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MATHEMATICAL MODELING AND ANALYSIS OF RABIES TRANSMISSION DYNAMICS WITH VACCINATION

[1]Isiyaku, M., *[1]Abdullahi, M., [2]Husseini, A., [3]Song, I. M., [3]Bade, S. H., [4]Abubakar, A. and [5]Gidado, U.

[1]Department of Mathematics, Modibbo Adama University, Yola, Nigeria.
[2]Department of Mathematics, Nigerian Army University, Biu, Nigeria.
[3]Department of Mathematics, Federal College of Education, Yola, Nigeria.
[4]Department of Mathematics Education, Adamawa State Polytechnic, Yola, Nigeria.
[5]Government Day Senior Secondary School Hammawa Toungo, Yola, Nigeria.

*Corresponding authors' email: mabdallah@mau.edu.ng

ABSTRACT

Rabies remains a fatal zoonotic disease that poses a significant public health challenge, particularly in low- and middle-income countries. In this study, we develop and analyze a deterministic compartmental model based on ordinary differential equations to investigate the transmission dynamics of rabies between humans and dogs. Basic properties of the model such as positivity, boundedness, and the existence of equilibria are established, and the model is well-posed mathematically and biologically. The basic reproduction number, R_D, is derived using the next-generation matrix method, and stability analysis reveals that the rabies-free equilibrium is locally and globally asymptotically stable when $R_D < 1$. The model exhibits a unique endemic equilibrium, when $R_D > 1$, which is also globally stable whenever $R_D < 1$. Sensitivity analysis using both normalized forward sensitivity indices and partial rank correlation coefficients (PRCC) identifies the most influential parameters on R_D. Numerical simulations demonstrate that vaccination, particularly in the dog population is the most effective control strategy in reducing the spread of rabies. The results emphasize the importance of prioritizing control interventions in the dog population to effectively manage and reduce the burden of rabies.

Keywords: Rabies, Reproduction Number, Vaccination, Stability, Sensitivity

INTRODUCTION

Rabies is a viral infection that impacts mammals, including humans, and is caused by the Rabies lyssa virus. The virus spreads from the point of entry to the brain, leading to inflammation and damage to the nervous system (Paola *et al.,* 2022, Kumar *et al.,* 2023). Although dogs are the main carriers of the rabies virus responsible for over 99% of human cases globally other animals like bats, raccoons, skunks, and foxes can also transmit the virus through bites or scratches (Pallvi *et al.,* 2023).

Rabies symptoms typically appear between 20 days and 3 months after exposure and may include fever, headache, fatigue, agitation, anxiety, hallucinations, fear of water (hydrophobia), difficulty swallowing, and paralysis. The onset period can range from 1 week to 1 year post-exposure, depending on factors such as the site of viral entry and the amount of virus introduced. In rare instances, the incubation period may extend up to 7 years. Without timely and appropriate medical intervention, such as vaccination, the infection can progress to coma and eventually result in death (Demsis *et al.,* 2022).

Rabies causes approximately 60,000 human deaths worldwide each year (Bilal *et al.,* 2021). In low- and middle-income countries (LMICs) across Asia and Africa, effective control efforts are often challenged by the absence of timely and reliable data on rabies cases in both humans and animals. The true number of deaths resulting from rabies virus (RABV) infections in low- and middle-income countries is likely underreported, and the behavior and dynamics of rabid dog populations remain insufficiently understood (Tian *et al.,* 2018).

Research by Sambo *et al.,* (2013) found that the actual human rabies mortality rate in the United Republic of Tanzania was substantially higher than the figures officially reported. Based on an analysis of active surveillance data on bite incidents, the study estimated an annual rabies mortality rate of 1,499 deaths, with a 95% confidence interval ranging from 891 to 2,238 deaths. This corresponds to an annual death rate of 4.9 per 100,000 people, with a range of 2.9 to 7.2 per 100,000.

Mathematical modeling has played a crucial role in improving the understanding and control of infectious diseases like rabies. These analytical tools have been valuable in forecasting disease trends and guiding healthcare professionals in developing effective management strategies. A wide range of mathematical models has been formulated and examined to explore the transmission dynamics of rabies in human and canine populations (Hassan & Abdulmajid, 2021; Amaoko *et al.,* 2021; Fredrick *et al.,* 2022). These studies have highlighted key factors influencing the spread of the disease and have proposed various control strategies.

According to Bohrer *et al.,* (2002), in desert regions where host population sizes fluctuate over time, a non-uniform distribution of oral rabies vaccination can, under certain conditions, be more effective than the standard uniform approach. The effectiveness of such a targeted strategy partially depends on the movement patterns of the host species. Their findings also indicate that in warmer environments, rabies may persist in certain high-density areas that are surrounded by populations with densities below the critical threshold needed for sustained transmission.

Levin *et al.,* (2012) developed a model to investigate the immune response to rabies virus in bats. Coyne et al. (1989) introduced an SEIR framework, which was later applied to study the local transmission dynamics of rabies among raccoons in the United States. Similarly, Childs et al. (2000) examined rabies outbreaks in raccoon populations with seasonal birth pulses, employing an SEIRS model integrated with optimal control techniques to capture population dynamics. Hampson *et al.,* (2007) observed that rabies epidemics in African dog populations exhibit cyclical patterns every 3 to 6 years, leading them to formulate an SEIV (Susceptible-Exposed-Infectious-Vaccinated) model

incorporating an intervention variable that demonstrated strong synchrony in epidemic behavior.

Carroll *et al.,* (2010) employed compartmental models to examine rabies transmission in dog populations and assessed the effectiveness of three control strategies: routine vaccination, pulse vaccination combined with fertility control, and culling. Additionally, studies (Wangand & Lou, 2008; Yang & Lou, 2009) utilized ordinary differential equation models to describe the transmission dynamics of rabies between humans and dogs. Zinsstag *et al.,* (2009) advanced existing rabies transmission models by incorporating dog-to-human transmission, concluding that combining dog vaccination campaigns with human post-exposure prophylaxis (PEP) offers a more cost-effective long-term control strategy. In a related effort, Ding *et al.,* (2007) developed a spatially structured, discrete-time epidemic model for rabies in raccoons. Their study focused on identifying optimal vaccine bait distribution strategies aimed at minimizing both disease spread and control costs.

Asamoah *et al.*, (2017) proposed that vaccinating domestic animals and administering both pre-exposure and post-exposure prophylaxis are effective measures for controlling the spread of rabies. In regions such as China and parts of Africa including Ghana's Upper East and West dog meat is considered a delicacy. Notably, despite extensive studies on rabies transmission and control, the role of dog consumption by humans in the disease's spread remains largely unexplored. This study aims to investigate the combined impact of treatment and vaccination strategies within human and dog populations.

