -\mu_D & 0 & -\frac{\eta}{N_D^0} & \sigma_D \\ 0 & 0 & 0 & 0 & 0 & 0 & \kappa S_D^0 & 0 & -k_7 & \frac{\eta}{N_D^0} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \in_D & -k_8 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega_D & -k_9 \end{pmatrix} \tag{20}$$

The Jacobian (20) reduces to (21) as the $\lambda_1 = -\mu_H, \lambda_2 = -\mu_D$

$$J_{LFE} = \begin{pmatrix} k_1 & 0 & 0 & 0 & 0 & \psi S_H^0 & 0 & \frac{\iota}{N_D^0} & 0 \\ q\in_H & k_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ (1-q)\in_H & 0 & k_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \delta_1 & \delta_2 & k_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \omega_H & k_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & k_6 & 0 & \phi & 0 \\ 0 & 0 & 0 & 0 & 0 & \kappa S_D^0 & k_7 & \frac{\eta}{N_D^0} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \in_D & k_8 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega_D & k_9 \end{pmatrix} \tag{21}$$

where

$$\left.\begin{aligned} &k_1 = (\in_H + \mu_H),\ k_2 = (\mu_H + \mu_1 + \delta_1),\ k_3 = (\mu_H + \mu_2 + \delta_2), \\ &k_6 = (\tau + \in),\ k_7 = (\in_D + \mu_D), k_8 = (\mu_D + \mu_4 + \omega_D) \end{aligned}\right\}$$

The characteristic equation of equation (21), given by $\left|FV^{-1} - \lambda I\right| = 0$ is:

Using the approach in Podder and Gumel, (2009), the characteristic equation (21) at the associated DFE, E_0 is given by

$$J(E_0) = [G^*_{6\times 10} \quad G^{**}_{4\times 10}] \tag{22}$$

The following characteristic equation are obtained

$$\left(\lambda^6 + B_1\lambda^5 + B_2\lambda^4 + B_3\lambda^3 + B_4\lambda^2 + B_5\lambda^1 + B_6\right)\left(\lambda^4 + P_1\lambda^3 + P_2\lambda^2 + \lambda P_3 + P_4\right) = 0 \tag{23}$$

where

$$\left.\begin{aligned} a_1 &= k_5 + k_4 + k_3 + k_2 + k_1 \\ a_2 &= \left(k_2 + k_3 + k_4 + k_5\right)k_1 + \left(k_3 + k_4 + k_5\right)k_2 + \left(k_4 + k_5\right)k_3 + k_4k_5 \\ a_3 &= ((k_3 + k_4 + k_5)k_2 + (k_4 + k_5)k_3 + k_4k_5)k_1 + ((k_4 + k_5)k_2 + k_3k_4k_5 \\ a_4 &= (((k_4 + k_5)k_3 + k_4k_5)k_2 + k_3k_4k_5)k_1 + k_2k_3k_4k_5 \\ a_5 &= k_5k_4k_3k_2k_1 \end{aligned}\right\} \tag{24}$$

And

$$\left.\begin{aligned} b_0 &= 1 \\ b_1 &= (k_9 + k_8 + k_7 + k_6) \\ b_2 &= (k_9 + k_8 + k_7)k_6 + (k_8 + k_9)k_7 - \in_D d_4 + k_9 k_8 \\ b_3 &= ((k_7 + k_8)k_9 - \in_D d_4 + k_7 k_8)k_6 + (k_7 k_8 - \in_D d_4)k_9 - \phi d_3 \in_D \\ b_4 &= k_9(k_7 k_8 - d_4 \in_D)k_6 - \phi d_3 \in_D \end{aligned}\right\} \quad (25)$$

Applying Routh-Hurwitz Criterion which state that all roots of the polynomial (23) have negative real part if and only if the coefficients B_i and P_i are positive and the determinant of the matrices $H_i > 0$ for $i = 0, 1, 2, 3, ..., 5.$ .and $H_j > 0$ for $j = 0, 1, 2, 3, 4.$ it was shown that all the eigenvalues of the polynomial (23) have negative real parts, implying that λ_i are less than zero, for $i = 1, 2, 3, ..., 11.$, when $R_0 < 1.$

Therefore, the conclusion was that the disease-free equilibrium point is Locally Asymptotically Stable (LAS).

4.1.6 Global stability of the Leptospirosis- free equilibrium point

In this section, the global stability of the disease free equilibrium for the model (1) was investigated using the theorem by (Castillo-Chavez and Song, 2004)

Lemma 1 Consider a model system written in the form

$$\left.\begin{aligned} \frac{dY}{dt} &= F(Y, Z) \\ \frac{dZ}{dt} &= G(Y, Z), (Y, 0) = 0 \end{aligned}\right\} \quad (26)$$

where $Y = (S_H, H_H, R_H, S_D, R_D)$ and $Z = (L_H, I_{AH}, I_{SH}, L_E, E_D, I_D)$ with the components of $Y \in \mathbb{R}^5$ denoting the uninfected population and the components of $Z \in \mathbb{R}^6$ denoting infected population.

The disease free equilibrium is now denoted as:

$$M_0 = \begin{pmatrix} \frac{\Lambda_H}{\mu_H} \\ 0 \\ 0 \\ \frac{\Lambda_D}{\mu_D} \\ 0 \end{pmatrix}$$

The following two conditions H_1 and H_2 must be met to guarantee a global asymptotic stability:

Assume that:

(H_1). For $\frac{dY}{dt} = F(Y,0)$ is globally asymptotically stable (27)

(H_2). $G(Y,Z) = AZ - \hat{G}(Y,Z), \hat{G}(Y,Z) \geq 0$, For $(X,Z) \in \Omega$ (28)

where $A = D_z G(X^0, 0)$ is a $M-$matrix (the off-diagonal elements of A are non-negative) and Ω is the region where the model makes biological sense. Then, the LFE, Y^* is globally asymptotically stable if $R_0 < 1$ (Castillo-Chavez et al. 2002).

Theorem 4: The Leptospirosis-free equilibrium point $M_0 = (Y^*, 0)$ of the system model (1) is globally asymptotically stable provided $R_0 < 1$ and the conditions (H1) and (H2) are satisfied.

Proof: from the model (1) we can get $F(X,Z)$ and $G(X,Z)$

$$\frac{dY}{dt} = F(Y,Z) \tag{29}$$

$$F(Y,Z) = \begin{pmatrix} \Lambda_H - \left(\frac{\psi L_E}{(1+\alpha I_D)} + \frac{\iota I_D}{N_D}\right) S_H - \mu_H S_H + \sigma_H R_H \\ \delta_1 I_{AH} + \delta_2 I_{SH} - k_4 H_H \\ \omega_H H_H - k_5 R_H \\ \Lambda_D - \left(\frac{\kappa L_E}{(1+\alpha I_D)} + \frac{\eta I_D}{N_D}\right) S_D - \mu_D S_D + \sigma_D R_D \\ \omega_D I_D - k_9 R_D \end{pmatrix} \tag{30}$$

$$G(Y,Z) = \begin{pmatrix} \left(\frac{\psi L_E}{(1+\alpha I_D)} + \frac{\iota I_D}{N_D}\right) S_H - k_1 L_H \\ q\varepsilon_H L_H - k_2 I_{AH} \\ (1-q)\varepsilon_H L_H - k_3 I_{SH} \\ \phi I_D - k_6 L_E \\ \left(\frac{\kappa L_E}{(1+\alpha I_D)} + \frac{\eta I_D}{N_D}\right) S_D - k_7 E_D \\ \varepsilon_D E_D - k_8 I_D \end{pmatrix} \tag{31}$$

At disease free equilibrium, equation (30) becomes

$$F(Y,0)=\begin{pmatrix}\Lambda_H-\mu_H S_H\\0\\0\\\Lambda_D-\mu_D S_D\\0\end{pmatrix} \tag{32}$$

From the system (32) above, it can be seen that

$M_0=\begin{pmatrix}\frac{\Lambda_H}{\mu_H} & 0 & 0 & \frac{\Lambda_D}{\mu_D} & 0\end{pmatrix}$ is Locally-Asymptotically Stable (LAS).

