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Modeling and Analysis of the Dynamics of Rabies using Nonstandard Finite Difference Approach with Optimal Control

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Abstract: Rabies is a viral disease caused by the neurotropic virus belonging to the Lyssavirus genus. It is a fatal disease affecting primarily the central nervous system of both humans and animals, resulting in inflammation of the brain and eventual death. In this paper, we formulated a deterministic model for rabies to better understand the dynamics of transmission and control strategies in the Ashanti Region of Ghana. Employing the nonstandard finite difference scheme, we establish key stability properties of the disease-free equilibrium $(\mathbb{E}_0)$ with epidemiological interpretations: Local asymptotic stability implies that if a small number of cases arise, the disease will eventually fade out when the basic reproduction number is kept below 1. Global asymptotic stability demonstrates that rabies can be eliminated even if it starts at high prevalence levels. The asymptotic nature of stability confirms that disease elimination requires continuous intervention implementation over extended periods. The rabies model was expanded to incorporate an optimal control strategy using treatment of exposed dogs, policy and education on good petting, and effective education and campaigns on rabies. By optimizing our objective function, our optimal control analysis demonstrates that integrating effective public education and awareness campaigns, with the treatment of exposed dogs offers a highly effective strategy with strong long-term potential to reduce rabies transmission in the Ashanti Region of Ghana.

Keywords: Rabies, Nonstandard finite difference, Optimal control, Global stability, Optimizing

1 Introduction

Rabies is among the earliest documented diseases, with instances recorded as far back as 4000 years ago. Throughout the majority of human history, a bite from a rabid animal was uniformly fatal. Many would commit suicide after being bitten by a potentially rabid animal in the past due to the extreme fear of rabies [1]. It is a viral disease brought on by the neurotropic rabies virus, which is a member of the Lyssavirus genus. The two components of the rabies virus belong to the Lyssavirus genus within the Rhabdoviridae virus family. The most typical way for the virus to spread is through the bite of an infected mammal, both domestic and wild. However, saliva can also transfer the virus through cuts in the skin or mucous membranes [2].

Rabies symptoms typically appear in stages after an incubation period, which can last anywhere from a few days to several months, depending on the location of the bite, viral load, and other factors. During this time, the host experiences initial symptoms like fever, hallucinations, paralysis, and eventually death [3]. It is known as a fatal disease once the symptoms appear and affects primarily the central nervous system of both humans and animals, leading to brain inflammation and eventual death.

According to research, rabies causes between 30,000 and 70,000 deaths annually, with less developed nations being more severely affected. There aren't many documented human cases in the US, yet it could be because post-exposure prophylaxis is so often used, and there are preventative initiatives in place. Only around

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10% of rabies cases in affluent nations have been transmitted by domesticated animals [4,5]. The primary focus of treatment is prevention, which includes education, monitoring, and vaccination programs for domestic animals [6,7,8].

By comparing many potential techniques, mathematical modeling aims to provide insight into the cause of a disease, forecast its progression, and develop some of the most effective strategies for controlling the infectious disease [9,10]. Deterministic models increase the general knowledge of the disease spread by offering a theoretical frame that underlines elements that account for the spread and control of diseases [11,12]. Optimal control of deterministic models have shown to be the best combating strategies, as they determine the best optimal control mechanism and the most effective cost minimization of infections diseases [13,14].

To understand the dynamics of rabies virus transmission in dogs and predict the best control measures, [15] proposed two mathematical models for her study. A susceptible, exposed and infected (SEI) in the absence of vaccination. And a susceptible, expose, infected and recovered compartmental model in the presence of vaccines. Their finding shows the disease can be controlled by increasing and expanding dog vaccination to a higher percentage and reducing annual new birth of puppies simultaneously. The model of [16] was formulated to deduce the effects of rabies and its control in three populations, jackal, dog and human beings. Their proposed strategies for mitigating the disease was human vaccination, dog vaccination, culling of dogs, dog sterilization and vaccination of jackals. Their results shows the simultaneous implementation of dog culling and dog vaccination is the most effective way of suppressing the spread of the disease in Nepal.

[17] develop an SIR model to identified public education on administration of both pre and post exposure prophylaxis of rabies vaccine and practicing of responsible dog ownership as the best way of controlling rabies in Kenya. They argue that proper implementation of public education can eliminate rabies in Kenya by 2030. [18] performed optimal control analysis by considering vaccination, controlling of annual birth rate of dogs and culling of dogs as their control measures. MATLAB ode45 was used to investigate the numerical simulations that were carried out. And their results shows increasing dog vaccination and culling of dogs has the most significant impact on the spread of the disease. The study of [19] confirmed the robustness of the Non Standard Finite Difference Scheme. [20] performed optimal control analysis on an SERIV model to describe the transmission dynamics of rabies in raccoons with birth pulse. The model accounts for loss of vaccines due to other factors other than raccoons eating the vaccine baits. It was observed from the simulation that the closer the detection of the infection is to the birth pulse, the longer the period of vaccine distribution should take.

Despite the extensive literature on mathematical modeling of rabies, deterministic models that incorporate wound treatment of exposed persons' as a way of minimizing the risk of contracting rabies remain scarce. This study aims at formulating a mathematical model using nonstandard finite difference approach to analyze the transmission dynamics of rabies and describe possible ways to reduce the spread of the virus.

2 Model formulation and description

Graph 1 and 2 show the incidence of rabies in the Ashanti Region of Ghana based on observed data from the Veterinary office, Kumasi.

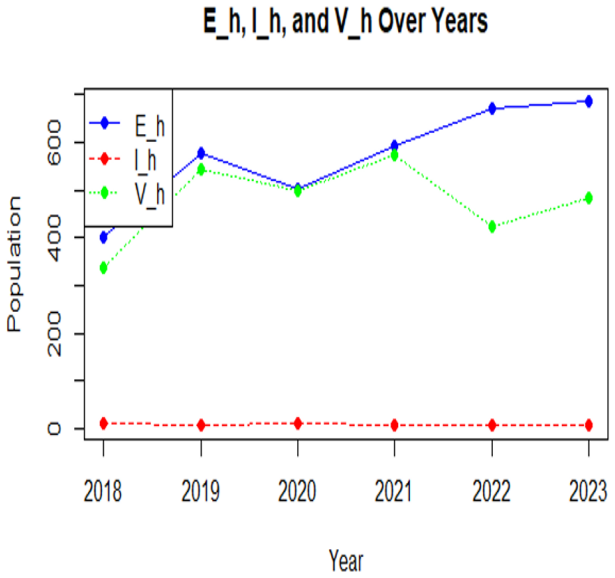

Fig. 1: Incidence of rabies among humans in Ashanti Region from 2018-2023. E_h: exposed humans, I_h: infectious humans, V_h: vaccinated humans. (Source: Veterinary Office, Kumasi)

Figure 3 shows the mode of transmissions of rabies disease among human beings and canines respectively in the Ashanti Region. This Figure 3, is important as it enlightens us about the rabies transmission dynamics in animals and humans. We split our model in half, considering both the total human population $N_h(t)$ and the total animal population $N_d(t)$. At any time, $N_h(t)$ is divided into six compartments and $N_d(t)$ into four compartments. Hence, the models consist of a total of ten compartments that represent the populations at any time t. These are, the compartments forming the human populations; susceptible $S_h(t)$, Exposed $E_h(t)$, Treated $T_h(t)$, Infected $I_h(t)$, Recovered $R_h(t)$, Vaccinated $V_h(t)$, and Susceptible S_d, Exposed E_d, Infected I_d, and

Vaccinated V_d , the compartments forming the dog populations.

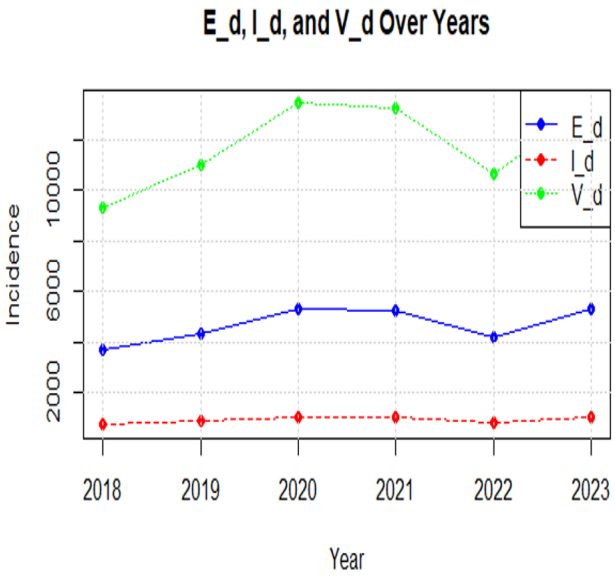

Fig. 2: Incidence of rabies among dogs in Ashanti Region from 2018-2023. E_d: exposed dogs, I_d: infectious dogs, V_d: vaccinated dogs. (Source: Veterinary Office, Kumasi)