Motivated by the projections of the Global Alliance for Rabies Control ("Global alliance for rabies control," 2016) and the aforementioned studies, this work aims to formulate a deterministic vaccination rabies model in humans and dog's population. The structure of this paper is as follows: Section 2 presents the model formulation, including the underlying assumptions, the flow diagram, model equations, and their basic properties. Section 3 provides the analytical results, covering the equilibrium points, the basic reproduction number, R_D, and the stability analysis of the equilibria. In Section 4, we outline the parameter values used to compute the basic reproduction number, perform a sensitivity analysis using Latin Hypercube Sampling (LHS), and display related numerical simulations. Finally, Section 5 discusses the findings and concludes the study.

MATERIALS AND METHODS

Model Formulation

In this section, a mathematical model for the transmission dynamics of rabies in human and dog population is developed. The total human population at time t, denoted by $N_H(t)$, is divided into compartments of susceptible ($S_H(t)$), vaccinated ($V_H(t)$), infected ($I_H(t)$), treated ($T_H(t)$) and recovered ($R_H(t)$) humans, so that:

$N_H(t) = S_H(t) + V_H(t) + I_H(t) + T_H(t) + R_H(t)$

Similarly, the population of dogs at time t, denoted by $N_D(t)$, is subdivided into compartments of susceptible ($S_D(t)$), vaccinated ($V_D(t)$) and infected ($I_D(t)$) dogs, so that:

$N_D(t) = S_D(t) + V_D(t) + I_D(t)$

In the rabies model system (1), $\Pi_H(\Pi_D)$ is the recruitment of humans (dogs) into the population of susceptible humans (dogs), $\beta_{DD}(\beta_{DH})$ represents rabies transmission rate from dogs-dogs (dogs-humans). Natural death for humans (dogs) is given by the parameter $\mu_H(\mu_D)$ which is assumed to occur in all humans (dogs) populations. Susceptible humans (dogs) gets vaccinated (pre-exposure prophylaxis) at a rate $t_H(m_D)$, the parameters $K_H(K_D)$ represents vaccine waning rate in humans (dogs) population. Infected humans get treated at a rate σ_H and progress to treated class. ρ_H represents the rate at which treated humans recover from rabies infection. Recovered humans loss their immunity (and revert back to susceptible class) at a rate r_H. Disease induced mortality (dog culling) is assumed to occur in humans (dogs) population at a rate $c_H(c_D)$. The flow diagram of the rabies model (1) is depicted in Figure 1, and the state variables and model parameters of model (1) are described in Table 1 and Table 2, respectively. The model is formulated based on the following assumptions:

i. Human and Dog population mixed homogeneously: it is assumed that the population of humans and dogs mixed homogeneously, and that every member of the community (human or dog) has equal likelihood of mixing with every other member of the community.
ii. Human-human transmission was not considered (i.e., rabies virus is transmitted through contact with infected dogs only).
iii. Culling of infected dogs was considered as one of the control strategies in curtailing rabies infection.

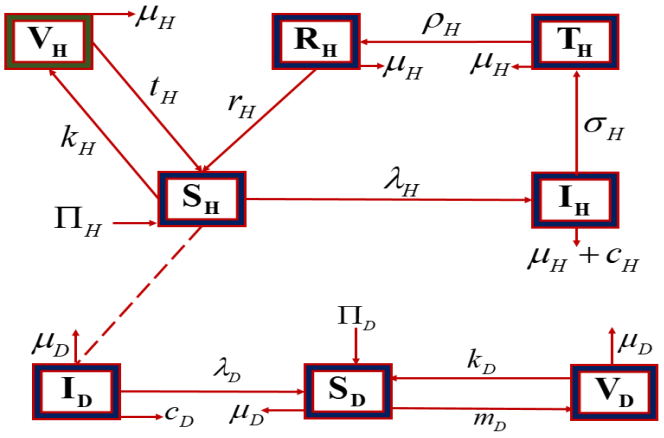

Figure 1: Schematic Diagram of the Model

The equations defining the dynamics of rabies virus in dogs – humans is given below:

$$\left.\begin{aligned}
\dot{S}_H &= \Pi_H - (\lambda_H + \mu_H + t_H)S_H + k_H V_H + r_H R_H \\
\dot{V}_H &= t_H S_H - (k_H + \mu_H)V_H \\
\dot{I}_H &= \lambda_H S_H - (\mu_H + \sigma_H + c_H)I_H \\
\dot{T}_H &= \sigma_H I_H - (\mu_H + \rho_H)T_H \\
\dot{R}_H &= \rho_H T_H - (r_H + \mu_H)R_H \\
\dot{S}_D &= \Pi_D - (\lambda_D + \mu_D + m_D)S_D + k_D V_D \\
\dot{V}_D &= m_D S_D - (k_D + \mu_D)V_D \\
\dot{I}_D &= \lambda_D S_D - (\mu_D + c_D)I_D
\end{aligned}\right\} \quad (1)$$

where $\lambda_H(\lambda_D)$ is the infection rate for humans (dogs) and are defined by:

$\lambda_H = \frac{\beta_{DH} I_D}{N_H}$ and $\lambda_D = \frac{\beta_{DD} I_D}{N_D}$

with $S_H(0) > 0, V_H(0) \geq 0, I_H(0) \geq 0, T_H(0) \geq 0, R_H(0) \geq 0, S_D(0) > 0, V_D(0) \geq 0, I_D(0) \geq 0$.

Table 1: State Variables of the Model

State Variable	Description
S_H	Susceptible human population
V_H	Vaccinated human population
I_H	Infected human population
T_H	Treated human population
R_H	Recovered human population
S_D	Susceptible dog population
V_D	Vaccinated dog population
I_D	Infected dog population

Table 2: Parameter Description of the Model

Parameter	Description
Π_H, Π_D	Human/Dog recruitment rate
μ_H, μ_D	Natural death rate of Humans/Dogs
β_{DH}, β_{DD}	Human/Dog transmission rate
c_H, c_D	Disease induced death in Humans/culling effects in Dogs
t_H, m_D	Vaccination rate in Humans/Dogs
k_H, k_D	Vaccine waning rate in Humans/Dogs
r_H	Loss of immunity rate in Humans
σ_H	Treatment rate in Humans
ρ_H	Humans recovery rate

Basic Properties of the Model

Since the model system (1) monitors human and dog populations, all its associated parameters are non-negative. Further, the following non-negativity result holds:

Theorem 2.2.1: The variables of the model system (1) are non-negative for all time $t > 0$. In other words, the solution of the model system (1) with positive initial data will remain positive for all time $t > 0$.