This can be verified from the solutions, namely:

$$\begin{aligned}S_H(t)&=\frac{\Lambda_H}{\mu_H}+\left[S_H(0)-\frac{\Lambda_H}{\mu_H}\right]e^{-u_H t}\\H_H(0)&=H_H(0)e^{-(\mu_H+\mu_3+\omega_H)t}\\R_H(t)&=R_H(0)e^{-(\mu_H+\sigma_H)t}\\S_D(t)&=\frac{\Lambda_D}{\mu_D}+\left[S_D(0)-\frac{\Lambda_D}{\mu_D}\right]e^{-u_D t}\\R_D(t)&=R_D(0)e^{-(\mu_D+\sigma_D)t}\end{aligned} \tag{33}$$

As $t\to\infty$, the solutions

$$S_H(\infty),V_H(\infty),R(\infty),S_D(\infty),R_D(\infty)\to\begin{pmatrix}\frac{\Lambda_H}{\mu_H} & 0 & 0 & \frac{\Lambda_D}{\mu_D} & 0\end{pmatrix}$$

which implies a global convergence of the system (1) in Ω ,and this satisfied condition H_1.

The second condition demands that:

$$\hat{G}(Y,Z)=AZ-G(Y,Z) \tag{34}$$

and that $\hat{G}(Y,Z)\geq 0,\ \ \forall\ (Y,Z)\ \in\Omega$

we already know that from equation (31),

$$G(Y,Z)=\begin{pmatrix}\left(\frac{\psi L_E}{(1+\alpha I_D)}+\frac{\iota I_D}{N_D}\right)S_H-k_1L_H\\ q\varepsilon_H L_H-k_2I_{AH}\\ (1-q)\varepsilon_H L_H-k_3I_{SH}\\ \phi I_D-k_6L_E\\ \left(\frac{\kappa L_E}{(1+\alpha I_D)}+\frac{\eta I_D}{N_D}\right)S_D-k_7E_D\\ \varepsilon_D E_D-k_8I_D\end{pmatrix}$$

$$\hat{G}(Y,Z)=AZ-G(Y,Z).$$

where A is an $n\times n$ matrix, Z is a column vector and $G(Y,Z)$) is a column vector formed from the infectious compartments.

By computing the Jacobian of equation (31) and evaluating at disease-free equilibrium, gives

$$A_{LFE}=\begin{pmatrix}-k_1 & 0 & 0 & d_1 & 0 & d_2\\ q\in_H & -k_2 & 0 & 0 & 0 & 0\\ (1-q)\in_H & 0 & -k_3 & 0 & 0 & 0\\ 0 & 0 & 0 & -k_6 & 0 & 0\\ 0 & 0 & 0 & d_3 & -k_7 & d_4\\ 0 & 0 & 0 & 0 & \in_D & -k_8\end{pmatrix} \tag{35}$$

It can be observed that the matrix A is a metzler matrix because all its off-diagonal elements are non-negative. Thus multiplying the matrix A and Z gives

$$AZ=\begin{pmatrix}-k_1 & 0 & 0 & d_1 & 0 & d_2\\ q\in_H & -k_2 & 0 & 0 & 0 & 0\\ (1-q)\in_H & 0 & -k_3 & 0 & 0 & 0\\ 0 & 0 & 0 & -k_6 & 0 & 0\\ 0 & 0 & 0 & d_3 & -k_7 & d_4\\ 0 & 0 & 0 & 0 & \in_D & -k_8\end{pmatrix}\begin{pmatrix}L_H\\ I_{AH}\\ I_{SH}\\ L_E\\ E_D\\ I_D\end{pmatrix} \tag{36}$$

Evaluating equation (36), gives

$$AZ = \begin{pmatrix} \frac{\psi\Lambda_H L_E}{\mu_H} + \frac{\iota I_D}{N_D^0} - k_1 L_H \\ q\varepsilon_H L_H - k_2 I_{AH} \\ (1-q)\varepsilon_H L_H - k_3 I_{SH} \\ \phi I_D - k_6 L_E \\ \frac{\kappa\Lambda_H L_E}{\mu_H} + \frac{\eta I_D}{N_D^0} - k_7 E_D \\ \varepsilon_D E_D - k_8 I_D \end{pmatrix} \tag{37}$$

Now, using $\hat{G}(Y,Z) = AZ - G(Y,Z)$ one obtains the following

$$\hat{G}(X,Z) = \begin{pmatrix} \psi L_E\left(S_H^0 - \frac{S_H}{(1+\alpha I_D)}\right) + \iota I_D\left(S_H^0 - \frac{S_H}{N_D}\right) \\ 0 \\ 0 \\ 0 \\ \kappa L_E\left(S_D^0 - \frac{S_D}{(1+\alpha I_D)}\right) + \eta I_D\left(S_D^0 - \frac{S_D}{N_D}\right) \\ 0 \end{pmatrix} \tag{38}$$

In the system (1), the total population is bounded by $\left(\frac{\Lambda_H}{\mu_H}, \frac{\Lambda_D}{\mu_D}\right)$ and $(L_H, I_{AH}, I_{SH}, L_E, E_D, I_D) \le (S_H^0, S_D^0)$.

Hence, it is clear that the matrix $\hat{G}(X,Z) \ge 0$ since $S_H^0 \ge \frac{S_H}{(1+\alpha I_D)}$, $, S_H^0 \ge \frac{S_H}{N_D}, S_D^0 \ge \frac{S_D}{N_D}, S_D^0 \ge \frac{S_D}{(1+\alpha I_D)}$. Also, it is obvious that the matrix A is an M-matrix which satisfies the conditions H_1 and H_2, we conclude that the system (1) is Globally Asymptotically Stable, (GAS) if $R_0 < 1$.

4.4 Sensitivity Analysis

In this section, sensitivity analysis of the parameters of the reproduction number of leptospirosis model was examined. This helps in checking and identifying the parameters that have impact on the basic reproductive number. To determine the impact, R_0 was partially differentiated with respect to the model parameters in equation (1). In an

attempt to compute the sensitivity index, the technique outlined by Blower and Dowlatabadi (1994) was adopted. This technique develops a formula to obtain the sensitivity index of all the basic parameters, defined as;

$$\Delta_x^{R_0^{HSV}} = \frac{\partial R_0^{HSV}}{\partial x} \times \frac{x}{R_0^{HSV}} \tag{39}$$

where X represents all the basic parameters to be tested.

$$\in_D, \xi, \eta, \phi, \tau, \in, \omega_D, \mu_4$$

The sensitivity of R_0 to each of the 12 parameters presented in Table 2 is calculated using the effective reproduction number given below;

$$R_0 = \frac{\kappa \Lambda_D \phi \in_D}{\mu_D(\tau + \in)(\in_D + \mu_D)(\mu_D + \mu_4 + \omega_D)} + \frac{\eta \mu_D \in_D}{\Lambda_D(\in_D + \mu_D)(\mu_D + \mu_4 + \omega_D)}$$

Sensitivity index implied the parameters values obtained in Table 3, we can see that the parameters have both positive and negative effects on R_0 .Positive SI values, such as $\varepsilon_D, \kappa, \eta, \phi$ as shown in Table 3 reveal that an increase in these parameters values increases R_0 , which brings about the infection attacking the population. While the parameters $\tau, \varepsilon, \omega_D, \mu_4$ have negative SI, that is, an increase in these parameters values decrease R_0 , and as a result, the infection gradually fades from the population.

