Mathematical Analysis of Rabies Transmission Dynamics and Control

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Abstract: *Rabies is a dangerous disease that kills many people than any other communicable disease and yet it is underrated. This results from the little knowledge on the myriad ways of transmission of the virus. A deterministic model is proposed to study the spread of the rabies virus in both domestic dogs (Canis familiaries) and humans (Homo sapiens). We elaborately studied the spread of the rabies virus from dogs-to-dogs, dogs-to-humans and for the first time, humans-to-humans. Sensitivity analysis is performed to determine the influence of various parameters on the transmission of rabies the most. The rabies-free equilibrium and the endemic equilibrium points were determined and the conditions under which the equilibria are stable were also obtained. The stability conditions provide the conditions under which the disease will persist or get to be eradicated. Numerical solutions of the model were obtained using the ode45 routine in MATLAB. The study demonstrated that for rabies to be eradicated, the rate at which dogs are recruited must be decreased, culling of exposed and infected dogs should be increased and mass vaccination of the dog population should be targeted.*

Keywords: Rabies virus, Pre-exposure prophylaxis, Post-exposure prophylaxis, Stability analysis, Sensitivity analysis

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1 Introduction

Rabies is a transmittable disease that affects mammals [9]. It is a ribonucleic acid (RNA) virus of various shapes of genus Lyssavirus, family *Rhabdoviridae* and order *Mononegavirales* [12]. The encumbrance of rabies is on the rise as it has been estimated that 55,000 humans die each year [11], more than yellow fever, dengue fever, or encephalitis [10]. Rabies has been a burden since ancient times because of its myriad death cases compared to many transmittable diseases [1, 16]. In Asia, Africa, and Latin America, about 59,000 people are killed by rabies yearly [7]. Mathematical models have become a prominent instrument for providing interventions in eliminating diseases [17, 18]. In modeling, only one type of animals are used

and this makes it difficult to take decision for all animals because distinct transmission dynamics exist between them [13]. However, each model has its unique disease pathways which provides enough features to understand all the characteristics of animals [13]. Rhodes et al. [14] used a deterministic model equations to examine the flow of rabies in jackals *(Canis adustus)* and how these animals transmits the rabies virus to dogs. They showed that rabies is transmitted to jackals by infected dogs leaving in communities nearby but never included any control measure. The most appreciated work of Russell et al. [15] on the different ways of how raccoon-related rabies showed that vaccines should not be given to raccoons beyond rivers so as to limit the endemic region. Subsequently, raccoon rabies will be eliminated. The research however did not considered the younger raccoons that must travel for longer distances to the river which results in increasing death rate of the raccoons. Hampson et al. [8] presented a systematic way to ensure whether the eradication of dog-mediated rabies from the world is totally attainable. The research used an elaborative scientific method to study infected animals by taking into account very needy features in Tanzania. The deductions of the research showed that the idea of eliminating animal rabies can be achieved in Africa through vaccination and public education. The increase in recruitment of dogs has become a greater burden to put their findings into reality, notwithstanding, the fact that dogs have been proven to be the major causes of rabies in Africa. Zinssttaga [21] proposed a nuique deterministic rabies transmission model to determine the cost associated with eradicating rabies in the developing world. They concluded that, among various ways of eliminating rabies, dog vaccination is the most expensive but the best method to attain total rabies eradication in Africa for more than 6 years. Sornsong et al. [18] developed an $SEIR$ model of rabies with control measures and observed that, if the isolation value of infected dogs is increased and the eradication value of infected dogs is also increased, then the number of infected dogs will decrease. The research however, did not take into consideration the failure of vaccination and treatment in humans and dogs and the best optimal control strategy. Zhang et al. [20] proposed a model to describing how rabies spread amongst dogs and humans using ordinary differential equations and concluded that managing dog recruitment rate and vaccination of dogs are the competent ways of eradicating rabies in the world and isolation of many number of healthy dogs should be substituted with vaccination in order to accomplish the aim of eradicating rabies in the world. Taylor and colleagues [19] showed how domestic dogs should be properly treated to avoid getting the disease in the first place. Another interesting research of rabies was conducted to study the spread of rabies in a population of humans that take dog meat as a delicacy [3]. The insightful work of Asamoah and partners [2] studied the optimal control model of rabies and concluded that, using more vaccination to control the rabies virus among dogs and humans. There is the existence of the transmission of the disease from one human to the other, which has not been considered in previous models. Motivated by the research in [2] and the aim of global alliance to curb rabies, there is the need for more research into rabies by considering all the possible pathways of the spread of the disease together with the strategy to eliminate it as well.

In the next section, the model under consideration is formulated. In section 3, qualitative properties of

the model are discussed. The model is numerically solved in section 4 to simulate the impact of some epidemiological factors on the dynamics of rabies. Important conclusions of the study are drawn in section 5

2 Model Formulation

For this study, the dog compartment $N_G(t)$ is segregated into four compartments: Susceptible dogs $S_G(t)$, dogs exposed to rabies virus $E_G(t)$, rabies-infected dogs $I_G(t)$ and dog recovered from rabies infection $R_G(t)$ respectively. Thus, the total dog compartment is $N_G(t) = S_G(t) + E_G(t) + I_G(t) + R_G(t)$.

The total human compartment is $N_H(t)$ is likewise divided into four compartments of susceptible humans $S_H(t)$, humans exposed to rabies virus $E_H(t)$, rabies-infected humans $I_H(t)$ and recovered humans from rabies contagion $R_H(t)$ respectively. The entire human compartment yields $N_H(t) = S_H(t) + E_H(t) + I_H(t) + R_H(t)$.

There is an effective contact between infected and susceptible dogs to the susceptible humans. The susceptible dog population, $S_G(t)$, is increased by a birth rate of A_G. Also, recruitment into the human population by and birth or immigration is considered to be at rate A_H into the susceptible human population. Susceptible dogs which are exposed to rabies virus by an effective contact in the exposed and infectious dogs at a rate of $(1-\theta_G)\,\beta_{GG}\,(E_G + I_G)\,S_G$ per unit time, with β_{GG} being the per capita effective contact rate between susceptible dogs and exposed/infected dogs. We assume that educational campaigns towards rabies disease eradication lead to a discounting of the infection by a factor $1-\theta_G$, where θ_G is the effectiveness of the public health education in reducing rabies among dogs. Dogs that get exposed to the rabies virus are assigned to γ_G. If exposed dogs are treated (post-exposure prophylaxis) at the rate of ω_G, then $(1-\omega_G)\gamma_G E_G$ represents the movement of E_G to the infected compartment while $\omega_G\gamma_G E_G$ is the flow of E_G to the recovered class. Through pre-exposure prophylaxis, Susceptible dogs flow to the recovered compartment at ν_G per unit time. Since post-exposure recovery does not confer permanent immunity, we take ε_G to be the rate of waning of immunity or the rate at which the recovered dogs become susceptible. Exposed dogs are culled at the rate of C_G. The mortality rate amongst dogs and rabies-induced mortality among infected animals are taken to be n_G and μ_G respectively. The parameters θ_H, γ_H, ω_H, ε_H, n_H and μ_H are similarly defined for the human population as they were defined for the dog population. While human-to-human flow of rabies is uncommon, it is still possible especially through organ transplant (which has been recorded albeit only twice). Therefore, we take β_{HH} to be the probability of infection through human-to-human transmission.