Proof: Let $t_1 = sup\{t > 0: S_H > 0, V_H \geq 0, I_H \geq 0, T_H \geq 0, R_H \geq 0, S_D > 0, V_D \geq 0, I_D \geq 0\}$. Thus, $t_1 > 0$. It follows from the first equation of model system (1) that:

$\dot{S}_H = \Pi_H - (\lambda_H + \mu_H + t_H)S_H(t) + r_H R_H(t) + k_H V_H(t) \geq \Pi_H - (\lambda_H + \mu_H + t_H)S_H(t)$

So that

$S_H(t_1) \geq S_H(0)\, exp\left[-(\mu_H + t_H)t_1 - \int_0^{t_1} \lambda_H(u)du\right] + \left\{exp\left[-(\mu_H + t_H)t_1 - \int_0^{t_1} \lambda_H(u)du\right]\right\} \int_0^{t_1} \Pi_H\, exp[(\mu_H + t_H)x + \int_0^x \lambda_H(u)du]\, dx > 0$

A similar approach can be used to show that $V_H(t) \geq 0, I_H(t) \geq 0, T_H(t) \geq 0, R_H(t) \geq 0, S_D(t) > 0,$ $V_D(t) \geq 0, I_D(t) \geq 0$ for all time $t > 0$. Hence, all non-negative initial solutions of model system (1) remain non-negative for all time t. We claim the following result.

Theorem 2.2.2: Consider the closed sets

$E_H = \{(S_H, V_H, I_H, T_H, R_H) \in \mathbb{R}_+^5\}$ and $E_D = \{(S_D, V_D, I_D) \in \mathbb{R}_+^3\}$

The region

$E = E_H \cup E_D$

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

Proof: Adding the first five and the last three equations of model (1), we have

$\frac{dN_H}{dt} = \Pi_H - \mu_H N_H - c_H I_H$ (1)

and

$\frac{dN_D}{dt} = \Pi_D - \mu_D N_D - c_D I_D$ (2)

Since all parameters of model (1) are non-negative, it follows from (1.1) and (1.2) that

$\frac{dN_H(t)}{dt} \leq \Pi_H - \mu_H N_H(t)$ (3)

and

$\frac{dN_D(t)}{dt} \leq \Pi_D - \mu_D N_D(t)$ (4)

Consequently, it follows from (2.3) and (2.4), using the comparison theorem as used by Hale (1969), that:

$N_H(t) \leq N_H(0)e^{-\mu_H(t)} + \frac{\Pi_H}{\mu_H}\left[1 - e^{-\mu_H(t)}\right]$

Similarly, from (2.4), it follows that:

$N_D(t) \leq N_D(0)e^{-\mu_D(t)} + \frac{\Pi_D}{\mu_D}\left[1 - e^{-\mu_D(t)}\right]$

In particular, $N_H(t) \leq \frac{\Pi_H}{\mu_H}$ if $N_H(0) \leq \frac{\Pi_H}{\mu_H}$ and $N_D(t) \leq \frac{\Pi_D}{\mu_D}$ if $N_D(0) \leq \frac{\Pi_D}{\mu_D}$, respectively. Thus, if $N_H(0) > \frac{\Pi_H}{\mu_H}$, then either the solution of the human component of the model enters E_H in finite time, or $\frac{N_H(t)\to\Pi_H}{\mu_H}$. Hence, the feasible region E_H is invariant and attracting. Similarly, it can be seen that if $N_D(0) > \frac{\Pi_D}{\mu_D}$, then either the solution of the dog component of the model enters E_D in finite time, or $N_D(t) \to \frac{\Pi_D}{\mu_D}$. Hence, E_D is also positively invariant and attracting. It follows that E is positively invariant and attracting with respect to the model.

Model Analysis

Existence and Stability of Rabies-Free Equilibrium

The model (1) has a unique rabies-free equilibrium (RFE) given by:

$\Gamma_0 = \left[S_H^0, V_H^0, I_H^0, T_H^0, R_H^0, S_D^0, V_d^0, I_D^0\right] = \left[S_H^0, V_H^0, 0,0,0, S_D^0, V_d^0, 0\right]$

where:

$$\left.\begin{aligned} S_H^0 &= \frac{\Pi_H(k_H+\mu_H)}{\mu_H(k_H+\mu_H+t_H)} \\ V_H^0 &= \frac{t_H\Pi_H}{\mu_H(k_H+\mu_H+t_H)} \\ S_D^0 &= \frac{\Pi_D(k_D+\mu_D)}{\mu_D(k_D+\mu_D+m_D)} \\ V_D^0 &= \frac{m_D\Pi_D}{\mu_D(k_D+\mu_D+m_D)} \end{aligned}\right\}$$

Using the next-generation matrix method, the rabies-free equilibrium of model (1) is locally asymptotically stable if the spectral radius of matrix FV^{-1} is less than one, where matrices F and V are given by:

$$F = \begin{bmatrix} 0 & \beta_{DH}\left(\frac{S_H^0}{N_H^0}\right) \\ 0 & \beta_{DD}\left(\frac{S_D^0}{N_D^0}\right) \end{bmatrix}, \text{ and } V = \begin{bmatrix} \mu_H + \sigma_H + c_H & 0 \\ 0 & \mu_D + c_D \end{bmatrix}$$

we define the reproduction number of the model as:

$R_D = \rho(FV^{-1}) = \frac{\beta_{DD}\Pi_D(k_D+\mu_D)}{\mu_D(\mu_D+c_D)(k_D+\mu_D+m_D)}$

The basic reproduction number R_D, of model (1), represents the average number of new cases (in humans or dogs) generated by a typical infectious dog if introduced in a completely susceptible population of both humans and dogs.

Theorem 3.1 *The rabies-free equilibrium of model (1) is locally asymptotically stable whenever* $R_D < 1$ and unstable if $R_D > 1$.

The epidemiological implication of Theorem 2.1 is that small influx of infected dogs in well-mixed dogs-human population (when $R_D < 1$) will not generate a significant rabies virus outbreak in the human-dog community if the initial number of infected humans or camels is small enough.