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Susceptible individuals are recruited by Λ and the rate of human recovery from vaccination is κ . Rate of natural mortality in human population is μ, with rabies induced death rate in I_h compartment δ. The exposed individuals treat their wounds at a rate τ whiles the treated persons receive vaccination at Σ rate. The exposed individuals become infected at a rate ν, and get vaccinated at η. The rate of interaction between susceptible and exposed humans is β. The treated individuals become infected at ρ, and recovery of treated individuals from their injuries is ψ. Susceptible dogs are recruited by A, mass vaccination at a rate n and the rate of their interaction with exposed dogs is B. Dogs die at a natural death rate of m, with rabies induced death rate in infected dogs f. Exposed dogs become infected at a rate r and receive post-exposure prophylaxis at q. Hence the total population $N(t) = N_h(t) + N_d(t)$, where

$$N_h(t) = S_h(t) + E_h(t) + T_h(t) + I_h(t) + R_h(t) + V_h(t) \quad (1)$$

and

$$N_d(t) = S_d(t) + E_d(t) + I_d(t) + V_d(t). \quad (2)$$

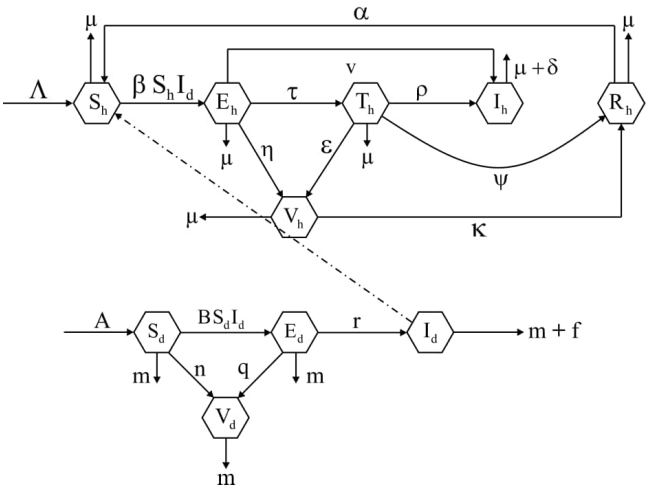

Fig. 3: Schematic diagram of the formulated rabies transmissions dynamics of rabies disease among human beings and canines

2.0.1 Model assumptions

- –The model assume, dogs and human beings interact homogeneously because most dogs are free roaming dogs and most people in the region use the streets and other open spaces.
- –Both dogs and humans are recruited by birth.
- –Natural death rate is constant for dogs in all compartments and humans in all compartments.
- –All infected humans and dogs die without recovery, but vaccinated humans do recover from adverse effects of the vaccine.
- –Age, sex, breed and weather conditions do not affect the disease spread.

The system of model equations from Figure 3 are given below:

$$\begin{cases} S_h'(t) = \lambda + (\alpha)R_h - \beta S_h I_d - \mu S_h \\ E_h'(t) = \beta S_h I_d - (\nu + \eta + \tau + \mu)E_h \\ T_h'(t) = (\tau)E_h - (\Sigma + \rho + \psi + \mu)T_h \\ I_h'(t) = (\rho)T_h + (\nu)E_h - (\mu + \delta)I_h \\ R_h'(t) = (\psi)T_h + (\kappa)V_h - (\alpha + \mu)R_h \\ V_h'(t) = (\eta)E_h + (\Sigma)T_h - (\kappa + \mu)V_h \\ S_d'(t) = A - (BI_d + n + m)S_d \\ E_d'(t) = BS_d I_d - (r + q + m)E_d \\ I_d'(t) = (r)E_d - (m + f)I_d \\ V_d'(t) = (n)S_d + (q)E_d - (m)V_d \end{cases} \quad (3)$$

3 Model analysis

3.1 Existence and uniqueness of solutions

To ensure the validity of our rabies transmission model, we establish the existence and uniqueness of solutions to

the system of differential equations. Let the system (3) be written in the compact form

$$\frac{d\mathbf{X}}{dt} = \mathbf{F}(t,\mathbf{X}(t)), \quad \mathbf{X}(0) = \mathbf{X}_0, \tag{4}$$

where $\mathbf{X}(t) = (S_h, E_h, T_h, I_h, R_h, V_h, S_d, E_d, I_d, V_d)^T \in \mathbb{R}^{10}$, and $\mathbf{F} : [0,T] \times \mathbb{R}^{10} \to \mathbb{R}^{10}$ is the vector field determined by the right-hand side of the model equations (3).

We consider the Banach space $C([0,T],\mathbb{R}^{10})$ of continuous functions from $[0,T]$ to $\mathbb{R}^{10}$ equipped with the supremum norm:

$$\|\mathbf{X}\| = \sup_{t\in[0,T]} \|\mathbf{X}(t)\|_\infty, \text{where } \|\mathbf{X}(t)\|_\infty = \max_{1\le i\le 10} |X_i(t)|. \tag{5}$$

The function $\mathbf{F}(t,\mathbf{X})$ is composed of continuously differentiable (polynomial or bilinear) terms, which implies that it is locally Lipschitz in $\mathbf{X}$. Therefore, for any closed and bounded domain $D \subset \mathbb{R}^{10}$, there exists a constant $L > 0$ such that:

$$\|\mathbf{F}(t,\mathbf{X}_1) - \mathbf{F}(t,\mathbf{X}_2)\|_\infty \le L\|\mathbf{X}_1 - \mathbf{X}_2\|_\infty, \ \forall\, \mathbf{X}_1, \mathbf{X}_2 \in D, \tag{6}$$

and $\mathbf{F}(t,\mathbf{X})$ is Lipschitz continuous in $\mathbf{X}$. We want to show that by applying the Banach fixed point theorem, there exists a unique fixed point $\mathbf{X}(t) \in C([0,T],\mathbb{R}^{10})$, which is the unique solution to system (3). Let define the operator $\mathscr{T}$ on $C([0,T],\mathbb{R}^{10})$ by:

$$(\mathscr{T}\mathbf{X})(t) = \mathbf{X}_0 + \int_0^t \mathbf{F}(s,\mathbf{X}(s))\,ds. \tag{7}$$

Then for any $\mathbf{X}, \mathbf{Y} \in C([0,T],\mathbb{R}^{10})$, we have:

$$\begin{aligned}
&\|(\mathscr{T}\mathbf{X})(t) - (\mathscr{T}\mathbf{Y})(t)\|_\infty \\
&= \left\| \int_0^t [\mathbf{F}(s,\mathbf{X}(s)) - \mathbf{F}(s,\mathbf{Y}(s))]\,ds \right\|_\infty \\
&\le \int_0^t \|\mathbf{F}(s,\mathbf{X}(s)) - \mathbf{F}(s,\mathbf{Y}(s))\|_\infty\, ds \\
&\le L\int_0^t \|\mathbf{X}(s) - \mathbf{Y}(s)\|_\infty\, ds \\
&\le LT\|\mathbf{X} - \mathbf{Y}\|.
\end{aligned}$$

Thus, $\|\mathscr{T}\mathbf{X} - \mathscr{T}\mathbf{Y}\| \le LT\|\mathbf{X} - \mathbf{Y}\|$. Choosing $T > 0$ sufficiently small such that $LT < 1$, the operator $\mathscr{T}$ becomes a contraction on the closed ball in $C([0,T],\mathbb{R}^{10})$. Hence, by the Banach Fixed Point Theorem, $\mathscr{T}$ admits a unique fixed point in $C([0,T],\mathbb{R}^{10})$, which corresponds to the unique solution of the system on $[0,T]$.

Lemma 1. *Existence of solution*
Let $\mathbf{F}(t,\mathbf{X})$ be Lipschitz continuous in $\mathbf{X}$ as defined in (6) such that $\mathbf{X}(t) \in C([0,T],\mathbb{R}^{10})$ is a unique solution. Then a solution exists to the system (3) bounded in the domain D.

Proof. Given $\mathbf{F}(t,\mathbf{X})$ the right hand side of the model equation (3). We show that $\frac{\partial \mathbf{F}_i(t,\mathbf{X})}{\partial \mathbf{X}_j}\ \forall\, i,j = 1,2,\cdots 10$ is continuous and bounded. From system (3),

$$\begin{cases}
\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial S_h} = -(\beta I_d + \mu) \text{ and } \left|\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial S_h}\right| = |\beta I_d + \mu| < \infty, \\
\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial I_d} = -(\beta S_h) \text{ and } \left|\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial I_d}\right| = |\beta S_h| < \infty, \\
\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial R_h} = \alpha \text{ and } \left|\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial R_h}\right| = |\alpha| < \infty, \\
\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial E_h} = \frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial T_h} = \frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial V_h} = \frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial S_d} = \frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial E_d} = \\
\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial I_d} = \frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial V_d} = 0, \text{ and} \\
\left|\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial E_h}\right| = \left|\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial T_h}\right| = \left|\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial V_h}\right| = \left|\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial S_d}\right| = \\
\left|\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial E_d}\right| = \left|\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial I_d}\right| = \left|\frac{\partial \mathbf{F}_1(t,\mathbf{X})}{\partial V_d}\right| < \infty.
\end{cases}$$

Similar approach shows $\forall\, i,j = 1,2,\cdots 10$, $\left|\frac{\partial \mathbf{F}_i(t,\mathbf{X})}{\partial \mathbf{X}_j}\right| < \infty$. Thus following [21], we have verified that $\forall\ i,j = 1,2,\cdots 10$, $\frac{\partial \mathbf{F}_i(t,\mathbf{X})}{\partial \mathbf{X}_j}$ is bounded and continuous. Hence, according to the Banach fixed point theorem, there exists a unique solution of system (3) in the domain D.