Table 3: Sensitivity indices for the reproduction number, R_0

Parameter	Baseline values	References	Sensitivity Index	Remarks
ε_D	0.0018	Hiver, Phineas & Isaac, (2022)	+0.5000	Positive
κ	0.0018	Hiver, Phineas & Isaac, (2022)	+0.9999	Positive
η	0.0078	(7)	+0.0000451	Positive
ϕ	0.00357	Hiver, Phineas & Isaac, (2022)	+0.9999	Positive
τ	0.0055	Hiver, Phineas & Isaac, (2022)	-0.3333	Negative
ε	0.011	(3)	-0.6666	Negative
ω_D	0.3000	Hiver, Phineas & Isaac, (2022)	-0.7143	Positive
μ_4	0.6000	(14)	-0.1429	Negative

The partial ranking correlation coefficient for the sensitivity indices for the reproduction number is presented in figure 3

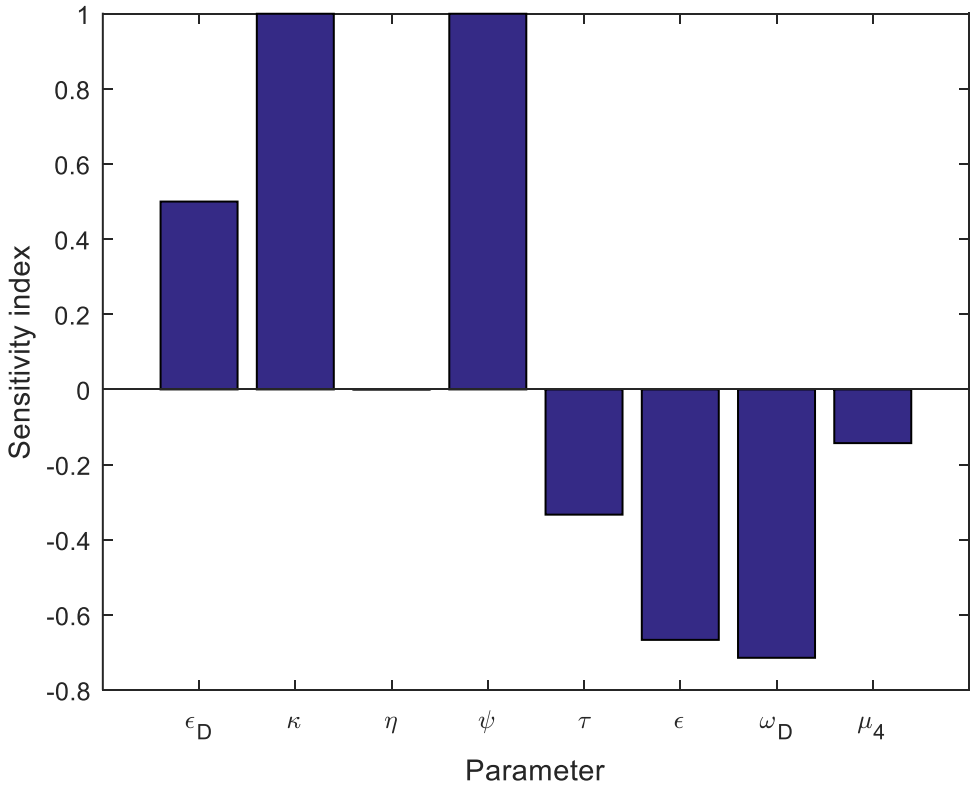

Figure 3: Showing the sensitivity indices for parameters in R_0

The sensitivity indices show the effects of the variation of parameters in relation to the reproduction number. An increase in those parameters with positive values will lead to a corresponding increase in the reproduction number. While an increase in those parameters with negative values will have a reducing effect on the reproduction number.

4.5 Computational results

We performed numerical simulation of the model equations using the inbuilt RK–ode45 function in MATLAB R2021a to study the behavior of the system and effects of the contact rates of both human and animal on the dynamics of leptospirosis infection. The initial condition employed are:

$$S_H(0)=100, L_H(0)=50, I_{AH}(0)=25, I_{SH}(0)=15, H_H(0)=15, R_H(0)=15,$$
$$L_E(0)=1500, S_D(0)=50, E_D(0)=20, I_D(0)=10, R_D(0)=30,$$

Figures 4.1 to 4.18 are the graphs generated from numerical simulations carried out on the model equations.

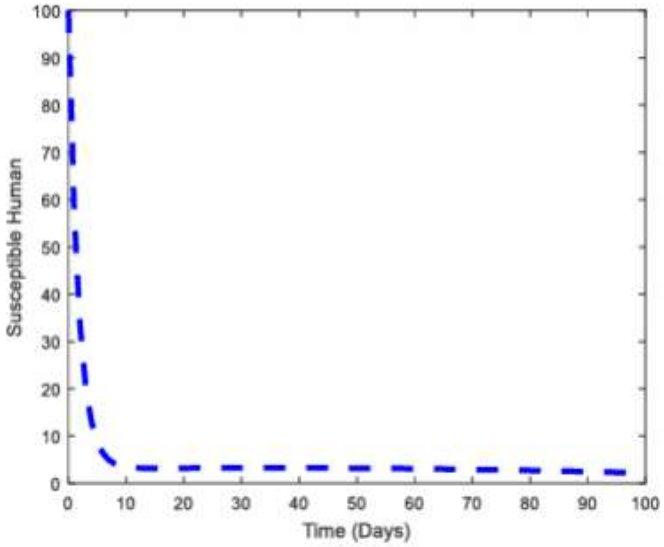

Figure 4.2: Susceptible Humans as a function of time.

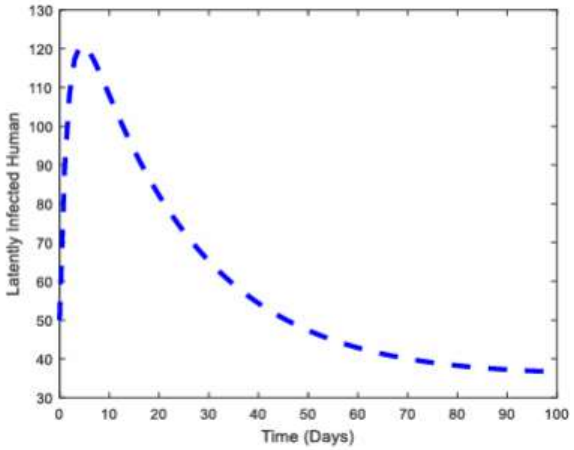

Figure 4.3: Suspected contact with latently infected humans as a function of time

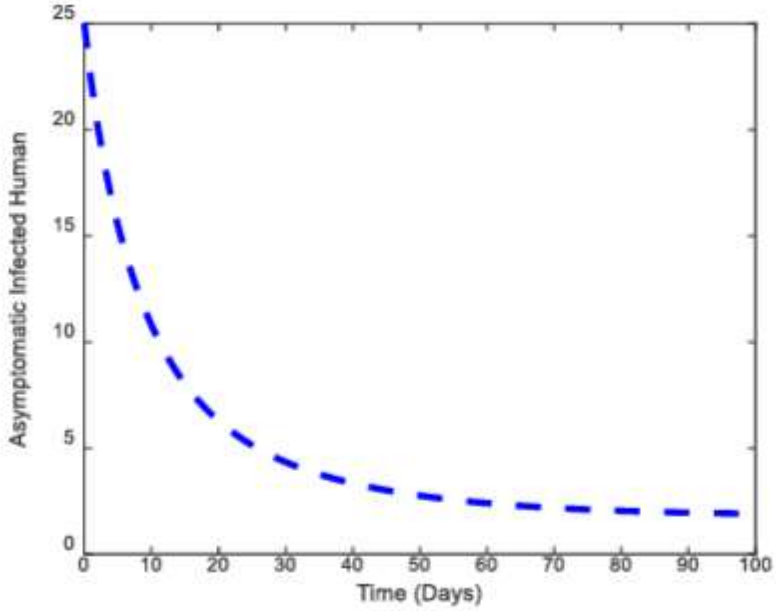

Figure 4.4: Asymptomatic Infected Humans as a function of time t.