Endemic Equilibrium Point

The endemic equilibrium point denoted by Γ_1 which describes contact between infected dogs and humans is given as

$\Gamma_1 = \left[S_H^*, V_H^*, I_H^*, T_H^*, R_H^*, S_D^*, V_d^*, I_D^*\right]$

where:

$S_H^* = \frac{\Pi_H + r_H R_H^* + k_H V_H^*}{\beta_{DD} I_D^* + (\mu_H + t_H)}$,

$V_H^* = \frac{t_H S_H^*}{k_H+\mu_H}$, $I_H^* = \frac{\beta_{DD} I_D^* S_H^*}{\mu_H+\sigma_H+c_H}$,

$T_H^* = \frac{\sigma_H I_H^*}{\mu_H+\rho_H}$, $R_H^* = \frac{\rho_H T_H^*}{r_H+\mu_H}$,

$S_D^* = \frac{\mu_D + c_D}{\beta_{DD}}$

$V_D^* = \frac{m_D(\mu_D+c_D)}{\beta_{DD}(k_D+\mu_D)}$, $I_D^* = \frac{\mu_D(k_D+\mu_D+m_D)}{\beta_{DD}(\mu_D+k_D)}[R_D - 1]$

which exists whenever $R_D > 1$.

Global Stability of Rabies-Free Equilibrium Point

The global stability analysis of the rabies-free equilibrium point will be carried out using Lyapunov function as adapted in Musa *et al.,* (2024). We claim the following;

Theorem 3.2 The rabies-free equilibrium point, Γ_0 is globally asymptotically stable (GAS) whenever $R_D < 1$.

Proof: Consider the following Lyapunov function:

$K = g_1 I_D$

with Lyapunov derivative given by

$\dot{K} = g_1 \dot{I}_D = g_1[\beta_{DD} I_D S_D - (\mu_D + c_D) I_D]$

$= g_1\left[\frac{\beta_{DD}\Pi_D(k_D+\mu_D)}{\mu_D(k_D+m_D+\mu_D)} - (\mu_D + c_D)\right] I_D$

choose $g_1 = \mu_D$, so that

$\dot{K} = \mu_D\left[\frac{\beta_{DD}\Pi_D(k_D+\mu_D)}{\mu_D(k_D+m_D+\mu_D)} - (\mu_D + c_D)\right] I_D$

$= \mu_D(\mu_D + c_D)\left[\frac{\beta_{DD}\Pi_D(k_D+\mu_D)}{\mu_D(\mu_D+c_D)(k_D+m_D+\mu_D)} - 1\right] I_D$

$= \mu_D(\mu_D + c_D) I_D[R_D - 1] \leq 0 \quad \text{for} \quad R_D < 1$

since all the model parameters are assumed to be non-negative, it follows that $\dot{K} \leq 0$ if $R_D < 1$ with $\dot{K} = 0$ only if $I_D = 0$. Hence, $\dot{K}$ is a Lyapunov function on E. This result shows that rabies virus can be eliminated from the community within a certain period of time if the threshold $R_D < 1$.

Global Stability of Endemic Equilibrium Point

The endemic equilibrium point, Γ_1 is globally asymptotically stable (GAS) whenever $R_D > 1$.

Proof: Let $R_D > 1$ and consider a non-linear Lyapunov function of Goh-Volterrra type given by

$J = d_1\left[S_H - S_H^* - S_H^* ln\left(\frac{S_H}{S_H^*}\right)\right] + d_2\left[V_H - V_H^* - V_H^* ln\left(\frac{V_H}{V_H^*}\right)\right] + d_3\left[I_H - I_H^* - I_H^* ln\left(\frac{I_H}{I_H^*}\right)\right] + d_4\left[T_H - T_H^* - T_H^* ln\left(\frac{T_H}{T_H^*}\right)\right] + d_5\left[R_H - R_H^* - R_H^* ln\left(\frac{R_H}{R_H^*}\right)\right] + d_6\left[S_D - S_D^* - S_D^* ln\left(\frac{S_D}{S_D^*}\right)\right] + d_7\left[V_D - V_D^* - V_D^* ln\left(\frac{V_D}{V_D^*}\right)\right] + d_8\left[I_D - I_D^* - I_D^* ln\left(\frac{I_D}{I_D^*}\right)\right]$ (5)

taking the time derivative of J from (3.5), we have

$\dot{J} = d_1\left[1 - \frac{S_H^*}{S_H}\right]\dot{S}_H + d_2\left[1 - \frac{V_H^*}{V_H}\right]\dot{V}_H + d_3\left[1 - \frac{I_H^*}{I_H}\right]\dot{I}_H + d_4\left[1 - \frac{T_H^*}{T_H}\right]\dot{T}_H + d_5\left[1 - \frac{R_H^*}{R_H}\right]\dot{R}_H + d_6\left[1 - \frac{S_D^*}{S_D}\right]\dot{S}_D + d_7\left[1 - \frac{V_D^*}{V_D}\right]\dot{V}_D + d_8\left[1 - \frac{I_D^*}{I_D}\right]\dot{I}_D$ (6)

where $d_i (i = 1,2, \ldots, 8)$ are equal to 1. At steady state, the following relations holds:

$$\begin{aligned} &\Pi_H = \lambda_H^* S_H^* + (\mu_H + t_H)S_H^*, \ (\mu_H + k_H) = \frac{t_H S_H^*}{V_H^*}, \ (\mu_H + \sigma_H + c_H) = \frac{\lambda_H^* S_H^*}{I_H^*} \\ &(\mu_H + \rho_H) = \frac{\sigma_H I_H^*}{T_H^*}, \quad (r_H + \mu_H) = \frac{\rho_H T_H^*}{R_H^*}, \ \Pi_D = \lambda_D^* S_D^* + (\mu_D + m_D)S_D^* \\ &(k_D + \mu_D) = \frac{m_D S_D^*}{V_D^*}, \quad (\mu_D + c_D) = \frac{\lambda_D^* S_D^*}{I_D^*} \end{aligned} \quad (7)$$

Following the approach in Yang *et al.,* (2017), let the function $J = 1 - x + ln\, x$, then if $x > 0$, it leads to $J(x) \leq 0$. And if $x = 1$, then $J(x) = 0$ so that $x \geq 1 + ln\, x$ for any $x > 0$. Then, substituting the RHS of model equation (1) into (3.6), we have;

$$\dot{J} = \left[1 - \frac{S_H^*}{S_H}\right](\Pi_H - \lambda_H S_H - (\mu_H + t_H)S_H) + \left[1 - \frac{V_H^*}{V_H}\right](t_H S_H - (k_H + \mu_H)V_H)$$
$$+ \left[1 - \frac{I_H^*}{I_H}\right](\lambda_H S_H - (\mu_H + \sigma_H + c_H)I_H) + \left[1 - \frac{T_H^*}{T_H}\right](\sigma_H I_H - (\mu_H + \rho_H)T_H)$$
$$+ \left[1 - \frac{R_H^*}{R_H}\right](\rho_H T_H - (r_H + \mu_H)R_H) + \left[1 - \frac{S_D^*}{S_D}\right](\Pi_D - \lambda_D S_D - (\mu_D + m_D)S_D)$$
$$+ \left[1 - \frac{V_D^*}{V_D}\right](m_D S_D - (k_D + \mu_D)V_D) + \left[1 - \frac{I_D^*}{I_D}\right](\lambda_D S_D - (\mu_D + c_D)I_D) \quad (8)$$

