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Modeling the effects of screening, vaccination, treatment, and non-pharmaceutical controls on leptospirosis transmission dynamics

Janeth Michael[1,2†], Herieth Rwezaura[1], Goodluck Mlay[1], Nyimvua Shaban Mbare[1]

[1]*Department of Mathematics, University of Dar es Salaam, P.O. Box 35062, Dar es Salaam, Tanzania.*
[2]*National Institute of Transport, P.O. Box 705, Dar es Salaam, Tanzania.*

Keywords

Leptospirosis; *Leptospira* Bacteria; Stability Analysis; Effective Reproduction Number; Non-pharmaceutical controls.

Abstract

Leptospirosis is the world's most common zoonotic disease and remains a significant public health challenge, particularly in regions with poor sanitation and high exposure risks. This study aims to evaluate the effectiveness of combined interventions: screening, vaccination, treatment, and non-pharmaceutical controls in reducing leptospirosis transmission in human and domestic animal populations. The study develops and analyzes a deterministic compartmental model with eleven compartments representing human, domestic animal, and bacterial populations. Stability analysis reveals that the Disease-Free Equilibrium (DEF) is globally asymptotically stable when the effective reproduction number is less than a unit ($R_e < 1$), ensuring disease eradication under these conditions, whereas the Endemic Equilibrium Point (EEP) remains stable when $R_e > 1$. The study employs the normalized forward sensitivity index to determine the most influential parameters affecting disease dynamics and shows that an increase in the transmission of infections from a contaminated environment to susceptible domestic animals, transmission of infections from a contaminated environment to susceptible humans, contact between infected animals and susceptible humans, and contact among domestic animals ($\beta_1, \beta_2, \beta_3, \beta_4$), respectively with humans recruitment rate (ϖ_h), significantly contributes to a higher basic reproduction number R_0, leading to a higher induced infection rate as more people become infected. While parameters related to bacterial decay $(\mu \| b)$, natural mortality rates (μ_h, μ_d), and disease-induced mortality (δ_h) were found to be negatively influencing R_0. Furthermore, numerical simulations demonstrate that implementing a combination of screening, vaccination, treatment, and non-pharmaceutical interventions substantially reduces the reproduction number and disease prevalence compared to implementing single interventions. The findings emphasize that controlling leptospirosis requires an integrated, multi-faceted strategy, including environmental sanitation, reservoir control, fencing, the use of proper protective equipment (PPE), and public health education to reduce exposure risks. The study provides a robust mathematical foundation to support policymakers in developing integrated public health strategies aimed at achieving long-term disease reduction.

†† Corresponding author(s). E-mail(s): janeth.mtabazi2021@gmail.com
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Introduction

Leptospirosis is a zoonotic disease caused by pathogenic spirochetes of the genus *Leptospira*. It affects both humans and animals and remains a major public health challenge worldwide. Transmission occurs through direct contact with the urine of infected animals or indirectly via contaminated water, soil, or food, where the bacteria can persist for weeks to months (Adler and de la Peña Moctezuma 2010, Paisanwarakiat and Thamchai 2021). Seasonal patterns of rainfall and flooding amplify its spread by creating favorable environmental conditions for bacterial survival and dissemination (WHO 2009, Lau et al. 2010, Desvars et al. 2011, Mughini-Gras 2014). Entry into the human body typically occurs through broken skin or mucous membranes of the eyes, nose, or mouth (Paisanwarakiat and Thamchai 2021), with an incubation period ranging from 7 to 12 days (Gomes-Solecki et al. 2017)with high occupational exposure such as agricultural workers in rice and sugar plantations, veterinarians, slaughterhouse staff, sewer cleaners, rescue workers, fishermen, and military personnel face greater risk (Paisanwarakiat and Thamchai 2021, Okosun et al. 2016, WHO 2009). Clinical presentation in humans varies from mild influenza-like symptoms (fever, muscle pain, headache, nausea, diarrhea, abdominal pain, and skin rashes) to severe manifestations, including jaundice, meningitis, renal and hepatic failure, hemorrhage, and respiratory distress (Khan et al. 2014). In domestic animals, leptospirosis causes reduction in milk yield, abortions, stillbirths, weak offspring, and low growth rate (Engida et al. 2022, Sykes et al. 2022, Cilia 2020). These effects encompass companion animals (dogs, cats), farmed livestock (cattle, sheep, goats), and draft animals (horses, donkeys, camels). Globally, leptospirosis accounts for approximately one million cases and nearly 60,000 deaths annually, with incidence rates reaching 10–100 cases per 100,000 people in tropical regions (De Vries et al. 2014, Felix et al. 2020). High-burden regions include South and Southeast Asia, the Caribbean, Latin America, and sub-Saharan Africa (Holt et al. 2006). In Tanzania, cases are concentrated in areas of intense human-livestock-wildlife interaction (Maze et al. 2018, Mgode et al. 2021, Katandukila et al. 2023). A notable outbreak occurred in July 2022 in Lindi, with 20 confirmed cases and three deaths (Masunga et al. 2022).

Despite its burden, an effective universal vaccine for leptospirosis has not been achieved. Bacterin-based vaccines are available but provide only serovar-specific protection (Koizumi and Watanabe 2005). Therefore, control strategies rely on treatment, early screening, and non-pharmaceutical measures such as public health education, reservoir control, protective gear (rubber boots, waterproof gloves, goggles), and hygiene improvement (Engida et al. 2022). Socioeconomic conditions strongly influence transmission dynamics: poor sanitation, inadequate drainage, overcrowding, and limited awareness in low-income communities increase risk and delay treatment access (Baca-Carrasco et al. 2015, Ojo et al. 2021, Batchelor 2012, Zaman et al. 2012). Rodent proliferation in such environments further exacerbates spread. Several mathematical models have been developed to study leptospirosis dynamics and interventions, often focusing separately on humans or animal reservoirs. For instance, Okosun et al. (2016) evaluated prevention, vaccination, and treatment among livestock, concluding that while animal vaccination and treatment are effective, they may not be cost-effective. Eguda et al. (2023) extended this work by advocating for human vaccination to prevent reinfection. Aslan (2019) assessed cattle interventions and found that biannual vaccination was more effective for eradication than annual measures, particularly when targeting adult cattle. Felix et al. (2020) reviewed emerging human vaccine candidates but did not explore vector-focused approaches. While these studies provide valuable insights, most restrict their analysis to either the human or vector (livestock, rodents, dogs) population. Few models integrate interventions across humans, domestic animals, and environmental

reservoirs simultaneously. To address gaps in existing models, this study develops a novel deterministic model extending the frameworks of Okosun et al. (2016), Bhalraj (2019), Engida et al. (2022) and Eguda et al. (2023). This model simultaneously evaluates the impact of screening, vaccination, treatment, and non-pharmaceutical controls on human and domestic animal populations, incorporating environmental pathogen concentrations and interactions using systems of ordinary differential equations.

Model Formulation

Using a system of nonlinear differential equations, this study builds up a compartmental mathematical model for the transmission process of the leptospirosis epidemic, considering the basic model in (Engida et al. 2022). The model includes human, vector (domestic animal), and bacterial populations. The model describes division of human population at time t, denoted by $N_h(t)$ into susceptible $S_h(t)$, vaccinated $V_h(t)$, exposed $E_h(t)$, asymptomatic $A_h(t)$, symptomatic $I_h(t)$ and recovered $R_h(t)$, so that $N_h(t)=S_h(t)+V_h(t)+E_h(t)+A_h(t)+I_h(t)+$. Similarly, the population of domestic animals $N_d(t)$ is divided into susceptible animals $S_d(t)$, vaccinated $V_d(t)$, infected animals $I_d(t)$ and those who have recovered from the disease $R_d(t)$, implying that $N_d(t)=S_d(t)+V_d(t)+I_d(t)+R_d(t)$. An additional compartment $B(t)$ representing the concentration of bacteria in the environment has also been incorporated in the model. Contents in $B(t)$ can increase by the shading of pathogens from infected humans and infectious domestic animals at the rates ϕ_h and ϕ_d respectively. Susceptibles from the humans and animals are vaccinated at the rates θ_h and θ_d respectively. Non-pharmaceutical interventions denoted by u are also put in place to minimize the spread of the disease. Non-pharmaceutical controls include public health education campaigns like controls of reservoir animal population, using appropriate protective gears (rubber boots, waterproof, goggles, and gloves), and general hygiene. It is also assumed that infected humans and domestic animals are treated at the rates α_h and $\alpha_{d,}$ respectively. Asymptomatic individuals become symptomatic by screening at the rate ξ_h. The force of infection for humans is

$$\lambda_h=\lambda_{hd}+\lambda_{hB}=\frac{\beta_3(1-u)I_d}{N_d}+\frac{\beta_2(1-u)B}{(B+b)},$$

since $\lambda_{hd}=\frac{\beta_3(1-u)I_d}{N_d}$, and $\lambda_{hB}=\frac{\beta_2(1-u)B}{(B+b)}$. Similarly force of infection for domestic animals is given by λ_d

$$=\lambda_{dd}+\lambda_{dB}=\frac{\beta_4(1-u)I_d}{N_d}+\frac{\beta_1(1-u)B}{(B+b)},$$

since $\lambda_{dd}=\frac{\beta_4(1-u)I_d}{N_d}$, and $\lambda_{dB}=\frac{\beta_1(1-u)B}{(B+b)}$. Parameter b represents the concentration of bacteria, β_2 is the transmission of infections from a contaminated environment to susceptible humans, and β_3 represents the transmission of infections from infected domestic animals to susceptible humans. β_1 represents transmission of infections from a contaminated environment to susceptible domestic animals, and β_4 represents transmission of infections among domestic animals. Parameters ϵ_h and ϵ_d represent vaccination efficacy in human and domestic animals, implying that vaccinated humans and animals may eventually contract the infections at the reduced rates of $(1-\epsilon_h)\lambda_h$ and $(1-\epsilon_d)\lambda_d$ respectively.

In formulating the model, we assume all parameters are non-negative. Susceptible humans enter the population via migration at a constant rate ϖ_h, whereas susceptible

domestic animals are recruited through births at rate ϖ_d. Humans acquire infection through two routes; direct contact with infected domestic animals and exposure to contaminated environmental sources under a homogeneous mixing framework between human and domestic animal populations. Because human-to-human transmission of leptospirosis is rare, it is neglected. For domestic animals, once infected they either die of the disease or remain infected unless treated; thus, recovery occurs only through treatment. We further consider a free-range management system for domestic animals and do not include animal migration.

Other parameters in model system 1 are described in Table 1.

