2024-06-15

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

Masanja, Verdiana

Heliyon

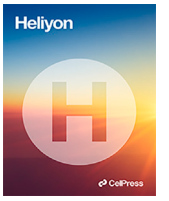
https://doi.org/10.1016/j.heliyon.2024.e32012

Heliyon 10 (2024) e32012

Contents lists available at ScienceDirect

Heliyon

journal homepage: www.cell.com/heliyon



Research article

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

Mfano Charles [a,b,*], Verdiana G. Masanja [a], Delfim F.M. Torres [c], Sayoki G. Mfinanga [d], G.A. Lyakurwa [a]

[a] School of Computational and Communication Science and Engineering, The Nelson Mandela African Institution of Science and Technology (NM-AIST), P.O. BOX 447, Arusha, Tanzania

[b] Department of ICT and Mathematics, College of Business Education (CBE), P.O. BOX 1968, Dar es Salaam, Tanzania

[c] Center for Research and Development in Mathematics and Applications (CIDMA), Department of Mathematics, University of Aveiro, 3810-193 Aveiro, Portugal

[d] NIMR Chief Research Scientist Fellow, Tanzania

ARTICLE INFO

Keywords:
Rabies disease
Mathematical model
Environment
Contact rate
Periodic transmission

ABSTRACT

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

1. Introduction

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

* Corresponding author at: School of Computational and Communication Science and Engineering, The Nelson Mandela African Institution of Science and Technology (NM-AIST), P.O. BOX 447, Arusha, Tanzania.

E-mail addresses: mfanoc@nm-aist.ac.tz (M. Charles), verdiana.masanja@nm-aist.ac.tz (V.G. Masanja), delfim@ua.pt (D.F.M. Torres), gsmfinanga@yahoo.com (S.G. Mfinanga), geminpeter.lyakurwa@nm-aist.ac.tz (G.A. Lyakurwa).

https://doi.org/10.1016/j.heliyon.2024.e32012
Received 17 October 2023; Received in revised form 23 May 2024; Accepted 27 May 2024

Available online 31 May 2024

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

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

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

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

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

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

2. Model formulation and model analysis

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

2.1. Model formulation

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

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

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

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

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

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

2.1.1. Description of model interaction

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

$$\chi_1 = \left(\tau_1 I_F + \tau_2 I_D + \tau_3 \lambda(M)\right) S_H, \tag{1}$$

where

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

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

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

$$\chi_2 = \left(\kappa_1 I_w + \kappa_2 I_D + \kappa_3 \lambda(M)\right) S_F. \tag{2}$$

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

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

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

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

Rabies virus is spread in the environment through shedding by infected free-range and domestic dogs, as well as humans, at rates denoted by ν_2, ν_3, and ν_1, respectively. Equation (4) can be used to estimate the virus recruitment in the environment:

$$\theta_4 = \left(\nu_1 I_H + \nu_2 I_F + \nu_3 I_D\right) M. \tag{4}$$

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

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

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

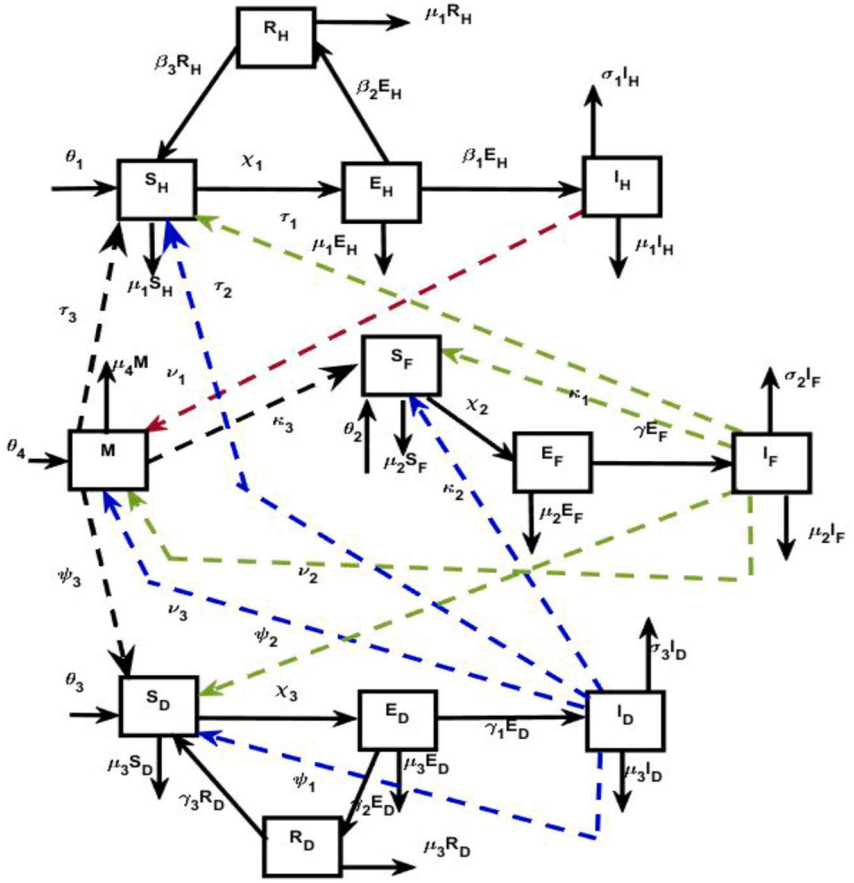


Fig. 1. Flow diagram for rabies transmission among human, free range and domestic dogs with parameters.

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

subject to the non-negative conditions

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

2.2. Model analysis

2.2.1. Model's invariant region

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

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

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

$$\Omega_F = \left\{ \left(S_F, E_F, I_F\right) \in \mathbb{R}_+^3 : 0 \le N_F \le \frac{\theta_2}{\mu_2} \right\},$$

$$\Omega_D = \left\{ \left(S_D, E_D, I_D, R_D\right) \in \mathbb{R}_+^4 : 0 \le N_D \le \frac{\theta_3}{\mu_3} \right\},$$

and Ω *is the positive invariant region.*

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

$$\frac{dN_H}{dt} = \frac{dS_H}{dt} + \frac{dE_H}{dt} + \frac{dI_H}{dt} + \frac{dR_H}{dt}. \tag{7}$$

Then, the equation (7) leads to

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

Thus, equation (8) becomes

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

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

$$\frac{dN_H}{dt} = \theta_1 - N_H\mu_1. \tag{10}$$

From the integrating factor we have

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

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

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

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

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

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

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

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

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

Since $N_H \le \frac{\theta_1}{\mu_1}$, $N_F \le \frac{\theta_2}{\mu_3}$ and $N_D \le \frac{\theta_3}{\mu_3}$, it follows that $I_H \le \frac{\theta_1}{\mu_1}$, $I_F \le \frac{\theta_2}{\mu_2}$ and $I_D \le \frac{\theta_3}{\mu_3}$.
Thus, equation (13) becomes

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

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

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

By using the integration factor, equation (15) becomes

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

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

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

Equation (17) gives

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

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

2.2.2. Positivity of the solution

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

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

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

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

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

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

Then, equation (19) yields

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

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

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

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

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

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

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

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

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

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

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

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

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

$$\frac{dx_i}{dt} = \mathcal{F}_i(x) - \left(\mathcal{V}_i^+(x) - \mathcal{V}_i^-(x)\right), \tag{23}$$

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

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

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

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

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

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

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

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

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

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

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

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

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

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

where

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

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

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

From equation (30), we obtained the eigenvalues as

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

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

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

Therefore, the spectral radius of the next generation Matrix is

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

2.2.5. Local sensitivity analysis

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

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

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

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

2.3. Existence of the steady state solution

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

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

with

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

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

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

where

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

The endemic equilibrium point of the rabies disease persists when $I_H,\ I_F,\ I_D,\ M>0$ and $\mathcal{R}_0\geq 1$, as summarized in Theorem 3.