Using the relation (3.7) in (3.8) gives that

$$\dot{J} = \left[1 - \frac{S_H^*}{S_H}\right](\lambda_H^* S_H^* + (\mu_H + t_H)S_h^* - \lambda_H S_H - (\mu_H + t_H)S_H)$$
$$+ \left[1 - \frac{V_H^*}{V_H}\right]\left(t_H S_H - \frac{t_H S_H^*}{V_H^*}V_H\right) + \left[1 - \frac{I_H^*}{I_H}\right]\left(\lambda_H S_H - \frac{\lambda_H^* S_H^*}{I_H^*}I_H\right)$$
$$+ \left[1 - \frac{T_H^*}{T_H}\right]\left(\sigma_H I_H - \frac{\sigma_H I_H^*}{T_H^*}T_H\right) + \left[1 - \frac{R_H^*}{R_H}\right]\left(\rho_H T_H - \frac{\rho_H T_H^*}{R_H^*}R_H\right)$$
$$+ \left[1 - \frac{S_D^*}{S_D}\right](\lambda_D^* S_D^* + (\mu_D + m_D)S_D^* - \lambda_D S_D - (\mu_D + m_D)S_D)$$
$$+ \left[1 - \frac{V_D^*}{V_D}\right]\left(m_D S_D - \frac{m_D S_D^*}{V_D^*}V_D\right) + \left[1 - \frac{I_D^*}{I_D}\right]\left(\lambda_D S_D - \frac{\lambda_D^* S_D^*}{I_D^*}I_D\right) \quad (9)$$

Simplifying, we obtain that;

$$\dot{J} \leq \lambda_H^* S_H^*\left[-ln\left(\frac{\lambda_H}{\lambda_H^*} \div \frac{S_H^*}{S_H}\right) - \frac{S_H^*}{S_H} + \frac{\lambda_H}{\lambda_H^*}\right] + t_H S_H^*\left[-ln\left(\frac{S_H}{S_H^*} \div \frac{V_H}{V_H^*}\right) + \frac{S_H}{S_H^*} - \frac{V_H}{V_H^*}\right]$$
$$+\lambda_H^* S_H^*\left[-ln\left(\frac{\lambda_H S_H}{\lambda_H^* S_H^*} \div \frac{I_H}{I_H^*}\right) + \frac{\lambda_H S_H}{\lambda_H^* S_H^*} - \frac{I_H}{I_H^*}\right] + \sigma_H I_H^*\left[-ln\left(\frac{I_H}{I_H^*} \div \frac{T_H^*}{T_H}\right) + \frac{I_H}{I_H^*} - \frac{T_H}{T_H^*}\right]$$
$$+\rho_H T_H^*\left[-ln\left(\frac{T_H}{T_H^*} \div \frac{R_H}{R_H^*}\right) + \frac{T_H}{T_H^*} - \frac{R_H}{R_H^*}\right] + \lambda_D^* S_D^*\left[-ln\left(\frac{\lambda_D}{\lambda_D^*} \div \frac{S_D^*}{S_D}\right) - \frac{S_D^*}{S_D} + \frac{\lambda_D}{\lambda_D^*}\right]$$
$$+m_D S_D^*\left[-ln\left(\frac{S_D}{S_D^*} \div \frac{V_D}{V_D^*}\right) + \frac{S_D}{S_D^*} - \frac{V_D}{V_D^*}\right] + \lambda_D^* S_D^*\left[-ln\left(\frac{\lambda_D S_D}{\lambda_D^* S_D^*} \div \frac{I_D}{I_D^*}\right) + \frac{\lambda_D S_D}{\lambda_D^* S_D^*} - \frac{I_D}{I_D^*}\right]$$

Hence,

$$\dot{J} \leq \lambda_H^* S_H^*\left[-ln\frac{\lambda_H}{\lambda_H^*} + \frac{\lambda_H}{\lambda_H^*} + ln\frac{S_H^*}{S_H} - \frac{S_H^*}{S_H}\right] + t_H S_H^*\left[-ln\frac{S_H}{S_H^*} + \frac{S_H}{S_H^*} + ln\frac{V_H}{V_H^*} - \frac{V_H}{V_H^*}\right]$$
$$+\lambda_H^* S_H^*\left[-ln\frac{\lambda_H S_H}{\lambda_H^* S_H^*} + \frac{\lambda_H S_H}{\lambda_H^* S_H^*} + \frac{I_H}{I_H^*} - \frac{I_H}{I_H^*}\right] + \sigma_H I_H^*\left[-ln\frac{I_H}{I_H^*} + \frac{I_H}{I_H^*} + ln\frac{T_H^*}{T_H} - \frac{T_H}{T_H^*}\right]$$
$$+\rho_H T_H^*\left[-ln\frac{T_H}{T_H^*} + \frac{T_H}{T_H^*} + ln\frac{R_H}{R_H^*} - \frac{R_H}{R_H^*}\right] + \lambda_D^* S_D^*\left[-ln\frac{\lambda_D}{\lambda_D^*} + \frac{\lambda_D}{\lambda_D^*} + ln\frac{S_D^*}{S_D} - \frac{S_D^*}{S_D}\right]$$
$$+m_D S_D^*\left[-ln\frac{S_D}{S_D^*} + \frac{S_D}{S_D^*} + ln\frac{V_D}{V_D^*} - \frac{V_D}{V_D^*}\right] + \lambda_D^* S_D^*\left[-ln\frac{\lambda_D S_D}{\lambda_D^* S_D^*} + \frac{\lambda_D S_D}{\lambda_D^* S_D^*} + ln\frac{I_D}{I_D^*} - \frac{I_D}{I_D^*}\right]$$

This shows that $\frac{dJ}{dt} \leq 0$ and $\frac{dJ}{dt} = 0$, if and only if $S_H = S_H^*$, $V_H = V_H^*, I_H = I_H^*, T_H = T_H^*$, $R_H = R_H^*, S_D = S_D^*, V_D = V_D^*, I_D = I_D^*$. Every solution of model (1) with the initial conditions approaches Γ_1 as $t \to \infty$; therefore, the largest compact invariant set in $\left\{(S_H, V_H, I_H, T_H, R_H, S_D, V_D, I_D) \in \text{E}: \frac{dJ}{dt} \leq 0\right\}$ is the singleton set $\{\Gamma_1\}$. Therefore, from Lassalle's invariant principle, it implies that the endemic equilibrium, Γ_1 is globally asymptotically stable in E whenever $R_D > 1$.

