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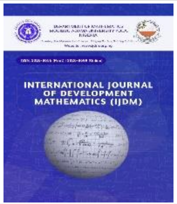

Dynamics of Rabies Transmission Model in Human and Dog Populations with Time Delay

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ABSTRACT

Rabies is a fatal zoonotic disease caused by a virus through bites or saliva of an infected animal. Dogs are the main reservoir of Rabies and are responsible for most Human cases worldwide. In this research, a delay differential equations model for assessing the effects of controls and time delay as an incubation period on the transmission dynamics of Rabies in human and dog populations is formulated and analyzed. Basic properties of the model as par theories of delay differential equations are established, and the model is well-posed mathematically and biologically. Analysis of the model shows that there is a locally and globally asymptotically stable rabies-free equilibrium whenever the epidemiological threshold (the control reproduction number,) is less than unity. Furthermore, the model has a unique endemic equilibrium point when the control reproduction number, exceeds unity. Time delay is found to have an influence on the endemicity of rabies. Treatment and Vaccination of humans and dogs, coupled with an annual crop of puppies, are imposed to curtail the spread of Rabies in the populations. Numerical experiments are conducted to illustrate the theoretical results and control strategies.

1. Introduction

Rabies is a zoonotic viral disease that is usually transmitted by an infected animal (such as foxes, raccoons, cats, coyotes, bats, skunks and dogs) through bites or scratches that introduces the virus into the blood of another animal or a human (World Health Organization, 2016). Once the virus enters the body, it either migrates directly to the brain via the peripheral nervous system to replicate there or stays in the muscles to replicate before migrating to the brain via the neuromuscular junctions. The infection becomes fatal upon reaching the brain since it produces acute inflammations of the brain, leading to coma and eventually death (Rupprecht, *et al*., 2010). The Rabiesvirus is mainly contained in the saliva of animals but may also be found in tears, urine, semen and other body fluids (Alan and Jackson, 2008). The virus infects the central nervous system and causes cerebral dysfunction in the brain, and once clinical symptoms develop, its mortality rate is certain. Moreover, Rabies has the highest case fatality rate of any conventional infectious disease, approaching the 100% mark (Chen, *et al*., 2015).

Dogs are the primary sources of Rabies in humans, and the main transmission route is through bites or scratches, especially by infected dogs (Jibat *et al*., 2017). Rabies can also be transmitted through direct contact with wound or mucosal surface (e.g. mouth, nose, and eye) that is contaminated with the saliva from a rabid dog (Chapwanya *et al*., 2016). Humans and other mammals can develop Rabies from aerosol transmission or through transplantation of tissues and organs (Alan and Jackson, 2008; Tenzin, 2012). At the early stage of infection, initial symptoms of Rabies resemble flu-like, which includes fever with pain and unusual or unexplained tingling, hypersalivation, sore throat, cough, nausea and vomiting, pricking, or burning sensation (paranesthesia) at the site of an animal bite (Stanley, 2000; WHO, 2019). After that, as the virus spreads to the central nervous system, progressive and fatal inflammation of the brain and spinal cord developed, resulting in hyperactivity and paralysis (Stanley, 2000).

Most cases of Rabies results from the bite of an infected dog. The consequence of exposure to RABV depends on several factors, including the severity of the wound, the location of the bite on the body, the quantity and variant (genotype) of virus inoculated into the wound(s), and the timeliness of post-exposure prophylaxis (PEP). Without PEP, the average probability of developing Rabies following a bite by a rabid animal to the head is 55%, the upper extremity 22%, the trunk 9% and the lower limb 12%.9 Virus load in the saliva of RABV-infected dogs

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varies during the disease and influences the risk of infection for human bite victims. RABV infection in rodents is very uncommon. No human Rabies cases from rodents bites have been reported (WHO, 2018).
RABV can be found in saliva, tears, urine and nervous tissues of human Rabies cases and, exposure to these body fluids and tissues carries a theoretical risk of transmission. RABV is not found in blood. Human-to-human transmission of RABV is extremely rare, and this very low risk should not hinder the care of patients. The only documented cases of human-to-human transmission occurred via tissue and organ transplants from 2 RABV-infected individuals and a single case of likely perinatal RABV transmission. Based on pathobiology and epidemiology, transmission by breastfeeding is unlikely to occur, although there is a paucity of evidence (WHO, 2018).