The assumptions made yielded the following system of eight(8) differential equations below:

$$\left.\begin{aligned}
\frac{\mathrm{d}S_G}{\mathrm{d}t} &= A_G - (1-\theta_G)\beta_{GG}\left(E_G + I_G\right)S_G + \varepsilon_G R_G - \left(n_G + \nu_G + \theta_G\right)S_G,\\
\frac{\mathrm{d}E_G}{\mathrm{d}t} &= (1-\theta_G)\beta_{GG}S_G\left(E_G + I_G\right) - \left(\gamma_G + n_G + C_G\right)E_G,\\
\frac{\mathrm{d}I_G}{\mathrm{d}t} &= (1-\omega_G)\gamma_G E_G - (n_G + \mu_G)I_G,\\
\frac{\mathrm{d}R_G}{\mathrm{d}t} &= \nu_G S_G + \gamma_G\omega_G E_G - (n_G + \varepsilon_G)R_G,\\
\frac{\mathrm{d}S_H}{\mathrm{d}t} &= A_H - (1-\theta_H)\left[\beta_{HH}(E_H + I_H) + \beta_{GH}\left(E_G + I_G\right)\right]S_H + \varepsilon_H R_H - (n_H + \nu_H + \theta_H)S_H,\\
\frac{\mathrm{d}E_H}{\mathrm{d}t} &= (1-\theta_H)\,S_H\left[\beta_{HH}(E_H + I_H) + \beta_{GH}\left(E_G + I_G\right)\right] - \left(\gamma_H + n_H\right)E_H,\\
\frac{\mathrm{d}I_H}{\mathrm{d}t} &= (1-\omega_H)\gamma_H E_H - (n_H + \mu_H)I_H,\\
\frac{\mathrm{d}R_H}{\mathrm{d}t} &= \nu_H S_H + \omega_H\gamma_H E_H - \left(n_H + \varepsilon_H\right)R_H,\\
&\text{with initial conditions being non-negative.}
\end{aligned}\right\} \quad (1)$$

The following conventions are applied where required in the succeeding discussions.

$$\left.\begin{aligned}
&k_1 = \left(n_G + \nu_G + \theta_G\right), \quad k_2 = \left(\gamma_G + n_G + C_G\right), \quad k_3 = n_G + \mu_G, \quad k_4 = n_G + \varepsilon_G,\\
&k_5 = (n_H + \nu_H + \theta_H), \quad k_6 = \gamma_H + n_H, \quad\quad\quad k_7 = n_H + \mu_H, \quad k_8 = n_H + \varepsilon_H.
\end{aligned}\right\} \quad (2)$$

3 Model Analysis

This section presents how the model equation is analyzed extensively.

3.1 Feasible region of the model

Summing the first-four together and also the last-four equations of model (1) and solving yields

$$N_G(t) \leq \frac{A_G}{n_G} \text{ and } N_H(t) \leq \frac{A_H}{n_H}.$$

The feasible region of the model equation therefore yields $\Gamma = \Gamma_G \times \Gamma_H$, where

$$\Gamma_G = \left\{(S_G, E_G, I_G, R_G) \in \mathbb{R}^4_+ | N_G = S_G + E_G + I_G + R_G \leq \frac{A_G}{n_G}\right\} \text{ and}$$

$$\Gamma_H = \left\{(S_H, E_H, I_H, R_H) \in \mathbb{R}^4_+ | N_H = S_H + E_H + I_H + R_H \leq \frac{A_H}{n_H}\right\}.$$

Then, every solution of model (1) alongside initial conditions in Γ remains in Γ for every $t > 0$. The neighbourhood Γ is a positively invariant region with regard to model (1). Hence the Model (1) is mathematically and epidemiologically well-posed. Since the model equations depict the population dynamics, all parameters and variables are required to be positive and therefore in the succeeding analysis, they are usurped to be positive.

The model (1) can be fragmented into sub-models of two, namely; Rabies-in-Dog-only model and Rabies-in-humans-only model. Therefore, in the subsequent sections, these two sub-models will be separately analyzed before the full-model is analyzed.

3.2 Qualitative properties of sub-models

The Dog-only model is given by

$$\left.\begin{array}{ll} \overbrace{\begin{array}{l}\frac{\mathrm{d}S_G}{\mathrm{d}t} = A_G - \lambda_{GG}S_G + \varepsilon_G R_G - k_1 S_G,\\ \frac{\mathrm{d}E_G}{\mathrm{d}t} = \lambda_{GG}S_G\left(E_G + I_G\right) - k_2 E_G,\\ \frac{\mathrm{d}I_G}{\mathrm{d}t} = (1-\omega_G)\gamma_G E_G - k_3 I_G,\\ \frac{\mathrm{d}R_G}{\mathrm{d}t} = \nu_G S_G + \gamma_G\omega_G E_G - k_4 R_G,\end{array}}^{\text{Dog-only model (DoM)}} & \overbrace{\begin{array}{l}\frac{\mathrm{d}S_H}{\mathrm{d}t} = A_H - \lambda_{HH}S_H + \varepsilon_H R_H - k_5 S_H,\\ \frac{\mathrm{d}E_H}{\mathrm{d}t} = \lambda_{HH}S_H - k_6 E_H,\\ \frac{\mathrm{d}I_H}{\mathrm{d}t} = (1-\omega_H)\gamma_H E_H - k_7 I_H,\\ \frac{\mathrm{d}R_H}{\mathrm{d}t} = \nu_H S_H + \omega_H\gamma_H E_H - k_8 R_H,\end{array}}^{\text{Human-Only Model (HoM)}} \\ \text{with non-negative initial conditions.} & \end{array}\right\} \quad (3)$$