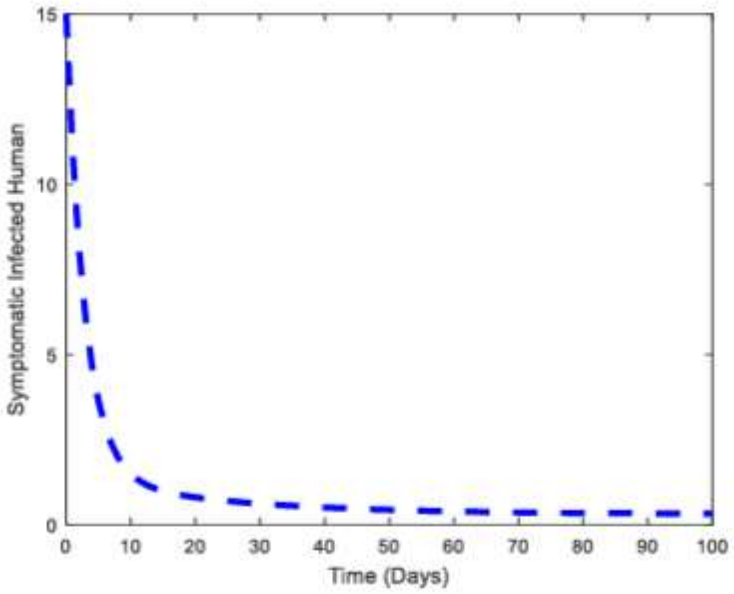

Figure 4.5: Symptomatic Infected Humans as a function of time.

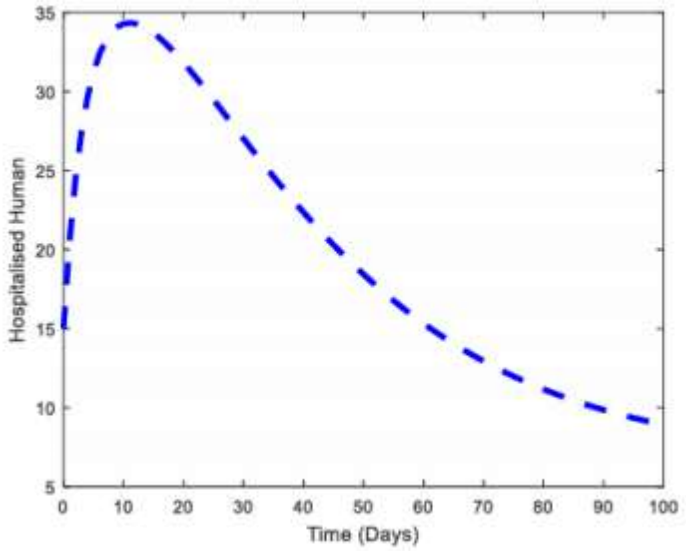

Figure 4.6: Hospitalised Humans as a function of time.

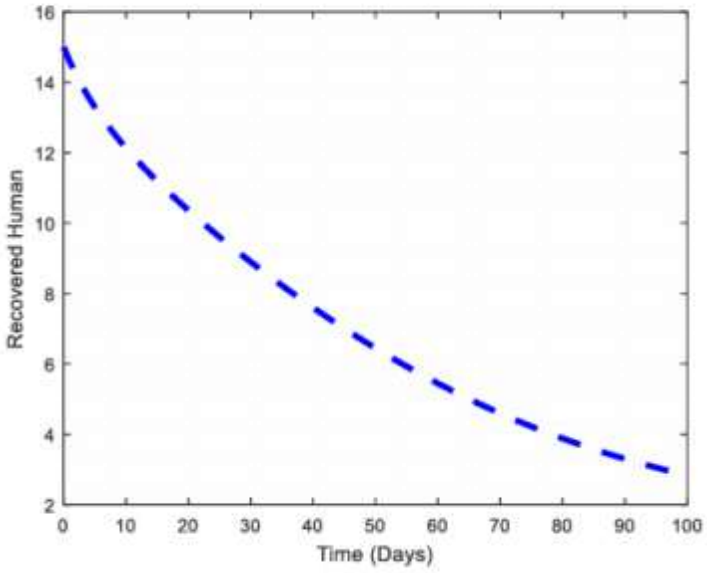

Figure 4.7: Recovered Humans as a function of time.

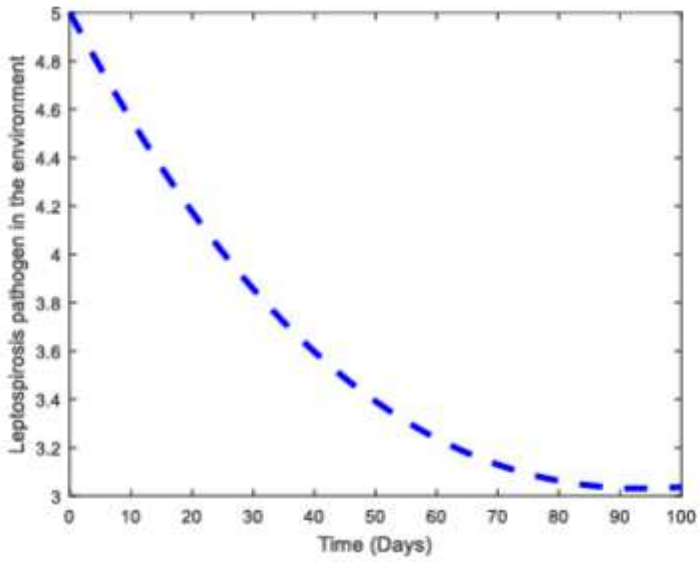

Figure 4.8: Concentration of leptospirosis pathogen in the environment as a function of time.

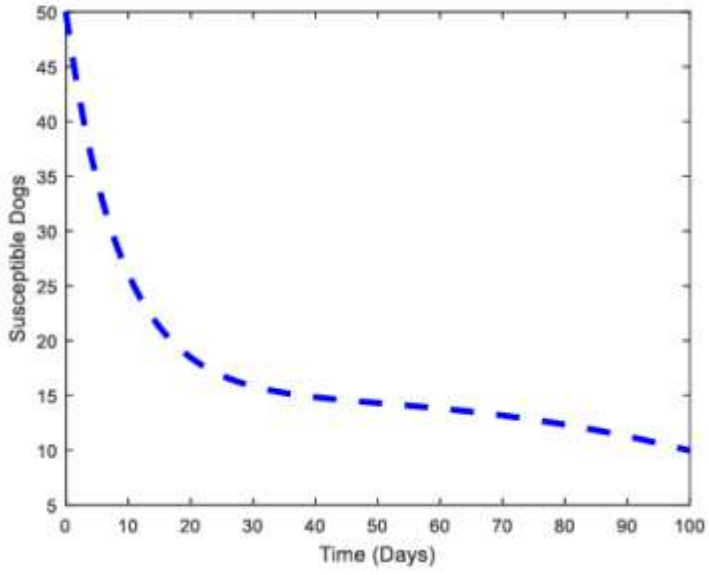

Figure 4.9: Susceptible Dogs as a function of time.

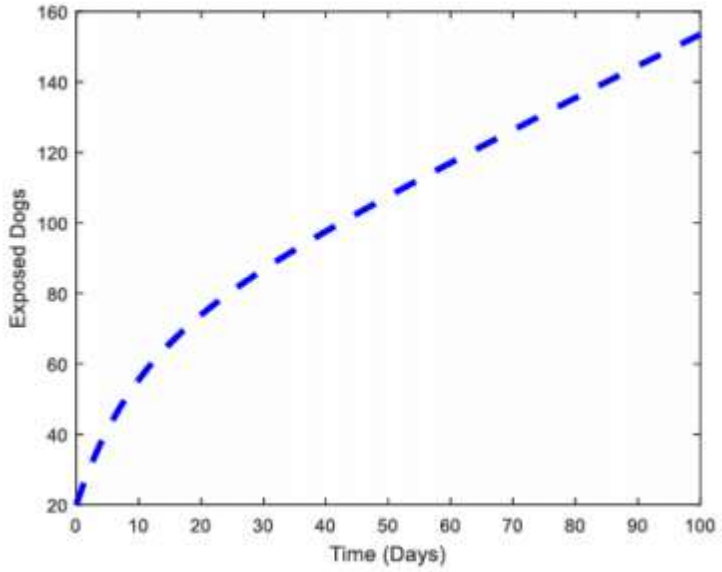

Figure 4.10: Exposed Dogs as a function of time.