The dynamics of the disease can be visualized in Figure 1:

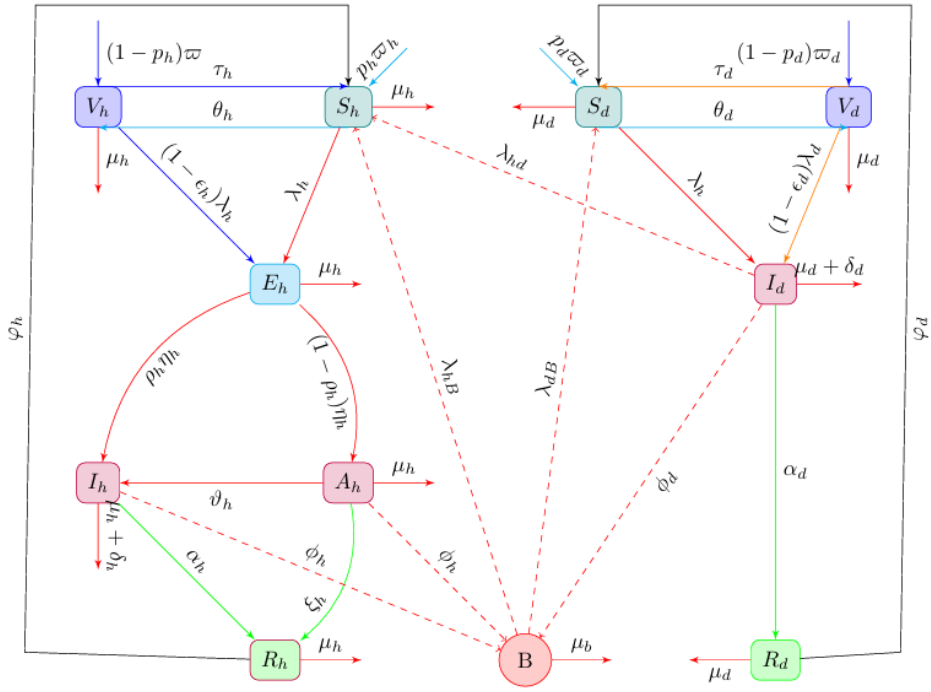


Figure 1: Transfer diagram for the leptospirosis disease model

Table1: Description of Parameters of Model (1)

Parameter	Description	Value	Unit	Source
ϖ_h, ϖ_d	Recruitment rates	1.74; 1.34	$Da\,y^{-1}$	Okosun et al. (2016)
p_h, p_d	Proportion of recruitment without vaccination	3×10^{-5}; 5	Dimensionless	Estimated
β_1	Transmission rate from contaminated environment to domestic animals	0.12	$Da\,y^{-1}$	Aslan (2019)
β_2	Transmission rate from contaminated environment to humans	0.004	$Da\,y^{-1}$	Baca-Carrasco et al. (2015)
β_3	Transmission rate from infected domestic animals to humans	0.033	$Da\,y^{-1}$	Engida et al. (2022)

Parameter	Description	Value	Unit	Source
β_4	Transmission rate among domestic animals	0.05	Day^{-1}	Baca-Carrasco et al. (2015)
α_h, α_d	Treatment rates	0.072; 0.064	Day^{-1}	Engida et al. (2022)
μ_h, μ_d, μ_b	Natural death rates for humans, domestic animals, and bacteria	0.062; 0.018; 0.11	Day^{-1}	Okosun et al. (2016); Aslan (2019)
b	Concentration of bacteria in the environment	10000	*CFU/mL*	Aslan (2019)
ϑ_h	Human screening rate	0.067	Day^{-1}	Estimated
δ_h, δ_d	Disease-induced death rates	0.04; 0.01	Day^{-1}	Okosun et al. (2016)
ϕ_h, ϕ_d	Shedding rate from infected individuals to the environment	0.8; 0.06	Day^{-1}	Engida et al. (2022)
φ_h, φ_d	Temporary recovery rates	0.089; 0.83	Day^{-1}	Engida et al. (2022)
θ_h, θ_d	Vaccination rates	0.02; 0.03	Day^{-1}	Estimated
τ_h, τ_d	Vaccination waning rates	0.004; 0.013	Day^{-1}	Eguda et al. (2023); Zaman et al. (2012)
$\varepsilon_h, \varepsilon_d$	Vaccination efficacy	0.7;0.5	Dimensionless	Estimated
ξ_h	Asymptomatic natural recovery rate	0.05	Day^{-1}	Estimated
η_h	Progression rate from exposed humans to asymptomatic and symptomatic stages	0.0902	Day^{-1}	Engida et al. (2022)
ρ_h	Proportion transitioning from exposed to infectious	0.00056	Dimensionless	Estimated
u	Non-pharmaceutical control rate	0.58	Dimensionless	Estimated

Based on the above descriptions and assumptions, the model is formulated as the following system of ordinary differential equations:

$$
\begin{aligned}
dS_h/dt &= p_h\omega_h + \varphi_h R_h + \tau_h V_h - (\mu_h + \lambda_h + \theta_h)S_h \\
dV_h/dt &= (1-p_h)\omega_h + \theta_h S_h - ((1-\varepsilon_h)\lambda_h + \mu_h + \tau_h)V_h \\
dE_h/dt &= \lambda_h S_h + (1-\varepsilon_h)\lambda_h V_h - (\eta_h + \mu_h)E_h \\
dA_h/dt &= (1-\rho_h)\eta_h E_h - (\mu_h + \xi_h + \vartheta_h)A_h \\
dI_h/dt &= \rho_h\eta_h E_h + \vartheta_h A_h - (\mu_h + \alpha_h + \delta_h)I_h \\
dR_h/dt &= \alpha_h I_h + \xi_h A_h - (\mu_h + \varphi_h)R_h \\
dS_d/dt &= p_d\omega_d + \varphi_d R_d + \tau_d V_d - (\mu_d + \lambda_d + \theta_d)S_d \\
dV_d/dt &= (1-p_d)\omega_d + \theta_d S_d - ((1-\varepsilon_d)\lambda_d + \mu_d + \tau_d)V_d \\
dI_d/dt &= \lambda_d S_d + (1-\varepsilon_d)\lambda_d V_d - (\mu_d + \alpha_d + \delta_d)I_d \\
dR_d/dt &= \alpha_d I_d - (\mu_d + \varphi_d)R_d \\
dB/dt &= \phi_h A_h + \phi_h I_h + \phi_d I_d - \mu_b B
\end{aligned}
\qquad (1)
$$

$S_h(0)>0, V_h(0)>0, E_h(0)\geq 0, A_h(0)\geq 0, I_h(0)\geq 0, R_h(0)\geq 0, S_d(0)>0, V_d>0, I_d(0)\geq 0, R_d(0)\geq 0, B(0)\geq 0.$

The initial conditions of the model are:

Model Analysis

Positive Invariant Region of the System

The focus of this section is to ensure the biological and mathematical relevance of the model system 1. We investigate all necessary state variables of the model to ensure that they remain positive throughout the whole duration of the model simulation $\forall\, t \geq 0$. In this part, the basic properties of the model system 1 were analyzed. The model is biologically and mathematically meaningful when its solutions are positive and bounded.

Theorem 1 (Positivity): Let the initial conditions of the model system be:

$S_h>0, V_h>0, E_h\geq 0, A_h\geq 0, I_h\geq 0, R_h\geq 0,$

$S_d>0, V_d>0, I_h\geq 0, R_d\geq 0, B\geq 0.$

Then, for all t > 0, the solutions $S_h, V_h, E_h, A_h, I_h, R_h, R_h, S_d, V_d, I_d, B$ of the model system (1) will remain positive.

Proof: Considering the equations of the model system (1):

The first equation is:

$dS_h/dt = p_h\omega_h + \phi_h R_h + \tau_h V_h - (\mu_h + \lambda_h + \theta_h) S_h$

This can be written as:

$$dS_h/dt \geq -(\mu_h+\lambda_h+\theta_h) S_h$$

Upon separating variables and integrating:

$$\int_0^t \frac{dS_h}{S_h} \geq -\int_0^t (\mu_h+\lambda_h(t)+\theta_h) S_h(t)\, dt$$

$$\ln \frac{S_h(t)}{S_h(0)} \geq -(\mu_h+\theta_h)t - \int_0^t \lambda_h\, d$$

Thus:

$$S_h(t) \geq S_h(0)\, e^{-(\mu_h+\theta_h)t - \int_0^t \lambda_h(s)\, ds}$$

Similarly, for:

$$dV_h/dt = (1-p_h)\varpi_h + \theta_h S_h - ((1-\varepsilon_h)\lambda_h + \mu_h + \tau_h) V_h$$ we get:

$$V_h(t) \geq V_h(0)\, e^{-(\mu_h+\tau_h)t - \int_0^t (1-\varepsilon_h)\lambda_h dt}$$

For the exposed human class:

$$dE_h/dt \geq -(\mu_h+\theta_h) E_h$$

we get:

$$E_h(t) \geq E_h(0)\, e^{-(\mu_h+\theta_h)t}$$

For the 7th and 8th equations:

$$S_d(t) \geq S_d(0)\, e^{-(\mu_d+\theta_d)t - \int_0^t \lambda_d(s) ds}$$

$$V_d(t) \geq V_d(0) e^{-(\mu_d+\tau_d)t - \int_0^t (1-\varepsilon_d)\lambda_d(s) ds}$$

Therefore, all solutions of the model system (1) with positive initial data will remain positive for all t > 0.