3.2.1 Rabies-free equilibrium points and basic reproduction numbers

The rabies-free equilibrium points of the Dog-only and Human-only sub-models are respectively given by $\mathcal{E}_D^0 = \left(\frac{k_4 A_G}{(k_1k_4-\nu_G\varepsilon_G)}, 0, 0, \frac{\nu_G A_G}{(k_1k_4-\nu_G\varepsilon_G)}\right)$ and $\mathcal{E}_H^0 = \left(\frac{k_8 A_H}{(k_5k_8-\nu_H\varepsilon_H)}, 0, 0, \frac{\nu_H A_H}{(k_5k_8-\nu_H\varepsilon_H)}\right)$.
Using the next generation matrix method [6], the basic reproduction number of the sub-models can be found to be a spectral radius when utilizing the next-generation matrices $\mathcal{F}_G\mathcal{V}_G^{-1}$ and $\mathcal{F}_H\mathcal{V}_H^{-1}$ are given as

$$\mathcal{F}_G = \begin{bmatrix} (1-\theta_G)\beta_{GG}S_G & (1-\theta_G)\beta_{GG}S_G \\ 0 & 0 \end{bmatrix}, \quad \mathcal{V}_G = \begin{bmatrix} k_2 & 0 \\ -(1-\omega_G)\gamma_G & k_3 \end{bmatrix},$$
$$\mathcal{F}_H = \begin{bmatrix} (1-\theta_H)\beta_{HH}S_H & (1-\theta_H)\beta_{HH}S_H \\ 0 & 0 \end{bmatrix}, \quad \mathcal{V}_H = \begin{bmatrix} k_6 & 0 \\ -(1-\omega_H)\gamma_H & k_7 \end{bmatrix}.$$

The basic reproduction number of the Dog-Only model and the humans-only sub-models are obtained as

$$\overbrace{\mathcal{R}_0^G = \frac{\beta_{GG}(1-\theta_G)\left[(1-\omega_G)\gamma_G + k_3\right]S_G^0}{k_2k_3}}^{\text{Basic Reproduction number of DoM}} \text{ and } \overbrace{\mathcal{R}_0^H = \frac{\beta_{HH}(1-\theta_H)\left[(1-\omega_H)\gamma_H + k_7\right]S_H^0}{k_6k_7}}^{\text{Basic Reproduction number of HoM}}.$$

The following result is therefore obtained from [6].

Theorem 1. *The rabies-free equilibrium points of the Dog-only and Human-only sub-models are locally asymptotically stable whenever the respective basic reproduction numbers are less than one.*

3.2.2 Endemic equilibrium points

The rabies-persistent equilibrium points of the sub-models are given by

$$\left.\begin{array}{ll} \overbrace{\begin{array}{l}S_G^* = \frac{k_4 A_G}{\mathcal{R}_0^G(k_1k_4-\nu_G\varepsilon_G)},\\ E_G^* = \frac{k_4 A_G}{(k_2k_4-\gamma_G\omega_G\varepsilon_G)}\left(1-\frac{1}{\mathcal{R}_0^G}\right),\\ I_G^* = \frac{k_4(1-\omega_G)\gamma_G A_G}{k_3(k_2k_4-\gamma_G\omega_G\varepsilon_G)}\left(1-\frac{1}{\mathcal{R}_0^G}\right),\\ R_G^* = \frac{\nu_G A_G}{\mathcal{R}_0^G(k_1k_4-\nu_G\varepsilon_G)} + \frac{\gamma_G\omega_G A_G}{(k_2k_4-\gamma_G\omega_G\varepsilon_G)}\left(1-\frac{1}{\mathcal{R}_0^G}\right).\end{array}}^{\text{Endemic Equilibrium of DoM}} & \overbrace{\begin{array}{l}S_H = \frac{k_8 A_H}{\mathcal{R}_0^H(k_5k_8-\nu_H\varepsilon_H)},\\ E_G = \frac{k_8 A_H}{(k_6k_8-\gamma_H\omega_H\varepsilon_H)}\left(1-\frac{1}{\mathcal{R}_0^H}\right),\\ I_H = \frac{k_8(1-\omega_H)\gamma_H A_H}{k_7(k_6k_8-\gamma_H\omega_H\varepsilon_H)}\left(1-\frac{1}{\mathcal{R}_0^H}\right),\\ R_H = \frac{\nu_H A_H}{\mathcal{R}_0^H(k_5k_8-\nu_H\varepsilon_H)} + \frac{\gamma_H\omega_H A_H}{(k_6k_8-\gamma_H\omega_H\varepsilon_H)}\left(1-\frac{1}{\mathcal{R}_0^H}\right).\end{array}}^{\text{Endemic Equilibrium of HoM}} \end{array}\right.,$$

The succeeding result is thus shown.

Lemma 2. *The rabies-persistent equilibrium points of the two sub-models exist only when the basic reproduction numbers are greater than one. That is, the equilibrium points of the sub-models are characterized as follows:*

$$\textit{Critical points of sub-models} = \begin{cases} \textit{Rabies-free equilibrium, if} & \mathcal{R}_0^G,\ \mathcal{R}_0^H \leq 1, \\ \textit{Endemic equilibrium, if} & \mathcal{R}_0^G,\ \mathcal{R}_0^H > 1. \end{cases}$$

3.2.3 Global stability of rabies-free equilibrium points

We define the following Lyapunov functions for the sub-models

$$\overbrace{\mathcal{L}^G = \left((1-\omega_G)\,\gamma_G + k_3\right) E_G + k_2 I_G}^{\text{Lyapunov function of the Dog-only sub-model}} \qquad \overbrace{\mathcal{L}^H = \left((1-\omega_H)\,\gamma_H + k_7\right) E_H + k_6 I_H}^{\text{Lyapunov function of the Human-only sub-model}}$$