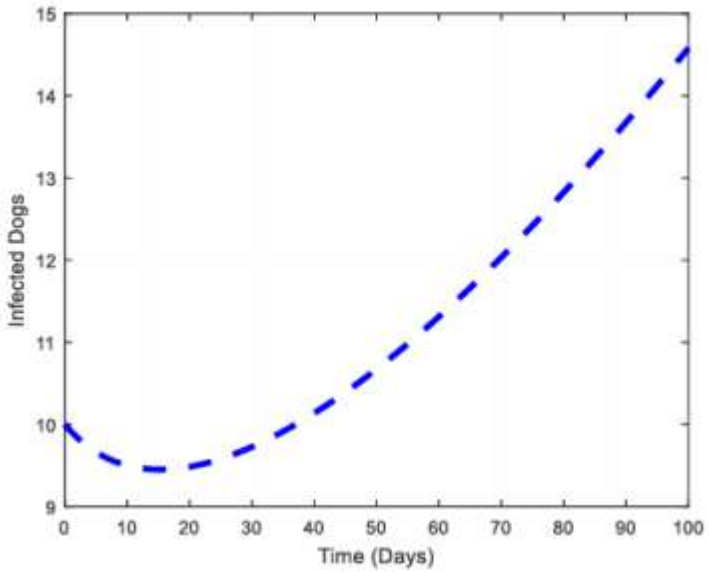

Figure 4.11: Infected Dogs as a function of time

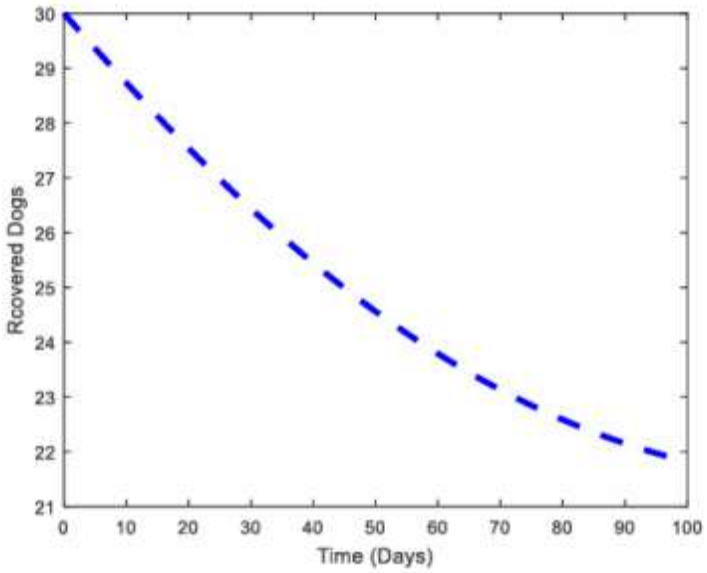

Figure 4.12: Recovered Dogs as a function of time .

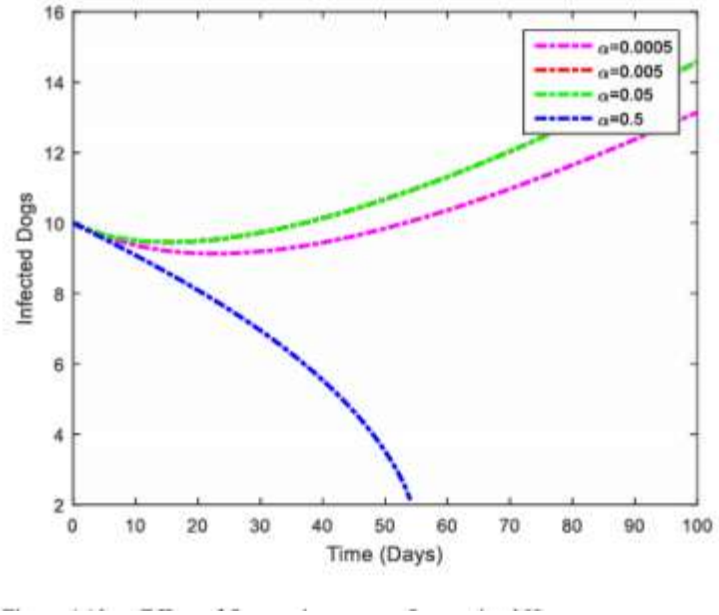

Figure 4.13: Effect of saturation parameters on infected dogs

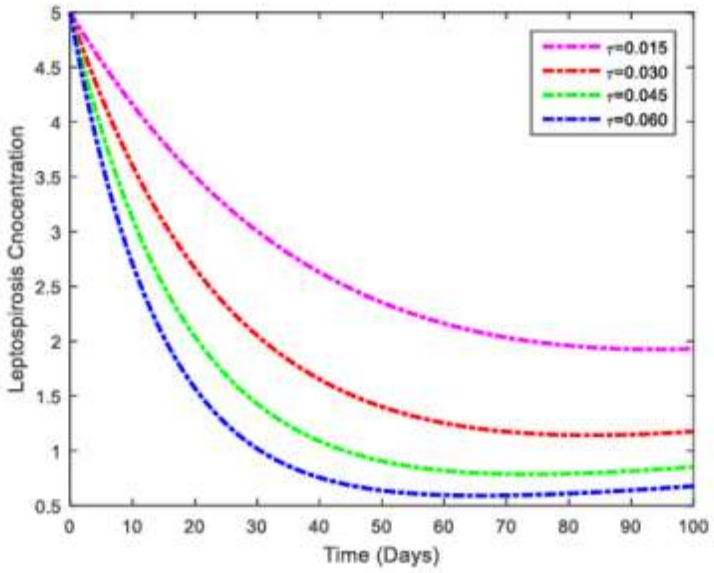

Figure 4.14: Effects of environmental sanitation on leptospirosis concentration

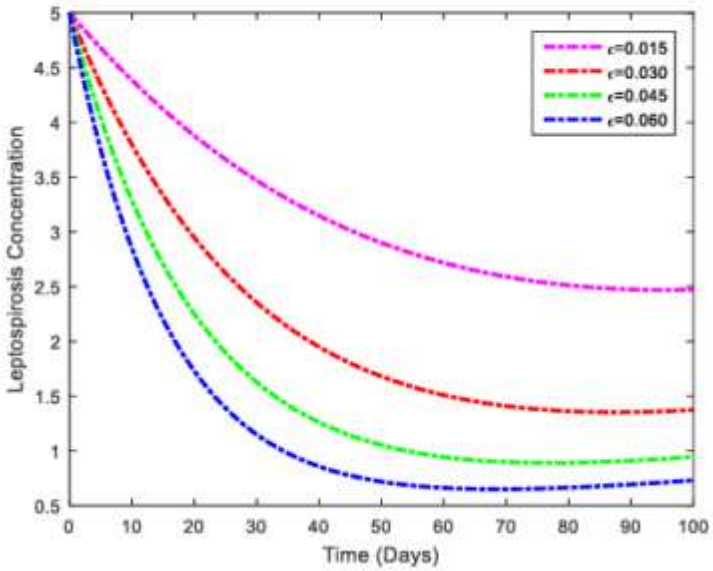

Figure 4.15: Effects of natural decay rate on leptospirosis concentration

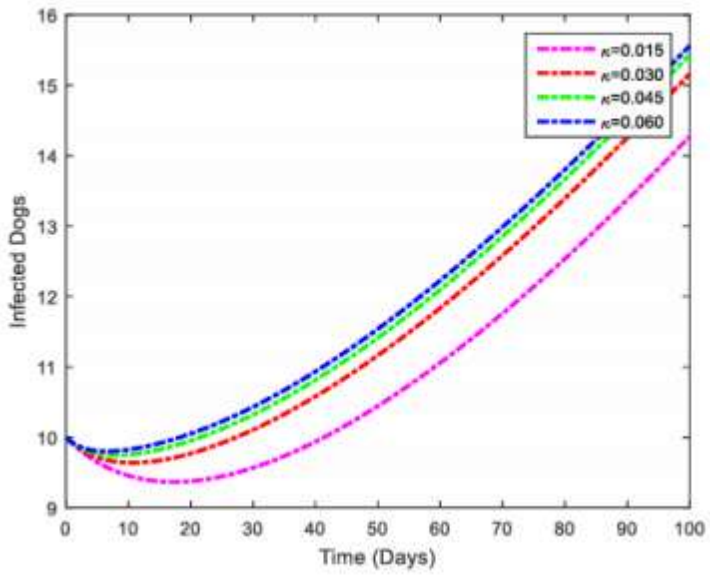

Figure 4.16: Effects of contact rate of susceptible dogs with contaminated environment on infected dogs