Invariant Region

We have to ensure that the model solutions are bounded within a biologically feasible region, ensuring the sustainability and long-term applicability of the leptospirosis transmission model. To prove the solution of model system (1) is bounded, Theorem 2, is stated and proved as follows:

Theorem 2. *All Possible Solutions of model system (1) are contained within a uniformly bounded region*

$$\Omega=\left\{\left(S_h,V_h,E_h,A_h,I_h,R_h,S_d,V_d,I_d,R_d,B\right)\in R_{+11}: N_h\le\frac{\varpi_h}{\mu_h},N_d\le\frac{\varpi_d}{\mu_d},B\le\left(\frac{\phi_h\varpi_h}{\mu_h\mu_b}+\frac{\phi_d\varpi_d}{\mu_d\mu_b}\right)\right\} \quad (2)$$

Proof. The method, as applied by Mwasunda *et al.* 2023, was used to prove this Theorem. The initial populations of humans and domestic animals are considered as $N_h(0)$ and $N_d(0)$ respectively, and the initial concentration of pathogens as $B(0)$. The first six equations of the model system 1 enable us to derive the total population size of human that gives:

$$\frac{dS_h}{dt}=\varpi_h-\mu_hN_h-\delta_hI_h, \quad (3)$$

$\Longrightarrow\frac{dS_h}{dt}\le\varpi_h-\mu_hN_h$. Solving the differential inequality, model system 1 yields the solution $N_h(t)\le\frac{\varpi_h}{\mu_h}+\left(N_h(0)-\frac{\varpi_h}{\mu_h}\right)e^{-\mu_ht}$. (4)

As $t\to\infty$, $N_h(t)\to\frac{\varpi_h}{\mu_{h,}}$ and hence we obtain $N_h(t)\le\frac{\varpi_h}{\mu_h}$. Also, from model 1 the overall dynamics of the domestic animal population is defined as

$$\frac{dN_d}{dt}=\varpi_d-\mu_dN_d-\delta_dI_d,\Longrightarrow\frac{dN_d}{dt}\le\varpi_d-\mu_dN_d \quad (5)$$

Solution of the differential inequality for $N_d(t)$ is $N_d(t)\le\frac{\varpi_d}{\mu_d}+\left(N_d(0)-\frac{\varpi_d}{\mu_d}\right)e^{-\mu_dt}$. When $t\to\infty$, $N_d(t)\to\frac{\varpi_d}{\mu_{d,}}$ which leads us to the conclusion that $N_d(t)\le\frac{\varpi_d}{\mu_d}$.

Similarly, model 1 gives the concentration of bacteria as

$$\frac{dB}{dt}=\left(A_h+I_h\right)\phi_h+\phi_dI_d-\mu_bB, \quad (6)$$

which can be written as: $\frac{dB}{dt}+\mu_bB=\left(A_h+I_h\right)\phi_h+\phi_dI_d$. Since the total human and domestic animal population is given as:

$N_h(t)=S_h(t)+V_h(t)+E_h(t)+A_h(t)+I_h(t)+R_h(t)\le\frac{\varpi_h}{\mu_h}$ and

$N_d(t)=S_d(t)+V_d(t)+I_d(t)+R_d(t)\le\frac{\varpi_d}{\mu_d}$, then it is obvious that $A_h(t)+I_h(t)\le\frac{\varpi_h}{\mu_h}$ and

$$I_d(t) \le \frac{\varpi_d}{\mu_d}.$$

Therefore, we have: $\frac{dB}{dt} + \mu_b B \le \frac{\phi_h \varpi_h}{\mu_h} + \frac{\phi_d \varpi_d}{\mu_d}$. Using the integrating factor and applying the initial condition, we get:

$$B(t) \le \left(\frac{\phi_h \varpi_h}{\mu_h \mu_b} + \frac{\phi_d \varpi_d}{\mu_d \mu_b}\right) + \left(B(0) - \left(\frac{\phi_h \varpi_h}{\mu_h \mu_b} + \frac{\phi_d \varpi_d}{\mu_d \mu_b}\right)\right) e^{-\mu_b t} \quad (7)$$

As $t \to \infty$, we obtain $B(t) \le \left(\frac{\phi_h \varpi_h}{\mu_h \mu_b} + \frac{\phi_d \varpi_d}{\mu_d \mu_b}\right)$.

Therefore, the solution of model system 1 is positive and bounded in the region

$$\Omega = \left\{ \left(S_h, V_h, E_h, A_h, I_h, R_h, S_d, V_d, I_d, R_d, B\right) \in R_{+\square^{11}} : N_h \le \frac{\varpi_h}{\mu_h}, N_d \le \frac{\varpi_d}{\mu_d}, B \le \left(\frac{\phi_h \varpi_h}{\mu_h \mu_b} + \frac{\phi_d \varpi_d}{\mu_d \mu_b}\right) \right\} \quad (8)$$

Using the standard techniques outlined in, the feasible region can be demonstrated to be positively invariant. Consequently, all solutions $\left(S_h, V_h, E_h, A_h, I_h, R_h, S_d, V_d, I_d, R_d, B\right)$ remain within the region Ω, ensuring that the model is both mathematically and epidemiologically well-posed.

Basic Model

Considering the basic model, the following system of nonlinear differential equations describes the transmission dynamics of leptospirosis under consideration:

$$\begin{aligned}
dS_h/dt &= \omega_h + \varphi_h R_h - (\mu_h + \lambda_h) S_h \\
dE_h/dt &= \lambda_h S_h - (\eta_h + \mu_h) E_h \\
dI_h/dt &= \eta_h E_h - (\mu_h + \xi_h + \delta_h) I_h \\
dR_h/dt &= \xi_h I_h - (\mu_h + \varphi_h) R_h \\
dS_d/dt &= \omega_d - (\mu_d + \lambda_d) S_d \\
dI_d/dt &= \lambda_d S_d - (\mu_d + \delta_d) I_d \\
dB/dt &= \phi_h I_h + \phi_d I_d - \mu_b B
\end{aligned} \quad (9)$$

$S_h(0) > 0$, $E_h(0) \ge 0$, $I_h(0) \ge 0$, $R_h(0) \ge 0$, $S_d(0) > 0$, $I_d(0) \ge 0$, $B(0) \ge 0$.

The forces of infection are given by: $\lambda_h = \frac{\beta_3 I_d}{N_d} + \frac{\beta_2 B}{(B+b)}$, $\lambda_d = \frac{\beta_4 I_d}{N_d} + \frac{\beta_1 B}{(B+b)}$

Disease Free Equilibrium and Basic Reproduction Number

When there are no infections in humans and domestic animal, the disease free equilibrium E_0 is given by:

$$S_h^0, E_h^0, I_h^0, R_h^0, S_d^0, I_d^0, B^0 = \left(\frac{\varpi_h}{\mu_h}, 0, 0, 0, \frac{\varpi_d}{\mu_d}, 0, 0\right) \quad (10)$$

The Basic Reproduction Number R_0 is defined as the average number of secondary cases produced by one infectious case in a completely susceptible population (Martcheva 2015, Van den Driessche and Watmough 2002).

It is also used to assess the stability of the disease-free equilibrium (DFE) and the endemic equilibrium (EE). In this work, the next-generation approach proposed by Van den Driessche and Watmough (2002) was used to compute R_0. The next-generation matrix is given by:

$$FV^{-1}=\frac{\partial\left(F_i\left(E_0\right)\right)}{\partial x_j}\times\frac{\partial\left(V_i^{-1}\left(E_0\right)\right)}{\partial x_j} \tag{10}$$

where F_i is the rate of appearance of infection in compartment i, $V_i = V_i^- - V_i^{+¿¿}$ is the transfer rate of individuals from one compartment i to another, V_i^- is the rate of transfer of individuals out of compartment i, $V_i^{+¿¿}$ is the rate of transfer of individuals into compartment i, and x_j represents the infected classes.

From the given model system (10)

$$F_i=\begin{pmatrix}\left(\frac{\beta_3 I_d}{N_d}+\frac{\beta_2 B}{(B+b)}\right)S_h\\0\\\left(\frac{\beta_4 I_d}{N_d}+\frac{\beta_1 B}{(B+b)}\right)S_d\\0\end{pmatrix},\qquad V_i=\begin{pmatrix}(\eta_h+\mu_h)E_h\\(\mu_h+\xi_h+\delta_h)I_h-\eta_h E_h\\(\mu_d+\delta_d)I_d\\\mu_b B-\phi_h I_h+\phi_h I_d\end{pmatrix},$$

The Jacobian matrices F and V at the disease-free equilibrium E_0 are given by:

$$F=\begin{pmatrix}0&0&\frac{\beta_3 S_h^{\,0}}{N_d}&\left|\frac{\beta_2 S_h^{\,0}}{b}\right|&0&|0|&0&|0|&0&|0|&\frac{\beta_4 S_d^{\,0}}{N_d}&\left|\frac{\beta_1 S_d^{\,0}}{b}\right|&0&|0|&0&|0|\end{pmatrix},\ V^{-1}=\begin{pmatrix}\frac{1}{a_1}&0&0&0\\\frac{\eta_h}{a_1 b_1}&\frac{1}{b_1}&0&0\\0&0&\frac{1}{c_1}&0\\0&\frac{\phi_h}{b_1\mu_b}&\frac{\phi_d}{c_1\mu_b}&\frac{1}{\mu_b}\end{pmatrix}$$

Where, $a_1=(\eta_h+\mu_h),\ b_1=(\delta_h+\mu_h+\xi_h),\ c_1=(\mu_d+\delta_d),\ S_h^{\,0}=\frac{\varpi_h}{\mu_h},\ S_d^{\,0}=\frac{\varpi_d}{\mu_d}.$

Thus, the basic reproduction number R_0 is given by:

$$R_0=\rho F V^{-1} \tag{11}$$

By using definition (12), the basic reproduction number R_0 is:

$$R_0=\frac{1}{2}\left(G_1+G_4+\sqrt{(G_1-G_4)^2+4G_2G_3}\right) \tag{12}$$

$$G_1=\frac{\varpi_h}{\mu_h}\left(\frac{\beta_3}{N_d(\mu_d+\delta_d)}+\frac{\beta_2\beta_2\sigma\phi_d}{b\mu_b}\right)+\frac{\beta_1\varpi_d}{b\mu_b\mu_d},\ G_2=\frac{\varpi_h}{\mu_h}\frac{\beta_2}{b\mu_b\mu_d},$$

$$G_3=\frac{\varpi_d}{\mu_d(\mu_d+\delta_d)}\left(\frac{\beta_4}{N_d}+\frac{\beta_1\phi_d}{b\mu_b}\right)$$

$$G_4 = \frac{\beta_1 \varpi_d}{\mu_d b \mu_b},\ 4\, G_2 G_3 = 4\left(\frac{\beta_2 \varpi_h \varpi_d}{\mu_d^{\,2}(\mu_d + \delta_d) b \mu_b \mu_h}\left(\frac{\beta_4}{N_d} + \frac{\beta_1 \phi_d}{b \mu_b}\right)\right)$$

To provide biological interpretation, the basic reproduction number R_0 is expressed in the form of:

$$R_0 = \frac{1}{2}\left(\frac{\varpi_h}{\mu_h}\cdot\left(\frac{\beta_3}{N_d(\mu_d+\delta_d)} + \frac{\beta_2\phi_d}{b\mu_b}\right) + \frac{\varpi_d}{\mu_d}\cdot\frac{\beta_1}{b\mu_b} + \frac{\varpi_d}{\mu_d}\cdot\frac{\beta_1}{b\mu_b} + \sqrt{\left(\frac{\varpi_h}{\mu_h}\cdot\left(\frac{\beta_3}{N_d(\mu_d+\delta_d)} + \frac{\beta_2\phi_d}{b\mu_b}\right) + \frac{\varpi_d}{\mu_d}\cdot\frac{\beta_1}{b\mu_b} - \frac{\varpi_d}{\mu_d}\cdot\frac{\beta_1}{b\mu_b}\right)^2 + 4\left(\frac{\varpi_d}{\mu_d}\cdot\frac{\varpi_h}{\mu_h}\frac{\beta_3}{(\mu_d+\delta_d)b\mu_b}\left(\frac{\beta_4}{N_d} + \frac{\beta_1\phi_d}{b\mu_b}\right)\right)}\right)$$

$\frac{\varpi_h}{\mu_h}$ and $\frac{\varpi_d}{\mu_d}$: Represent the average number of susceptible humans and domestic animals at equilibrium. $\frac{\beta_3}{N_d(\mu_d+\delta_d)}$ represents the risk of transmission from infected domestic animals to susceptible humans per contact, adjusted by the domestic animal population size and their removal (death + disease mortality) rates. $\frac{\beta_2\phi_d}{b\mu_b}$: Represents environment to human transmission. $\frac{\beta_1}{b\mu_b}$: Represents the environment for domestic animal transmission, involving. $\frac{\beta_4}{N_d}$: Represents animal to animal transmission, adjusted by the domestic animal population. $\frac{\beta_1\phi_d}{b\mu_b}$: Represents the effect of environmental contamination due to shedding by domestic animals on transmission to animals.