Theorem 3. *The system model (5) has a unique endemic equilibrium if $\mathcal{R}_0\geq 1$ and $I_H,\ I_F,\ I_D,\ M>0$.*

3. Stability analysis

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

3.1. Local stability of the disease free equilibrium point

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

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

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

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

where

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

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

The first, fourth, fifth, eighth, and twelfth columns of the matrix $(J(\mathcal{E}0))$ in equation (36) contain the diagonal terms. It is obvious from the eigenvalues $\lambda_1=-\mu_1$, $\lambda_2=-a_3$, $\lambda_3=-\mu_2$, $\lambda_4=-\mu_3$, and $\lambda_5=-\mu_4$, respectively. Thus, the matrix $\left(J(\mathcal{E}_0)\right)$ reduces to

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

Again, the second and seventh columns of the matrix $(J(\mathcal{E}0))$ in (37) contain the diagonal terms. It is obvious from the eigenvalues $\lambda_6=-a_2$ and $\lambda_7=-a_8$. Thus, the matrix $\left(J(\mathcal{E}_0)\right)$ reduces to

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

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

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

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

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

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

$$\lambda^4+C_1\lambda^3+C_2\lambda^2+C_3\lambda+C=0. \tag{40}$$

Equations (36), (37), and (38) evidence that λ_1, λ_2, λ_3, λ_4, λ_5, λ_6, λ_7, and λ_8 exhibit negative real parts. By applying the Routh–Hurwitz criterion, the other four eigenvalues of the matrix equation (39) will also have negative real parts if all coefficients in equation (40) are greater than zero. Then,

$$\begin{cases} C_1=a_7+a_6+a_5+a_4>0, \\ C_2=-\gamma\, b_1-\gamma\, b_4+a_5a_4+a_6a_4+a_7a_4+a_6a_5+a_7a_5+a_7a_6>0, \\ C_3=\left((a_6+a_7)a_5-\gamma\, b_4+a_7a_6\right)a_4+\left(-\gamma\, b_4+a_7a_6\right)a_5-\gamma\, b_1(a_6+a_7)>0, \\ C=\gamma^2b_1b_4-\gamma^2b_2b_3-\gamma a_4a_5b_4-\gamma a_6a_7b_1+a_4a_5a_6a_7>0, \end{cases}$$

where

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

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

3.2. Global stability of the DFE point

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

$$\left.\begin{aligned} \frac{dY_s}{dt} &= G_0\left(Y_s-Y\left(\mathcal{E}_0\right)\right)+G_1Y_i, \\ \frac{dY_i}{dt} &= G_2Y_i, \end{aligned}\right\}$$

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

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

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

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

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

$$G_2 = \begin{pmatrix} -\mu_1-\beta_1-\beta_2 & 0 & 0 & \frac{\tau_1\theta_1}{\mu_1} & 0 & \frac{\tau_2\theta_1}{\mu_1} & 0 \\ \beta_1 & -\sigma_1-\mu_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\mu_2-\gamma & \frac{\kappa_1\theta_2}{\mu_2} & 0 & \frac{\kappa_1\theta_2}{\mu_2} & 0 \\ 0 & 0 & \gamma & -\mu_2-\sigma_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\psi_1\theta_3}{\mu_3(1+\rho_1)} & -\mu_3-\gamma_1-\gamma_2 & \frac{\psi_2\theta_3}{\mu_3(1+\rho_1)} & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\mu_3-\sigma_3 & 0 \\ 0 & \nu_1 & 0 & \nu_2 & 0 & \nu_3 & -\mu_4 \end{pmatrix}.$$

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

4. Model fitting and parameter estimation

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

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

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

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

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

5. Numerical simulations

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

Table 2
Model parameters, their description and values.

Parameters	Description	Value (Year^{-1})	Source
θ_1	Recruitment rate S_H	2000	(Estimated)
τ_1	The rate that S_H gets infection from I_F	0.0004	[10]
τ_2	The rate that S_H gets infection from I_D	0.0004	[10]
τ_3	The rate that S_H gets infection from M	[0.0003 0.0100]	(Estimated)
β_1	Progression rate out of E_H to I_H	$\frac{1}{6}$	[10,32]
β_2	Recovery rate of E_H	[0.54 1]	[12,32]
β_3	Rate of immunity loss of humans	1	(Estimated)
μ_1	Natural death rate of humans	0.0142	[34,35]
σ_1	Disease induced death rate for I_H	1	[12,32]
θ_2	Recruitment rate of free-range dogs	1000	(Estimated)
κ_1	The rate that S_F gets infection from I_F	0.00006	(Estimated)
κ_2	The rate that S_F gets infection from I_D	0.00005	(Estimated)
κ_3	The rate that S_F gets infection from M	[0.00001 0.00003]	(Estimated)
γ	The rate that S_F gets infection from I_F	$\frac{1}{6}$	[10,12,32]
σ_2	Disease induced death rate of I_F	0.09	[32,36]
μ_2	Natural mortality rate of free-range dogs	0.067	(Estimated)
θ_3	Recruitment rate of domestic dog population	1200	(Estimated)
ψ_1	The rate that S_D gets infection from I_D	0.0004	[24,36]
ψ_2	The rate that S_D gets infection from I_F	0.0004	[7]
ψ_3	The rate that S_D gets infection from M	0.0003	(Estimated)
μ_3	Natural death rate for domestic dog population	0.067	(Estimated)
σ_3	Disease induced death rate for I_D	0.08	[32]
γ_1	The rate at which E_D becomes I_D	$\frac{1}{6}$	[10,32]
γ_2	Recovery rate of E_D	0.09	[32]
γ_3	Rate of loss of temporary immunity for R_D	0.05	(Estimated)
ν_1	Environmental virus shedding rate from I_H	0.001	(Estimated)
ν_2	Environmental virus shedding rate from I_F	0.006	(Estimated)
ν_3	Environmental virus shedding rate from I_D	0.001	(Estimated)
μ_4	Natural removal rate of rabies from the environment	0.08	(Estimated)
ρ_1	The deterrent coefficient of domestic dog from I_F	10	[37]
ρ_2	The deterrent coefficient of domestic dog from I_D	8	(Estimated)
ρ_3	The deterrent coefficient of domestic dog from M	15	(Estimated)
C	Concentration of rabies in the environment	0.003 (PFU)/mL	(Estimated)

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

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

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

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

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

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

Therefore, to incorporate and unify the changing dynamics of rabies, transmission rates τ_1, τ_2, τ_3, ψ_1, ψ_2, ψ_3, κ_1, κ_2, κ_3, ν_1, ν_2 and ν_3, as applied by [42], are considered as

$$\tau_i = \tau_{(\text{mean})}\left(1 + \text{A}_i\sin\left(\frac{2\pi \text{t}}{\text{T}} + \phi\right)\right), \quad \psi_i = \psi_{(\text{mean})}\left(1 + \text{A}_i\sin\left(\frac{2\pi \text{t}}{\text{T}} + \phi\right)\right),$$
$$\kappa_i = \kappa_{(\text{mean})}\left(1 + \text{A}_i\sin\left(\frac{2\pi \text{t}}{\text{T}} + \phi\right)\right), \quad \nu_i = \nu_{(\text{mean})}\left(1 + \text{A}_i\sin\left(\frac{2\pi \text{t}}{\text{T}} + \phi\right)\right),$$

for $i = 1$, 2 and 3.