Lemma 2. *Boundedness of solutions*
If Lemma (1) holds, where

$$\Big(S_h(0), E_h(0), T_h(0), I_h(0), R_h(0), V_h(0), S_d(0), E_d(0), I_d(0), V_d(0)\Big)^T$$

is the initial conditions to system (3), then it's solution remains bounded $\forall\, t > 0$.

Proof. Following [22], $\frac{dN_h}{dt} = \lambda - \mu N_h - \delta I_h$. And $\frac{dN_h}{dt} \le \lambda - \mu N_h$ in the absence of disease-induced death. Applying the method of integrating factors,

$$\begin{aligned}
\frac{d}{dt}\left(N_h e^{\mu t}\right) &\le \lambda e^{\mu t} \\
\int d\left(N_h e^{\mu t}\right) &\le \int \lambda e^{\mu t} dt \\
N_h &\le \frac{\lambda}{\mu}(1 - e^{-\mu t}) \\
N_h &\le \frac{\lambda}{\mu}, \text{ as } t \to \infty.
\end{aligned}$$

Thus,

$$\mathbf{X}_h = \left\{(S_h, E_h, T_h, I_h, R_h, V_h) \in \mathbb{R}^6_+ : S_h + E_h + T_h + I_h + R_h + V_h \le \frac{\lambda}{\mu}\right\} \tag{8}$$

A similar approach on the animal compartment shows $N_d \le \frac{A}{m}$, as $t \to \infty$ for

$$\mathbf{X}_d = \left\{(S_d, E_d, I_d, V_d) \in \mathbb{R}^4_+ : S_d + E_d + I_d + V_d \le \frac{A}{m}\right\} \tag{9}$$

Hence the solution set of the system (3) with initial conditions remains in $\mathbf{X}$, where $\mathbf{X} = \left\{(\mathbf{X}_h, \mathbf{X}_d) \in \mathbb{R}^6_+ \times \mathbb{R}^4_+\right\}, \forall\, t > 0$.

Lemma 3. *Non-negativity of solutions*
The solution of the model equation (3) is in $\mathbf{X}$*, if its initial conditions are in* $\mathbf{X} \in \mathbb{R}^{10}_{+}$.

Proof. The solution of equation (3) is an expression of the form;

$$\mathbf{X}(t) = \left\{ \begin{array}{l} S_h(t), E_h(t), T_h(t), I_h(t), R_h(t), \\ V_h(t), S_d(t), E_d(t), I_d(t), V_d(t) \end{array} \right\} \subset \mathbb{R}^{10}_{+} \quad (10)$$

Let $\mathbb{H} : \mathbf{X} \in \mathbb{R}^{10}_{+} \to \mathbb{H}(\mathbf{X}) \in \mathbb{R}^{10}_{+}$, where

$$\mathbb{H}(\mathbf{X}) = \left\{ \begin{array}{l} S_h'(t) = \lambda + (\alpha)R_h - \beta S_h I_d - \mu S_h \\ E_h'(t) = \beta S_h I_d - (\nu + \eta + \tau + \mu)E_h \\ T_h'(t) = (\tau)E_h - (\Sigma + \rho + \psi + \mu)T_h \\ I_h'(t) = (\rho)T_h + (\nu)E_h - (\mu + \delta)I_h \\ R_h'(t) = (\psi)T_h + (\kappa)V_h - (\alpha + \mu)R_h \\ V_h'(t) = (\eta)E_h + (\Sigma)T_h - (\kappa + \mu)V_h \\ S_d'(t) = A - (BI_d + n + m)S_d \\ E_d'(t) = BS_d I_d - (r + q + m)E_d \\ I_d'(t) = (r)E_d - (m + f)I_d \\ V_d'(t) = (n)S_d + (q)E_d - (m)V_d \end{array} \right\}.$$

From the model equation (3),

$$S_h'(t) = \lambda + (\alpha)R_h - \beta S_h I_d - \mu S_h$$

$$S_h'(t) \geq -(\beta I_d + \mu)S_h$$

Integrating both sides of the inequality gives;
$\ln S_h \geq -(\beta I_d + \mu)t + \kappa$

$$S_h \geq \mathbb{K} e^{-(\beta I_d + \mu)t}$$

At $t = 0$, $\mathbb{K} = S_h(0)$ and $S_h \geq S_h(0)\mathbb{K}e^{-(\beta I_d + \mu)t}$.

Thus $S_h(t) \geq 0$. Applying a similar approach to the remaining compartments can be used to establish their positivity. Therefore, for $\mathbb{H}(\mathbf{X}) \in \mathbb{R}^{10}_{+}$, $\frac{d\mathbf{X}}{dt} = \mathbb{H}(\mathbf{X})$, where $\mathbf{X}(0) \geq 0$. Hence, there exists a non-negative solution of the system (3) whenever its initial conditions are non-negative following the existence and uniqueness theorem established above.

3.2 Construction of nonstandard finite difference scheme (NSFDS)

In this section we formulate a non-standard finite difference scheme that preserves the discussed dynamics of our continuous rabies model in equation (3) as numerical approximation. As a numerical approximation, we change the continuous time $t \in [0, \infty)$ to $t_n = n\Delta t$ where $n = 1, 2, 3, \ldots$. and Δt is the time step-size. The non-standard finite difference scheme used in this study follows [23,24,25,26]. We shall enforce, two Mickens rules in the construction of our NSFDS. These are, the nonlinear terms are approximated in a non-local way. Next, the complex denominator function $\phi(\Delta t)$ is used in place of the standard denominator Δt of the continuous derivative. The justification and detailed application of these rules are thoroughly discussed in [24], which provides the theoretical foundation for their use in constructing structure-preserving nonstandard finite difference schemes. Hence, the NSFD scheme for the rabies model (3) is given by

$$\left\{ \begin{array}{l} \frac{S_h^{n+1} - S_h^n}{\phi_1(\Delta t)} = \lambda + (\alpha)R_h^{n+1} - \beta S_h^{n+1} I_d^n - \mu S_h^{n+1} \\ \frac{E_h^{n+1} - E_h^n}{\phi_2(\Delta t)} = \beta S_h^{n+1} I_d^n - (\nu + \eta + (\tau) + \mu)E_h^{n+1} \\ \frac{T_h^{n+1} - T_h^n}{\phi_3(\Delta t)} = (\tau)E_h^{n+1} - (\Sigma + \rho + \psi + \mu)T_h^{n+1} \\ \frac{I_h^{n+1} - I_h^n}{\phi_4(\Delta t)} = (\rho)T_h^{n+1} + (\nu)E_h^{n+1} - (\mu + \delta)I_h^{n+1} \\ \frac{R_h^{n+1} - R_h^n}{\phi_5(\Delta t)} = (\psi)T_h^{n+1} + (\kappa)V_h^{n+1} - (\alpha + \mu)R_h^{n+1} \\ \frac{V_h^{n+1} - V_h^n}{\phi_6(\Delta t)} = (\eta)E_h^{n+1} + (\Sigma)T_h^{n+1} - (\kappa + \mu)V_h^{n+1} \\ \frac{S_d^{n+1} - S_d^n}{\phi_7(\Delta t)} = A - (BI_d^n + n + m)S_d^{n+1} \\ \frac{E_d^{n+1} - E_d^n}{\phi_8(\Delta t)} = BS_d^{n+1} I_d^n - (r + q + m)E_d^{n+1} \\ \frac{I_d^{n+1} - I_d^n}{\phi_9(\Delta t)} = (r)E_d^{n+1} - (m + f)I_d^{n+1} \\ \frac{V_d^{n+1} - V_d^n}{\phi_{10}(\Delta t)} = (n)S_d^{n+1} + (q)E_d^{n+1} - (m)V_d^{n+1} \end{array} \right. \quad (11)$$