R_0 Increases with recruitment rates (ϖ_h, ϖ_d), leading to more susceptible humans and domestic animals. Transmission rates β_1, β_2, β_3, β_4 increasing the likelihood of spread between compartments. Shedding rates (ϕ_h, ϕ_d), increasing environmental contamination which enhance indirect transmission. R_0 also decreases with, natural mortality rates (μ_h, μ_d, μ_b) and disease-induced mortality (δ_h), which reduce the number of infectious and susceptible individuals, thereby decreasing transmission. Bacterial decay rate (μ_b) reduce environmental transmission. Thus, interpretation demonstrates that the derived R_0 quantifies the combined effects of direct transmission (animal to human, animal to animal) and indirect transmission (through environment) within the community. R_0>1 indicates potential for outbreak persistence, while R_0<1 suggests disease elimination under the modeled parameters, providing a rigorous foundation for targeted control measures in leptospirosis management.]

Model with Interventions

Disease Free Equilibrium (DFE) and the Effective Reproduction Number R_e

The disease-free equilibrium (DFE) of leptospirosis is in a stable state when there is no infection present. We denote the state by E_{01} and is given by;

$$E_{01}=\left(\begin{array}{c}\dfrac{\varpi_h\left(p_h\mu_h+\tau_h\right)}{\mu_h\left(\mu_h+\theta_h+\tau_h\right)},\dfrac{\varpi_h\left(\mu_h\left(1-p_h\right)+\theta_h\right)}{\mu_h\left(\mu_h+\theta_h+\tau_h\right)},0,0,0,0,\\ \dfrac{\varpi_d\left(p_h\mu_d+\tau_d\right)}{\mu_d\left(\mu_d+\theta_d+\tau_d\right)},\dfrac{\varpi_d\left(\mu_d\left(1-p_d\right)+\theta_d\right)}{\mu_d\left(\mu_d+\theta_d+\tau_d\right)},0,0,0\end{array}\right) \tag{13}$$

In epidemiology, the effective reproduction number often denoted as R_e is the average number of secondary cases per infectious individuals in a population including the effects of immunity (either from previous infections or vaccination), non-pharmaceutical controls (such as controls of reservoir animal population and using appropriate protective gears (rubber boots, water proof, goggles, gloves) and general hygiene) and other factors. The next generation matrix approach as applied by Mwasunda et al. (2021), Irunde et al. (2023) and Ndendya et al. (2023), was occasionally used to determine the effective reproduction number. This approach involves the formation of a matrix that reflects the interaction and transition of individuals among different compartments of the disease model.

The intervention measures are ineffective when $R_e>1$, and effective when $R_e<1$. The effective reproduction number $R_e\geq 1$,for model system (1) is given by:

$$R_e=\frac{1}{2}\left(R_B+\sqrt{R_B^2+4R_{dh}}\right), \tag{14}$$

$$R_B=\left(d_h+f_h\right)u_{11}^{\ 2}u_{22}^{\ 2}u_{33}^{\ 2}u_{44}u_{55}^{\ 2}\left[u_{21}u_{33}u_{52}+u_{22}u_{31}u_{53}-u_{21}u_{32}u_{53}\right]-\left(d_d+f_d\right)u_{11}u_{22}^{\ 3}u_{33}^{\ 3}u_{54}u_{55}^{\ 2}+\left(c_d+e_d\right)u_{11}^{\ 2}u_{22}^{\ 3}u_{33}^{\ 3}u_{55}^{\ 3}$$

,

where,

$$e_d=\frac{\beta_4\left(1-u\right)\left[\left(1-\epsilon_d\right)V_d^{\ 0}\right]}{N_d},\ e_h=\frac{\beta_3\left(1-u\right)\left[\left(1-\epsilon_d\right)V_d^{\ 0}\right]}{N_d},\ f_h=\frac{\beta_2\left(1-u\right)\left[\left(1-\epsilon_d\right)V_d^{\ 0}\right]}{b},$$

$$d_h=\frac{\beta_2\left(1-u\right)S_h^{\ 0}}{b},$$

$$c_d=\frac{\beta_4\left(1-u\right)S_d^{\ 0}}{N_d},\ d_d=\frac{\beta_1\left(1-u\right)S_d^{\ 0}}{b},\ f_d=\frac{\beta_1\left(1-u\right)\left[\left(1-\epsilon_d\right)V_d^{\ 0}\right]}{b},\ c_h=\frac{\beta_3\left(1-u\right)S_h^{\ 0}}{N_d}.$$

$$R_B=\frac{\left(\beta_2\left(1-u\right)\left[S_h^{\ 0}+\left(1-\epsilon_h\right)V_h^{\ 0}\right]u_{11}^{\ 2}u_{22}^{\ 2}u_{33}^{\ 2}u_{44}u_{55}^{\ 2}\left[u_{21}u_{33}u_{52}+u_{22}u_{31}u_{53}-u_{21}u_{32}u_{53}\right]\right)}{b}-\frac{\beta_1\left(1-u\right)\left[S_d^{\ 0}+\left(1-\epsilon_d\right)V_d^{\ 0}\right]u_{11}^{\ 2}u_{22}^{\ 3}u_{33}^{\ 3}u_{54}u_{55}^{\ 2}}{b}$$
$$\frac{+\beta_4\left(1-u\right)\left[S_d^{\ 0}+\left(1-\epsilon_d\right)V_d^{\ 0}\right]u_{11}^{\ 2}u_{22}^{\ 3}u_{33}^{\ 3}u_{55}^{\ 3}}{N_d}$$

,

$$u_{11}=\eta_h+\mu_h,\ u_{21}=-\left(1-\rho_h\right)\eta_h,\ u_{22}=\mu_h+\xi_h+\vartheta_h,\ u_{31}=-\rho_h\eta_h,\ u_{32}=-\vartheta_h,$$
$$u_{33}=\mu_h+\alpha_h+\delta_h,\ u_{44}=\mu_d+\alpha_d+\delta_d,\ u_{52}=¿\ u_{53}=-\phi_h,\ u_{54}=-\phi_d,\ u_{55}=-\mu_b$$

$$R_B=\frac{\left(\beta_2(1-u)\left[S_h^{\,0}+(1-\epsilon_h)V_h^{\,0}\right]A_1B_1\right)}{b}-\frac{\beta_1(1-u)\left[S_d^{\,0}+(1-\epsilon_d)V_d^{\,0}\right]C_1}{b}$$
$$\frac{+\beta_4(1-u)\left[S_d^{\,0}+(1-\epsilon_d)V_d^{\,0}\right]D_1}{N_d}, \qquad (15)$$

and

$$A_1=\left[u_{21}u_{33}u_{52}+u_{22}u_{31}u_{53}-u_{21}u_{32}u_{53}\right]=(1-\rho_h)(\mu_h+\alpha_h+\delta_h-\vartheta_h)+\rho_h(\mu_h+\xi_h+\vartheta_h)$$
$$B_1=u_{11}^{\,2}u_{22}^{\,2}u_{33}^{\,2}u_{44}u_{55}^{\,2}=(\eta_h+\mu_h)^2(\mu_h+\xi_h+\vartheta_h)^2(\mu_h+\alpha_h+\delta_h)^2(\mu_d+\alpha_d+\delta_d)\mu_b^{\,2},$$
$$A_1B_1=[(\mu_h+\alpha_h+\delta_h-\vartheta_h)+\rho_h(\xi_h+2\vartheta_h-\alpha_h-\delta_h)]$$
$$\times\left((\eta_h+\mu_h)^2(\mu_h+\xi_h+\vartheta_h)^2(\mu_h+\alpha_h+\delta_h)^2\right),$$
$$C_1=u_{11}^{\,2}u_{22}^{\,3}u_{33}^{\,3}u_{54}u_{55}^{\,2}=-(\eta_h+\mu_h)^2(\mu_h+\xi_h+\vartheta_h)^3(\mu_h+\alpha_h+\delta_h)^3\phi_d\mu_b^{\,2},$$
$$D_1=u_{11}^{\,2}u_{22}^{\,3}u_{33}^{\,3}u_{55}^{\,3}=(\eta_h+\mu_h)^2(\mu_h+\xi_h+\vartheta_h)^3(\mu_h+\alpha_h+\delta_h)^3\mu_b^{\,3}.$$
$$S_h^{\,0}=\frac{\varpi_h(p_h\mu_h+\tau_h)}{\mu_h(\mu_h+\theta_h+\tau_h)},\ V_h^{\,0}=\frac{\varpi_h(\mu_h(1-p_h)+\theta_h)}{\mu_h(\mu_h+\theta_h+\tau_h)},\ S_d^{\,0}=\frac{\varpi_d(p_h\mu_d+\tau_d)}{\mu_d(\mu_d+\theta_d+\tau_d)},$$
$$V_d^{\,0}=\frac{\varpi_d(\mu_d(1-p_d)+\theta_d)}{\mu_d(\mu_d+\theta_d+\tau_d)}$$