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

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

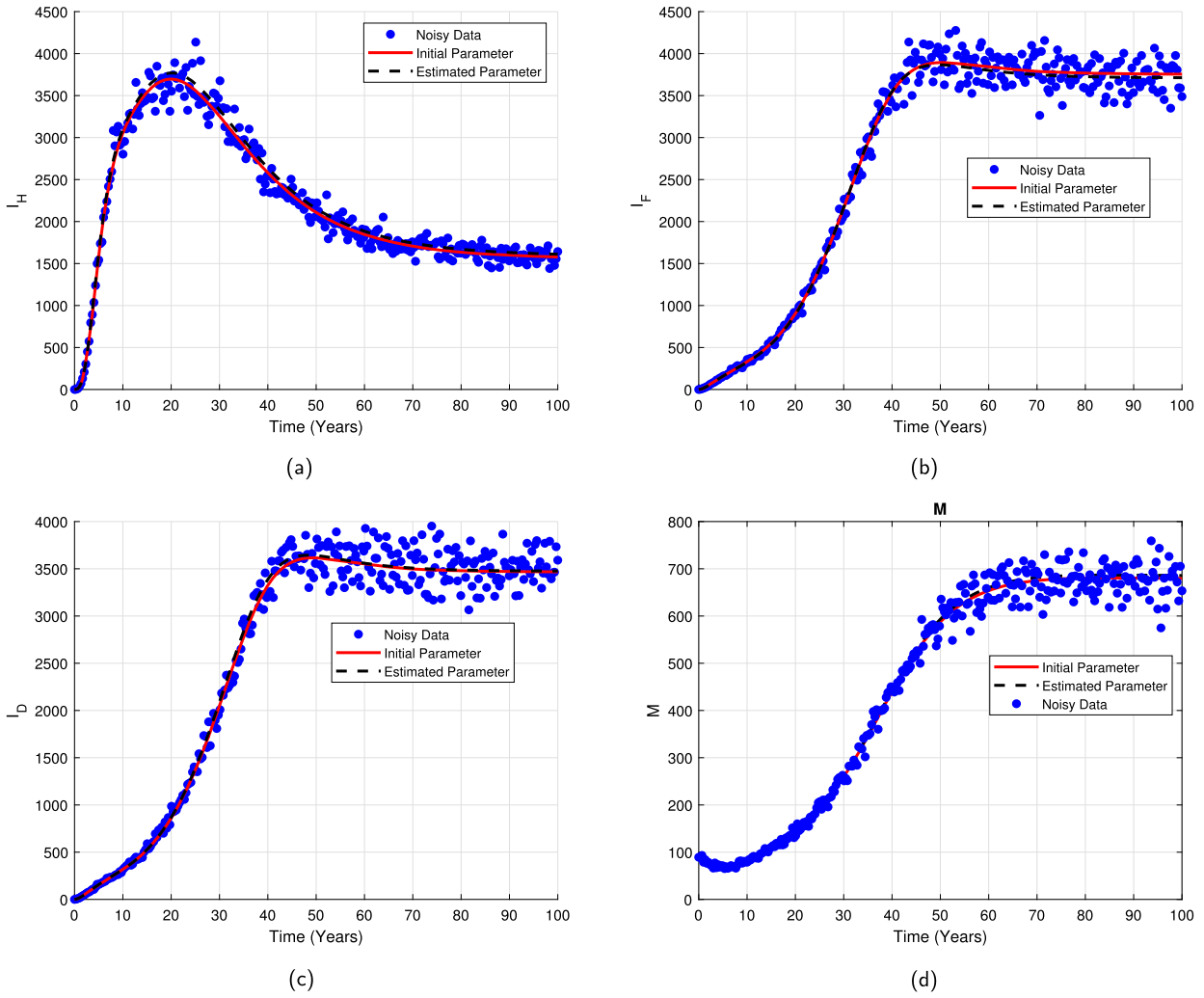


Fig. 2. Scatter estimated with standard deviation of 0.05 and numerical simulation (sold) with confidence interval of 95%.

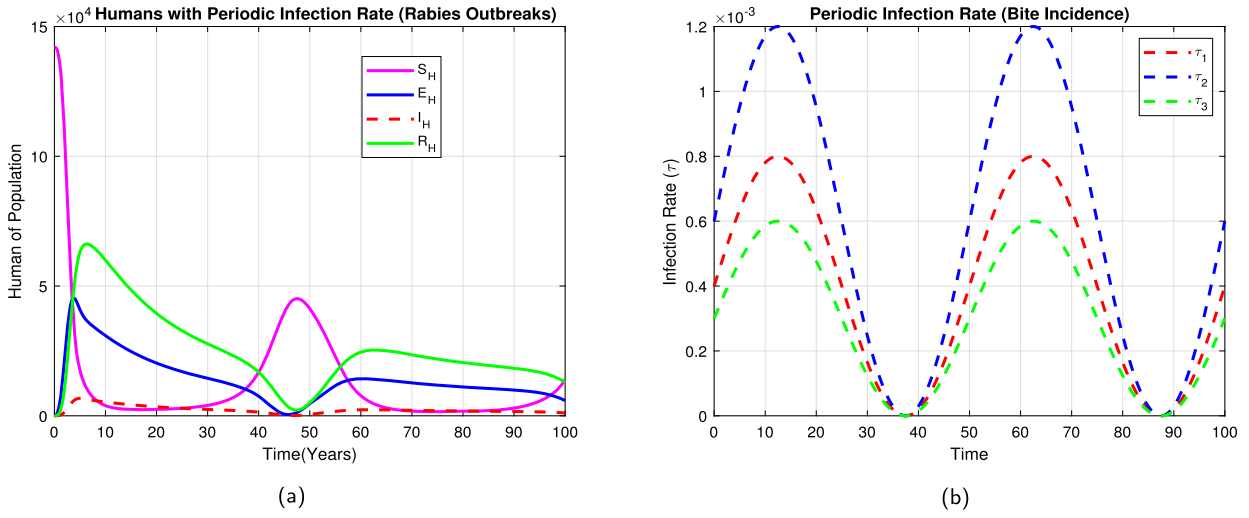


Fig. 3. The impact of human population bite incidence on the occurrence of periodic rabies outbreaks.

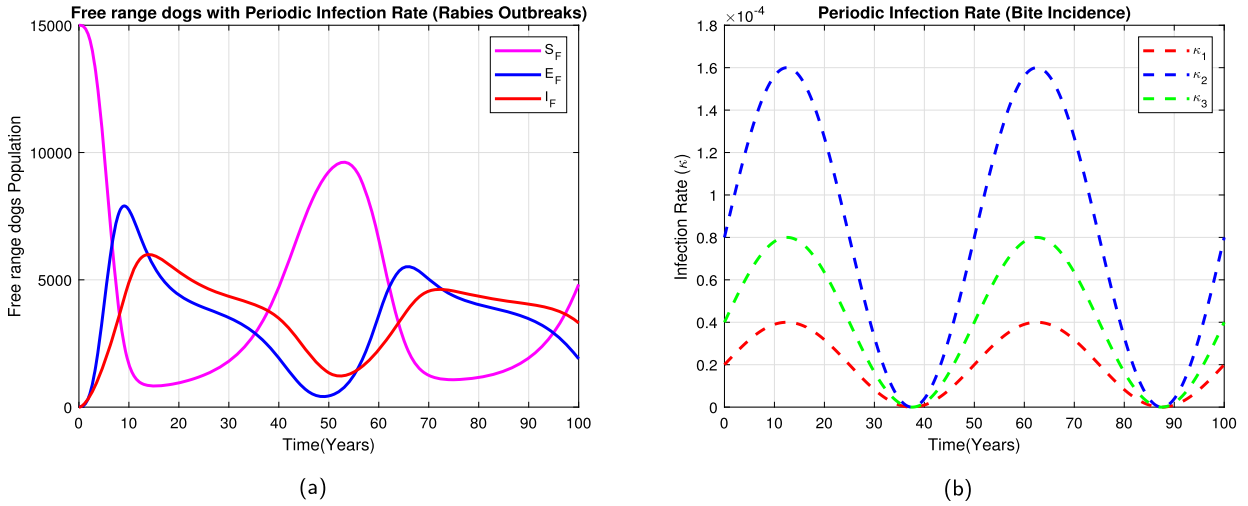


Fig. 4. The impact of free range dogs bite incidence on the occurrence of periodic rabies outbreaks.

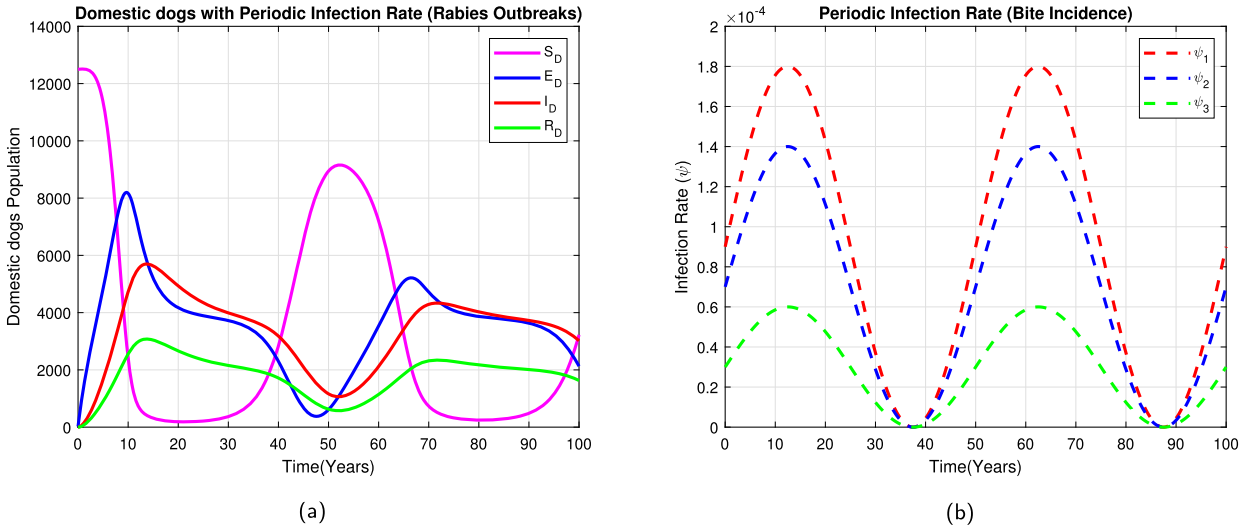


Fig. 5. The impact of domestic dogs bite incidence on the occurrence of periodic rabies outbreaks.