The functions $\phi(\Delta t)$ replaces dt, the denominator of the continuous system. And $\phi \equiv \phi(\Delta t) = \Delta t + \mathcal{O}(\Delta t^2)$. Thus, the nature of our model following the step-size function as seen in [25,24] has

$$\begin{array}{l} \phi_1(\Delta t) = \frac{1 - e^{-\mu \Delta t}}{\mu}, \\ \phi_2(\Delta t) = \frac{1 - e^{-(\mu + \nu + \eta + \tau)\Delta t}}{\mu + \nu + \eta + \tau}, \\ \phi_3(\Delta t) = \frac{1 - e^{-(\mu + \Sigma + \rho + \psi)\Delta t}}{(\mu + \Sigma + \rho + \psi)}, \\ \phi_4(\Delta t) = \frac{1 - e^{-\left|\left(\frac{\beta\lambda}{\mu} - \mu - \delta\right)\right|\Delta t}}{\left|\left(\frac{\beta\lambda}{\mu}\right) - \mu - \delta\right|}, \\ \phi_5(\Delta t) = \frac{1 - e^{-(\mu + \alpha)\Delta t}}{\mu + \alpha}, \\ \phi_6(\Delta t) = \frac{1 - e^{-(\mu + \kappa)\Delta t}}{\mu + \kappa}, \\ \phi_7(\Delta t) = \frac{1 - e^{-m\Delta t}}{m}, \\ \phi_8(\Delta t) = \frac{1 - e^{-(m + r + q)\Delta t}}{m + r + q}, \\ \phi_9(\Delta t) = \frac{1 - e^{-\left|\left(\frac{BA}{m} - m - f\right)\right|\Delta t}}{\left|\left(\frac{BA}{\mu}\right) - m - f\right|}, \\ \phi_{10}(\Delta t) = \frac{1 - e^{-m\Delta t}}{m}. \end{array}$$

The system of equations (11) can be written explicitly as

$$\begin{cases} S_h^{n+1} = \frac{\phi_1(\Delta t)\lambda+\phi_1(\Delta t)(\alpha)R_h^{n+1}+S_h^n}{1+\phi_1(\Delta t)(\beta I_d^n+\mu)} \\ E_h^{n+1} = \frac{(\phi_2(\Delta t))(\beta S_h^{n+1}I_d^n)+E_h}{1+\phi_2(\Delta t)((\nu+\eta+(\tau)+\mu))} \\ T_h^{n+1} = \frac{\phi_3(\Delta t)(\tau E_h^{n+1})+T_h^n}{1+\phi_3(\Delta t)(\Sigma+\rho+\psi+\mu)} \\ I_h^{n+1} = \frac{\phi_4(\Delta t)(\rho T_h^{n+1}+\nu E_h^{n+1})+I_h^n}{1+\phi_4(\Delta t)(\delta+\mu)} \\ R_h^{n+1} = \frac{\phi_5(\Delta t)(\psi T_h^{n+1}+\kappa V_h^{n+1})+R_h^n}{1+\phi_5(\Delta t)(\alpha+\mu)} \\ V_h^{n+1} = \frac{\phi_6(\Delta t)(\Sigma T_h^{n+1}+\eta E_h^{n+1})+V_h^n}{1+\phi_6(\Delta t)(\kappa+\mu)} \\ S_d^{n+1} = \frac{\phi_7(\Delta t)A+S_d^n}{1+\phi_7(\Delta t)(\beta I_d^n+m+n)} \\ E_d^{n+1} = \frac{(\phi_8(\Delta t))(BS_d^{n+1}I_d^n)+E_d^n}{1+\phi_8(\Delta t)(r+m+q)} \\ I_d^{n+1} = \frac{(\phi_9(\Delta t))(rE_d^{n+1})+I_d^n}{1+\phi_9(\Delta t)(m+f)} \\ V_d^{n+1} = \frac{(\phi_{10}(\Delta t))(qE_d^{n+1}+nS_d^{n+1})+V_d^n}{1+\phi_{10}(\Delta t)(m)} \end{cases} \tag{12}$$

At the steady state,

$$S_h^{n+1}=S_h^n, E_h^{n+1}=E_h^n, T_h^{n+1}=T_h^n, I_h^{n+1}=I_h^n, R_h^{n+1}=R_h^n,$$

$$V_h^{n+1}=V_h^n, S_d^{n+1}=S_d^n, E_d^{n+1}=E_d^n, I_d^{n+1}=I_d^n, V_d^{n+1}=V_d^n.$$

$$\begin{cases} S_h^n = \frac{\phi_1(\Delta t)\lambda+\phi_1(\Delta t)(\alpha)R_h^n+S_h^n}{1+\phi(\Delta t)(\beta I_d^n+\mu)} \\ E_h^n = \frac{(\phi_2(\Delta t))(\beta S_h^n I_d^n)+E_h}{1+\phi_2(\Delta t)((\nu+\eta+(\tau)+\mu))} \\ T_h^n = \frac{\phi_3(\Delta t)(\tau E_h^n)+T_h^n}{1+\phi_3(\Delta t)(\Sigma+\rho+\psi+\mu)} \\ I_h^n = \frac{\phi_4(\Delta t)(\rho T_h^n+\nu E_h^n)+I_h^n}{1+\phi_4(\Delta t)(\delta+\mu)} \\ R_h^n = \frac{\phi_5(\Delta t)(\psi T_h^n+\kappa V_h^n)+R_h^n}{1+\phi_5(\Delta t)(\alpha+\mu)} \\ V_h^n = \frac{\phi_6(\Delta t)(\Sigma T_h^n+\eta E_h^n)+R_h^n}{1+\phi_6(\Delta t)(\kappa+\mu)} \\ S_d^n = \frac{\phi_7(\Delta t)A+S_d^n}{1+\phi_7(\Delta t)(\beta I_d^n+m+n)} \\ E_d^n = \frac{(\phi_8(\Delta t))(BS_d^n I_d^n)+E_d}{1+\phi_8(\Delta t)(r+m+q)} \\ I_d^n = \frac{(\phi_9(\Delta t))(rE_d^n)+I_d^n}{1+\phi_9(\Delta t)(m+f)} \\ V_d^n = \frac{(\phi_{10}(\Delta t))(qE_d^n+nS_d^n)+V_d^n}{1+\phi_{10}(\Delta t)(m)} \end{cases}$$

3.3 Disease free equilibrium $(\mathbb{E}_0)$

At disease free equilibrium,

$E_h = T_h = I_h = V_h = R_h = E_d = I_d = 0.$

Given $S_h^n = \frac{\phi_1(\Delta t)\lambda+\phi_1(\Delta t)(\alpha)R_h^n+S_h^n}{1+\phi_1(\Delta t)(\beta I_d^n+\mu)}$, then

$$S_h^n = \frac{\phi_1(\Delta t)\lambda+\phi_1(\Delta t)(\alpha)R_h^n}{\phi_1(\Delta t)(\beta I_d^n+\mu))}$$

and consequently, $S_h^n = \frac{\lambda}{\mu}$.

Similarly $S_d = \frac{A}{n+m}$ and $V_d = \frac{An}{m(n+m)}$, hence

$$\mathbb{E}_0 = \left(\frac{\lambda}{\mu},0,0,0,0,0,\frac{A}{n+m},0,0,\frac{An}{m(n+m)}\right) \text{ is preserved.}$$

3.4 Local stability of the disease free equilibrium $(\mathbb{E}_0)$

The Jacobian at $(\mathbb{E}_0)$ for human population is given as in (Fig. 4) and that for the dog population as in (Fig. 5).

Let

$$\begin{cases} e = \frac{1}{1+\phi_1(\Delta t)(\beta I_d+\mu)}, & a = \frac{1}{1+\phi_2(\Delta t)(\nu+\eta+\tau+\mu)} \\ b = \frac{1}{1+\phi_3(\Delta t)(\Sigma+\rho+\tau+\psi+\mu)}, & f = \frac{1}{1+\phi_4(\Delta t)(\delta+\mu)} \\ g = \frac{1}{1+\phi_5(\Delta t)(\alpha+\mu)}, & h = \frac{1}{1+\phi_6(\Delta t)(\kappa+\mu)} \\ z = \frac{1}{1+\phi_7(\Delta t)(BI_d+n+m)}, & c = \frac{1}{1+\phi_8(\Delta t)(r+q+m)} \\ d = \frac{1}{1+\phi_9(\Delta t)(f+m)}, & x = \frac{1}{1+\phi_{10}(\Delta t)(m)} \end{cases}$$