$$R_{hd}=K.$$

where, $K=u_{11}^{\,3}u_{22}^{\,5}u_{33}^{\,5}u_{44}u_{55}^{\,5}u_{21}u_{52}$

$$K'=u_{11}^{\,3}u_{22}^{\,6}u_{33}^{\,5}u_{44}u_{55}^{\,5}u_{31}u_{53},$$
$$K''=u_{11}^{\,3}u_{22}^{\,5}u_{33}^{\,5}u_{44}u_{55}^{\,5}u_{21}u_{32}u_{53}$$
$$R_{hd}=K.+K'.\left[c\|d(d_h-d_d)+e_d(d_h+f_h)-e_h(d_d+f_h)-f_dc_h\right]+K''.$$

$$R_{hd}=K.\left[\frac{\beta_3(1-u)S_h^{\,0}}{N_d}.\left(\frac{\beta_1(1-u)S_d^{\,0}}{b}+\frac{\beta_2(1-u)S_h^{\,0}}{b}-\frac{\beta_1(1-u)(1-\epsilon_d)V_h^{\,0}}{b}\right),\frac{-\beta_3(1-u)(1-\epsilon_d)V_h^{\,0}}{N_d}\left(\frac{\beta_1(1-u)S_d^{\,0}}{b}+\frac{\beta_1(1-u)(1-\epsilon_d)S_h^{\,0}}{N_d}\right)+\frac{\beta_2(1-u)(1-\epsilon_d)V_d^{\,0}}{b}\left(\frac{\beta_4(1-u)S_d^{\,0}}{N_d}+\frac{\beta_4(1-u)(1-\epsilon_d)V_d^{\,0}}{N_d}\right)-\frac{\beta_2(1-u)S_h^{\,0}}{b}.\frac{\beta_4(1-u)(1-\epsilon_d)V_d^{\,0}}{N_d}\right]$$

$$\left[\frac{\beta_4(1-u)S_d^{\,0}}{N_d}\left(\frac{\beta_2(1-u)S_h^{\,0}}{b}-\frac{\beta_1(1-u)S_d^{\,0}}{b}\right)+\frac{\beta_4(1-u)(1-\epsilon_d)V_d^{\,0}}{N_d}.\left(\frac{\beta_2(1-u)S_h^{\,0}}{b}+\frac{\beta_2(1-u)(1-\epsilon_d)V_d^{\,0}}{N_d}\right)\right]$$
$$+\left[\frac{-\beta_3(1-u)(1-\epsilon_d)V_d^{\,0}}{N_d}\left(\frac{\beta_1(1-u)S_d^{\,0}}{b}+\frac{\beta_2(1-u)(1-\epsilon_d)V_d^{\,0}}{b}\right)-\left(\frac{\beta_3(1-u)S_h^{\,0}}{b}.\frac{\beta_1(1-u)(1-\epsilon_h)V_d^{\,0}}{b}\right)\right]$$
$$+K''.\left[\left(\frac{\beta_3(1-u)S_h^{\,0}}{N_d}+\frac{\beta_3(1-u)(1-\epsilon_d)V_d^{\,0}}{N_d}\right)\left(\frac{\beta_1(1-u)(1-\epsilon_d)V_d^{\,0}}{b}+\frac{\beta_1(1-u)S_d^{\,0}}{b}\right)-\left(\frac{\beta_4(1-u)S_d^{\,0}}{N_d}+\frac{\beta_4(1-u)(1-\epsilon_d)V_d^{\,0}}{N_d}\right)\left(\frac{\beta_2(1-u)(1-\epsilon_d)V_d^{\,0}}{b}+\frac{\beta_2(1-u)S_h^{\,0}}{b}\right)\right]$$
$$+K'. \qquad (16)$$

where, R_B represents infections acquired by humans and domestic animals through consumption of contaminated materials from the environment, while R_{hd}describes infections acquired by susceptible humans and domestic animals from infected domestic animals during the infectious period.

Global Stability of the Disease-Free Equilibrium Point (DFE)

This section applies the approach of Castillo-Chavez et al. (2002) to analyze the global stability of the disease-free equilibrium point of the model system (1). The model system (1) can be written in the form of:

$$\begin{cases} \dfrac{dP}{dt} & ¿F(P,Q) \\ \dfrac{dQ}{dt} & ¿G(P,Q), with\, G(P,0)=0 \end{cases} \tag{17}$$

In this system, P represents disease-free subgroups, which are $(S_h, V_h, R_h, S_d, V_d, R_d)$ and $Q=(E_h, A_h, I_h, I_d, B)$ represents infectious subgroups. If the following conditions (H_1) and (H_2) are satisfied, then the global stability of E_0 is guaranteed when $R_0<1$:

(H1) For dP/dt = F (P, 0), P* is globally asymptotically stable (g.a.s).

(H2) G (P, Q) = A Q – $\hat{G}$ (P, Q), where $\hat{G}$ (P, Q) ≥ 0 for (P, Q) ∈ Ω.

In this case, the Jacobian $A=\dfrac{\partial G}{\partial Q}(P^{\square},0)$ is a Metzler matrix (off-diagonal elements of A are non-negative) and Ω is the region where the model system yields a solution which is biological meaningful. If the model system1 satisfies the above conditions, then the following theorem holds;

Theorem 3. *The disease-free equilibrium* E_0 *, is globally asymptotically stable if* $R_e<1$ *and unstable otherwise.*

Proof. To prove Theorem 3, the method used by is applied. Then, it is required to show that the conditions (H_1) and (H_2) hold if $R_e<1$.

For condition (H_1).

The model system can be written in form of P, Q and

$$(P^{\square},0)=\left(\begin{array}{c} \dfrac{\varpi_h(p_h\mu_h+\tau_h)}{\mu_h(\mu_h+\theta_h+\tau_h)}, \dfrac{\varpi_h(\mu_h(1-p_h)+\theta_h)}{\mu_h(\mu_h+\theta_h+\tau_h)}, 0,0,0,0, \\ \dfrac{\varpi_d(p_h\mu_d+\tau_d)}{\mu_d(\mu_d+\theta_d+\tau_d)}, \dfrac{\varpi_d(\mu_d(1-p_d)+\theta_d)}{\mu_d(\mu_d+\theta_d+\tau_d)}, 0,0,0 \end{array}\right), \tag{18}$$

then,

$$\frac{dP}{dt}=\begin{pmatrix} p_h\varpi_h+\varphi_h R_h+\tau_h V_h-\left(\mu_h+\left(\frac{\beta_3(1-u)I_d}{N_h}+\frac{(\beta_2(1-u)B)}{(B+b)N_h}\right)+\theta_h\right)S_h \\ (1-p_h)\varpi_h+\theta_h S_h-\left((1-\varepsilon_h)\left(\frac{\beta_3(1-u)I_d}{N_h}+\frac{(\beta_2(1-u)B)}{(B+b)N_h}\right)+\mu_h+\tau_h\right)V_h \\ \alpha_h I_h+\xi_h A_h-(\mu_h+\varphi_h)R_h \\ p_d\varpi_d+\varphi_d R_d+\tau_d V_d-\left(\mu_d+\left(\frac{\beta_4(1-u)I_d}{N_d}+\frac{(\beta_1(1-u)B)}{(B+b)N_d}\right)+\theta_d\right)S_d \\ (1-p_d)\varpi_d+\theta_d S_d-\left((1-\varepsilon_d)\left(\frac{\beta_4(1-u)I_d}{N_d}+\frac{(\beta_1(1-u)B)}{(B+b)N_d}\right)+\mu_d+\tau_d\right)V_d \\ \alpha_d I_d-(\mu_d+\varphi_d)R_d \end{pmatrix} \tag{19}$$

At DFE $E_0=(P^{\square},0)$ yields,

$$\frac{dP}{dt}=F(P,0)=\begin{pmatrix} p_h\varpi_h+\tau_h V_h-(\mu_h+\theta_h)S_h \\ (1-p_h)\varpi_h+\theta_h S_h-(\mu_h+\tau_h)V_h \\ 0 \\ p_d\varpi_d+\tau_d V_d-(\mu_d+\theta_d)S_d \\ (1-p_d)\varpi_d+\theta_d S_d-(\mu_d+\tau_d)V_d \\ 0 \end{pmatrix}. \tag{20}$$

From the matrix we have, $\frac{dS_h(t)}{dt}=p_h\varpi_h+\tau_h V_h-(\mu_h+\theta_h)S_h$,

This is a linear first o.d.e and its solution can be found as:

$$\frac{dS_h(t)}{dt}+(\mu_h+\theta_h)S_h=p_h\varpi_h+\tau_h V_h.$$

By using the concept of integrating factor it yields, $I=\exp\int(\mu_h+\theta_h)dt \Longrightarrow \mathrm{I}e^{(\mu_h+\theta_h)t}$.

Multiply equation by integrating factor on both sides;

$$e^{(\mu_h+\theta_h)t}\left(\frac{dS_h(t)}{dt}+(\mu_h+\theta_h)S_h\right)=(p_h\varpi_h+\tau_h V_h)e^{(\mu_h+\theta_h)t},$$

$$\Longrightarrow\int_0^t\frac{d}{ds}\left(Se^{(\mu_h+\theta_h)s}\right)ds=(p_h\varpi_h+\tau_h V_h)\int_0^t e^{(\mu_h+\theta_h)s}ds,$$

$$\Longrightarrow S_h(t)=S(0)e^{-(\mu_h+\theta_h)t}+\left(\frac{p_h\varpi_h+\tau_h V_h}{\mu_h+\theta_h}\right)\left(1-e^{-(\mu_h+\theta_h)t}\right),$$

$$S_h(t)=\frac{p_h\varpi_h+\tau_h V_h}{\mu_h+\theta_h}-\left(\frac{p_h\varpi_h+\tau_h V_h}{\mu_h+\theta_h}-S_h(0)\right)e^{-(\mu_h+\theta_h)t}. \quad (21)$$

In the same way, upon integration by separating the variables and applying initial conditions for the remaining state variables gives;

$$V_h(t)=\frac{(1-p_h)\varpi_h+\theta_h S_h}{(\mu_h+\tau_h)}-\left(\frac{(1-p_h)\varpi_h+\theta_h S_h}{(\mu_h+\tau_h)}-V(0)\right)e^{-(\mu_h+\tau_h)t}. \quad (22)$$

$$S_d(t)=\frac{p_d\varpi_d+\tau_d V_d}{\mu_d+\theta_d}-\left(\frac{p_d\varpi_d+\tau_d V_d}{\mu_d+\theta_d}-S_d(0)\right)e^{-(\mu_d+\theta_d)t}. \quad (23)$$

$$V_d(t)=\frac{(1-p_d)\varpi_d+\theta_d S_d}{(\mu_d+\tau_d)}-\left(\frac{(1-p_d)\varpi_d+\theta_d S_d}{(\mu_d+\tau_d)}-V(0)\right)e^{-(\mu_d+\tau_h)t}. \quad (24)$$

From $S_h(t), V_h(t), R_h, S_d(t), V_d(t)$and $R_d(t)$, it is clearly that, $S_h(t)\to\frac{p_h\varpi_h+\tau_h V_h}{\mu_h+\theta_h}$, $V_h(t)\to\frac{(1-p_h)\varpi_h+\theta_h S_h}{(\mu+\tau)}, R_h=0, S_d(t)=\frac{p_d\varpi_d+\tau_d V_d}{\mu_d+\theta_d}, V_d(t)=\frac{(1-p_d)\varpi_d+\theta_d S_d}{(\mu_h+\tau_h)}$ and $R_d(t)=0$ as $t\to\infty$, regardless of the values of $S_h(0)$, $V_h(0)$, $R_h(t)$, $S_d(0)$, $V_d(0)$and $R_d(t)$. Thus $P^{\square}$ is globally asymptotically stable. Hence, condition (H_1) is satisfied.