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

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

5.2. *Effect of varying the most sensitive parameters*

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

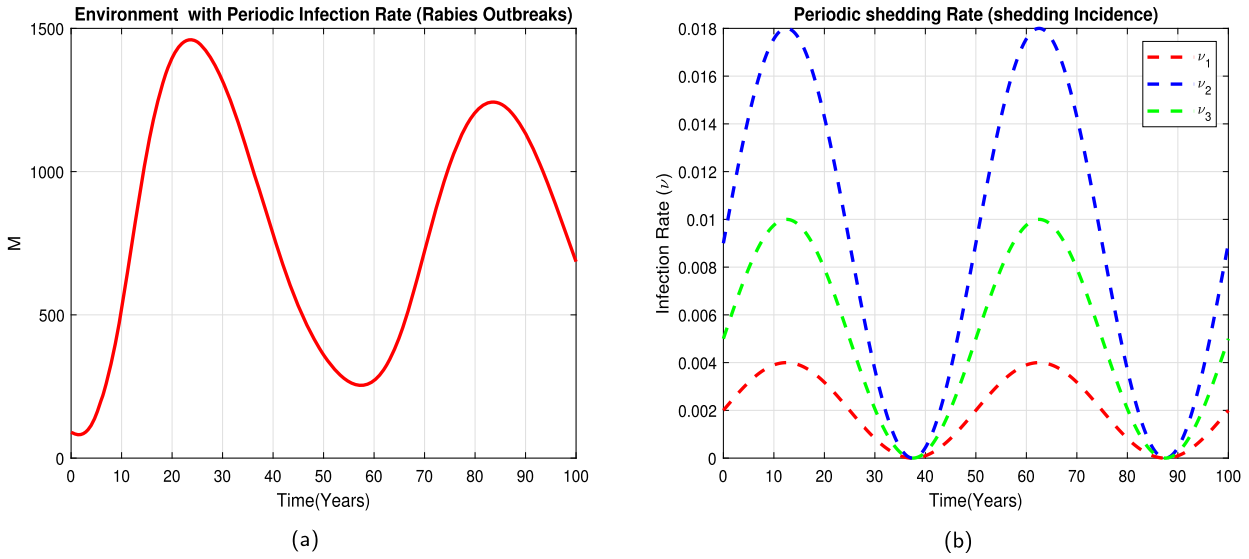
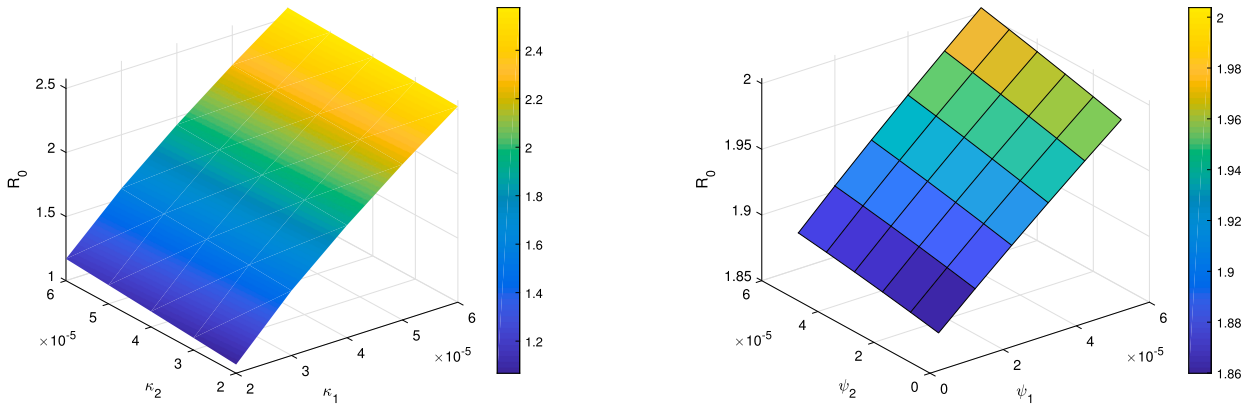


Fig. 6. The impact of environment shedding incidence on the occurrence of periodic rabies outbreaks.



(a) Effect of $\mathcal{R}_0$ with respect to κ_1 and κ_2.

(b) Effect of $\mathcal{R}_0$ with respect to ψ_1 and ψ_2.

Fig. 7. Effect of $\mathcal{R}_0$ with respect to κ_1, κ_2, ψ_1, and ψ_2.

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

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

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

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

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

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

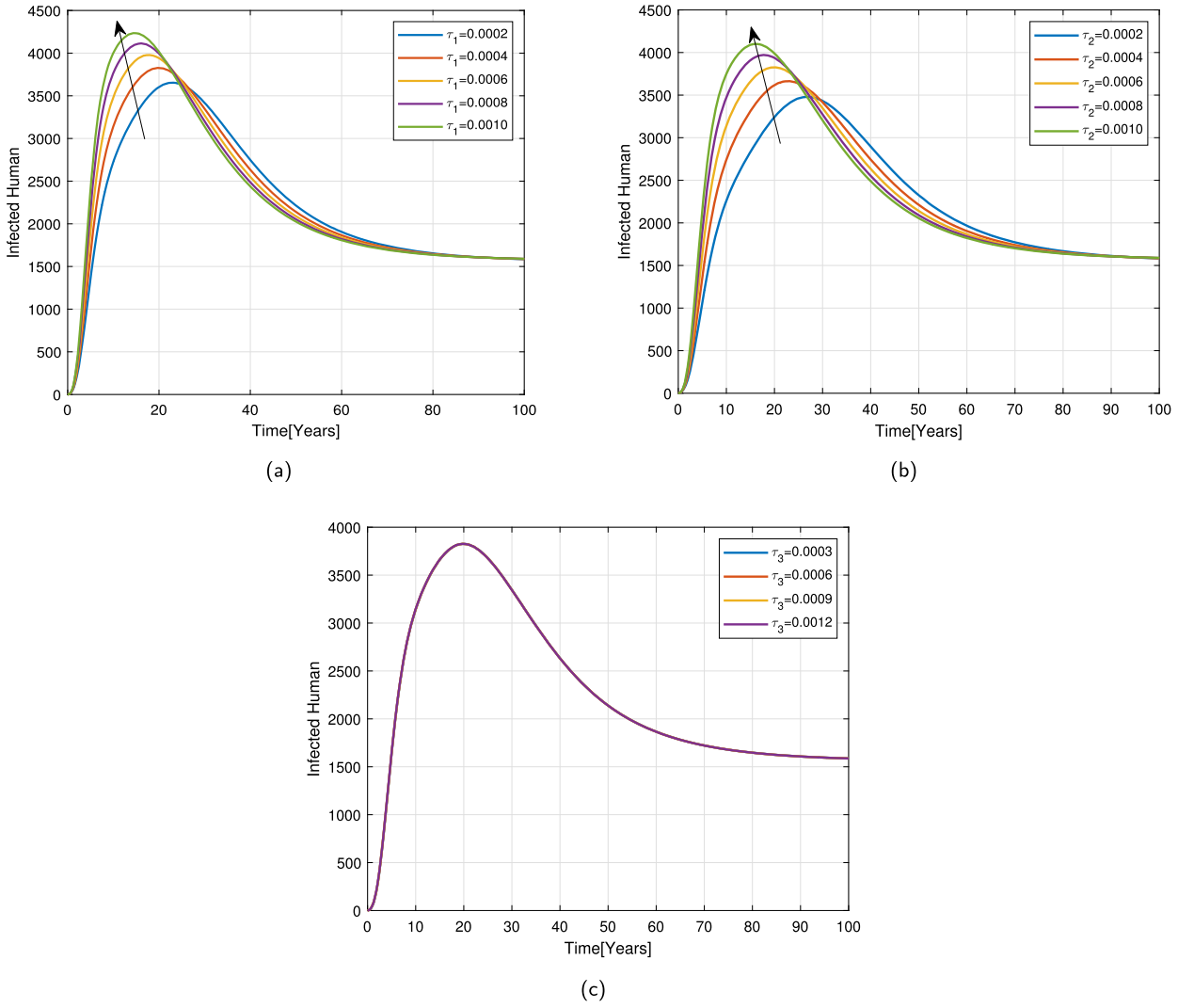


Fig. 8. Simulation results of model (5) for I_H with respect to τ_1, τ_2, and τ_3.