and

$$\begin{cases} p_1 = -\frac{\phi_1(\Delta t)\beta(\phi_1(\Delta t)(\lambda+\alpha R_h)+S_h)}{(1+\phi_1(\Delta t)(\beta I_d+\mu))^2} \\ p_2 = \frac{\phi_2(\Delta t)\beta I_d}{1+\phi_2(\Delta t)(\nu+\eta+\tau+\mu)}, & p_3 = \frac{\phi_2(\Delta t)\beta S_h}{1+\phi_2(\Delta t)(\nu+\eta+\tau+\mu)} \\ p_4 = \frac{\phi_3(\Delta t)\tau}{1+\phi_3(\Delta t)(\Sigma+\rho+\psi+\mu)}, & p_5 = \frac{\phi_4(\Delta t)\nu}{1+\phi_4(\Delta t)(\delta+\mu)} \\ p_6 = \frac{\phi_4(\Delta t)\rho}{1+\phi_4(\Delta t)(\delta+\mu)}, & p_7 = \frac{\phi_5(\Delta t)\psi}{1+\phi_5(\Delta t)(\alpha+\mu)} \\ p_8 = \frac{\phi_5(\Delta t)\kappa}{1+\phi_5(\Delta t)(\alpha+\mu)}, & p_9 = \frac{\phi_6(\Delta t)\eta}{1+\phi_6(\Delta t)(\kappa+\mu)} \\ p_{10} = \frac{\phi_6(\Delta t)\Sigma}{1+\phi_6(\Delta t)(\kappa+\mu)}, & p_{11} = -\frac{\phi_7(\Delta t)B(\phi_7(\Delta t)A+S_d)}{(1+(\phi_7(\Delta t)(BI_d+m+n)))^2} \\ p_{12}\frac{\phi_8(\Delta t)BI_d}{1+\phi_8(\Delta t)(r+m+q)}, & p_{13} = \frac{\phi_8(\Delta t)BS_d}{1+\phi_8(\Delta t)(r+m+q)} \\ p_{14} = \frac{\phi_9(\Delta t)r}{1+\phi_9(\Delta t)(m+f)}, & p_{15} = \frac{\phi_{10}(\Delta t)n}{1+\phi_{10}(\Delta t)m} \\ p_{16} = \frac{\phi_{10}(\Delta t)q}{1+\phi_{10}(\Delta t)m}, & \alpha = \frac{\phi_1(\Delta t)\alpha}{1+\phi_1(\Delta t)(\beta I_d+\mu)} \end{cases}$$

Evaluating the Jacobian at $\mathbb{E}_0$,

$$J_{\mathbb{E}_0} = \begin{pmatrix} e_1 & 0 & 0 & 0 & \alpha^* & 0 & 0 & 0 & p_1^* & 0 \\ 0 & a & 0 & 0 & 0 & 0 & 0 & 0 & p_3 & 0 \\ 0 & p_4 & b & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & p_5 & p_6 & f & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & p_7 & 0 & g & p_8 & 0 & 0 & 0 & 0 \\ 0 & p_9 & p_{10} & 0 & 0 & h & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & z_1 & 0 & p_{11}^* & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & c & p_{13} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & p_{14} & d & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & p_{15} & p_{16} & 0 & x \end{pmatrix}$$

where $p_1^* = \frac{\phi_1(\Delta t)\beta\frac{\lambda}{\mu}}{1+\phi_1(\Delta t)\mu}, p_{11}^* = \frac{\phi_7(\Delta t)B\frac{A}{m+n}}{1+\phi_7(\Delta t)(m+n)}, \alpha^* = \frac{\phi_1(\Delta t)\alpha}{1+\phi_1\mu}$

For the eigenvalues of our discretize system,

$$|J_{\mathbb{E}_0} - \lambda I| = \begin{pmatrix} e_1-\lambda & 0 & 0 & 0 & \alpha^* & 0 & 0 & 0 & p^*1 & 0 \\ 0 & a-\lambda & 0 & 0 & 0 & 0 & 0 & 0 & p_3 & 0 \\ 0 & p_4 & b-\lambda & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & p_5 & p_6 & f-\lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & p_7 & 0 & g-\lambda & p_8 & 0 & 0 & 0 & 0 \\ 0 & p_9 & p_{10} & 0 & 0 & h-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & z_1-\lambda & 0 & p_{11}^* & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & c-\lambda & p_{13} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & p_{14} & d-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & p_{15} & p_{16} & 0 & x-\lambda \end{pmatrix}$$

$$\begin{aligned} &\left[(c-\lambda)(d-\lambda)-p_{13}.p_{14}\right](e_1-\lambda)(a-\lambda) \\ &\left[(b-\lambda)(f-\lambda)(z_1-\lambda)(g-\lambda)(h-\lambda)(x-\lambda)\right] < 1, \end{aligned} \tag{13}$$

$$\begin{pmatrix} \frac{1}{1+\phi_1(\Delta t)(\beta I_d+\mu)} & 0 & 0 & 0 & 0 & \frac{\phi_1(\Delta t)\alpha}{1+\phi_1(\Delta t)(\beta I_d+\mu)} & 0\ 0\ p_1\ 0 \\ p_2 & \frac{1}{1+\phi_2(\Delta t)(\nu+\eta+\tau+\mu)} & 0 & 0 & 0 & 0 & 0\ 0\ p_3\ 0 \\ 0 & p_4 & \frac{1}{1+\phi_3(\Delta t)(\Sigma+\rho+\tau+\psi+\mu)} & 0 & 0 & 0 & 0\ 0\ 0\ 0 \\ 0 & p_5 & p_6 & \frac{1}{1+\phi_4(\Delta t)(\delta+\mu)} & 0 & 0 & 0\ 0\ 0\ 0 \\ 0 & 0 & p_7 & 0 & \frac{1}{1+\phi_5(\Delta t)(\alpha+\mu)} & p_8 & 0\ 0\ 0\ 0 \\ 0 & p_4 & p_{10} & 0 & 0 & \frac{1}{1+\phi_6(\Delta t)(\kappa+\mu)} & 0\ 0\ 0\ 0 \end{pmatrix}$$

Fig. 4: Jacobian for human population

$$\begin{pmatrix} 0\ 0\ 0\ 0\ 0\ 0 & \frac{1}{1+\phi_7(\Delta t)(BI_d+n+m)} & 0 & p_{11} & 0 \\ 0\ 0\ 0\ 0\ 0\ 0 & p_{12} & \frac{1}{1+\phi_8(\Delta t)(r+q+m)} & p_{13} & 0 \\ 0\ 0\ 0\ 0\ 0\ 0 & 0 & p-14 & \frac{1}{1+\phi_9(\Delta t)(f+m)} & 0 \\ 0\ 0\ 0\ 0\ 0\ 0 & p & p_{16} & 0 & \frac{1}{1+\phi_{10}(\Delta t)(m)} \end{pmatrix}$$

Fig. 5: Jacobian for dog population

where

$$\begin{cases} e_1 = \frac{1}{1+\phi_1(\Delta t)\mu}, a = \frac{1}{1+\phi_2(\Delta t)(\nu+\eta+\tau+\mu)} \\ b = \frac{1}{1+\phi_3(\Delta t)(\Sigma+\rho+\tau+\psi+\mu)}, f = \frac{1}{1+\phi_4(\Delta t)(\delta+\mu)} \\ g = \frac{1}{1+\phi_5(\Delta t)(\alpha+\mu)}, h = \frac{1}{1+\phi_6(\Delta t)(\kappa+\mu)}, z_1 = \frac{1}{1+\phi_7(\Delta t)(n+m)} \\ c = \frac{1}{1+\phi_8(\Delta t)(r+q+m)}, d = \frac{1}{1+\phi_9(\Delta t)(f+m)}, x = \frac{1}{1+\phi_{10}(\Delta t)(m)} \\ p_{13} = \frac{\phi_8(\Delta t)BS_d}{1+\phi_8(\Delta t)(r+m+q)}, p_{14} = \frac{\phi_9(\Delta t)r}{1+\phi_9(\Delta t)(m+f)} \end{cases}$$

Clearly all the λs in the first factor of our characteristic polynomial of equation (13) are less than one. The remaining eigenvalues can be obtain from

$$\lambda^2 + (d+c)\lambda + cd - p_{13}p_{14} < 1,$$

where

$$\lambda = \frac{-(d+c) \pm \sqrt{(d+c)^2 - 4(cd - p_{13}p_{14})}}{2} < 1.$$

$\lambda_{9,10} = \frac{-(\frac{1}{\mathscr{K}_1}+\frac{1}{\mathscr{K}_2})}{2} \pm \frac{\sqrt{\left(\frac{1}{\mathscr{K}_1}+\frac{1}{\mathscr{K}_2}\right)^2 - 4\left(\left(\frac{1}{\mathscr{K}_1}x\frac{1}{\mathscr{K}_2}\right) - \left(\frac{\phi_8(\Delta t)BS_d}{\mathscr{K}_1}x\frac{\phi_9(\Delta t)r}{\mathscr{K}_2}\right)\right)}}{2}$

where $\mathscr{K}_1 = 1 + \phi_8(\Delta t)(r+m+q)$, and $\mathscr{K}_2 = 1 + \phi_9(\Delta t)(m+f)$. Basic algebraic manipulations shows that λ_9 and λ_{10} are less than one.

Hence $\mathbb{E}_0$ is asymptotically stable for every value of Δt.

3.5 Global stability of the disease free fixed points

[25] stated that, $\mathbb{E}_0$ is globally asymptotically stable if conditions of local stability are satisfied and the sequence at all times converges to $\mathbb{E}_0$.