Then, the time-derivatives of the Lyapunov functions are given by

$$\begin{aligned} \overbrace{\frac{\mathrm{d}\mathcal{L}^G}{\mathrm{d}t}}^{\text{Lyapunov function of the Dog-only sub-model}} &= \left((1-\omega_G)\,\gamma_G + k_3\right)\frac{\mathrm{d}E_G}{\mathrm{d}t} + k_2\frac{\mathrm{d}I_G}{\mathrm{d}t}, \\ &= k_2k_3\left[\tfrac{\beta_{GG}((1-\omega_G)\gamma_G+k_3)(1-\theta_G)S_G}{k_2k_3} - 1\right](E_G+I_G), \\ &\leq k_2k_3\left(\tfrac{\mathcal{R}_0^G N_G}{S_G^0} - 1\right)(E_G+I_G), \\ &\leq k_2k_3\left(\tfrac{\mathcal{R}_0^G A_G}{n_G S_G^0} - 1\right)(E_G+I_G), \end{aligned} \qquad \begin{aligned} \overbrace{\frac{\mathrm{d}\mathcal{L}^H}{\mathrm{d}t}}^{\text{Lyapunov function of the Human-only sub-model}} &= \left((1-\omega_H)\,\gamma_H + k_7\right)\frac{\mathrm{d}E_H}{\mathrm{d}t} + k_6\frac{\mathrm{d}I_H}{\mathrm{d}t}, \\ &= k_6k_7\left[\tfrac{\beta_{HH}((1-\omega_H)\gamma_H+k_7)(1-\theta_H)S_H}{k_6k_7} - 1\right](E_H+I_H), \\ &\leq k_6k_7\left(\tfrac{\mathcal{R}_0^H N_H}{S_H^0} - 1\right)(E_H+I_H), \\ &\leq k_6k_7\left(\tfrac{\mathcal{R}_0^H A_H}{n_H S_H^0} - 1\right)(E_H+I_H). \end{aligned}$$

The following result therefore easily follows.

Theorem 3. *The rabies-free equilibrium points of the sub-models are globally asymptotically stable when the following respective conditions hold*

$$\underbrace{\mathcal{R}_0^G \leq \frac{k_4 n_G}{(k_1k_4 - \nu_G\varepsilon_G)}}_{\textbf{\textit{Dog-only sub-model}}}, \quad \textit{and} \quad \underbrace{\mathcal{R}_0^H \leq \frac{k_8 n_H}{(k_5k_8 - \nu_H\varepsilon_H)}}_{\textbf{\textit{Human-only sub-model}}}.$$

3.3 Qualitative analysis of the full rabies model

In this section, we present some qualitative properties of the full mode (1), which we restate as follows.

$$\left.\begin{aligned} \frac{\mathrm{d}S_G}{\mathrm{d}t} &= A_G - \lambda_{GG}S_G + \varepsilon_G R_G - k_1 S_G, \\ \frac{\mathrm{d}E_G}{\mathrm{d}t} &= \lambda_{GG}S_G - k_2 E_G, \\ \frac{\mathrm{d}I_G}{\mathrm{d}t} &= (1-\omega_G)\gamma_G E_G - k_3 I_G, \\ \frac{\mathrm{d}R_G}{\mathrm{d}t} &= \nu_G S_G + \gamma_G\omega_G E_G - k_4 R_G, \\ \frac{\mathrm{d}S_H}{\mathrm{d}t} &= A_H - \lambda_H S_H + \varepsilon_H R_H - k_5 S_H, \\ \frac{\mathrm{d}E_H}{\mathrm{d}t} &= \lambda_H S_H - k_6 E_H, \\ \frac{\mathrm{d}I_H}{\mathrm{d}t} &= (1-\omega_H)\gamma_H E_H - k_7 I_H, \\ \frac{\mathrm{d}R_H}{\mathrm{d}t} &= \nu_H S_H + \omega_H\gamma_H E_H - k_8 R_H, \\ &\text{with initial conditions being non-negative,} \end{aligned}\right\} \tag{4}$$

where $\lambda_{GG} = (1-\theta_G)\beta_{GG}\left(E_G + I_G\right)$ and $\lambda_H = (1-\theta_H)\left[\beta_{HH}(E_H + I_H) + \beta_{GH}\left(E_G + I_G\right)\right]$.

3.3.1 Basic reproduction number for the full model

The full model (4) has a rabies-free equilibrium point given by

$$\mathcal{E}^0 = \left(\frac{k_4 A_G}{(k_1k_4 - \nu_G\varepsilon_G)}, 0, 0, \frac{\nu_G A_G}{(k_1k_4 - \nu_G\varepsilon_G)}, \frac{k_8 A_H}{(k_5k_8 - \nu_H\varepsilon_H)}, 0, 0, \frac{\nu_H A_H}{(k_5k_8 - \nu_H\varepsilon_H)}\right).$$

With the aid of the Next-generation matrix method [6], we obtain the transmission and transition matrices as follows:

$$\mathcal{F} = \begin{bmatrix} (1-\theta_G)\,\beta_{GG}S_G^0 & (1-\theta_G)\,\beta_{GG}S_G^0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ S_H^0\,(1-\theta_H)\,\beta_{GH} & S_H^0\,(1-\theta_H)\,\beta_{GH} & (1-\theta_H)\,\beta_{HH}S_H^0 & (1-\theta_H)\,\beta_{HH}S_H^0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

and

$$\mathcal{V} = \begin{bmatrix} k_2 & 0 & 0 & 0 \\ -(1-\omega_G)\,\gamma_G & k_3 & 0 & 0 \\ 0 & 0 & k_6 & 0 \\ 0 & 0 & -(1-\omega_H)\,\gamma_H & k_7 \end{bmatrix},$$

so that the next-generation matrix $\mathcal{F}\mathcal{V}^{-1}$ is given by

$$\mathcal{F}\mathcal{V}^{-1} = \begin{bmatrix} \mathcal{R}_G^0 & \frac{(1-\theta_G)\beta_{GG}S_G^0}{k_3} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ \frac{S_H^0(1-\theta_H)\beta_{GH}\mathcal{R}_G^0}{(1-\theta_G)\beta_{GG}S_G^0} & \frac{S_H^0(1-\theta_H)\beta_{GH}}{k_3} & \mathcal{R}_H^0 & \frac{(1-\theta_H)\beta_{HH}S_H^0}{k_7} \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$

The basic reproduction which is defined as a spectral radius when utilizing the next-generation matrix is given by

$$\mathcal{R}_0 = \max\left\{\mathcal{R}_G^0,\ \mathcal{R}_H^0\right\}.$$

The following result is established.

Theorem 4. *The rabies-free equilibrium point $\mathcal{E}_0$ is locally asymptotically stable whenever $\mathcal{R}_0 < 1$ and unstable otherwise.*

Proof.