6. Discussion

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

7. Conclusion

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

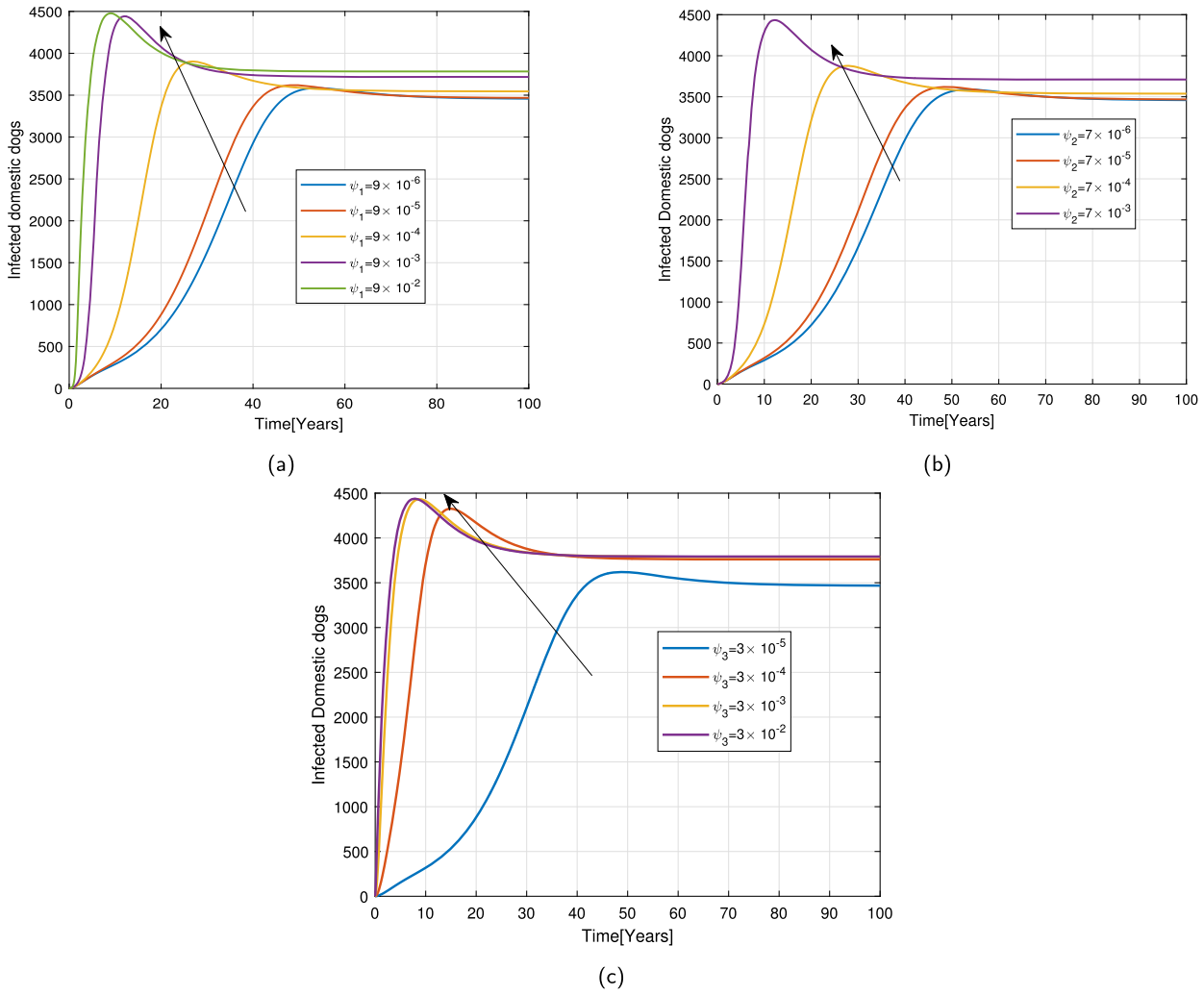


Fig. 9. Simulation results of model (5) for I_D with respect to ψ_1, ψ_2, and ψ_3.

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

Funding statement

Torres is supported by the Portuguese Foundation for Science and Technology (FCT), project UIDB/04106/2020 (CIDMA).

CRediT authorship contribution statement

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

Declaration of competing interest

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

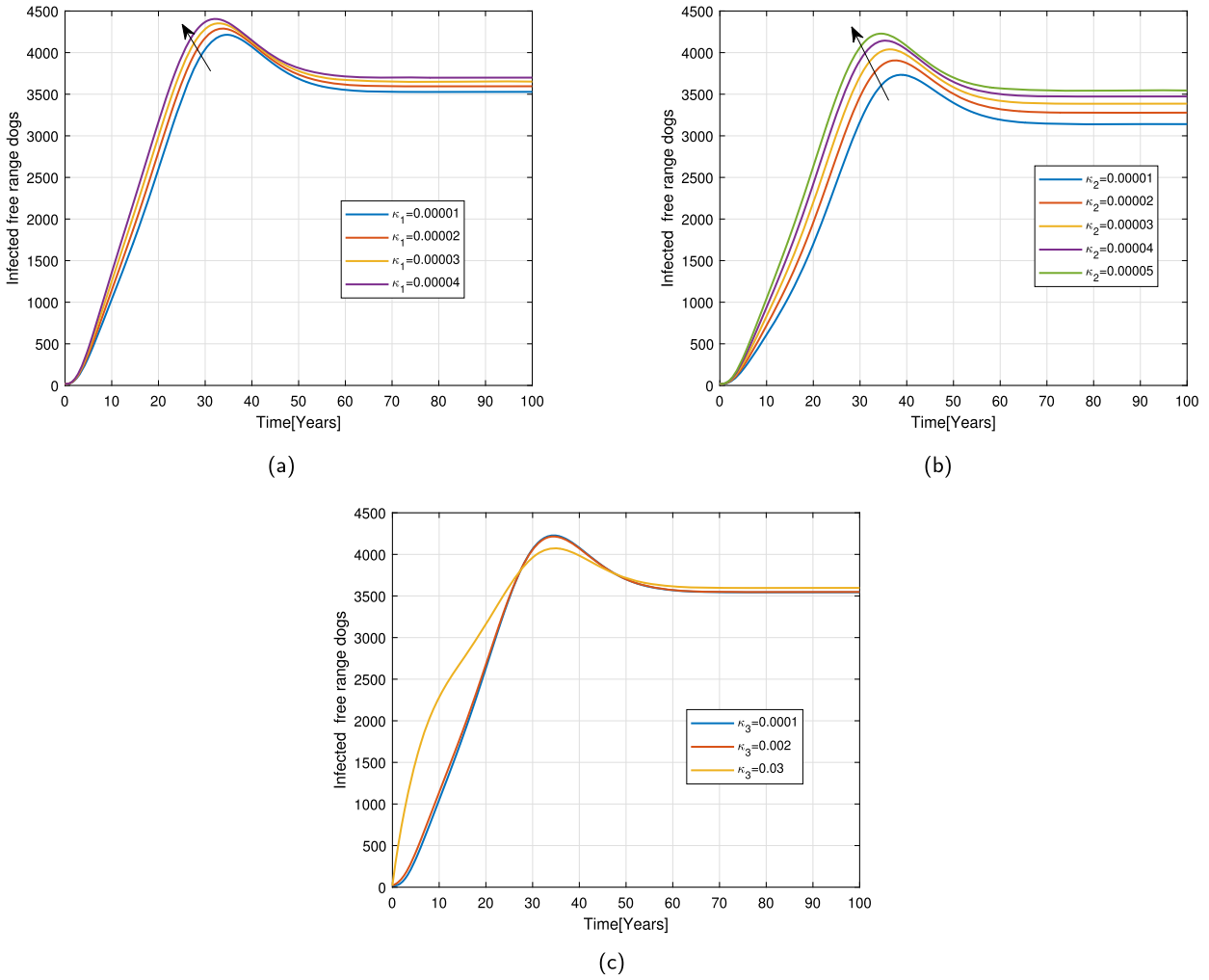


Fig. 10. Simulation results of model (5) for I_F with respect to τ_1, τ_2, and τ_3.

Data availability

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

Acknowledgements

The authors thank the handling editor and reviewer for helpful suggestions that improved this paper's quality. We acknowledge The Nelson Mandela African Institution of Science and Technology (NM-AIST) and the College of Business Education (CBE) for providing a conducive environment.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

$$\begin{aligned}\frac{d\mathcal{L}}{dt} &= G_1\left(1-\frac{S_H^*}{S_H}\right)\left(\theta_1+\beta_3 R_H-\mu_1 S_H-\chi_1\right)+G_2\left(1-\frac{E_H^*}{E_H}\right)\left(\chi_1-\left(\mu_1+\beta_1+\beta_2\right)E_H\right)\\ &+G_3\left(1-\frac{I_H^*}{I_H}\right)\left(\beta_1 E_H-\left(\sigma_1+\mu_1\right)I_H\right)+G_4\left(1-\frac{R_H^*}{R_H}\right)\left(\beta_2 E_H-\left(\beta_3+\mu_1\right)R_H\right)\\ &+G_5\left(1-\frac{S_F^*}{S_F}\right)\left(\theta_2-\chi_2-\mu_2 S_F\right)+G_6\left(1-\frac{E_F^*}{E_F}\right)\left(\chi_2-\left(\mu_2+\gamma\right)E_F\right)\\ &+G_7\left(1-\frac{I_F^*}{I_F}\right)\left(\gamma E_F-\left(\mu_2+\sigma_2\right)I_F\right)+G_8\left(1-\frac{S_D^*}{S_D}\right)\left(\theta_3-\mu_3 S_D-\chi_3+\gamma_3 R_D\right)\\ &+G_9\left(1-\frac{E_D^*}{E_D}\right)\left(\chi_3-\left(\mu_3+\gamma_1+\gamma_2\right)E_D\right)+G_{10}\left(1-\frac{I_D^*}{I_D}\right)\left(\gamma_1 E_D-\left(\mu_3+\delta_3\right)I_D\right)\\ &+G_{11}\left(1-\frac{R_D^*}{R_D}\right)\left(\gamma_2 E_D-\left(\mu_3+\gamma_3\right)R_D\right)+G_{12}\left(1-\frac{M^*}{M}\right)\left(\left(\nu_1 I_H+\nu_2 I_F+\nu_3 I_D\right)-\mu_4 M\right).\end{aligned} \tag{45}$$