The incubation period of Rabies in humans is usually about 1 to 3 months after exposure but can range from less than 7 days to over one year (Johnson *et al*., 2010). The longest incubation period of 27 years was reported in the Philippines (Dimaano, *et al*., 2011). However, in Rabies-endemic areas, there is always a possibility of recurrent unrecognized natural infection. The incubation period depends on various factors including location and severity of bite wounds, the amount of virus inoculated into five the bite wound, the degree of innervations at the bite site, and host factors. The bites on the face, neck, and hand (nearer to the brain) have a higher risk and shorter incubation than bites on the extremities (Jackson, 2009). The incubation period of Rabies in dogs is 3-8 weeks on average but may vary from 10 days to as long as 6 months, but is rarely more than four months. In general, rabid animals of all species commonly exhibit typical signs of central nervous system disturbances with behavioral changes. A rabid dog may show either the furious or the paralytic (dumb) form of rabies. The major clinical signs in dogs are aggression, abnormal barking, biting unusual things like sticks and stones, roaming, laryngeal paralysis, excessive salivation, tremors, ataxia, and generalized seizures (Niezgoda *et al*., 2002).

Although Rabies in humans almost inevitably ends in death, six survivors of Rabies have been documented between 1970 and 2004 in the USA (Willoughby *et al*., 2005), Argentina (Porras *et al*., 1976), Mexico (Alvarez *et al*., 1994), and India (Madhusudana *et al*., 2002). Five of the six patients had received post-exposure Rabies vaccine series before the onset of clinical symptoms. All showed nervous signs but recovered gradually with some neurological disorder. (Warrell and Warrell, 2004). The sixth patient who survived Rabies was reported in 2004 in Wisconsin, USA (Willoughby *et al*., 2005). In recent years (between 2008 and 2009) three more survival cases of Rabies have been reported following bat bites in Brazil, Colombia and the USA (CDC, 2010). It should be noted that the four survivor cases using the Milwaukee protocol (since 2004) involved Rabies virus strain of bat origin, while none survived with canine Rabies virus strains, indicating that canine strains are more virulent than bat strains (Rubin, *et al*., 2009).

Most cases occur in Africa and Asia, with approximately 40% of cases in children aged 15. All mammals are susceptible to infection by the Rabies virus (RABV). Transmission of RABV by dogs is responsible for up to 99% of human Rabies cases in rabies-endemic regions, with a small proportion due to transmission via wildlife (such as foxes, wolves, jackals, bats, racoons, skunks or mongoose) (WHO 2018). After Asia, Africa is the second most affected continent, with an estimated 23,000 human Rabies deaths reported every year (Knobel, *et al*., 2005). Rabies is endemic throughout the African continent – Northern, Southern, Eastern, Western and Central Africa (Harrak, 2011; Nel, 2011; Hayman *et al*., 2011). The cosmopolitan Rabies lineage is thought to have been introduced into sub-Saharan Africa from North Africa and Eurasia following patterns of human colonization (Talbi *et al*., 2009). Rabies deaths in Africa are linked to poverty, lack of awareness among people and medical practitioners, and lack of infrastructure for the management of Rabies exposure (Harrak, 2011). However, studies by Lembo *et al*., (2010) have confirmed that there are no insurmountable problems with canine Rabies control in most of Africa, and Rabies elimination is epidemiologically and practically feasible through mass dog Vaccination.

Mass Vaccination campaigns targeting dogs constitute the principal strategy for Rabies control by interrupting RABV transmission between dogs and reducing transmission to humans and other mammals. This strategy has been effective in different settings in Africa, Asia, Europe and the Americas. As dog-mediated Rabies incidence declines as a result of effective control programs, Rabies from other sources, although rare, becomes more prominent, as currently noted in the Americas. Wild carnivore species and bats (Carnivora and Chiroptera) represent a higher risk for RABV transmission than other wildlife, as they are reservoirs for RABV. Human-to-human transmission of Rabies has never been confirmed, except extremely rarely as a result of infected tissue and organ transplantation (Rupprecht *et al*., 2016; WHO 2018). Chapwanya *et al*. (2016) formulated yet another SEI Rabies model in human and dog populations with an additional vaccinated compartment in humans. They proposed the discrete counterpart using the nonstandard finite difference scheme. Wang and Lou (2008) developed an ODE model to study the impact of combinations of culling and Vaccination on the control of Rabies transmission. They observed that Vaccination alone was a better strategy, whilst culling was worst in the control of Rabies transmission.