The Jacobian of the model evaluated at $\mathcal{E}_0$ is given by

$$\mathcal{J} = \begin{bmatrix} \mathcal{J}_1 & \mathcal{J}_2 \end{bmatrix}.$$

where

$$\mathcal{J}_1 = \begin{bmatrix} -k_1 & -(1-\theta_G)\beta_{GG}S_G^0 & -(1-\theta_G)\beta_{GG}S_G^0 & \varepsilon_G \\ 0 & (1-\theta_G)\beta_{GG}S_G^0 - k_2 & (1-\theta_G)\beta_{GG}S_G^0 & 0 \\ 0 & (1-\omega_G)\gamma_G & -k_3 & 0 \\ \nu_G & \gamma_G\omega_G & 0 & -k_4 \\ 0 & -S_H^0(1-\theta_H)\beta_{GH} & -S_H^0(1-\theta_H)\beta_{GH} & 0 \\ 0 & S_H^0(1-\theta_H)\beta_{GH} & S_H^0(1-\theta_H)\beta_{GH} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

$$\mathcal{J}_2 = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ -k_5 & -(1-\theta_H)\beta_{HH}S_H^0 & -(1-\theta_H)\beta_{HH}S_H^0 & \varepsilon_H \\ 0 & (1-\theta_H)\beta_{HH}S_H^0 - k_6 & (1-\theta_H)\beta_{HH}S_H^0 & 0 \\ 0 & (1-\omega_H)\gamma_H & -k_7 & 0 \\ \nu_H & \gamma_H\omega_H & 0 & -k_8 \end{bmatrix}.$$

whose characteristic polynomial can be written as follows:

$$\Phi_1(X)\Phi_2(X)\Phi_3(X)\Phi_4(X) = 0 \tag{5}$$

where

$$\begin{aligned} \Phi_1(X) &= X^2 + (k_5+k_8)X + k_5k_8 - \nu_H\varepsilon_H, \\ \Phi_2(X) &= X^2 + (k_1+k_4)X + k_1k_4 - \nu_G\varepsilon_G, \\ \Phi_3(X) &= X^2 + \left[k_6 + k_7 - (1-\theta_H)\beta_{HH}S_H^0\right]X + k_6k_7\left(1-\mathcal{R}_0^H\right), \\ \Phi_4(X) &= X^2 + \left[k_2 + k_3 - (1-\theta_G)\beta_{GG}S_G^0\right]X + k_2k_3\left(1-\mathcal{R}_0^G\right). \end{aligned}$$

Clearly, four of the eigenvalues of $\mathcal{J}$ are negative since the two roots of each of $\Phi_1(X)$ and $\Phi_2(X)$ are negative. The remaining eigenvalues of $\mathcal{J}$ are the roots of $\Phi_3(X)$ and $\Phi_4(X)$. It is easy to show that, all coefficients of $\Phi_3(X)$ and $\Phi_4(X)$ are positive if $\mathcal{R}_0 \leq 1$. This guarantees that all zeros of $\Phi_3(X)$ and $\Phi_4(X)$ and consequently the eigenvalues of $\mathcal{J}$ will be negative. This completes the proof of the theorem. □

3.3.2 Rabies-persistent equilibrium point of full model

$$\left.\begin{aligned}
S_G^{**} &= \tfrac{k_4 A_G}{\mathcal{R}_0^G(k_1k_4-\nu_G\varepsilon_G)},\\
E_G^{**} &= \tfrac{k_4 A_G}{(k_2k_4-\gamma_G\omega_G\varepsilon_G)}\left(1-\tfrac{1}{\mathcal{R}_0^G}\right),\\
I_G^{**} &= \tfrac{k_4(1-\omega_G)\gamma_G A_G}{k_3(k_2k_4-\gamma_G\omega_G\varepsilon_G)}\left(1-\tfrac{1}{\mathcal{R}_0^G}\right),\\
R_G^{**} &= \tfrac{\nu_G A_G}{\mathcal{R}_0^G(k_1k_4-\nu_G\varepsilon_G)}+\tfrac{\gamma_G\omega_G A_G}{(k_2k_4-\gamma_G\omega_G\varepsilon_G)}\left(1-\tfrac{1}{\mathcal{R}_0^G}\right),\\
S_H^{**} &= \tfrac{k_8 A_H}{\left(k_5k_8-\varepsilon_H\nu_H+k_8\lambda_H^*\right)}+\tfrac{\varepsilon_H\omega_H\gamma_H E_H^{**}}{\left(k_5k_8-\varepsilon_H\nu_H+k_8\lambda_H^*\right)},\\
E_H^{**} &= \tfrac{k_8 A_H\lambda_H^*}{\left[k_5k_6k_8-\varepsilon_H\nu_Hk_6+(k_6k_8-\varepsilon_H\omega_H\gamma_H)\lambda_H^*\right]},\\
I_H &= \tfrac{(1-\omega_H)\gamma_H}{k_7}E_H^{**}\\
R_H &= \tfrac{\nu_v A_H}{\left(k_5k_8-\varepsilon_H\nu_H+k_8\lambda_H^*\right)}+\omega_H\gamma_H\left(\tfrac{k_5+\lambda_H^*}{\left(k_5k_8-\varepsilon_H\nu_H+k_8\lambda_H^*\right)}\right)E_H^{**}
\end{aligned}\right\}$$

where λ_H^* satisfies

$$(\lambda_H^*)^2+\left\{k_6\left(k_5k_8-\varepsilon_H\nu_H\right)\left(\tfrac{1}{(k_6k_8-\varepsilon_H\omega_H\gamma_H)}-\mathcal{R}_0^H\right)-\tfrac{\beta_{GH}(1-\theta_H)k_2(k_1k_4-\nu_G\varepsilon_G)}{\beta_{GG}(1-\theta_G)(k_2k_4-\gamma_G\omega_G\varepsilon_G)}\left(\mathcal{R}_0^G-1\right)\right\}\lambda_H^*$$
$$-\tfrac{\beta_{GH}(1-\theta_H)k_2k_6(k_1k_4-\nu_G\varepsilon_G)(k_5k_8-\varepsilon_H\nu_H)}{\beta_{GG}(1-\theta_G)(k_2k_4-\gamma_G\omega_G\varepsilon_G)(k_6k_8-\varepsilon_H\omega_H\gamma_H)}\left(\mathcal{R}_0^G-1\right)=0.$$

The following results follows from applying Routh-Hurwitz criterion of the above polynomial equation.

Theorem 5.

1. *Whenever $\mathcal{R}_0^G>1$, then a unique rabies-endemic equilibrium point exists.*
2. *Whenever $\mathcal{R}_0^G<1$, then either of the following holds:*
   (a) *a unique rabies-endemic equilibrium point exists when*
   $$k_6\left(k_5k_8-\varepsilon_H\nu_H\right)\left(\frac{1}{(k_6k_8-\varepsilon_H\omega_H\gamma_H)}-\mathcal{R}_0^H\right)-\frac{\beta_{GH}\left(1-\theta_H\right)k_2\left(k_1k_4-\nu_G\varepsilon_G\right)}{\beta_{GG}\left(1-\theta_G\right)\left(k_2k_4-\gamma_G\omega_G\varepsilon_G\right)}\left(\mathcal{R}_0^G-1\right)<0;$$
   (b) *There is no rabies-endemic equilibrium point otherwise.*

The stability of the DFE of the full model is determined by obtaining the eigenvalues of $\mathcal{J}(\mathcal{E}_0)$. If the entire eigenvalues constitute only negative real parts, then the $\mathcal{E}_0$ will be locally asymptotically stable and unstable otherwise.