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

$$\begin{aligned}\frac{d\mathcal{L}}{dt} &= G_1\left(1-\frac{S_H^*}{S_H}\right)\Bigg(\left(\tau_1 I_F^*+\tau_2 I_D^*+\tau_3\lambda(M^*)\right)S_H^*+\mu_1 S_H^*-\beta_3 R_H^*-\mu_1 S_H\\ &\quad-\left(\tau_1 I_F+\tau_2 I_D+\tau_3\lambda(M)\right)S_H+\beta_3 R_H\Bigg)\\ &+G_2\left(1-\frac{E_H^*}{E_H}\right)\left(\left(\tau_1 I_F+\tau_2 I_D+\tau_3\lambda(M)\right)S_H-\frac{\left(\tau_1 I_F^*+\tau_2 I_D^*+\tau_3\lambda(M^*)\right)S_H^* E_H}{E_H^*}\right)\\ &+G_3\left(1-\frac{I_H^*}{I_H}\right)\left(\beta_1 E_H-\frac{\beta_1 E_H^* I_H}{I_H^*}\right)+G_4\left(1-\frac{R_H^*}{R_H}\right)\left(\beta_2 E_H-\frac{\beta_2 E_H^* R_H}{R_H^*}\right)\\ &+G_5\left(1-\frac{S_F^*}{S_F}\right)\Bigg(\left(\kappa_1 I_F^*+\kappa_2 I_D^*+\kappa_3\lambda(M^*)\right)S_F^*+\mu_2 S_F^*-\mu_2 S_F\\ &\quad-\left(\kappa_1 I_F+\kappa_2 I_D+\kappa_3\lambda(M)\right)S_F\Bigg)\\ &+G_6\left(1-\frac{E_F^*}{E_F}\right)\left(\left(\kappa_1 I_F+\kappa_2 I_D+\kappa_3\lambda(M)\right)S_F-\frac{\left(\tau_1 I_F^*+\kappa_2 I_D^*+\kappa_3\lambda(M^*)\right)S_F^* E_F}{E_F^*}\right)\\ &+G_7\left(1-\frac{I_F^*}{I_F}\right)\left(\gamma E_F-\frac{\gamma E_F^* I_F}{I_F^*}\right)\\ &+G_8\left(1-\frac{S_D^*}{S_D}\right)\Bigg(\left(\frac{\psi_1 I_F^*}{1+\rho_1}+\frac{\psi_2 I_D^*}{1+\rho_2}\frac{\psi_3\lambda(M^*)}{1+\rho_3}\right)S_D^*+\mu_3 S_D^*-\gamma_3 R_D^*\\ &\quad-\left(\frac{\psi_1 I_F}{1+\rho_1}+\frac{\psi_2 I_D}{1+\rho_2}\frac{\psi_3\lambda(M)}{1+\rho_3}\right)S_D-\mu_3 S_D+\gamma_3 R_D\Bigg)\\ &+G_9\left(1-\frac{E_D^*}{E_D}\right)\left(\left(\frac{\psi_1 I_F}{1+\rho_1}+\frac{\psi_2 I_D}{1+\rho_2}\frac{\psi_3\lambda(M)}{1+\rho_3}\right)S_D-\frac{\left(\frac{\psi_1 I_F^*}{1+\rho_1}+\frac{\psi_2 I_D^*}{1+\rho_2}\frac{\psi_3\lambda(M^*)}{1+\rho_3}\right)S_D^* E_D}{E_D^*}\right)\\ &+G_{10}\left(1-\frac{I_D^*}{I_D}\right)\left(\gamma_1 E_D-\frac{\gamma_1 E_D^* I_D}{I_D^*}\right)+G_{11}\left(1-\frac{R_D^*}{R_D}\right)\left(\gamma_2 E_D-\frac{\gamma_2 E_D^* R_D}{R_D^*}\right)\\ &+G_{12}\left(1-\frac{M^*}{M}\right)\left(\nu_1 I_H+\nu_2 I_F+\nu_3 I_D-\frac{\left(\nu_1 I_H^*+\nu_2 I_F^*+\nu_3 I_D^*\right)M}{M^*}\right).\end{aligned} \tag{46}$$

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

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

Equation (47) can be written as

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

where

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

and

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

To simplify (48), let

$$
\begin{aligned}
&a=\frac{S_H}{S_H^*},\ b=\frac{E_H}{E_H^*},\ c=\frac{I_H}{I_H^*},\ d=\frac{R_H}{R_H^*},\ e=\frac{S_F}{S_F^*},\ f=\frac{E_F}{E_F^*},\ g=\frac{I_F}{I_F^*},\\
&h=\frac{S_D}{S_D^*},\ r=\frac{E_D}{E_D^*},\ n=\frac{I_D}{I_D^*},\ m=\frac{\lambda(M)}{\lambda(M^*)},\ l=\frac{R_D}{R_D^*},\ \text{and } k=\frac{M}{M^*}.
\end{aligned}
$$

One gets from (48) that

$$
\begin{aligned}
\mathcal{Q} &= \tau_1 S_H I_F\left(1-\frac{1}{a}\right)\left(\frac{1}{ab}-1\right)+\tau_2 S_H I_D\left(1-\frac{1}{a}\right)\left(\frac{1}{an}-1\right)+\tau_3 S_H\lambda(M)\left(1-\frac{1}{a}\right)\left(\frac{1}{am}-1\right)\\
&+\beta_3 R_H\left(1-\frac{1}{a}\right)\left(1-\frac{1}{d}\right)+\tau_1 S_H I_F\left(1-\frac{1}{b}\right)\left(1-\frac{b}{af}\right)+\tau_2 S_H I_D\left(1-\frac{1}{b}\right)\left(1-\frac{b}{an}\right)\\
&+\tau_3 S_H\lambda(M)\left(1-\tfrac{1}{b}\right)\left(1-\frac{b}{am}\right)+\beta_1 E_H\left(1-\frac{1}{c}\right)\left(1-\frac{b}{c}\right)+\beta_2 E_H\left(1-\frac{1}{d}\right)\left(1-\frac{b}{d}\right)\\
&+\kappa_1 S_F I_F\left(1-\frac{1}{e}\right)\left(\frac{1}{ef}-1\right)+\kappa_2 S_F I_D\left(1-\frac{1}{e}\right)\left(\frac{1}{ne}-1\right)+\kappa_3 S_F\lambda(M)\left(1-\frac{1}{e}\right)\left(\frac{1}{me}-1\right)\\
&+\kappa_1 S_F I_F\left(1-\frac{1}{f}\right)\left(1-\frac{f}{en}\right)+\kappa_2 S_F I_D\left(1-\frac{1}{f}\right)\left(1-\frac{f}{en}\right)+\kappa_3 S_F\lambda(M)\left(1-\frac{1}{f}\right)\left(1-\frac{f}{me}\right)\\
&+\gamma E_F\left(1-\frac{1}{f}\right)\left(1-\frac{g}{f}\right)+\frac{\psi_1 S_D I_F}{(1+\rho_1)}\left(1-\frac{1}{h}\right)\left(\frac{1}{hg}-1\right)+\frac{\psi_2 S_D I_F}{(1+\rho_2)}\left(1-\frac{1}{h}\right)\left(\frac{1}{hn}-1\right)\\
&+\frac{\psi_3 S_D\lambda(M)}{(1+\rho_3)}\left(1-\frac{1}{h}\right)\left(\frac{1}{mh}-1\right)+\gamma_3 R_D\left(1-\tfrac{1}{h}\right)\left(1-\frac{1}{l}\right)\frac{\psi_1 S_D I_F}{(1+\rho_1)}\left(1-\frac{1}{r}\right)\left(1-\frac{r}{hg}\right)\\
&+\frac{\psi_2 S_D I_F}{(1+\rho_2)}\left(1-\frac{1}{r}\right)\left(1-\frac{r}{hn}\right)+\frac{\psi_3 S_D I_F}{(1+\rho_3)}\left(1-\frac{1}{r}\right)\left(1-\frac{r}{mh}\right)+\gamma_1 E_D\left(1-\frac{1}{n}\right)\left(1-\frac{n}{r}\right)\\
&+\gamma_2 E_D\left(1-\frac{1}{l}\right)\left(1-\frac{l}{r}\right)+\nu_1 I_H\left(1-\tfrac{1}{k}\right)\left(1-\tfrac{k}{c}\right)+\nu_2 I_F\left(1-\tfrac{1}{k}\right)\left(1-\tfrac{k}{g}\right)+\nu_3 I_D\left(1-\tfrac{1}{k}\right)\left(1-\tfrac{k}{n}\right).
\end{aligned}
\tag{49}
$$