Theorem 1.
The sequence $\left(S_h^n, E_h^n, T_h^n, I_h^n, R_h^n, V_h^n, S_d^n, E_d^n, I_d^n, V_d^n\right)$ *converges to* $\mathbb{E}_0$ *for any positive initial condition whenever conditions of [24,25] are satisfied for every value of* Δt.

Proof. Suppose for $n > 0$, $\left(S_h^n, E_h^n, T_h^n, I_h^n, R_h^n, V_h^n, S_d^n, E_d^n, I_d^n, V_d^n\right)$ converges to $\mathbb{E}_0$, then $\left(S_h^{n+1}, E_h^{n+1}, T_h^{n+1}, I_h^{n+1}, R_h^{n+1}, V_h^{n+1}, S_d^{n+1}, E_d^{n+1}, I_d^{n+1}, V_d^{n+1}\right)$ converges to $\mathbb{E}_0$.

Considering the discretized system of equations in (12), it can be proven that $\mathbb{E}_0$ satisfies the global asymptotic conditions in [26].

$$S_h^{n+1} = \frac{\phi_1(\Delta t)\lambda + \phi_1(\Delta t)(\alpha)R_h^{n+1} + S_h^n}{1 + \phi_1(\Delta t)(\beta I_d^n + \mu)}$$

$$\text{as} \quad n \to \infty,\ n = n+1 \equiv \infty$$

$$(S_h^n + S_h^n\phi_1(\Delta t)(\beta I_d^n + \mu)) = \phi_1(\Delta t)\lambda + \phi_1(\Delta t)(\alpha)R_h^{n+1} + S_h^n$$

$$S_h^n\phi_1(\Delta t)(\beta I_d^n + \mu)) = \phi_1(\Delta t)\lambda + \phi_1(\Delta t)(\alpha)R_h^{n+1}$$

$$S_h^n(\beta I_d^n + \mu) = \lambda + (\alpha)R_h^{n+1}$$

But $R_h = I_d = 0$ at $\mathbb{E}_0$. Hence $S_h^{n+1} \to \frac{\lambda}{\mu}$, as $n \to \infty$.

$$S_d^{n+1} = \frac{\phi_7(\Delta t)A + S_d^n}{1 + \phi_7(\Delta t)(\beta I_d^n + m + n)}$$

Then $S_d^{n+1} \to \frac{A}{n+m}$, as $n \to \infty$.

$$V_d^{n+1} = \frac{(\phi_{10}(\Delta t))(qE_d^n + nS_d^n) + V_d^n}{1 + \phi_{10}(\Delta t)(m)}$$

Then $V_d^{n+1} \to \frac{An}{m(m+n)}$, as $n \to \infty$.

Hence, the discrete solution generated by the NSFD scheme converges to the disease-free equilibrium $\mathbb{E}_0$ as $n \to \infty$ for all values of Δt. Since the NSFD scheme is constructed to preserve the qualitative dynamics of the continuous system, and the conditions in [24,25] ensure the global asymptotic stability of $\mathbb{E}_0$, we conclude that $\mathbb{E}_0$ remains globally asymptotically stable.

4 Optimal control

In this section, we shall have a look at some possible ways of controlling rabies in both populations by formulating an optimal control for our rabies model. This will be done by adding time-dependent controls of : u_1, representing effective education and campaign on rabies, u_2, representing policy and education on good petting and u_3, representing the treatment of exposed dogs to kill the the virus before infections. The optimal control model studies the importance of the selected control measures to reduce the additions carried out by various individuals in the compartments. Thus, taking into account the controls implemented, the non-linear model becomes;

$$\begin{cases} S_h'(t) &= \lambda + (\alpha)R_h - (1-u_1)(\beta I_d + \mu)S_h \\ E_h'(t) &= (1-u_1)\beta S_h I_d - (\nu+\eta+(\tau)+\mu)E_h \\ T_h'(t) &= (\tau)E_h - (\Sigma+\rho+\psi+\mu)T_h \\ I_h'(t) &= (\rho)T_h + (\nu)E_h - (\mu+\delta)I_h \\ R_h'(t) &= (\psi)T_h + (\kappa)V_h - (\alpha+\mu)R_h \\ V_h'(t) &= (\eta)E_h + (\Sigma)T_h - (\kappa+\mu)V_h \\ S_d'(t) &= A - (1-u_2)(BI_d + n + m)S_d \\ E_d'(t) &= (1-u_2)BS_d I_d - (r+q+m)(1-u_3)E_d \\ I_d'(t) &= (1-u_3)(r)E_d - (m+f)I_d \\ V_d'(t) &= (n)S_d + (q)E_d - (m)V_d \end{cases} \tag{14}$$

The purpose of the control strategies is to reduce the number of exposed individuals and animals, infected ones while reducing the cost of treatment at the same time. With this the cost incurred in the three control measures are denoted by C_1, C_2 and C_3 and we set our objective functional as;

$$\zeta(u_1,u_2,u_3) = \int_0^T (L_1 S_h + L_2 S_d + L_3 E_d + \frac{1}{2}\sum_1^3 C_i u_i^2)dt \tag{15}$$

where L_1 ,L_2 ,L_3 , and C_1 ,C_2 ,L_3 , are all positive weight constants.

The terms; $C_1 u_1^2$ represents the cost of education that protects susceptible individuals from coming into contact with infected dogs, $c_2 u_2^2$ the cost of education and policy implementation on proper petting, and $c_3 u_3^2$ the cost for the proper treatment of wounds from exposed dogs before they become infected.

We are interested in an optimal functions $\{u_1^*, u_2^*, u_3^*\}$, $\ni$

$$\zeta\{(u_1^*,u_2^*,u_3^*)\} = min\{\zeta(u_1^*,u_2^*,u_3^*) : u_1^*,u_2^*,u_3^* \in \mathbf{N}\}$$

where

$$\mathbf{N} = \{u_i = 0 \le u_i(t) \le 1, \in [0,T] \text{ lebesgue measurable}\} \tag{16}$$

$\forall\, i = 1,2,3$ called the controls [27,28,29].

Theorem 2. *There exist $U^* = (u_1^*, u_2^*, u_3^*) \in U$ an optimal control $\ni$*

$$\zeta(u_1^*,u_2^*,u_3^*) = \min_U \zeta(u_1,u_2,u_3), \tag{17}$$

s.t to equations (14) with the initial conditions.

Proof. Following [12], we demonstrate the existence of an optimal control for our extended rabies model. It can easily be seen that, all variables including control variables are non-negatives. Additionally, it is clear that the objective function's required and convex properties in u_1, u_2, and u_3 are met when the control system is minimized. The space $U = \{u | u_1, u_2, u_3$ are measurable, $0 \le u_1, u_2, u_3 \le u_{max} < \infty$, $t \in [0,T]\}$ according to [11] is closed and convex. The control system is bounded and that suffice the compactness required for the presence of an optimal control.

Equation (15) has an integrand that is convex on the control u. Thus, we observe a constant $q > 1$ and positive numbers u_1, u_2 and u_3 exist, $\ni$ $\zeta(u_1,u_2,u_3) \ge u_1 \left(|u_1|^2 + |u_2|^2 + |u_3|^2\right)\frac{q}{2} - u^2$. Hence, there is an existence of the optimal control.

4.1 Pontryagin's maximum principle application

Here we shall use the Pontryagin's Maximum Principle, [30] to identify the prerequisites for the optimality. The hamiltonian (H) with respect to (u_1,u_2,u_3) is given by;

$$\begin{cases} H = [(L_1 S_h + L_2 S_d + L_3 E_d + \frac{1}{2}(c_1 u_1^2 + c_2 u_2^2 + c_1 3 u_3^2) \\ +\lambda_1[\lambda + (\alpha)R_h - (1-u_1)\beta I_d S_h + \mu S_h] \\ +\lambda_2[(1-u_1)\beta S_h I_d - (\nu+\eta+(\tau)+\mu)E_h] \\ +\lambda_3[(\tau)E_h - (\Sigma+\rho+\psi+\mu)T_h] \\ +\lambda_4[(\rho)T_h + (\nu)E_h - (\mu+\delta)I_h] \\ +\lambda_5[(\psi)T_h + (\kappa)V_h - (\alpha+\mu)R_h] \\ +\lambda_6[(\eta)E_h + (\Sigma)T_h - (\kappa+\mu)V_h] \\ +\lambda_7[A - (1-u_2)BI_d S_d + (n+m)S_d] \\ +\lambda_8[(1-u_2)BS_d I_d - (1-u_3)rE_d + (q+m)E_d] \\ +\lambda_9[(1-u_3)(r)E_d - (m+f)I_d] \\ +\lambda_{10}[(n)S_d + (q)E_d - (m)V_d] \end{cases} \tag{18}$$