Numerical Simulation

In this section, we performed numerical simulation of model (1) to illustrate some of the theoretical results. Parameter values used in carrying out the simulation is given in Table 3.

Table 3: Parameter Values

Parameter	Value	Source
Π_H	$0.0314y^{-1}$	[3]
Π_D	$5 \times 10^6 y^{-1}$	Assumed
μ_H	$0.0074y^{-1}$	[3]
μ_D	$0.056y^{-1}$	[3]
β_{DH}	$2.29 \times 10^{-12}y^{-1}$	[3]
β_{DD}	$1.58 \times 10^{-5}y^{-1}$	Assumed
c_H	$1y^{-1}$	[3]
c_D	$0.042y^{-1}$	Assumed
t_H	$0.054y^{-1}$	[3]
m_D	$0.75y^{-1}$	Assumed
k_H	$0.46y^{-1}$	Assumed
k_D	$0.025y^{-1}$	Assumed
r_H	$1y^{-1}$	[3]
σ_H	$0.3y^{-1}$	Assumed
ρ_H	$0.06y^{-1}$	Assumed

Sensitivity Analysis

To identify the parameters that most significantly influence rabies transmission, we employed two sensitivity analysis techniques: the normalized forward sensitivity index as described by Martcheva (2015), and Latin Hypercube Sampling (LHS) following the method of Zhang *et al.,* (2015). Using a sample size of $n = 1000$, we assessed the independence and influence of parameters in R_D through Partial Rank Correlation Coefficients (PRCC), with the results for six key parameters displayed in Figure 2. In Figure 2, longer bars indicate a stronger statistical influence of the corresponding parameters on changes in R_D. Additionally, the normalized forward sensitivity indices, calculated using the parameter values listed in Table 2, are presented in Table 3 along with the direction of their influence. A positive sign indicates a direct (positive) relationship with R_D, while a negative sign reflects an inverse (negative) relationship.

$\Upsilon_{R_D}^{\beta_{DD}} = \frac{\partial R_D}{\partial \beta_{DD}} \frac{\beta_{DD}}{R_D} = 1,$ $\Upsilon_{R_D}^{\Pi_D} = \frac{\partial R_D}{\partial \Pi_D} \frac{\Pi_D}{R_D} = 1,$ $\Upsilon_{R_D}^{c_D} = \frac{\partial R_D}{\partial c_D} \frac{c_D}{R_D} = -\frac{c_D}{(\mu_D + c_D)} = -0.47727,$

$\Upsilon_{R_D}^{k_D} = \frac{\partial R_D}{\partial k_D} \frac{k_D}{R_D} = \frac{k_D m_D}{(\mu_D + k_D)(\mu_D + m_D + k_D)} = 0.04842,$ $\Upsilon_{R_D}^{m_D} = \frac{\partial R_D}{\partial m_D} \frac{m_D}{R_D} = -\frac{m_D}{(\mu_D + m_D + k_D)} = -0.93926,$

$$\Upsilon_{R_D}^{\mu_D} = \frac{\partial R_D}{\partial \mu_D} \frac{\mu_D}{R_D} = \frac{-2\mu_D^3 - \mu_D^2(c_D + 4k_D + m_D) - 2k_D\mu_D(c_D + k_D + m_D) - c_D k_D(k_D + m_D)}{(k_D + \mu_D)(\mu_D + c_D)(\mu_D + m_D + c_D)}$$
$$= -0.63188$$

Therefore, from Table 2 it shows that an addition or a reduction in the values of β_{DD}, Π_D and k_D will have an increase or decrease in the spread of the rabies virus. For example, $\Upsilon_{R_D}^{\beta_{DD}} = 1$ indicates that increasing or reducing the transmission rate by a certain percentage may increase or reduce the number of secondary infection by that percentage.