We express the equation (49) as

$$\left.\begin{aligned}
\mathcal{Q} &= \tau_1 S_H I_F\left(1-\frac{b}{ac}+\frac{1}{ac}-\frac{1}{b}\right)+\tau_2 S_H I_D\left(\frac{1}{an}-1+\frac{1}{a^2n}+\frac{1}{a}\right)+\tau_3 S_H \lambda(M)\left(\frac{1}{am}-1+\frac{1}{a^2m}+\frac{1}{a}\right)\\
&+\beta_3 R_H\left(1-\frac{1}{d}-\frac{1}{a}+\frac{1}{ad}\right)+\tau_1 S_H I_F\left(1-\frac{b}{af}-\frac{1}{b}+\frac{1}{af}\right)+\tau_2 S_H I_D\left(1-\frac{b}{an}-\frac{1}{b}+\frac{1}{an}\right)\\
&+\tau_3 S_H \lambda(M)\left(1-\frac{b}{af}-\frac{1}{b}+\frac{1}{af}\right)+\tau_2 S_H I_D\left(1-\frac{b}{am}-\frac{1}{b}+\frac{1}{am}\right)+\beta_1 E_H\left(1-\frac{b}{c}-\frac{1}{c}+\frac{b}{c^2}\right)\\
&+\beta_2 E_H\left(1-\frac{b}{d}-\frac{1}{d}+\frac{b}{d^2}\right)+\kappa_1 S_F I_F\left(\frac{1}{ef}-1-\frac{1}{e^2f}+\frac{1}{e}\right)+\kappa_2 S_F I_D\left(\frac{1}{en}-1-\frac{1}{e^2n}+\frac{1}{e}\right)\\
&+\kappa_3 S_F \lambda(M)\left(\frac{1}{em}-1-\frac{1}{e^2m}+\frac{1}{e}\right)+\kappa_1 S_F I_F\left(1-\frac{f}{en}-\frac{1}{f}+\frac{1}{en}\right)+\kappa_2 S_F I_D\left(1-\frac{f}{en}-\frac{1}{f}+\frac{1}{en}\right)\\
&+\kappa_3 S_F \lambda(M)\left(1-\frac{f}{me}-\frac{1}{f}+\frac{1}{me}\right)+\gamma E_F\left(1-\frac{g}{f}-\frac{1}{f}+\frac{g}{f^2}\right)+\frac{\psi_1 S_D I_F}{(1+\rho_1)}\left(1-\frac{1}{h}-\frac{1}{h^2g}+\frac{1}{h}\right)\\
&+\frac{\psi_2 S_D I_F}{(1+\rho_2)}\left(1-\frac{1}{h}-\frac{1}{h^2g}+\frac{1}{h}\right)+\frac{\psi_3 S_D \lambda(M)}{(1+\rho_3)}\left(\frac{1}{mh}-1-\frac{1}{h^2m}+\frac{1}{h}\right)+\gamma_3 R_D\left(1-\frac{1}{l}-\frac{1}{h}+\frac{1}{hl}\right)\\
&+\frac{\psi_1 S_D I_F}{(1+\rho_1)}\left(1-\frac{r}{hg}-\frac{1}{r}+\frac{1}{hg}\right)+\frac{\psi_2 S_D I_F}{(1+\rho_2)}\left(1-\frac{r}{hn}-\frac{1}{r}+\frac{1}{hn}\right)+\frac{\psi_3 S_D I_F}{(1+\rho_3)}\left(1-\frac{r}{hm}-\frac{1}{r}+\frac{1}{hm}\right)\\
&+\gamma_1 E_D\left(1-\frac{l}{r}-\frac{1}{l}+\frac{1}{r}\right)+\gamma_2 E_D\left(1-\frac{r}{hg}-\frac{1}{r}+\frac{1}{hg}\right)+\nu_1 I_H\left(1-\frac{k}{c}-\frac{1}{k}+\frac{1}{c}\right)+\nu_2 I_F\left(1-\frac{k}{g}-\frac{1}{k}+\frac{1}{g}\right)\\
&+\nu_3 I_D\left(1-\frac{k}{n}-\frac{1}{k}+\frac{1}{n}\right).
\end{aligned}\right\} \tag{50}$$

Now we make use of the following basic inequality.

Proposition 6. *If $\epsilon(y)=1-y+\ln y$, then $\epsilon(y)\le 0$ such that $1-y\le -\ln y$ if and only if $y>0$.*

From equation (50), we have

$$1-\frac{1}{d}-\frac{1}{a}+\frac{1}{ad}=\left(1-\frac{1}{d}\right)+\left(1-\frac{1}{a}\right)-\left(1-\frac{1}{ad}\right). \tag{51}$$

Using Proposition 6 and the concept of geometric mean, equation (51) can be written as

$$\begin{aligned}
\left(1-\frac{1}{d}\right)+\left(1-\frac{1}{a}\right)-\left(1-\frac{1}{ad}\right) &\le -\ln\left(\frac{1}{d}\right)-\ln\left(\frac{1}{a}\right)+\ln\left(\frac{1}{ad}\right)\\
&\le \ln\left(a\times d\times\frac{1}{ad}\right)=\ln(1)=0.
\end{aligned} \tag{52}$$

Following similar procedures in (52), we get

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

From equation (47), the global stability holds only if $\frac{d\mathcal{L}}{dt}\le 0$. Now, if $\mathcal{R}<\mathcal{Q}$, then $\frac{d\mathcal{L}}{dt}$ will be negative definite, which implies that $\frac{d\mathcal{L}}{dt}<0$ and $\frac{d\mathcal{L}}{dt}=0$ only at the endemic equilibrium point $\mathbb{E}^*$. Hence, by LaSalle's invariance principle [28], the only invariant set in $\left\{\left(S_H(t),E_H(t),I_H(t),R_H(t),S_F(t),E_F(t),I_F(t),S_D(t),E_D(t),I_D(t),R_H(t)\right)\in\mathbb{R}_+^{12}\right\}$: $\left\{\left(S_H(t),E_H(t),I_H(t),R_H(t),S_F(t),E_F(t),I_F(t),S_D(t),E_D(t),I_D(t),R_H(t)\right)\to\mathbb{E}^*\right\}$ is the singleton endemic point $\mathbb{E}^*$. Thus, any solution to the rabies model (5) which intersect the interior $\mathbb{R}_+^{12}$ limits to $\mathbb{E}^*$ is globally asymptotically stable whatever $\mathcal{R}_0>1$. □

References

[1] Paola De Benedictis, Stefania Leopardi, Wanda Markotter, Andres Velasco-Villa, The importance of accurate host species identification in the framework of rabies surveillance, control and elimination, Viruses 14 (3) (2022) 492.

[2] Anil Kumar, Sonam Bhatt, Ankesh Kumar, Tanmoy Rana, Canine rabies: an epidemiological significance, pathogenesis, diagnosis, prevention and public health issues, Comp. Immunol. Microbiol. Infect. Dis. (2023) 101992.

[3] Pallvi Slathia, Riya Abrol, Satuti Sharma, Sakshi Sharma, Rabies: a review on clinical signs, prevention and control, Pharma Innov. J. 12 (5) (2023) 1675–1680.

[4] Barry J. McMahon, Serge Morand, Jeremy S. Gray, Ecosystem change and zoonoses in the Anthropocene, Zoonoses Public Health 65 (7) (2018) 755–765.

[5] Andrea Julia Nigg, Pamela L. Walker, Overview, prevention, and treatment of rabies, Pharmacother. J. Hum. Pharmacol. Drug Ther. 29 (10) (2009) 1182–1195.

[6] Nicholas Johnson, Adam F. Cunningham, Anthony R. Fooks, The immune response to rabies virus infection and vaccination, Vaccine 28 (23) (2010) 3896–3901.