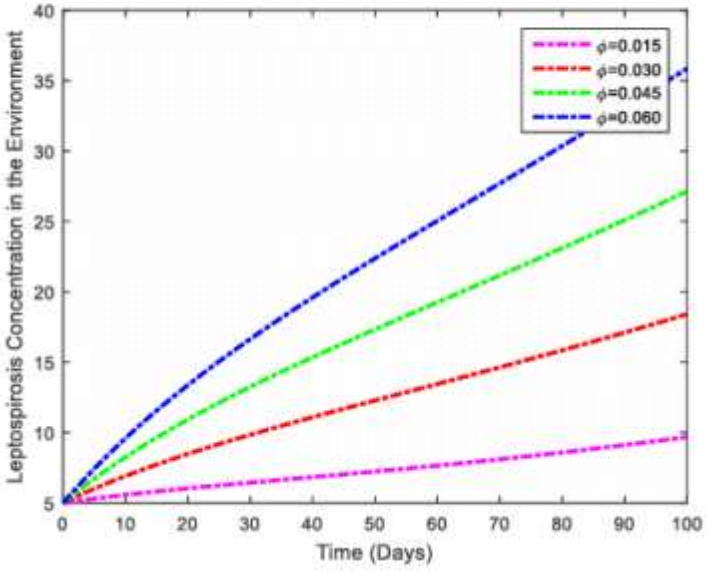

Figure 4.17: Effects of bacteria shedding rate from infected dogs on leptospirosis contami- nation in the environment

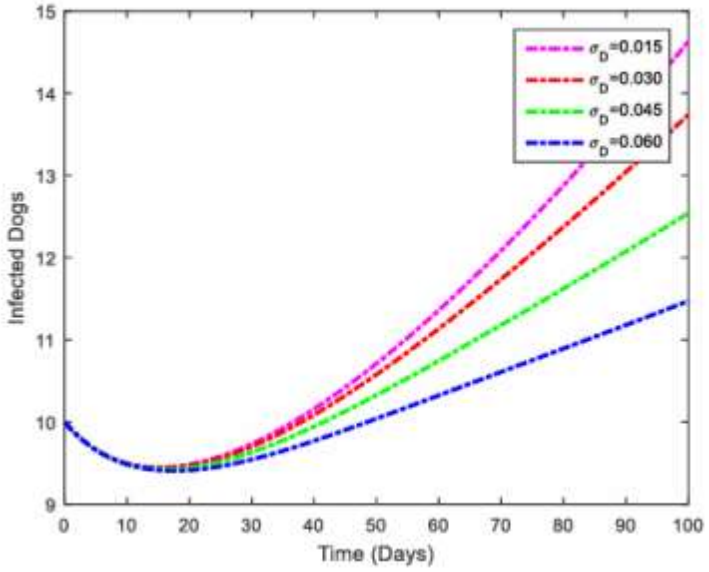

Figure 4.18: Effects of loss of immunity rate in recovered dogs on infected dogs

4.6 Numerical Simulations and Policy Implications

Figures 4.1-4.11 illustrate the temporal dynamics of the human and dog populations together with the environmental pathogen concentration. The susceptible human population (Figure 4.1) decreases rapidly before approaching a steady state, indicating intense disease transmission during the early phase of the outbreak. Biologically, this reflects the rapid depletion of individuals at risk of infection as exposure to contaminated environments and infected dogs increases. The latent human population (Figure 4.2) also declines sharply, suggesting a relatively short incubation period and rapid progression to infectious stages. This implies that infected individuals quickly become capable of transmitting the disease, thereby accelerating the spread of infection.

The asymptomatic infected population (Figure 4.3) and symptomatic infected population (Figure 4.4) both decline rapidly over time, indicating progression to hospitalization or recovery, together with the effects of disease-induced mortality and control interventions. Biologically, this demonstrates that infectious individuals do not remain in these compartments for extended periods, thereby reducing the duration of active transmission when effective treatment and control measures are implemented. Hospitalized individuals (Figure 4.5) increase sharply during the early stages of the outbreak before gradually declining. This early surge reflects the burden placed on healthcare facilities during an epidemic, while the subsequent decline indicates successful treatment, reduced disease transmission, or a decrease in the number of severe infections requiring hospitalization.

The recovered human population (Figure 4.6) decreases steadily over time, suggesting waning immunity and the gradual return of recovered individuals to the susceptible class. From a biological perspective, this indicates that

immunity is temporary, allowing reinfection to occur and creating conditions for recurrent outbreaks if transmission persists. Similarly, the environmental pathogen concentration (Figure 4.7) declines continuously, which may be attributed to natural pathogen decay, environmental sanitation, and reduced bacterial shedding as the number of infectious hosts decreases. Biologically, this reduction lowers the risk of indirect transmission and demonstrates the critical role of environmental management in interrupting the disease cycle.

For the dog population, susceptible dogs (Figure 4.8) decrease rapidly owing to infection pressure from contaminated environments. This indicates that dogs serve as an important reservoir capable of sustaining disease transmission within the population. The exposed dog population (Figure 4.9) initially increases as susceptible dogs become infected and subsequently stabilizes as transmission approaches equilibrium. Biologically, this reflects the continuous recruitment of newly infected dogs into the latent stage before the epidemic reaches a stable state.

The infected dog population (Figure 4.10) exhibits an initial decline followed by a slight resurgence, indicating the possible effects of reinfection, waning immunity, or reduced effectiveness of intervention measures. This behavior suggests that, without sustained control strategies, infection can persist within the reservoir population, thereby maintaining the risk of spillover transmission to humans. The recovered dog population (Figure 4.11) gradually declines, indicating temporary immunity. Biologically, this demonstrates that recovered dogs eventually become susceptible again, replenishing the susceptible reservoir and contributing to the long-term persistence of the disease.

Figures 4.12–4.17 present the sensitivity of the model to changes in selected epidemiological parameters. An increase in the saturation parameter (Figure 4.12) accelerates disease transmission, resulting in earlier epidemic peaks and faster infection spread. Biologically, this indicates that higher effective contact rates increase the speed at which susceptible individuals acquire infection, making outbreaks more difficult to contain.

Increasing the environmental sanitation rate (Figure 4.13) substantially reduces pathogen concentration, demonstrating the effectiveness of sanitation in controlling environmental contamination. This finding highlights environmental sanitation as a key public health intervention capable of reducing indirect transmission and protecting both humans and dogs. Likewise, increasing the natural pathogen decay rate (Figure 4.14) accelerates pathogen removal from the environment, thereby reducing the probability of infection following environmental exposure. Biologically, this suggests that environmental conditions that shorten pathogen survival can significantly suppress disease persistence.

Reducing the contact rate between dogs and contaminated environments, (Figure 4.15) significantly lowers the number of infected dogs. This emphasizes the importance of restricting animal access to contaminated water sources, waste disposal sites, or other environmental reservoirs to interrupt the transmission cycle. Conversely, increasing the bacterial shedding rate (Figure 4.16) elevates environmental pathogen concentration and intensifies disease transmission. Biologically, this indicates that highly infectious hosts disproportionately contaminate the environment, increasing exposure risk and sustaining community transmission.

Finally, increasing the loss of immunity rate (Figure 4.17) causes recovered dogs to return more rapidly to the susceptible class, thereby increasing the pool of susceptible hosts and promoting sustained disease transmission. This

demonstrates that long-lasting immunity is essential for reducing disease persistence and that interventions capable of prolonging immunity, such as effective vaccination, could substantially reduce future outbreaks and enhance disease control.