Theorem 6. *(GAS of $\mathcal{E}_0$).*
The condition $\mathcal{R}_0\leq 1$ does not guarantee global asymptotic stability of the rabies -free equilibrium point $\mathcal{E}_0$ of the full model (4).

Proof. The theorem of Castillo-Chavez et al. [4] is employed to examine this theorem. To do this, let $X=(S_G,\ R_G,\ S_H,\ R_H)$ and $Z=(E_G,I_G,E_H,I_H)$ so that, the full-model (1) can be demonstrated differently as

$$\left.\begin{aligned}\frac{\mathrm{d}X}{\mathrm{d}t}&=F(X,Z)\\ \frac{\mathrm{d}Z}{\mathrm{d}t}&=G(X,Z).\end{aligned}\right\}\tag{6}$$

where $F(X,Z) = \begin{pmatrix} \frac{\mathrm{d}S_G}{\mathrm{d}t} \\ \frac{\mathrm{d}R_G}{\mathrm{d}t} \\ \frac{\mathrm{d}S_H}{\mathrm{d}t} \\ \frac{\mathrm{d}R_H}{\mathrm{d}t}. \end{pmatrix}$ and $G(X,Z) = \begin{pmatrix} \frac{\mathrm{d}E_G}{\mathrm{d}t} \\ \frac{\mathrm{d}I_G}{\mathrm{d}t} \\ \frac{\mathrm{d}E_H}{\mathrm{d}t} \\ \frac{\mathrm{d}I_H}{\mathrm{d}t} \end{pmatrix}$.

The reduced non-infected system given by

$$\left.\frac{\mathrm{d}X}{\mathrm{d}t}\right|_{Z=0} = \begin{pmatrix} A_G + \varepsilon_G R_G - k_1 S_G \\ \nu_G S_G - k_4 R_G \\ A_H + \varepsilon_H R_H - k_5 S_H \\ \nu_H S_H - k_8 R_H \end{pmatrix}. \tag{7}$$

Clearly, $X^0 = \left(\frac{k_4 A_G}{(k_1 k_4 - \nu_G \varepsilon_G)}, \frac{\nu_G A_G}{(k_1 k_4 - \nu_G \varepsilon_G)}, \frac{k_8 A_H}{(k_5 k_8 - \nu_H \varepsilon_H)}, \frac{\nu_H A_H}{(k_5 k_8 - \nu_H \varepsilon_H)}\right)$ is a globally asymptotically stable equilibrium point of (7).

Now, let

$$\mathcal{L} = \begin{bmatrix} (1-\theta_G)\beta_{GG}S_G^0 - k_2 & (1-\theta_G)\beta_{GG}S_G^0 & 0 & 0 \\ (1-\omega_G)\gamma_G & -k_3 & 0 & 0 \\ S_H^0(1-\theta_H)\beta_{GH} & S_H^0(1-\theta_H)\beta_{GH} & (1-\theta_H)\beta_{HH}S_H^0 - k_6 & (1-\theta_H)\beta_{HH}S_H^0 \\ 0 & 0 & (1-\omega_H)\gamma_H & -k_7 \end{bmatrix}.$$

Then G(X,Z) can be written differently as $G(X,Z) = \mathcal{L}Z - \hat{G}(X,Z)$, where

$$\hat{G}(X,Z) = \begin{pmatrix} (1-\theta_G)\beta_{GG}\left(S_G - S_G^0\right)(E_G + I_G) \\ 0 \\ (1-\theta_H)\left(S_H - S_H^0\right)\left[\beta_{HH}(E_H + I_H) + \beta_{GH}(E_G + I_G)\right] \\ 0 \end{pmatrix}.$$

Now, the second condition (**H2**) of Castillo-Chavez et al. [5] is satisfied if and only if $S_G \geq S_G^0$ and $S_H \geq S_H^0$. Therefore, the global asymptotic stability of $\mathcal{E}_0$ is not guaranteed when $\mathcal{R}_0 \leq 1$ unless the initial populations are sufficiently large. This concludes the proof. □

3.3.3 Sensitivity analysis

Sensitivity analysis is conducted to help determine the impact of different epidemiological factors on the possible spread of disease. Since rabies eradication or persistence is connected with the basic reproduction number, we conduct the sensitivity analysis on the basic reproduction numbers of the basic sub-models. The normalized forward sensitivity index of a quantity ζ which differentiably depends on a parameter ι is given by

$$\Upsilon_\zeta^\iota = \frac{\partial \zeta}{\partial \iota} \times \frac{\iota}{\zeta}$$

The values of the parameters in Table 2 are utilized to calculate the sensitivity indexes of the two basic reproduction numbers and presented in Table 1. From Table 1, it can be seen that recruitment rates (A_G

Table 1: Sensitivity Indexes of Basic Reproduction Numbers of Sub-Models

$\mathcal{R}_0^H$		$\mathcal{R}_0^H$	
Par	Sensitivity Index	**Par**	Sensitivity Index
A_G	1	A_H	1
β_{GG}	1	β_{HH}	1
C_G	- 0.5739795918	ε_H	0.0003329027404
ε_G	0.008135079132	n_H	- 0.05968695692
n_G	- 0.3161938417	μ_H	- 0.2248438953
μ_G	- 0.1739420842	θ_H	- 2.160063760
θ_G	- 1.143308298	ω_H	- 0.02535239834
ω_G	- 0.04835589942	γ_H	- 0.7293159674
γ_G	- 0.1254539533	ν_H	- 0.0003353662206
ν_G	- 0.008590643562		

and A_H), contact rates (β_{GG} and β_{HH}) have the highest impact in increasing the spread of rabies as they increase the basic reproduction numbers. Also, effectiveness of campaigns towards reducing contact of rabies virus sources (θ_G and θ_H) has the highest impact in reducing the spread of rabies.

4 Numerical Simulations and Discussions

With the aid of *ode45* in MATLAB, the numerical simulation of the model equation was conducted using the parameter values in Table 2. Some important simulation results are presented in Figure 1 to Figure 3.

Table 2: Description and Values of Parameters of Rabies Model (1) .