The adjoint solutions are also given by,

$$\begin{cases} \frac{\partial\lambda_1}{\partial t} = -\frac{\partial H}{\partial S_h} = -L_1 + \lambda_1(1-u_1)\beta I_d + \mu - \lambda_2(1-u_1)(\beta I_d) \\ \frac{\partial\lambda_2}{\partial t} = -\frac{\partial H}{\partial E_h} = \lambda_2(\nu+\eta+\tau+\mu) - \lambda_3\tau - \lambda_4\nu - \lambda_6\eta \\ \frac{\partial\lambda_3}{\partial t} = -\frac{\partial H}{\partial T_h} = \lambda_3(\Sigma+\rho+\psi+\mu) - \lambda_4\rho - \lambda_5\psi - \lambda_6\Sigma \\ \frac{\partial\lambda_4}{\partial t} = -\frac{\partial H}{\partial I_h} = \lambda_4(\mu+\delta) \\ \frac{\partial\lambda_5}{\partial t} = -\frac{\partial H}{\partial R_h} = -\lambda_1\alpha + \lambda_5(\alpha+\mu) \\ \frac{\partial\lambda_6}{\partial t} = -\frac{\partial H}{\partial V_h} = -\lambda_5\kappa + \lambda_6(\kappa+\mu) \\ \frac{\partial\lambda_7}{\partial t} = -\frac{\partial H}{\partial S_d} = -L_2 + \lambda_7(1-u_2)BI_d + (n+m) - \lambda_8(1-u_2)BI_d - \lambda_{10}n \\ \frac{\partial\lambda_8}{\partial t} = -\frac{\partial H}{\partial E_d} = -L_3 + \lambda_8(1-u_3)r + (q+m) - \lambda_9(1-u_3)r - \lambda_{10}q \\ \frac{\partial\lambda_9}{\partial t} = -\frac{\partial H}{\partial I_d} = \lambda_1(1-u_1)(\beta S_h) - \lambda_2(1-u_1)(\beta S_h) + \lambda_7(1-u_2)BS_d \\ -\lambda_8(1-u_2)BS_d + \lambda_9(m+f) \\ \frac{\partial\lambda_{10}}{\partial t} = -\frac{\partial H}{\partial V_d} = \lambda_{10}m \end{cases} \tag{19}$$

and satisfies the boundary conditions

$$\lambda_i(t) = 0,\ \mathrm{i} = 1,2,\ldots,10 \tag{20}$$

4.2 Characterization of optimal control

We obtained the optimal set characterization by solving the system of partial differential equations $\frac{\partial H}{\partial u_1} = 0$, $\frac{\partial H}{\partial u_2} = 0$, $\frac{\partial H}{\partial u_3} = 0$, for u_1, u_2, u_3.

$$\begin{cases} \frac{\partial H}{\partial u_1} = c_1u_1 + \lambda_1\beta I_d S_h - \lambda_2\beta S_h I_d \\ u_1 = \frac{(\lambda_2-\lambda_1)\beta I_d S_h}{c_1} \\ \frac{\partial H}{\partial u_2} = c_2u_2 + \lambda_7 BI_d s_d - \lambda_8 BS_d I_d \\ u_2 = \frac{(\lambda_8-\lambda_7)BI_d s_d}{c_2} \\ \frac{\partial H}{\partial u_3} = c_3u_3 - \lambda_8 rE_d + \lambda_9 rE_d \\ u_3 = \frac{(\lambda_8-\lambda_9)rE_d}{c_1} \end{cases}$$

If the controls are bounded, then we have ∀= 1,2,3,

$$u_i^* = \begin{cases} 0 & \text{if } \hat{u}_i \le 0 \\ \hat{u}_i & \text{if } 0 \le \hat{u}_i \le 1 \\ 1 & \text{if } \hat{u}_i \ge 1 \end{cases}$$

The optimal control vector u_1^*, u_2^* and u_3^* that minimizes ζ is given by

$$\begin{cases} u_1^* = min\left\{1, max\left\{0, \left(\frac{(\lambda_2-\lambda_1)\beta I_d S_h}{c_1}\right)\right\}\right\} \\ u_2^* = min\left\{1, max\left\{0, \left(\frac{(\lambda_8-\lambda_7)BI_d s_d}{c_2}\right)\right\}\right\} \\ u_3^* = min\left\{1, max\left\{0, \left(\frac{(\lambda_8-\lambda_9)rE_d}{c_1}\right)\right\}\right\} \end{cases} \tag{21}$$

The λ_i are found by solving simultaneously equations (19) and (20). Putting in the controls u_1^* u_2^* *and* u_3^* in the control system gives

$$\begin{cases} S_h'(t) = \lambda + (\alpha)R_h - (1 - min\left\{1, max\left\{0, \left(\frac{(\lambda_2-\lambda_1)\beta I_d S_h}{c_1}\right)\right\}\right\})\beta I_d S_h + \mu S_h \\ E_h'(t) = (1 - min\left\{1, max\left\{0, \left(\frac{(\lambda_2-\lambda_1)\beta I_d S_h}{c_1}\right)\right\}\right\})\beta S_h I_d - (\nu+\eta+(\tau)+\mu)E_h \\ T_h'(t) = (\tau)E_h - (\Sigma+\rho+\psi+\mu)T_h \\ I_h'(t) = (\rho)T_h + (\nu)E_h - (\mu+\delta)I_h \\ R_h'(t) = (\psi)T_h + (\kappa)V_h - (\alpha+\mu)R_h + u_3 I_h \\ V_h'(t) = (\eta)E_h + (\Sigma)T_h - (\kappa+\mu)V_h \\ S_d'(t) = A - (1 - min\left\{1, max\left\{0, \left(\frac{(\lambda_8-\lambda_7)BI_d s_d}{c_2}\right)\right\}\right\})BI_d S_d + (n+m)S_d \\ E_d'(t) = (1 - min\left\{1, max\left\{0, \left(\frac{(\lambda_8-\lambda_7)BI_d s_d}{c_2}\right)\right\}\right\})BS_d I_d \\ -(1 - min\left\{1, max\left\{0, \left(\frac{(\lambda_8-\lambda_9)rE_d}{c_1}\right)\right\}\right\})rE_d + (q+m)E_d \\ I_d'(t) = (1 - min\left\{1, max\left\{0, \left(\frac{(\lambda_8-\lambda_9)rE_d}{c_1}\right)\right\}\right\})(r)E_d - (m+f)I_d \\ V_d'(t) = (n)S_d + (q)E_d - (m)V_d \end{cases} \tag{22}$$

5 Numerical analysis and discussion

Table 1: Rabies model parameters and description based on source data. The choice of parameter values was informed by fitting a model to observed data from the Veterinary service, Kumasi.

Parameter	values	Reference
Λ	2000	fitted
β	0.000301	fitted
μ	0.02	fitted
τ	0.80253	fitted
Σ	0.448464	fitted
ψ	0.498419	fitted
δ	0.9999	fitted
ρ	0.00727	fitted
ν	0.008766	fitted
η	0.140391	fitted
α	0.01	fitted
κ	0.603571	fitted
A	300	fitted
B	0.219972	fitted
r	0.200024	fitted
n	0.555589	fitted
q	0.150029	fitted
m	0.0312	fitted
f	0.999	fitted

We perform numerical analysis on the control strategies to curb the rabies model using the parameters in table (1). This was conducted by solving the state equations, adjoint solutions, and the boundary conditions using the ?desolve" library in Rsoftware for the solutions of the optimal control problem and ggplot2 for the graphs, guessing the controls over a simulated time. Several combination of controls were considered, and we assessed carefully the simulations for each considered strategy. Our parameter values table (1), are fitted data we obtained from the Veterinary service, Kumasi. We iterate till a stopping criterion is reached, and it terminates when the values of unknown variables from the previous iteration approach those in the current iteration. [25,30].

5.0.1 Strategy 1: u_1 only

The first control measure we implemented in our attempt to curb the spread of rabies virus is education and awareness creation on rabies. Figure (6) shows the effectiveness in minimizing the spread in human population as the control has reduced infected humans from 5 in the fifth year to 1. The reason is that, awareness only prevents human exposure to the disease and thus the control will have no effect on the dog population.

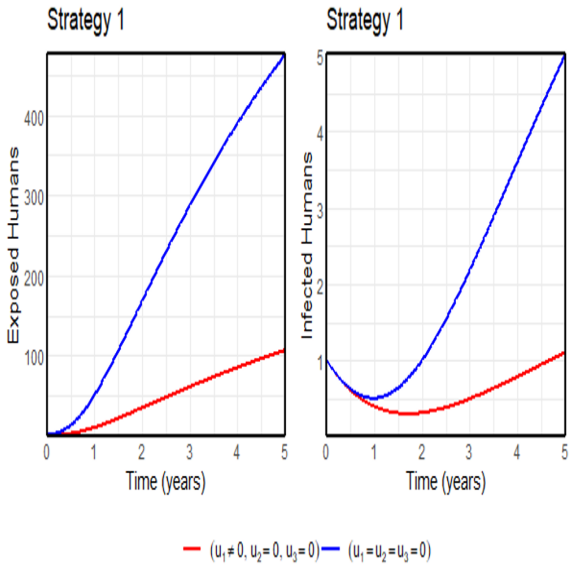

Fig. 6: Dynamics of infected and exposed humans population with and without controls.