Again, for condition (H_2) gives:

$$G(P,Q)$$

$$\begin{pmatrix}\left(\frac{\beta_3(1-u)I_d}{N_h}+\frac{(\beta_2(1-u)B)}{(B+b)N_h}\right)S_h+(1-\varepsilon_h)\left(\frac{\beta_3(1-u)I_d}{N_h}+\frac{(\beta_2(1-u)B)}{(B+b)N_h}\right)V_h-(\eta_h+\mu_h)E_h\\(1-\rho_h)\eta_h E_h-(\mu_h+\xi_h+\vartheta_h)A_h\\\rho_h\eta_h E_h+\vartheta_h A_h-(\mu_h+\alpha_h+\delta_h)I_h\\\left(\frac{\beta_4(1-u)I_d}{N_d}+\frac{(\beta_1(1-u)B)}{(B+b)N_d}\right)S_d+(1-\varepsilon_d)\left(\frac{\beta_4(1-u)I_d}{N_d}+\frac{(\beta_1(1-u)B)}{(B+b)N_d}\right)V_d-(\mu_d+\alpha_d+\delta_d)I_d\\\phi_h A_h+\phi_h I_h+\phi_d I_d-\mu_b B\end{pmatrix} \quad (25)$$

Recall that $A=\frac{\partial G}{\partial Q}(P^{\square},0)$. Since, it is clearly the Matrix A is a Metzler matrix (its off-diagonal elements are non-negative). Then, $\widehat{G}(P,Q)=AQ-G(P,Q)$. That is,

$$\widehat{G}(P,Q)=\begin{pmatrix} -(a_1) & 0 & 0 & \frac{(1-u)\beta_3}{N_h}\left(S_h^{\square}+V_h^{\square}(1-\varepsilon_h)\right) & \frac{(1-u)\beta_2}{N_h b}\left(S_h^{\square}+V_h^{\square}(1-\varepsilon_h)\right) \\ a_2 & -a_3 & 0 & 0 & 0 \\ \eta_h\rho_h & \vartheta_h & -a_5 & 0 & 0 \\ 0 & 0 & 0 & \frac{+(1-u)\beta_3}{N_d}\left(S_h^{\square}V_d^{\square}(1-\varepsilon_d)\right)-a_5 & \frac{(1-u)\beta_2}{N_d b}\left(S_d^{\square}+V_d^{\square}(1-\varepsilon_d)\right) \\ 0 & \phi_h & \phi_h & \phi_d & -\mu_b, \end{pmatrix}$$

$$\begin{pmatrix} E_h \\ A_h \\ I_h \\ I_d \\ B \end{pmatrix}-\begin{pmatrix} \lambda_h S_h+(1-\varepsilon_h)\lambda_h V_h-(\eta_h+\mu_h)E_h \\ (1-\rho_h)\eta_h E_h-(\mu_h+\xi_h+\vartheta_h)A_h \\ \rho_h\eta_h E_h+\vartheta_h A_h-(\mu_h+\alpha_h+\delta_h)I_h \\ \lambda_d S_d+(1-\varepsilon_d)\lambda_d V_d-(\mu_d+\alpha_d+\delta_d)I_d \\ \phi_h A_h+\phi_h I_h+\phi_d I_d-\mu_b B \end{pmatrix} \tag{26}$$

$$\widehat{G}(P,Q)=\begin{pmatrix} \frac{(1-u)\beta_3(S_h^{\square}-S_h)}{N_h}+\frac{(1-\varepsilon_h)(1-u)\beta_3(V_h^{\square}-V_h)}{N_h}+\frac{(S_h^{\square}-S_h)(1-u)\beta_2}{N_h b}+\frac{(V_h^{\square}-V_h)(1-\varepsilon_h)(1-u)\beta_2}{N_h b} \\ 0 \\ 0 \\ \frac{(1-u)\beta_4(S_d^{\square}-S_d)}{N_d}+\frac{(1-u)\beta_4(1-\varepsilon_d)(V_d^{\square}-V_d)}{N_d}+\frac{(1-u)\beta_1(S_d^{\square}-S_d)}{N_d b}+\frac{(1-u)\beta_1(1-\varepsilon_d)(V_d^{\square}-V_d)}{N_d b} \\ 0 \end{pmatrix}. \tag{27}$$

Since $S_h^{\square}>S_h$, $V_h^{\square}>V_h$, $S_d^{\square}>S_d$ and $V_d^{\square}>V_d$ it is clear that $\widehat{G}(P,Q)\geq 0$ and

$$P^{\square}=\left(\frac{p_h\mu_h+\tau_h V_h}{(\mu_h+\theta_h)},\frac{\varpi_h(\mu_h(1-p_h)+\theta_h)}{\mu_h(\mu_h+\theta_h+\tau_h)},0,0,0,0,\frac{\varpi_d(p_h\mu_d+\tau_d)}{\mu_d(\mu_d+\theta_d+\tau_d)},\frac{\varpi_d(\mu_d(1-p_d)+\theta_d)}{\mu_d(\mu_d+\theta_d+\tau_d)},0,0,0\right) \tag{28}$$

is a global asymptomatically stable of $\frac{dP}{dt}=F(P,0)$. Hence the theorem is proved.

This global asymptotic stability of the disease-free equilibrium (DFE) implies that, under the given conditions, leptospirosis will eventually be eliminated from both human and domestic animal populations if the effective reproduction number is maintained below one. Hence, field implementation of interventions such as vaccination, screening, treatment, and non-pharmaceutical controls (use of PPE, hygiene improvements, and habitat management) are not only effective in reducing transmission but can lead to complete eradication of the disease if applied consistently and at sufficient coverage levels.

Endemic Equilibrium Point (EEP)

Global Stability of Endemic Equilibrium

Theorem 4. *The endemic equilibrium point $E_{\square}$ is globally asymptotically stable if the effective reproduction number $R_e>1$ and unstable otherwise.*

Proof. In this work, a logarithmic Lyapunov function was used to investigate the stability of the endemic equilibrium. The Lyapunov function is constructed by using the general form:

$$F=\sum_{i=1}^{n}\left(x_i(t)-x_i^{\square}\ln x_i(t)\right). \tag{29}$$

Now, the system can write as;

$$\begin{aligned}F=&\left(S_h(t)-S^{\square}\ln S_h(t)\right)+\left(V_h(t)-V^{\square}\ln V_h(t)\right)+\left(E_h(t)-E_h^{\square}\ln E_h(t)\right)+\left(A_h(t)-A_h^{\square}\ln A_h(t)\right)\\&+\left(I_h(t)-I_h^{\square}\ln I_h(t)\right)+\left(R_h(t)-R_h^{\square}\ln R_h(t)\right)+\left(S_d^{\square}(t)-S^{\square}\ln S_d(t)\right)+\left(V_d(t)-V^{\square}\ln V_d(t)\right)\\&+\left(I_d^{\square}(t)-I_d^{\square}\ln I_d(t)\right)+\left(R_d(t)-R_d^{\square}\ln R_d(t)\right)+\left(B(t)-B^{\square}\ln B(t)\right).\end{aligned}$$

The time derivative of a function of F gives:

$$\begin{aligned}\frac{dF}{dt}=&\left(1-\frac{S_h^{\square}}{S_h}\right)\frac{dS_h}{dt}+\left(1-\frac{V_h^{\square}}{V_h}\right)\frac{dV_h}{dt}+\left(1-\frac{E_h^{\square}}{E_h}\right)\frac{dE_h}{dt}+\left(1-\frac{A_h^{\square}}{A_h}\right)\frac{dA_h}{dt}+\left(1-\frac{I_h^{\square}}{I_h}\right)\frac{dI_h}{dt}\\&+\left(1-\frac{R_h^{\square}}{R_h}\right)\frac{dR_h}{dt}+\left(1-\frac{S_d^{\square}}{S_d}\right)\frac{dS_d}{dt}+\left(1-\frac{V_d^{\square}}{V_d}\right)\frac{dV_d}{dt}+\left(1-\frac{I_d^{\square}}{I_d}\right)\frac{dI_d}{dt}+\left(1-\frac{R_d^{\square}}{R_d}\right)\frac{dR_d}{dt}\\&+\left(1-\frac{B^{\square}}{B}\right)\frac{dB}{dt}.\end{aligned} \tag{30}$$

Upon substitution and simplification, gives:

$$\frac{dF}{dt}\leq-\left(1-\frac{S_h^{\square}}{S_h}\right)^2(\mu_h+\theta_h)S_h-\left(1-\frac{V_h^{\square}}{V_h}\right)^2(\mu_h+\tau_h)V_h-\left(1-\frac{S_d^{\square}}{S_d}\right)^2(\mu_d+\theta_d)S_d \tag{31}$$

where, $F=\{(S_h,V_h,E_h,A_h,I_h,R_h,S_d,V_d,I_d,R_d,B)\}>0$ This implies that, $\frac{dF}{dt}=0$ if $S_h=S_h^{\square},V_h=V_h^{\square},E_h=E_h^{\square},A_h=A_h^{\square},I_h=I_h^{\square},R_h=R_h^{\square},S_d=S_d^{\square},V_d=V_d^{\square},I_d=I_d^{\square},R_d=R_d^{\square},B=B^{\square}$. It is clear that $\frac{dF}{dt}\leq 0$. Then $\frac{dF}{dt}=0$ for all $S_h,V_h,E_h,A_h,I_h,R_h,S_d,V_d,I_d,R_d,B>0$. This implies that endemic equilibrium point $E_{\square}$ is globally asymptotically stable if $R_e>1$ and unstable otherwise. From an epidemiological perspective, the above analysis clearly indicates that leptospirosis will persist and spread within the population when the reproduction number (R_e) exceeds one. In such a scenario, the disease becomes uncontrollable, resulting in considerable mortality. Without the implementation of effective control measures, eradication becomes unfeasible, and both the disease burden and related deaths are likely to increase in the society.