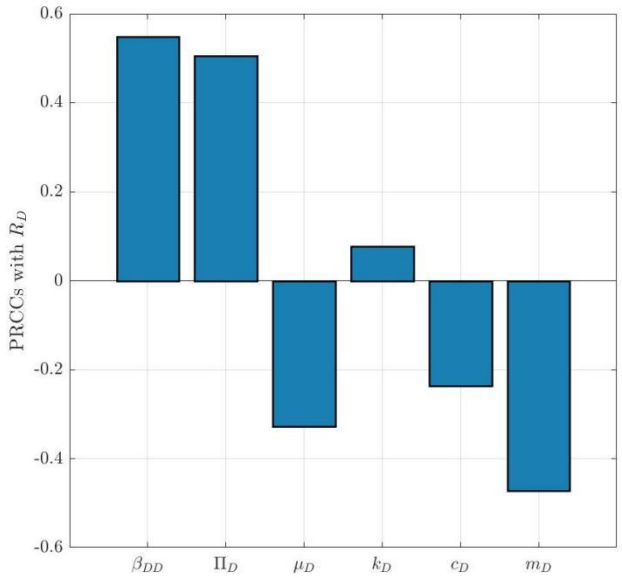

Figure 2(a): PRCCs plot for the parameters in R_D

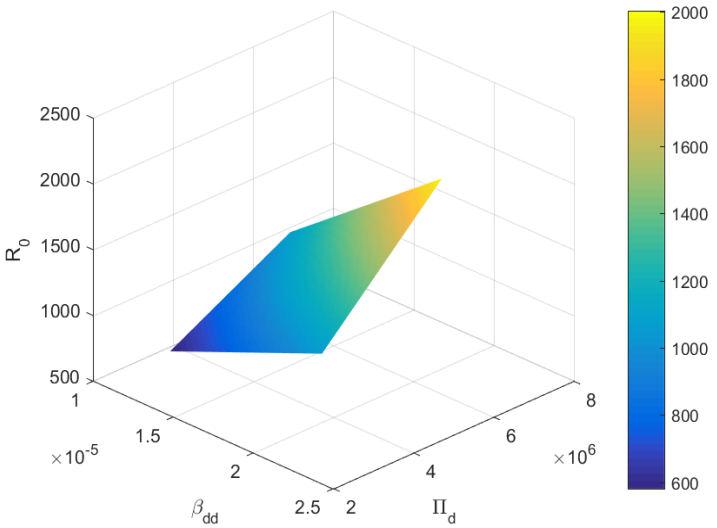

Figure 2(b): 3D plot of R_D to β_{DD} and Π_D

A negative sign in Table 2 indicates that an increase in the corresponding parameter will lead to a decrease in the basic reproduction number., R_D, An increase in the values of those parameters leads to a decrease in the basic reproduction number. Conversely, a reduction in the values of β_{DD}, Π_D and $k_D$may result in a rise in the number of secondary infections. The PRCC results indicate that μ_D and k_D exert minimal influence on the transmission rate of rabies. In contrast, the figure highlights β_{DD} as the most impactful parameter driving the spread of infection, followed by Π_D. Therefore, an increase in Π_D and β_{DD} leads to a direct rise in the spread of the rabies virus. The figure further illustrates that culling infected dogs has a limited effect in curbing transmission compared to vaccinating susceptible dogs. This suggests that vaccination of susceptible dogs is the most effective strategy for controlling rabies within the dog population. Figure 2(b) illustrates that Π_D and β_{DD} are positively correlated with the basic reproduction number, indicating that increases in these parameters enhance rabies transmission. Figure 3(a) demonstrates that increasing the human vaccination rate effectively boosts the vaccinated human population. Consequently, as shown in Figure 3(b), this leads to a reduction in the susceptible human population. Similarly, Figures 3(c) and 3(d) highlight the impact of dog vaccination—an increase in vaccination results in a larger vaccinated dog population while significantly decreasing the number of susceptible dogs. Additionally, Figure 3(e) illustrates the impact of dog vaccination on the infected dog population, clearly showing that increased vaccination efforts lead to a significant decline in the number of infected dogs over time. Therefore, dog vaccination m_D emerges as the most effective strategy for controlling the rabies virus, surpassing both human vaccination and treatment in effectiveness.

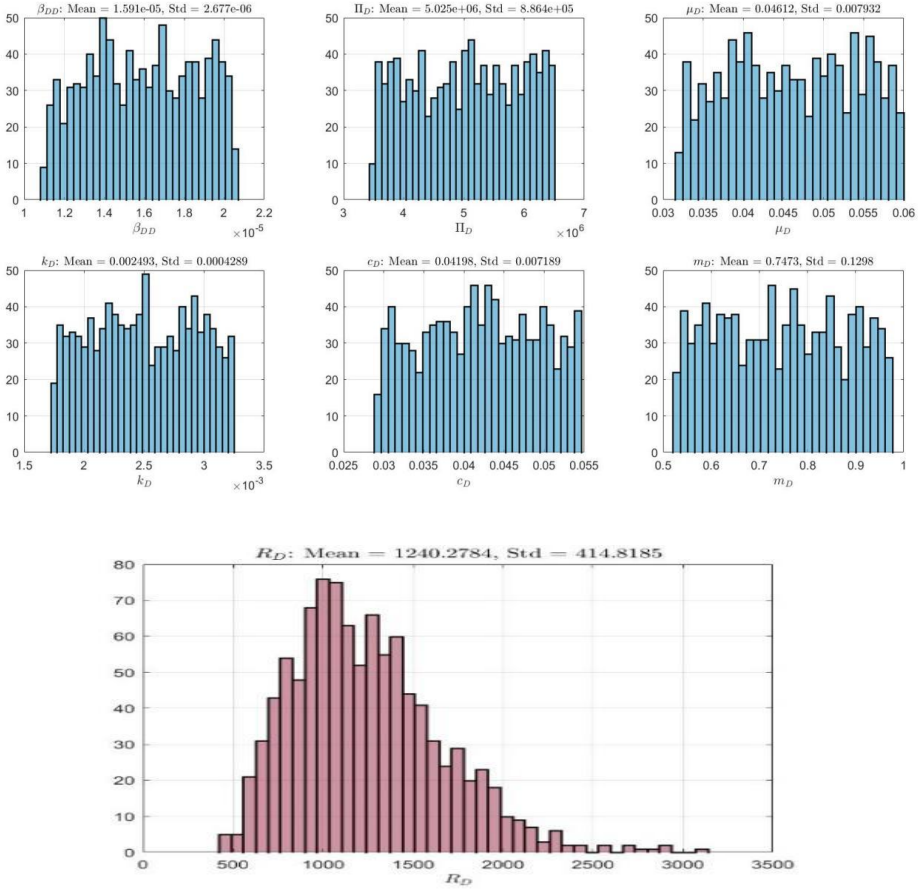

Figure 2(c): Distribution of parameters of the model (2.1) and the response function (R_D) generated from the uncertainty analysis. parameter values used are given by the baseline values and ranges in Table 2

Table 4: PRCC values of the parameters (6 of 15) of the model (2.1), with R_D chosen as the response function

Parameter	Baseline value	Range	PRCC
Π_D	5×10^6	$3.5 \times 10^6 - 6.5 \times 10^6$	0.52788
μ_D	0.046	$0.0322 - 0.0598$	-0.30059
β_{DD}	1.58×10^{-5}	$1.106 \times 10^{-5} - 2.054 \times 10^{-5}$	0.52887
c_D	0.042	$0.0294 - 0.0546$	-0.25896
k_D	0.0025	$0.00175 - 0.00325$	0.02614
m_D	0.75	$0.525 - 0.975$	-0.48241