Par.	Description	Baseline Value /year	Source
A_G	Birth rate of dogs	3×10^6	[20]
A_H	Recruitment rate of humans	0.0314	[2]
β_{GG}	Transmision flow in dogs	1.58×10^{-7}	[20]
β_{GH}	dog-humans transmission rate	2.29×10^{-12}	[20]
β_{HH}	human-human transmission rate	3.58×10^{-9}	assumed
C_G	Mortality of dogs assigned to culling	0.3	[2]
ε_G	Loss of immunity in dogs	1.0	[20]
ε_H	Loss of immunity in humans	1.0	[20]
n_G	Mortality of dogs	0.056	[20]
n_H	Natural death rate in humans	0.0074	[20]
μ_G	Disease induced mortality in dogs	1.0	[20]
μ_H	Disease induced mortality in humans	1.0	[2]
θ_G	Pre-exposure prophylaxis for dogs	0.25	[2]
θ_H	Pre-exposure prophylaxis for humans	0.54	[20]
ω_G	Post-exposure prophylaxis for dogs	0.2	[20]
ω_H	Post-exposure prophylaxis for humans	0.1	[20]
γ_G	Incubation rate in dogs	$\frac{1}{6}$	[20]
γ_H	Incubation rate in humans	$\frac{1}{6}$	[20]

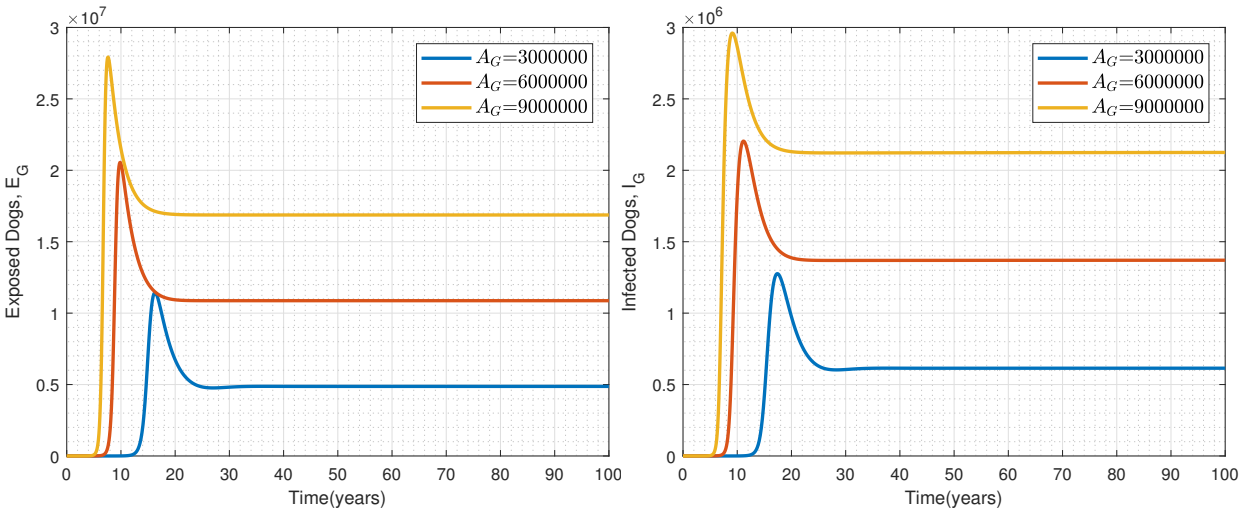

Figure 1: Impact of varying A_G on model variables.

In Figure 1, as the parameter values of the recruitment rate A_G, is decreased, the exposed and infected dogs are also decreased. This shows that, as A_G is decreased, rabies will totally be eliminated in the dog

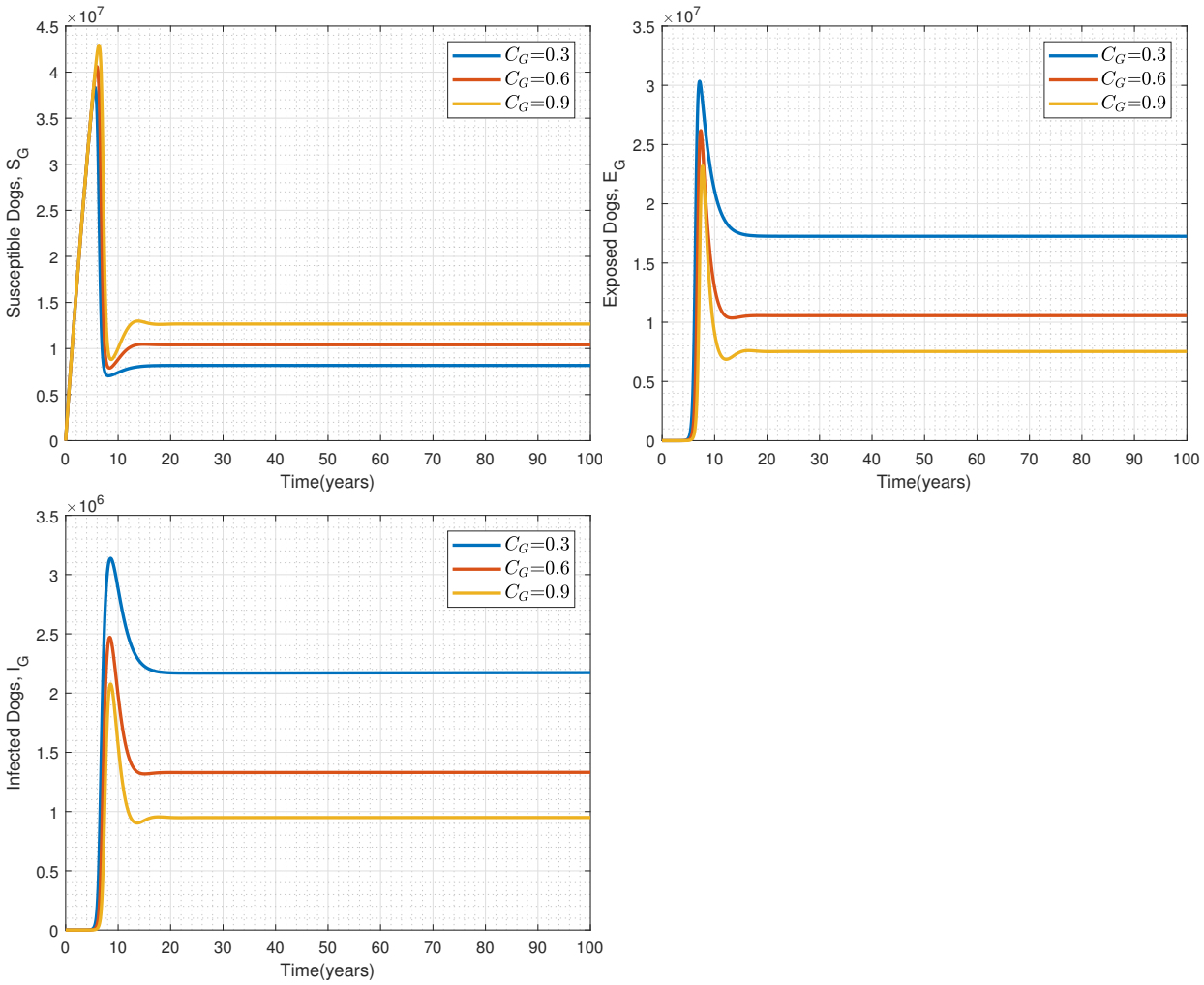

Figure 2: Impact of varying C_G on model variables.