5.0.2 Strategy 2: u_2 only

This control measure considers the implementation of a government policy that ensures all canines are vaccinated with serious consequences for defaulters. Our analysis, Figure (7) shows this control is not effective and cost worthy. This is because the changes brought in by the control in both populations is very negligible.

5.0.3 Strategy 3: u_3 only

The application of treatment of infected dogs before the virus finds its way into the blood stream of an exposed dog is the third control. Figure (8) shows the efficacy of the control in human population by reducing the infected humans from 5 to just above 2 in the fifth year. This intervention is effective for the dog population since the number of infected dogs dropped below 75 in the fifth year as seen in Figure (8).

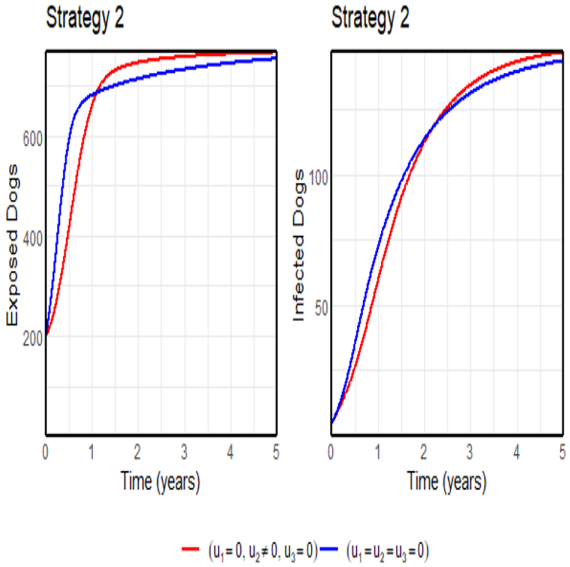

Fig. 7: Dynamics of exposed and infected dogs population with and without controls.

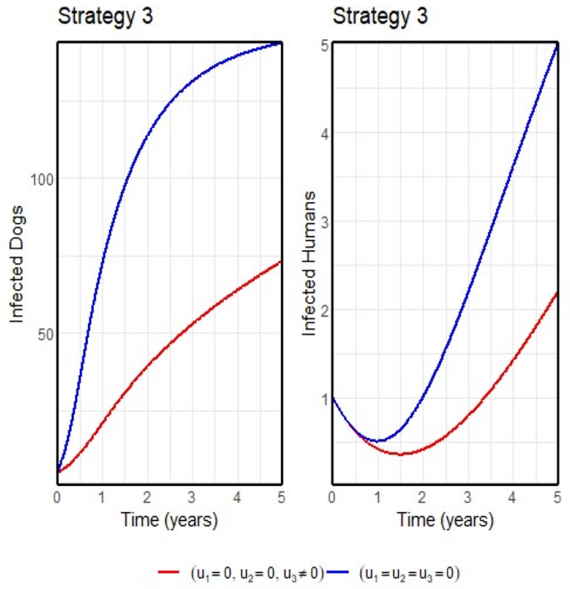

Fig. 8: Dynamics of infected dogs and humans population with and without controls.

5.0.4 Strategy 4: u_1 and u_2

Strategy 4 is the implementation of u_1 and u_2 to examine their impact on the model's dynamics. Figure (9), shows the infected human population increased from 1 to 5 within the five year period after an initial decline within the first one and half year period when no control is applied. The implementation of the strategy reduced the

number of infected and exposed persons drastically within the five year period. The outcome shows the strategy is efficient for both population.

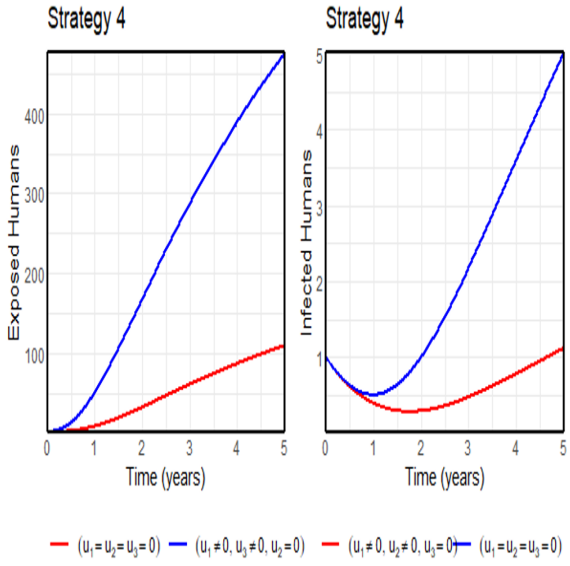

Fig. 9: Dynamics of exposed and infected humans population with and without controls.

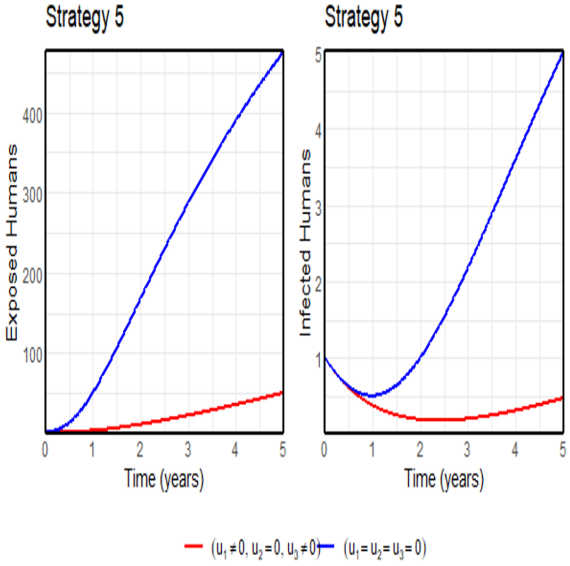

Fig. 10: Dynamics of exposed and infected humans population with and without controls.

5.0.5 Strategy 5:u_1 and u_3

We combined u_1 and u_3, in our quest to lower the number of infections. Without controls both populations rise swiftly in the five years though the infected human population has an initial decline. Figure (10) shows with the controls, the infected humans fell sharply blow 1 in the five year intervention period. The exposed humans on the other hand decreased smoothly to about 75 in the entire period. The strategy is therefore very effective for both humans.

5.0.6 Strategy 6:u_2 and u_3

We considered u_2 and u_3 to assess their impact on the infection rate in Figure (11). The controls reduced the number of infected dogs and humans in the five year period. The graphs of uncontrolled populations were far higher than the graphs with control strategies at all times. An indication that the strategy is effective in minimizing infections in both dogs and humans.

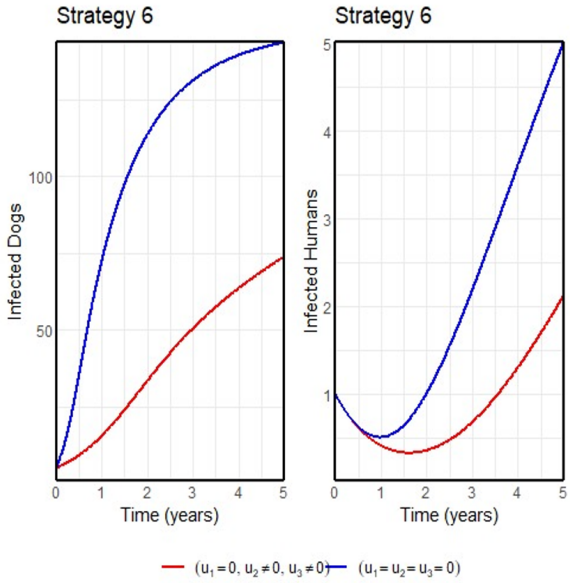

Fig. 11: Dynamics of infected dogs and humans population with and without controls.

Thus, effectively utilizing the best combination of interventions will help mitigate the spread of rabies within these populations and significantly impact human health.

6 Conclusion

A deterministic model that demonstrates the dynamic transmission of rabies disease among human beings and canines was investigated. Using the non-standard finite difference scheme for boundedness and non-negativity of solutions, $\mathbb{E}_0$, and its local and global stability, an in-depth investigation of the rabies model was carried out. The rabies model was expanded to incorporate treatment of exposed dogs, policy and education on good petting, and effective education and campaigns on rabies for optimal control. By optimizing our objective function, we found that integrating effective public education and

awareness campaigns with the treatment of exposed dogs offers a highly effective strategy with strong long-term potential to reduce rabies transmission in the Ashanti Region of Ghana.

Data Availability Statement

The parameter values employed to support the outcomes of this analysis are described in Section 2 and utilized in Section 5. The choice of parameter values was informed by fitting a model to observed data from the Veterinary service, Kumasi.

Declaration of Competing Interest

The authors declare that they have no competing interests.

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