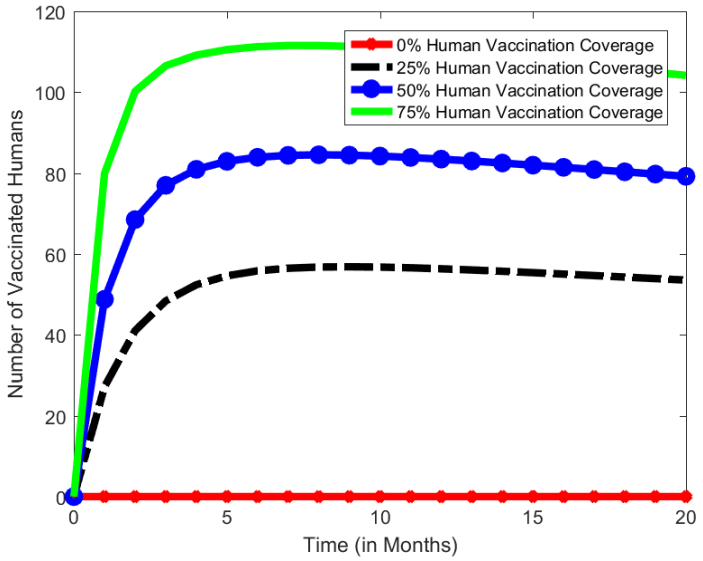

Figure 3(a): Effect of Human Vaccination on Vaccinated Humans Population

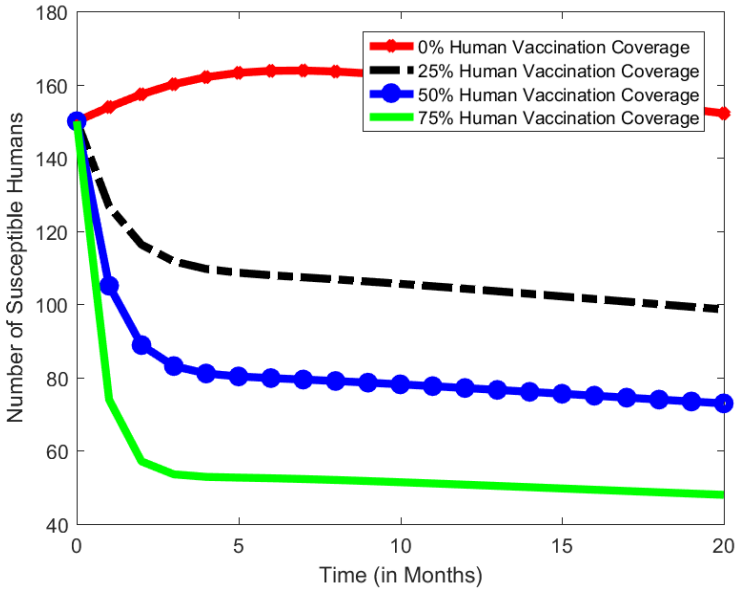

Figure 3(b): Effect of Human Vaccination on Susceptible Humans Population

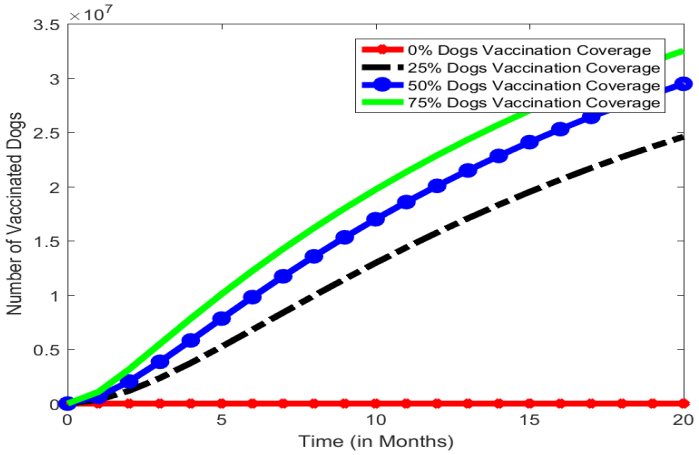

Figure 3(c): Effect of Dog Vaccination on Vaccinated Dogs Population

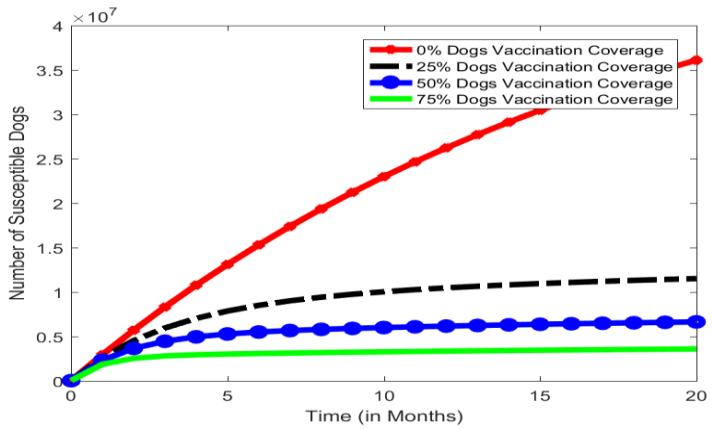

Figure 3(d): Effect of Dog Vaccination on Susceptible Dogs Population

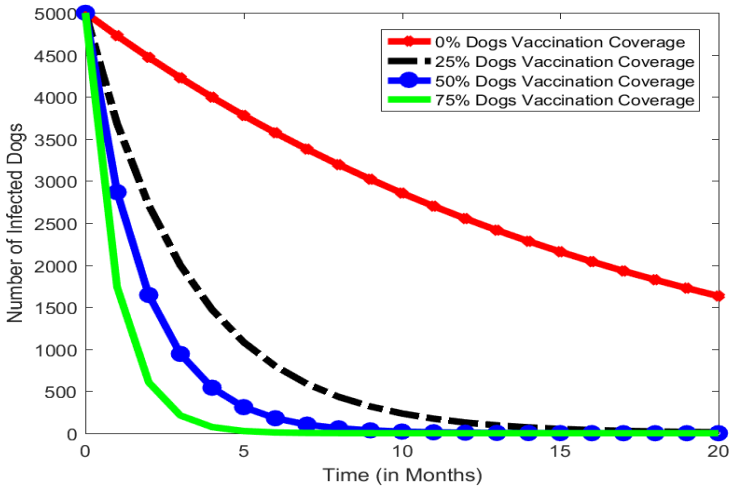

Figure 3(e): Effect of Dog Vaccination on Infected Dogs Population

CONCLUSION

This study proposed a novel mathematical model to describe the transmission dynamics of the rabies virus. The model, formulated as a deterministic system of nonlinear differential equations, was thoroughly analyzed to explore its qualitative behavior. In particular, the analysis addressed the boundedness and invariance of solutions, as well as the local asymptotic stability of the rabies-free equilibrium. It was established that the rabies-free equilibrium is locally asymptotically stable when the basic reproduction number, R_D, is less than one. Furthermore, a global sensitivity analysis employing Latin Hypercube Sampling (LHS) and Partial Rank Correlation Coefficients (PRCC) was conducted to identify the parameters that most significantly influence the response function, R_D. The two most influential parameters identified through PRCC analysis were: (a) the transmission rate from infected dogs to susceptible dogs (β_{D_D}), and (b) the recruitment rate of dogs (Π_D). The magnitude and negative signs of their PRCC values, as shown in Table 2, suggest that reducing these parameters particularly through widespread dog vaccination can effectively lower the basic reproduction number (R_D) and consequently reduce the rabies burden. Model simulations further demonstrated that vaccination efforts, both in humans and dogs, play a critical role in curbing the rise in susceptible humans, susceptible dogs, and infected dog populations. In Figures 3(a) to 3(d), vaccination coverage is applied across four compartments: vaccinated humans, susceptible humans, vaccinated dogs, and susceptible dogs, respectively. Figure 3(e) illustrates the impact of dog vaccination on the infected dog population, showing a marked decline over time. These results underscore the importance of prioritizing rabies control measures within the dog population, particularly through moderate to high vaccination coverage, rather than focusing solely on human intervention. In conclusion, the study recommends that control efforts and resources be directed more toward managing the disease in dogs, where they are likely to be more effective.

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