[7] Demsis Dejene Hailemichael, Geremew Kenassa Edessa, Purnachandra Rao Koya, Effect of vaccination and culling on the dynamics of rabies transmission from stray dogs to domestic dogs, J. Appl. Math. (2022) 2022.

[8] A. Bilal, Rabies is a zoonotic disease: a literature review, Occup. Med. Health Aff. 9 (2) (2021).

[9] Céline Mbilo, Andre Coetzer, Bassirou Bonfoh, Angélique Angot, Charles Bebay, Bernardo Cassamá, Paola De Benedictis, Moina Hasni Ebou, Corneille Gnanvi, Vessaly Kallo, et al., Dog rabies control in west and central Africa: a review, Acta Trop. 224 (2021) 105459.

[10] Huaiyu Tian, Yun Feng, Bram Vrancken, Bernard Cazelles, Hua Tan, Mandev S. Gill, Qiqi Yang, Yidan Li, Weihong Yang, Yuzhen Zhang, et al., Transmission dynamics of re-emerging rabies in domestic dogs of rural china, PLoS Pathog. 14 (12) (2018) e1007392.

[11] Maganga Sambo, Sarah Cleaveland, Heather Ferguson, Tiziana Lembo, Cleophas Simon, Honorati Urassa, Katie Hampson, The burden of rabies in Tanzania and its impact on local communities, PLoS Negl. Trop. Dis. 7 (11) (2013) e2510.
[12] Shafiu Abdulmajid, Adamu Shitu Hassan, Analysis of time delayed rabies model in human and dog populations with controls, Afr. Math. 32 (5–6) (2021) 1067–1085.
[13] Y.A. Amoako, P. El-Duah, A.A. Sylverken, M. Owusu, R. Yeboah, R. Gorman, T. Adade, J. Bonney, W. Tasiame, K. Nyarko-Jectey, et al., Rabies is still a fatal but neglected disease: a case report, J. Med. Case Rep. 15 (1) (2021) 1–6.
[14] Fredrick M. Abrahamian, Charles E. Rupprecht, Rhabdovirus: rabies, in: Viral Infections of Humans: Epidemiology and Control, Springer, 2022, pp. 1–49.
[15] Shigui Ruan, Modeling the transmission dynamics and control of rabies in china, Math. Biosci. 286 (2017) 65–93.
[16] Abayomi Ayotunde Ayoade, Olumuyiwa James Peter, Tokunbo Aderemi Ayoola, A.A. Victor, A saturated treatment model for the transmission dynamics of rabies, Malays. J. Comput. 4 (1) (2019) 201–213.
[17] Michael Chapwanya, Phindile Dumani, Environment considerations on the spread of rabies among African wild dogs (Lycaon pictus) with control measures, Math. Methods Appl. Sci. 45 (8) (2022) 4124–4139.
[18] H. Kadowaki, K. Hampson, K. Tojinbara, A. Yamada, K. Makita, The risk of rabies spread in Japan: a mathematical modelling assessment, Epidemiol. Infect. 146 (10) (2018) 1245–1252.
[19] Abayomi Ayotunde Ayoade, Mohammed Olanrewaju Ibrahim, Modeling the dynamics and control of rabies in dog population within and around Lagos, Nigeria, Eur. Phys. J. Plus 138 (5) (2023) 397.
[20] Aberu Mengistu Tulu, Purnachandra Rao Koya, The impact of infective immigrants on the spread of dog rabies, Am. J. Appl. Math. 5 (3) (2017) 68.
[21] Buddhi Pantha, Sunil Giri, Hem Raj Joshi, Naveen K. Vaidya, Modeling transmission dynamics of rabies in Nepal, Infect. Dis. Model. 6 (2021) 284–301.
[22] Tesfaye Tadesse Ega, Livingstone Luboobi, Dmitry Kuznetsov, Abraham Haile Kidane, Sensitivity analysis and numerical simulations for the mathematical model of rabies in human and animal within and around Addis Ababa, Asian J. Math. Appl. (2015) ama0271, 23 pp.
[23] Michelle Marie Esposito, Sara Turku, Leora Lehrfield, Ayat Shoman, The impact of human activities on zoonotic infection transmissions, Animals 13 (10) (2023) 1646.
[24] Katie Hampson, Francesco Ventura, Rachel Steenson, Rebecca Mancy, Caroline Trotter, Laura Cooper, Bernadette Abela-Ridder, Lea Knopf, Moniek Ringenier, Tenzin Tenzin, et al., The potential effect of improved provision of rabies post-exposure prophylaxis in Gavi-eligible countries: a modelling study, Lancet Infect. Dis. 19 (1) (2019) 102–111.
[25] Maria Cristina Rulli, Paolo D'Odorico, Nikolas Galli, David T.S. Hayman, Land-use change and the livestock revolution increase the risk of zoonotic coronavirus transmission from rhinolophid bats, Nature Food 2 (6) (2021) 409–416.
[26] Hao Peng, Qing Yang, Xinhong Zhang, Daqing Jiang, Transmission dynamics of a high dimensional rabies epidemic model in a markovian random environment, Qual. Theory Dyn. Syst. 21 (2) (2022) 46.
[27] Linda JS Allen, Pauline van den Driessche, The basic reproduction number in some discrete-time epidemic models, J. Differ. Equ. Appl. 14 (10–11) (2008) 1127–1147.
[28] Joseph P. LaSalle, Stability theory and invariance principles, in: Dynamical Systems, Elsevier, 1976, pp. 211–222.
[29] Hyun Mo Yang, The basic reproduction number obtained from jacobian and next generation matrices–a case study of dengue transmission modelling, Biosystems 126 (2014) 52–75.
[30] Sangeeta Saha, Guruprasad Samanta, Dynamics of an epidemic model under the influence of environmental stress, Math. Biol. Bioinform. 16 (2) (2021) 201–243.
[31] Samath Dharmaratne, Supun Sudaraka, Ishanya Abeyagunawardena, Kasun Manchanayake, Mahen Kothalawala, Wasantha Gunathunga, Estimation of the basic reproduction number (r0) for the novel coronavirus disease in Sri Lanka, J. Virol. 17 (2020) 1–7.
[32] Juan Zhang, Zhen Jin, Gui-Quan Sun, Tao Zhou, Shigui Ruan, Analysis of rabies in china: transmission dynamics and control, PLoS ONE 6 (7) (2011) e20891.
[33] Joshua Kiddy, K. Asamoah, Francis T. Oduro, Ebenezer Bonyah, Baba Seidu, et al., Modelling of rabies transmission dynamics using optimal control analysis, J. Appl. Math. (2017) 2017.
[34] World Health Organization, et al., Working to Overcome the Global Impact of Neglected Tropical Diseases: First WHO Report on Neglected Tropical Diseases, World Health Organization, 2010.
[35] World Health Organization, WHO Expert Consultation on Rabies: Second Report, vol. 982, World Health Organization, 2013.
[36] Kwaku Mari Addo, An SEIR Mathematical Model for Dog Rabies. Case Study: Bongo District, Ghana, PhD thesis, Kwame Nkrumah University of Science and Technology, 2012.
[37] Shigui Ruan, Spatiotemporal epidemic models for rabies among animals, Infect. Dis. Model. 2 (3) (2017) 277–287.
[38] Jean Claude Kamgang, Gauthier Sallet, Computation of threshold conditions for epidemiological models and global stability of the disease-free equilibrium (DFE), Math. Biosci. 213 (1) (2008) 1–12.
[39] Michael Y. Li, An Introduction to Mathematical Modeling of Infectious Diseases, vol. 2, Springer, 2018.
[40] In Jae Myung, Tutorial on maximum likelihood estimation, J. Math. Psychol. 47 (1) (2003) 90–100.
[41] Rihana Taher Abdulmoghni, Ahmed Hasan Al-Ward, Khaled Abdullah Al-Moayed, Mohammed Abdullah Al-Amad, Yousef S. Khader, Incidence, trend, and mortality of human exposure to rabies in Yemen, 2011-2017: observational study, JMIR Public Health Surveill. 7 (6) (2021) e27623.
[42] Protyusha Dutta, Guruprasad Samanta, Juan J. Nieto, Periodic transmission and vaccination effects in epidemic dynamics: a study using the SIVIS model, Nonlinear Dyn. (2024) 1–29.
[43] Shaibu Osman, Getachew Teshome Tilahun, Seleshi Demie Alemu, Winnie Mokeira Onsongo, Analysis of the dynamics of rabies in North Shewa, Ethiopia, Ital. J. Pure Appl. Math. 48 (2022) 877–902.
[44] Jane S. Musaili, Isaac Chepkwony, A mathematical model of rabies transmission dynamics in dogs incorporating public health education as a control strategy-a case study of Makueni county, J. Adv. Math. Comput. Sci. 35 (1) (2020) 1–11.