Sensitivity Analysis

In this section, we compute the sensitivity indices of the basic reproduction number using the normalized toward sensitivity approach of Chitnis et al. (2008) and Mwasunda et al. (2021). This method defines the sensitivity index of a variable with respect to a parameter as the ratio of relative change in the variable to the relative change in the parameter. When the variable is a differentiable function of the parameter, the index can alternatively be expressed using partial derivatives (Martcheva 2015). Our analysis focus on the response of the basic reproduction number to variations in model parameters, with the normalized forward sensitivity index quantifying these changes as:

$$Y_p^{R_0}=\frac{\partial R_0}{\partial p}\times\frac{p}{R_0}, \tag{32}$$

where p stands for any parameter in effective reproduction number R_e.

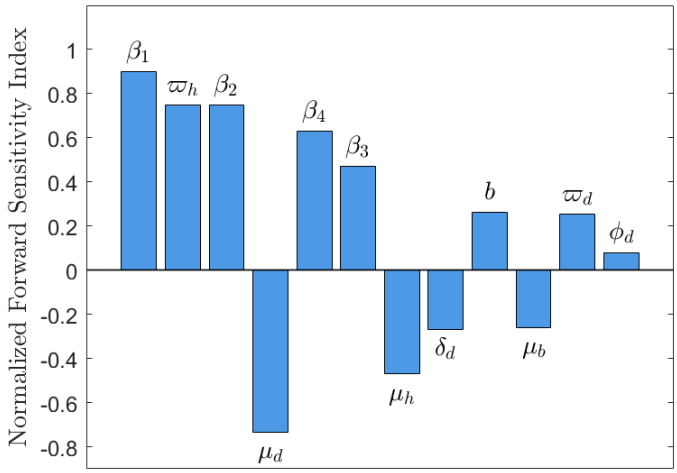


Figure 2: Sensitivity Analysis

Parameter	Index Value	Parameter	Index Value
β_4	0.6307119	β_3	0.470424
ϖ_d	0.25300	b	0.261038
μ_d	-0.25300	μ_h	-0.470677
δ_h	-0.712776	μ_b	-0.26038
ϕ_d	0.080164	δ_d	-0.26700
β_2	0.747	ϖ_h	0.318769

Table 2: Numerical Values of Sensitivity Indices of R_0

The sensitivity analysis in Figure 2 reveals that parameters from Table 2 with positive indices ($\varpi_h, \beta_2, \beta_4, \phi_d, \varpi_d, \beta_1, \beta_3, b$) increase R_0 and thus amplify transmission, with environmental infection probability (β_2) being especially critical. In contrast, parameters with negative indices ($\mu_d, \mu_h, \mu_b, \delta_d$) from Table 2 decrease R_0 when increased, reflecting the role of mortality and bacterial decay in lowering transmission. Since increasing human mortality is unrealistic, effective interventions should focus on environmental sanitation to boost bacterial decay and on managing domestic animal exposure. Overall, the strong influence of contact parameters (β_1, β_2 and β_3), significantly contributes to a higher R_0, leading to a higher induced infection rate as more people become infected. Therefore, it is necessary to employ interventions in order to curb the disease.

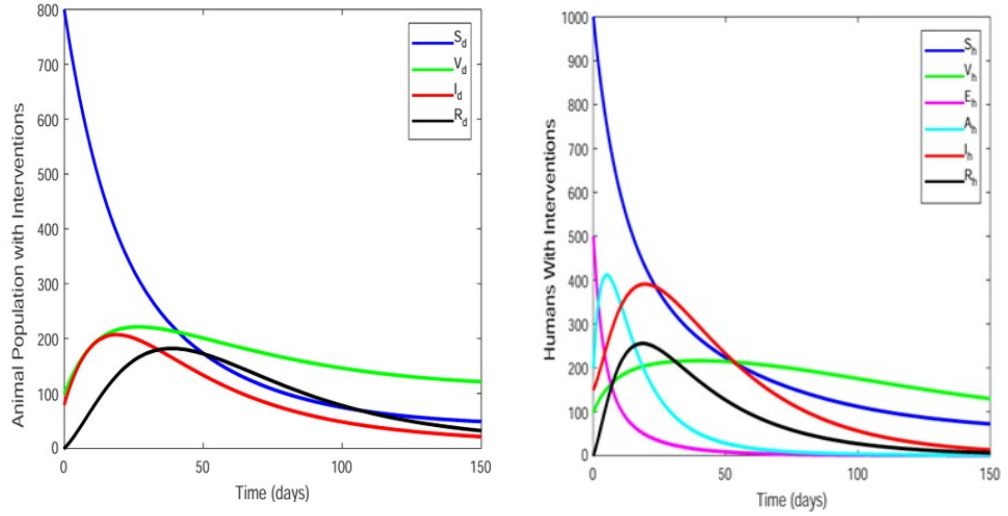
Numerical Simulations

The leptospirosis transmission model system (1), which captures interactions among human hosts, domestic animal reservoirs, and environmental contamination, is simulated using epidemiological parameter values provided in Table 1. Numerical simulations and graphical analyses are performed using MATLAB to investigate the dynamic behavior of the model. Parameter values, including the initial conditions of the state

variables, were obtained from the existing literature, while others were estimated. The initial population sizes for the respective compartments are set as follows: $S_h = 1000, V_d = 100, E_h = 600, A_h = 300,$. These values represent biologically and epidemiologically plausible scenarios reflecting real-world transmission patterns of leptospirosis between humans, domestic animals, and contaminated environments. The simulations aim to evaluate the effects of various demographic and epidemiological parameters on disease spread and to support the validation of both numerical and analytical results of the model.

Variation of Different Parameters on the Dynamics of the Leptospirosis Model

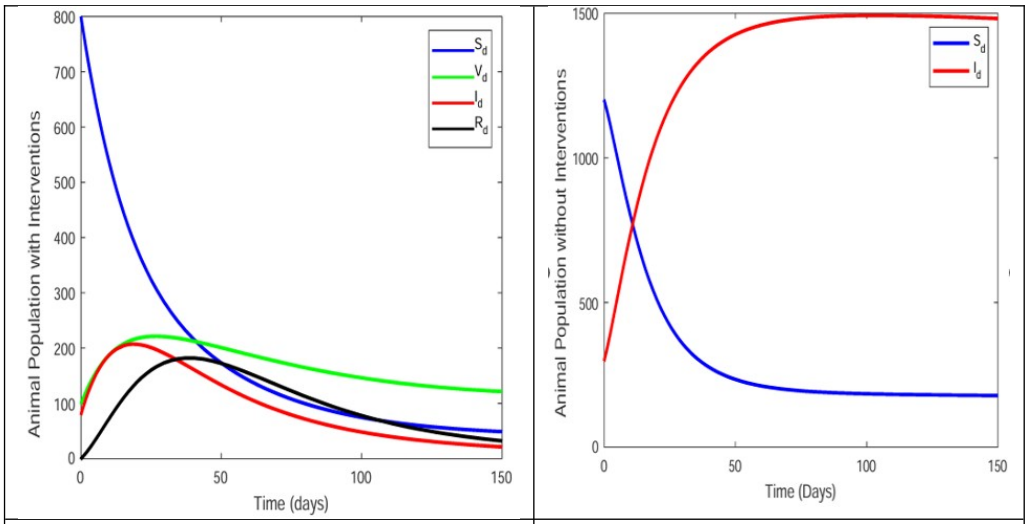
Figure 3(a) shows that without intervention, susceptible steadily decline as infections rise and settle at an endemic level; ongoing shedding of *Leptospira* sustains transmission and increases environmental bacterial concentration (Figure 5(a)). By contrast, Figure 3(b) demonstrates that screening, vaccination, treatment and non-pharmaceutical controls markedly reduce the infectious population, which lowers the environmental load (Figure 5(b)). Interventions also boost the recovered class; however, as immunity wanes, some individuals gradually return to susceptibility. Overall, these findings highlight that sustained implementation of control strategies can reduce symptomatic and asymptomatic cases, lower environmental contamination, and potentially eliminate leptospirosis from the population.



(a) (b)

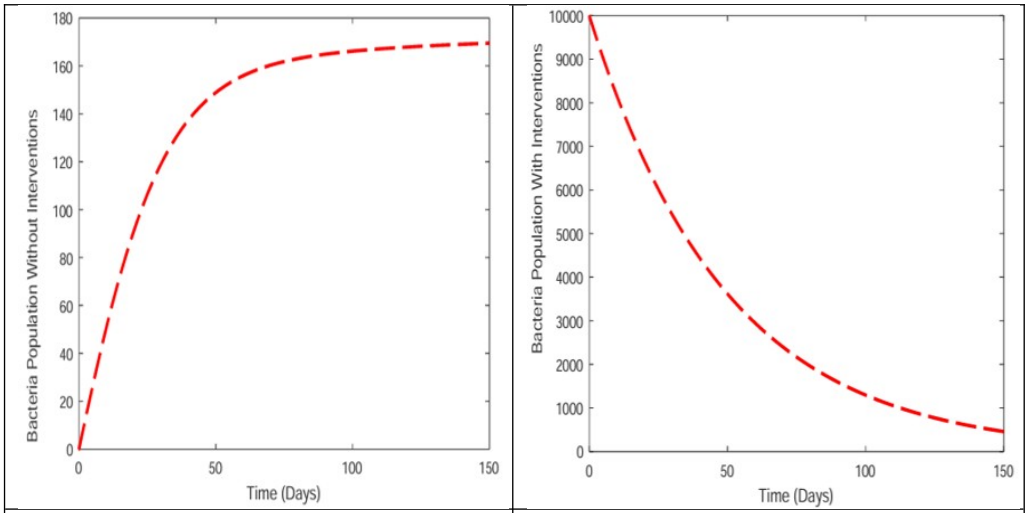
Figure. 3: General variation of the human population (a) without interventions and (b) with interventions in different classes

Figure 4(a) shows that without interventions, susceptible domestic animals decline as infections rise and settle at an endemic level, which elevates environmental *Leptospira* (Figure 5(a)). With vaccination, treatment, and non-pharmaceutical controls (Figure 4 (b)) infections drops sharply, lowering environmental bacterial load (Figure 5(b)). Vaccination is especially impactful, pushing animal prevalence toward zero; recoveries rise initially but later fall as immunity wanes. Overall, combined measures yield R_0= 0.037837, Indicating markedly suppressed transmission and highlighting the value of sustained, integrated interventions.