Asamoah *et al*. (2017) also suggested that Vaccination of pets and use of pre-exposure and post-exposure prophylaxis could help control Rabies spread. In China and some parts of Africa (for example, the Upper East and West regions of Ghana), dog meat is a delicacy. It is noteworthy that while so much research has been carried out on the transmission and control of rabies, the impact of predation of dogs by humans has not been studied in the transmission of rabies. Smith and Cheeseman (2002) show that culling could be more effective than Vaccination, given the same efficacy of control. Zinsstag *et al*. (2009) further extended the existing models on Rabies transmission between dogs to include dog-to-human transmission and concluded that human post-exposure prophylaxis (PEP) with a dog Vaccination campaign was more cost-effective in controlling the disease in the long run.

Furthermore, Ding *et al*. (2007) formulated an epidemic model for Rabies in raccoons with discrete time and spatial features. Their goal was to analyze the strategies for optimal distribution of vaccine baits to minimize the spread of the disease and the cost of carrying out the control. Zhang *et al*. (2011) also presented a mathematical model of Rabies transmission in dogs and from the dog population to the human population in China. Their study did not consider the use of optimal control analysis to the study of the Rabies virus in dogs and from the dog population to the human population. The introduction of time delay in mathematical modelling has shown significant impacts on the dynamics of the system and disease burden. Research has demonstrated the existence of a time delay between infections and infectiousness Levin *et al*. (2012). Time delay can cause the equilibria of models to change from stable to unstable or conditionally stable, thereby generating periodic solutions with delay as a bifurcating parameter (Greenhalgh, 2010).

2. Materials and Methods

2.1 Model formulation

The model consists of two populations: humans and dogs living in the same environment. At any time t, the human population is divided into six sub-populations of susceptible humans $S_h(t)$, infected humans $I_h(t)$, vaccinated humans $V_h(t)$, treated humans $T_h(t)$, and recovered humans $R_h(t)$. Hence, the total population of humans denoted by $N_h(t)$ is given by

$$N_h(t) = S_h(t) + I_h(t) + V_h(t) + T_h(t) + R_h(t)$$

Similarly, at any time t, the dog population is subdivided into three sub-populations of susceptible dogs $S_d(t)$, infected dogs $I_d(t)$, and vaccinated dogs $V_d(t)$ so, the total population of dogs is denoted by $N_d(t)$ is given by

$$N_d(t) = S_d(t) + I_d(t) + V_d(t)$$

Recruitment into the susceptible human population $S_h(t)$ is done by an increase in human annual birth at rate Λ_h, non-effectiveness (failure) of vaccination in humans at a rate d_h, and the population of humans who recovered from RABV after loss of immunity at a rate δ_h. This population is decreased by administering Vaccination at a rate υ_h, natural death μ_h, and sufficient contact between susceptible humans and infected dogs at a rate $\beta_{dh} S_h I_d e^{-\mu_d \tau}$, where $\tau > 0$ is the time lag (delay) between infection and infected stage, $e^{-\mu_d \tau}$ is the probability that a rabid dog survived the natural death over the period $[0, \tau]$, and β_{dh} is the transmission rate of RABV from dogs to humans.

The infected human population $I_h(t)$ is increased by sufficient contact between susceptible humans and infected dogs at a rate $\beta_{dh}S_hI_de^{-\mu_d\tau}$, and decreased by natural deaths, deaths due to RABV, and treatment at a rate μ_h, m_h and α_h respectively.

The vaccinated human population $V_h(t)$ is increased by vaccine administered to susceptible humans at a rate υ_h, it is decreased by natural death and non-effectiveness (failure) of Vaccination in humans at a rate μ_h and d_h, respectively.

The treated human population $T_h(t)$ is increased by treatment at a rate α_h, and decreased by natural death and rate at which treated humans recover from RABV at a rate μ_h and ρ_h respectively. The Recovered human population $R_h(t)$ is increased by the recovery rate due to treatment at a rate ρ_h. The class is further decreased by natural death and rate at which recovered humans from RABV revert to susceptible at a rate μ_h and δ_h respectively.

Similarly, susceptible dog population $S_d(t)$ is increased by dog annual birth rate Λ_d, failure of Vaccination in dogs at a rate c_d, the population is decreased by administering Vaccination in dogs, natural death, and sufficient contact between susceptible and infected dogs at a rate κ_d, μ_d and $\beta_{dd}S_dI_de^{-\mu_d\tau}$ respectively.