4.7 Biological and Public Health Implications

These findings demonstrate that effective control of leptospirosis requires a comprehensive One Health approach that simultaneously targets humans, animal reservoirs, and the environment. The simulation results indicate that improving environmental sanitation and waste management substantially reduces environmental pathogen concentration, thereby lowering the risk of indirect transmission. Similarly, increasing the natural decay of leptospiral bacteria in the environment shortens pathogen survival and further limits disease persistence.

The results also show that reducing contact between dogs and contaminated environments through responsible animal management, restricted roaming, proper waste disposal, and improved hygiene practices significantly decreases disease transmission among dogs and, consequently, the risk of human infection. Furthermore, reducing bacterial shedding through early diagnosis, prompt treatment, and effective management of infected dogs minimizes environmental contamination and interrupts the transmission cycle.

The observed effects of waning immunity suggest that maintaining long-term immunity in the dog population is essential for preventing recurrent outbreaks. Consequently, sustained vaccination programmes and regular veterinary surveillance should be considered key components of leptospirosis control strategies. For the human population, timely diagnosis, improved access to treatment, health education, and occupational protection for high-risk groups, and community awareness campaigns can reduce disease transmission, severe infections, and hospitalization.

Overall, the study highlights that isolated interventions are unlikely to eliminate leptospirosis. Instead, coordinated environmental, veterinary, and public health measures are required to achieve sustainable disease control and reduce the burden of leptospirosis in endemic communities.

5 Conclusion

A deterministic compartmental model describing the transmission dynamics of leptospirosis among humans, dogs, and the contaminated environment was developed and analysed. The model successfully captured the interactions between susceptible, exposed, infectious, hospitalized, and recovered human populations, the corresponding dog populations, and the environmental reservoir of leptospiral bacteria.

The disease-free equilibrium was established, and the basic reproduction number, R_0, was derived using the next-generation matrix approach. Local and global stability analyses showed that the disease-free equilibrium is asymptotically stable whenever $R_0 < 1$, implying that leptospirosis can be eliminated if each infectious individual generates, on average, less than one secondary infection. The endemic equilibrium was also shown to be globally asymptotically stable when $R_0 > 1$, indicating persistent disease transmission once the infection becomes established in the population.

Sensitivity analysis identified the parameters that exert the greatest influence on disease transmission. In particular, parameters associated with environmental contamination, bacterial shedding, contact between dogs and contaminated environments, sanitation, pathogen decay, and loss of immunity were found to play significant roles in determining the magnitude and persistence of the outbreak. These findings identify the most effective targets for disease control and provide quantitative guidance for prioritizing intervention strategies.

Numerical simulations further demonstrated the temporal behaviour of the epidemic and confirmed that interventions aimed at reducing environmental contamination, limiting exposure to contaminated environments, decreasing bacterial shedding, and maintaining immunity substantially reduce disease prevalence in both humans and dogs. The results also emphasize the critical role of the environmental reservoir in sustaining transmission, thereby reinforcing the importance of environmental management alongside veterinary and public health interventions.

In conclusion, the proposed model provides valuable insights into the epidemiological dynamics of leptospirosis and offers a quantitative framework for evaluating potential control strategies. The study supports the implementation of integrated One Health interventions that combine environmental sanitation, veterinary surveillance and vaccination, responsible dog management, early diagnosis and treatment, and public health education. Such coordinated measures are expected to reduce disease transmission, minimize environmental contamination, and ultimately lessen the public health and economic burden of leptospirosis in endemic regions. The impact of parameters on the reproduction number: raising a parameter with a positive sensitivity index increases the risk of a disease outbreak, while raising a parameter with a negative index reduces the likelihood of an outbreak. Numerical simulations demonstrate some of the measures that could be taken in order to eradicate the disease from society. The general conclusion of this work is that, to eradicate leptospirosis completely from the system, the best measures to employ are adequate environmental sanitation and proper treatment in both humans and animals.

References

Adler, B. (2015). History of leptospirosis and Leptospira. Current Topics in Microbiology and Immunology, 387, 1–9. https://doi.org/10.1007/978-3-662-45059-8 1

Adler, B., & dela Pen(˜)a Moctezuma, A. (2010). Leptospira and leptospirosis. Veterinary Microbiology, 140(3–4), 287–296. https://doi.org/10.1016/j.vetmic.2009.03.012

Ayoade A. A. A., Oyedepo, T. & Agunbiade, S. (2023). Mahamatical modeling of Tox- oplasma Gundii between the environment and Cat Population under vaccination and Sanitation. *Journal of Fractional Calculus and Applications*, 14(1), 75-87,

Bharti, A. R., Nally, J. E., Ricaldi, J. N., Matthias, M. A., Diaz, M. M., Lovett, M. A.,

. . . Vinetz, J. M. (2003). Leptospirosis: A zoonotic disease of global importance. *The Lancet Infectious Diseases,* 3(12), 757–771. Retrieved from https://doi.org/10.1016/S1473- 3099(03)00830-2

Birkhoff, G., Rota, G. C., & Birkhoff, G. (1989). Ordinary differential equations (4thed.). Wiley.

Blower, S. M. & Dowlatabadi, H. (1994). Sensitivity and Uncertanty Analysis of Com- plex models of Disease Transmission: An HIV model as an Example. *International Statistical Review* 62(2), 229-243. https://doi.org/10.1002/14651858.CD007342.pub2

Brhane, K. W., Ahmad, A. G., Hina, H., & Emadifar, H. (2024). Mathematical modeling of cholera dynamics with intrinsic growth considering constant interventions. *Scientific Reports*, 14, 4616. https://doi.org/10.1038/s41598-024-55240-0

Britton, N. F. (2003). Essential Mathematical Biology. [Springer]. [London].

Castillo-Chavez, C., & Song, B. (2004). Dynamical models of tuberculosis and their applications. *Mathematical Biosciences and Engineering*, 1(2), 361–404. https://doi.org/10.3934/mbe.2004.1.361

Castillo-Chavez, C., Blower, S., van den Driessche, P., Kirschner, D., & Yakubu, A. A. (2002). Mathematical approaches for emerging and reemerging infectious diseases: An introduction. Springer.

Costa, F., Hagan, J. E., Calcagno, J., Kane, M., Torgerson, P., Martinez-Silveira, M. S., . . . Ko, A. I. (2015). Global morbidity and mortality of leptospirosis: A systematic review. *PLoS Neglected Tropical Diseases,* 9(9), e0003898. https://doi.org/10.1371/journal.pntd.0003898

Coutinho, M. L., Choy, H. A., Kelley, M. M., Matsunaga, J., Babbitt, J. T., Lewis, M. S., Aleixo, J. A. G., & Haake, D. A. (2011). A LigA three-domain region protects hamsters from lethal infection by Leptospira interrogans. *PLoS Neglected Tropical Diseases,* 5*(12), e1422. https://doi.org/10.1371/journal.pntd.0001422DiStefano, J. J., III, Stubberud, A. R., & Williams, I. J. (2010). Schaum's outline of feedback and control systems (2nd ed.). McGraw-Hill.

Ellis, W. A. (2014). Animal leptospirosis. In B. Adler (Ed.), *Leptospira and leptospirosis* (pp. 99–137). Springer. https://doi.org/10.1007/978-3-662-45059-8_6

El-Marhomy, A., E. & Abdel Sattar, N. E. (2004). Stability analysis of rotor-bearing sys- tems via Routh-Hurwitz Criterion. [Applied Energy, 77(3), 287-308].

Engida, H. A., Theuri, D. M., Gathungu, D., Gachohi, J., & Alemneh, H. T. (2022). A mathematical model analysis for the transmission dynamics of leptospirosis disease in human and rodent populations. *Computational and Mathematical Methods in Medicine*, 2022*, Article 1806585. https://doi.org/10.1155/2022/1806585].