(b) (a)

Figure 4: General variation of the animal population (a) without interventions and (b) with interventions in different classes



(a) (b)

Figure 5: General variation of the bacteria population (a) without interventions, (b) with interventions

Effects of Multi- Interventions on Human and Domestic Animal Populations

In this subsection, simulations were performed to assess the impact of a single intervention and then the integrated interventions on the infected classes, on the dynamics of leptospirosis disease.

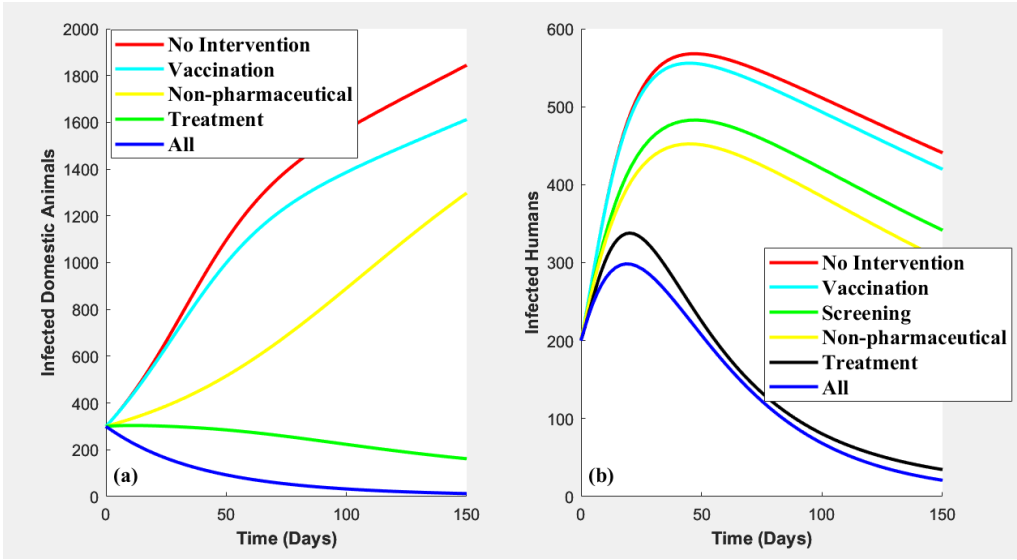
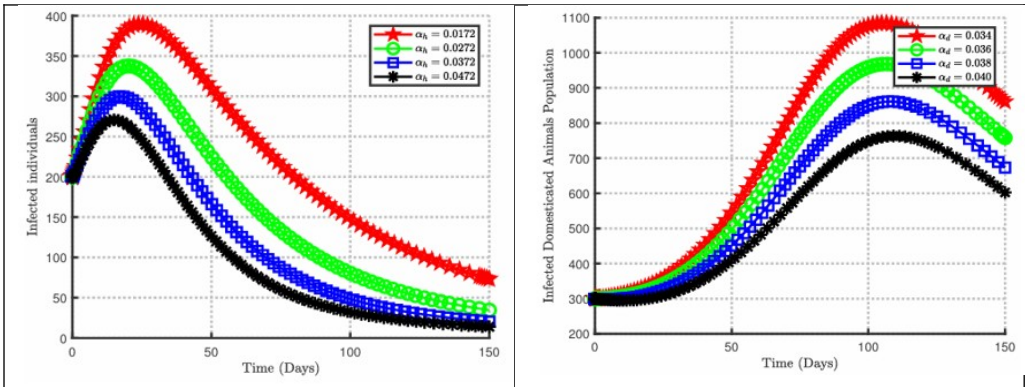


Figure 6: Effects of interventions on infected humans and domestic animals

Across both populations, integrated interventions; vaccination, screening (humans), treatment, and non-pharmaceutical controls substantially curb leptospirosis in Figures 6, combining measures outperforms any single strategy, rapidly suppressing transmission and prevalence. Treatment emerges as the strongest ever in both human and domestic animals, while vaccination, screening, and community level non pharmaceutical actions (Hygiene education, proper PPE, habitat modifications) provide vital complementary reductions. Together timely therapy plus sustained preventive measures offer the most reliable path toward control and potential elimination of leptospirosis of leptospirosis.

Effects of Varying Parameter Values of Interventions on Human and Domestic Animal Populations

This section presents simulations assessing the effects of human treatment (α_h), domestic animal treatment (α_d), and non-pharmaceutical controls (u) on infection dynamics. With other parameters held constant, varying α_h, α_d, and u showed that increasing treatment rates reduces the number of infective humans and animals, thereby lowering leptospirosis transmission in the community (Figures 6a–b).



(a) (b)

Figure 7: Variation of population under different values of (a) treatment (αh) on infected humans and (b) treatment α_d on infected domestic animals

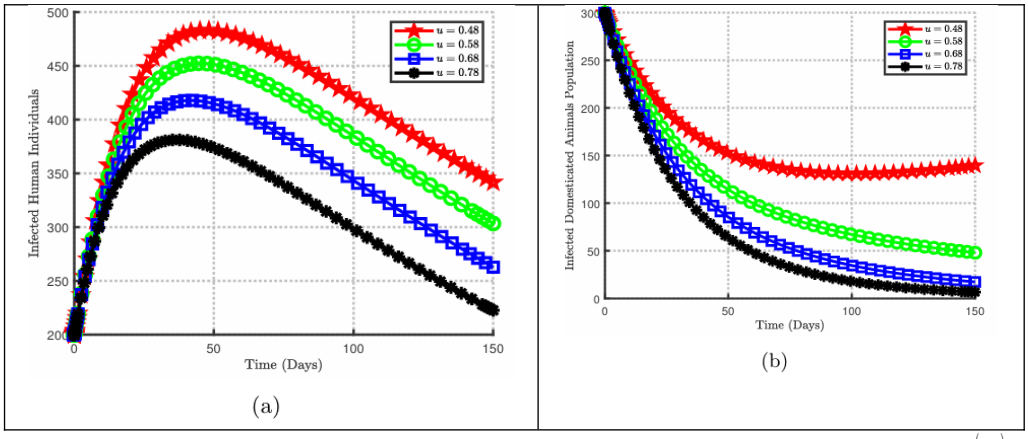


Figure 8: Variation of populations under different values of non-pharmaceutical control (u): (a) Infected humans and (b) Infected domestic animals.

Figures 8(a) and (b) show that increasing non-pharmaceutical control measures (u) reduces infective human and domestic animal populations. By varying their effectiveness while holding other parameters constant, the results highlight the role of environmental management, physical barriers, and public health education in limiting exposure to reservoir animals and contaminated environments. Collectively, these interventions lower both symptomatic and asymptomatic cases, helping to curb leptospirosis transmission

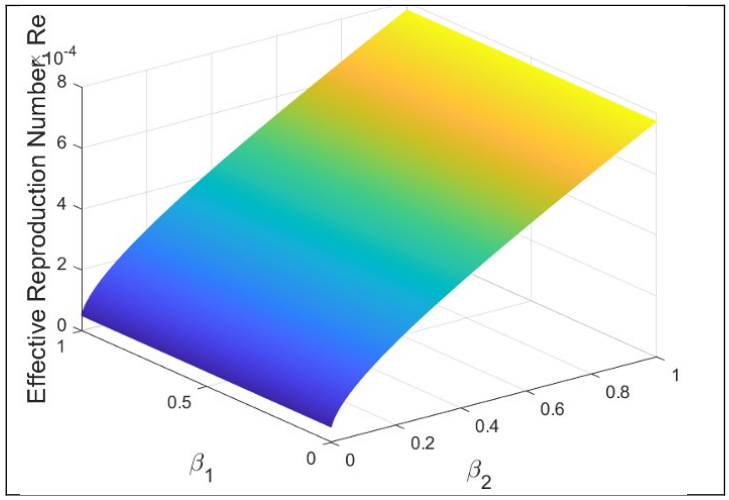


Figure 9: Effects of varying parameters β_1 and β_2 on the effective reproduction number, R_e

Figure 9 shows how the effective reproduction number R_e, depends on environmental infection of domestic animals (β_1) and environmental infection of humans (β_2). The surface plots indicate a nonlinear, compounding rise in R_e, as either or especially both rates increase, highlighting their joint role in sustain transmission. This

underscores that effective control requires simultaneously reducing environmental exposure for both humans and domestic animal

Discussion

Leptospirosis is a global public health concern, especially in developing urban areas where poor hygiene and overcrowding increase transmission risk. The disease also reduces livestock productivity and community economic stability. While several models exist, comprehensive frameworks that integrate screening, vaccination, treatment, and non-pharmaceutical controls in both humans and animals remain limited. Evidence from previous studies and our results confirm that human and animal treatment, enhanced by vaccination and environmental interventions, effectively reduce transmission. Sensitivity analysis highlights the most positively sensitive parameters which are transmission of infection from environment to domestic animal rate, β_1, human recruitment, ϖ_h, transmission probabilities, (β_2, β_4) and $(\beta \| 3)$, threshold contamination rate b and domestic animals' recruitment, ϖ_d . The natural mortality rates in domestic animals and humans) as well as the natural decay rate of *Leptospira* in the environment), remain the most negatively sensitive parameters. These findings align with earlier research (Guernier et al. 2018, Minter et al. 2022, Costa et al. 2015) emphasizing environmental and domestic animal reservoirs as major transmission drivers. Overall, our simulations qualitatively match observed patterns, supporting the relevance of integrated interventions for real-world leptospirosis control.

Conclusion and Recommendations

This study developed a deterministic compartmental mode of leptospirosis incorporating screening, vaccination, treatment, and non-pharmaceutical interventions for humans and domestic animals. The analysis showed that the disease dies out when $R_0 < 1$ and persist when $R_0 >$ 1. Sensitivity analysis identified environmental -to-human and environmental -to-domestic animals transmission probabilities $(\beta \| 2, \beta_1)$, recruitment rates (ϖ_h, ϖ_d) and contamination threshold (b) as a major driver of transmission, while human, domestic animal and decay rates (μ_h, μ_d, μ_b) had a negative effect on R_0. Simulations demonstrate that combined interventions are far more effective than single measures in lowering transmission and prevalence. The study concludes that integrated strategies treatment, vaccination, sanitation, reservoir control, fencing, use of PPE, and public health education are essential for controlling leptospirosis. These findings provide evidence base guidance for policy makers to design effective, context specific programs that can reduce disease burden, protect livestock productivity, and strengthen community stability, with future work recommended on seasonal effects, cost-effectiveness and empirical validation

Declaration of Competing Interests.

The authors declare that they have no competing interests or personal relationships that could have influenced the work reported in this paper.

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