compartment. If this happens, rabies will be eradicated in both dogs and humans, since dogs are the main agent that transmits rabies virus to humans. The results in Figure 2 demonstrate that rabies can be eradicated through culling C_G, of exposed and infected dogs. As the parameter values of the culling rate C_G, is increased, the total number of susceptible dogs increases, the total number of exposed and infected dogs decreases as well. This shows that rabies can be eliminated by increasing the culling rate C_G, of exposed and infected dogs. Figure 3, shows that, the eradication of rabies can completely be accomplished through mass vaccination and education on the various pathways in which rabies can be acquired. As the parameter values of vaccination θ_G, increases, the total number of susceptible and removed dogs increases and the total number of exposed and infected dogs decreases. This shows that, rabies can be eliminated totally through vaccination of dogs and education on how the rabies virus spread.

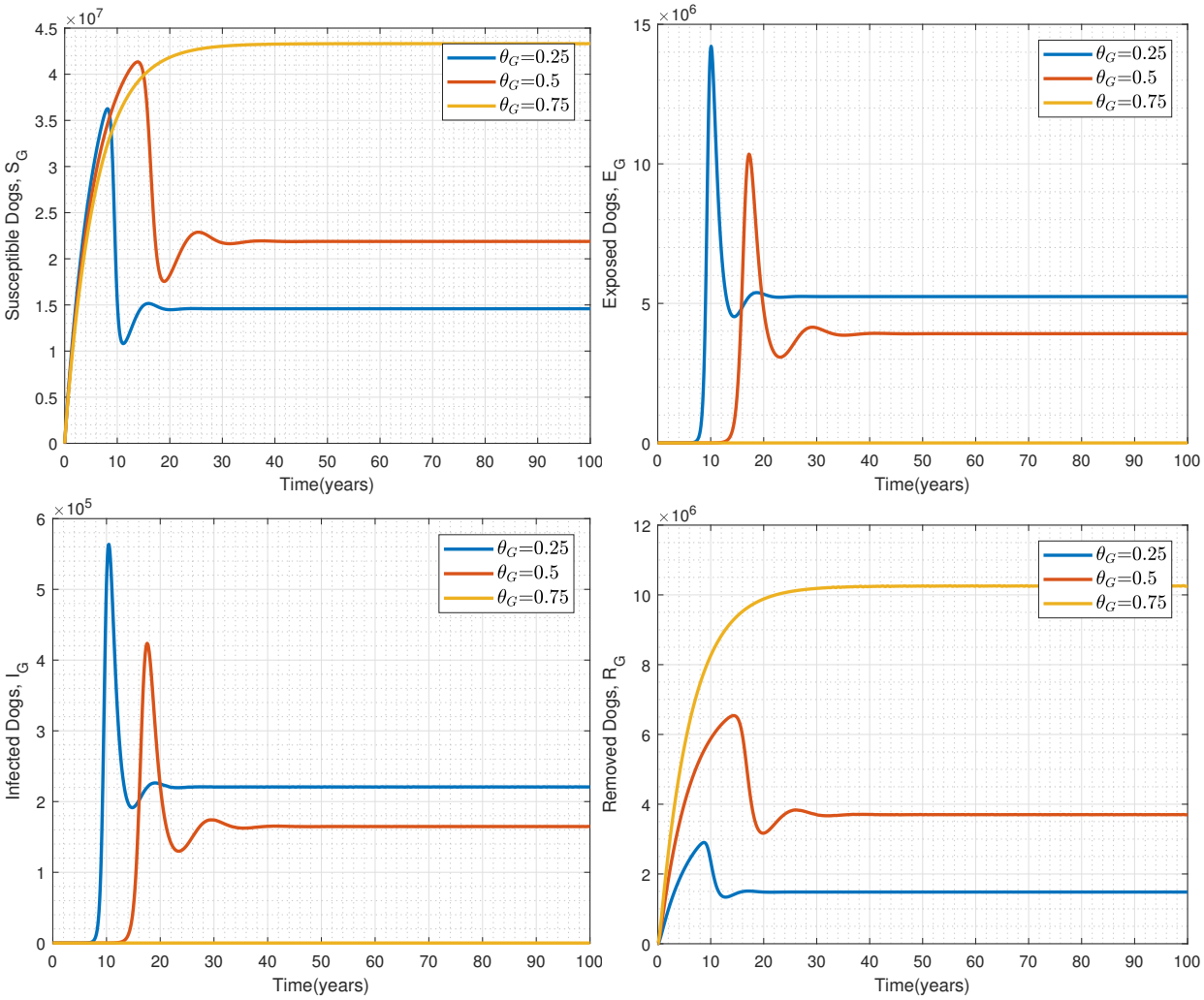

Figure 3: Impact of varying θ_G on model variables.

5 Conclusions

In this paper, a deterministic ordinary differential equations model was proposed to study the dynamics of rabies in an interacting populations of dogs and humans. The impact of human-to-human transmission of rabies, which has not been studied in previous mathematical models was considered. The model was split into two sub-models, namely; the dog-only model and the human-only model. Some qualitative properties of the sub-models and the full model were presented. The sub-models are shown to be globally asymptotically stable whenever their associated basic reproduction numbers are below some thresholds. The full model is shown to exhibit a a unique rabies-endemic equilibrium whenever the basic reproduction is above unity. Also, when the basic reproduction number is below unity, rabies-endemic equilibrium may exist under some conditions. It is observed that the basic reproduction being below unity does not guarantee global stability of the rabies-free equilibrium point. Numerical simulations revealed that contact rates, culling rate, recruitment rates, efficacy of educational campaigns against the spread of rabies are key factors that

should be targeted to control the spread of the disease.

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