Ganoza, C. A., Matthias, M. A., Saito, M., Cespedes, M., Gotuzzo, E., & Vinetz, J. M. (2006). Asymptomatic leptospiruria in periurban slum communities in Iquitos, Peru. *International Journal of Infectious Diseases*, 10(6), 501–507. https://doi.org/10.1016/j.ijid.2006.02.005

Torgerson, P. R., Hagan, J. E., Costa, F., Calcagno, J., Kane, M., Martinez-Silveira, M. S., Goris, M. G. A., Stein, C., Ko, A. I., & Abela-Ridder, B. (2015). Global burden of leptospirosis: Estimated in terms of disability adjusted life years. *PLoS Neglected Tropical Diseases,* 9(10), e0004122. https://doi.org/10.1371/journal.pntd.0004122Haake, D. A., & Levett, P. N. (2015). Leptospirosis in humans. Current Topics in Microbi- ology and Immunology, 387, 65–97. https://doi.org/10.1007/978-3-662-45059-8 5

Haake, D. A., & Levett, P. N. (2015). Pathogenesis of leptospirosis. *Current Topics in Mi- crobiology and Immunology,* 387, 141–163. https://doi.org/10.1007/978-3-662-45059- 8 7

Haake, D. A., Suchard, M. A., Kelly, M. M., Dundoo, M., Alt, D. P. & Zuerner, R. L. (2004). Molecular Evolution and Mosaicsm of Leptospiral outer membrane proteins involves horizontal DNA transfer. *Journal of Bacteriology,* 186(9), , 2818-2828.

Hiver, D. N., Phineas, R. K. & Isaac, C. (2022). A Fractional Order Model of Leptospirosis Transmission Dynamic with Enviromental Compartment. *Global Journal of Pure and Applied Mathematics*. 18(1), 18–110.

Hussaini, M. Y. (2010). Applied differential equations: The primary course. Springer.

Ko, A. I., Goarant, C., & Picardeau, M. (2009). Leptospira: The dawn of the molecular genetics era for an emerging zoonotic pathogen. *Nature Reviews Microbiology,* 7(10), 736–747. https://doi.org/10.1038/nrmicro2208

Lau, C. L., Smythe, L. D., Craig, S. B., & Weinstein, P. (2010). Climate change, flooding, urbanisation and leptospirosis: Fuelling the fire? Transactions of the Royal Society of Tropical Medicine and Hygiene, 104(10), 631–638. https://doi.org/10.1016/j.trstmh.2010.07.002

Levett, P. N. (2001). Leptospirosis Clinical Microbiology Reviews, 14(2), 296–326. https://doi.org/10.1128/CMR.14.2.296-326.2001

Levett, P. N., & Haake, D. A. (2015). Leptospira species (Leptospirosis). In J. E. Ben- nett, R. Dolin, & M. J. Blaser (Eds.), Mandell, Douglas, and Bennett'sprinciples and practice of infectious diseases (8thed., pp. 3055–3061). Elsevier.

LaSalle, J. P. (1986). The Stability and Control of Discrete Processes. Springer.

Li, M.-T., Sun, G.-Q., Zhang, W.-Y., & Jin, Z. (2017). Model-based evaluation of strategies to control brucellosis in China. *International Journal of Environmental Research and Public Health*, 14(3), 295. https://doi.org/10.3390/ijerph14030295Maze,

Meeyam, T., Tablerk, P., Petchanok, B., Pichpol, D., & Padungtod, P. (2006). Seropreva- lence and risk factors

associated with leptospirosis in dogs. Southeast Asian Journal of Tropical Medicine and Public Health, 37(1), 148–153.

Mwachui, M. A., Crump, L., Hartskeerl, R., Zinsstag, J., & Hattendorf, J. (2015). Envi- ronmental and behavioural determinants of leptospirosis transmission: A systematic review. *PLoS Neglected Tropical Diseases*, 9(9), e0003843. https://doi.org/10.1371/journal.pntd.0003843

Nur, W., Trisilowati, Suryanto, A., & Kusumawinahyu, W. M. (2021). Schistosomiasis model incorporating snail predator as biological control agent. *Mathematics,* 9(16), 1858. https://doi.org/10.3390/math9161858.

Panaphut, T., Domrongkitchaiporn, S., & Thinkamrop, B. (2003). Prognostic factors of death in leptospirosis: A prospective study in Khon Kaen, Thailand. *International Journal of Infectious Diseases*, 7(2), 111–114. https://doi.org/10.1016/S1201-9712(03)90005-2

Picardeau, M. (2013). Diagnosis and epidemiology of leptospirosis. *M´(e)decine et Maladies Infectieuses,* 43(1), 1–9. https://doi.org/10.1016/j.medmal.2012.11.005

Pimpunchat, B., Wake, G. C., Modchang, C., Triampo, W., & Babylon, A. M. (2013). Mathematical modeling of leptospirosis: Linearization Solutions and Stability Analy- sis. [Applied Mathematics, 4(10B)), 77-84].

Podder, C. N., & Gumel, A. B. (2009). Mathematical study of a model for the dynamics of tuberculosis. *Applied Mathematical Modelling*, 33(9), 4158–4173. https://doi.org/10.1016/j.apm.2008.12.016

Rambaud-Althaus, C., Althaus, F., Genton, B., & D'Acremont, V. (2015). Clinical features for diagnosis of pneumonia in children younger than 5 years: A systematic review and meta-analysis. *The Lancet Infectious Diseases,* 15(4), 439–450. https://doi.org/10.1016/S1473-3099(15)70017-4.

Reis, R. B., Ribeiro, G. S., Felzemburgh, R. D., Santana, F. S., Mohr, S., Melendez, A. X., . . . Ko, A. I. (2008). Impact of environment and social gradient on Leptospira infection in urban slums. *PLoS Neglected Tropical Diseases,* 2(4), e228. https://doi.org/10.1371/journal.pntd.0000228

Sohail, M. L., Khan, M. S., Ijaz, M., Naseer, O., Fatima, Z., Ahmad, A. S., & Ahmad, W. (2018). Seroprevalence and risk factor analysis of human leptospirosis in distinct climatic regions of Pakistan. *Acta Tropica, 181*, 79–83. https://doi.org/10.1016/j.actatropica.2018.01.021Strogatz, S. H. (1988). *Collective Dynamics of Coupled Non-linear Oscilators*: [Physica D, 31, 143-168].

Takafuji, E. T., Kirkpatrick, J. W., Miller, R. N., Karwacki, J. J., Kelley, P. W., Gray, M. R., . . . Sanchez, J. L. (1984). An efficacy trial of doxycycline prophylaxis against leptospirosis. *New England Journal of Medicine,* 310(8), 497–500. https://doi.org/10.1056/NEJM198402233100804

Taylor, A. J., Paris, D. H., & Newton, P. N. (2020). A systematic review of the mortality from untreated leptospirosis. *PLoS Neglected Tropical Diseases*, 14(8), e0008628.

https://doi.org/10.1371/journal.pntd.0008628

Torgerson, P. R., Hagan, J. E., Costa, F., Calcagno, J., Kane, M., Martinez-Silveira, M. S., . . . Ko, A. I. (2015). Global burden of leptospirosis: Estimated in terms of disability-adjusted life years. *PLoS Neglected Tropical Diseases*, 9(10), e0004122. https://doi.org/10.1371/journal.pntd.0004122.

Trueba, G., Zapata, S., Madrid, K., Cullen, P., & Haake, D. (2004). Cell aggregation: A mechanism of pathogenic Leptospira to survive in fresh water. *International Microbiology*, 7(1), 35–40.

Van den Driessche, P., & Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences*, 180(1–2), 29–48. https://doi.org/10.1016/S0025-5564(02)00108-6

Victoriano,A. F. B., Smythe,L. D., Gloriani-Barzaga, N., Cavinta,L. L., Kasai,T., Limpakarn- janarat, K., . . . Yanagihara, Y. (2009). Leptospirosis in the Asia Pacific region. *BMC Infectious Diseases*, 9, 147. https://doi.org/10.1186/1471-2334-9-147

Vijayachari, P., Sugunan, A. P., & Shriram, A. N. (2008). Leptospirosis: An emerging global public health problem. *Journal of Biosciences*, 33(4), 557–569. https://doi.org/10.1007/s12038-008-0074-z