The infected dog population $I_d(t)$ is increased by infection after sufficient contact between susceptible dogs and infected dogs at a rate $\beta_{dd}S_dI_de^{-\mu_d\tau}$, and is decreased by the rate at which the infected dogs die naturally and death due to RABV at a rate μ_d and m_d respectively. The vaccinated dog population $V_d(t)$ is increased by vaccine administered to susceptible dogs at a rate κ_d, it is decreased by natural death and non-effectiveness (failure) of Vaccination in humans at a rate μ_d and c_d respectively.

The biological assumptions of the model are as follows:

i. The dogs mix homogenously.

ii. All infected dogs die because dogs showing symptoms of sickness are clubbed to death, and their meat are used as meal.

iii. Age, sex and type of the dog, coupled with the climatic conditions, do not affect the probability of a dog being infected.

iv. There are failures in Vaccinations in human and dog populations so that vaccinated populations revert to susceptible.

Therefore, based on the above description and assumptions, the basic RABV Model leads to the following system of non-linear delay differential equations, the schematic diagram Figure 1 below, and the state variables and parameters indicated in the diagram are explained in Table 1.

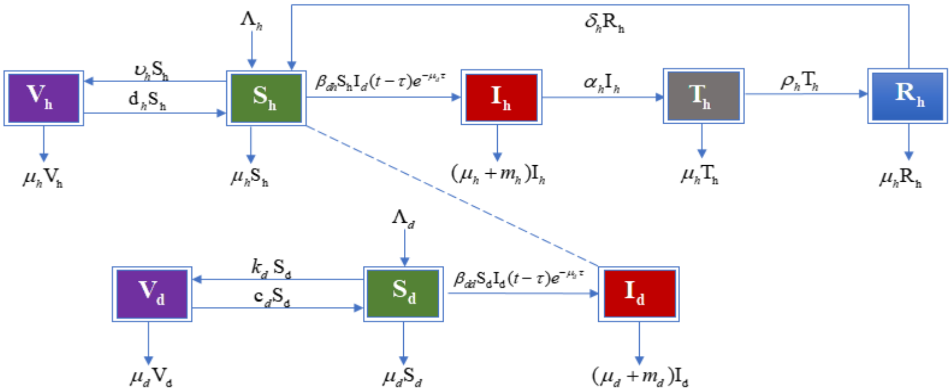


Figure 1: Flow diagram for the Rabiesmodel

Table 1: Description of the State Variables and Parameters of the System (1)

State Var/Par	**Description**
S_h	Susceptible Human Population at time t
I_h	Infected Human Population at time t
V_h	Vaccinated Human Population at time t
T_h	Treated Human Population at time t
R_h	Recovered Human Population at time t
S_d	Susceptible Dog Population at time t
I_d	Infected Dog Population at time t
V_d	Vaccinated Dog Population at time t
$\Lambda_h,\ \Lambda_d$	Human annual birth, Dog annual crop of newborn puppies
$\mu_h,\ \mu_d$	Natural death rates of Humans, Natural death rates of Dogs
$m_h,\ m_d$	Human death rate due to RABV, Dogs death rate due to RABV
$\upsilon_h,\ \kappa_d$	Human Vaccination rate, Dog Vaccination rate
β_{dh}, β_{dd}	Dog-human transmission rate, Dog-Dog transmission rate
$d_h,\ c_d$	Vaccine failure rate in Humans, Vaccination failure rate in Dogs
τ	Time lag between latency and infected stage
α_h	Treatment rate
ρ_h	Recovery rate
δ_h	Rate at which recovered humans becomes susceptible

The following system of non-linear delay differential equations gives the model:

$$
\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \beta_{dh} S_h I_d\left(t-\tau\right) e^{-\mu_d \tau} - \left(\mu_h + \upsilon_h\right) S_h + d_h V_h + \delta_h R_h \\
\frac{dI_h}{dt} &= \beta_{dh} S_h I_d\left(t-\tau\right) e^{-\mu_d \tau} - \left(\mu_h + m_h + \alpha_h\right) I_h \\
\frac{dV_h}{dt} &= \upsilon_h S_h - \left(d_h + \mu_h\right) V_h \\
\frac{dT_h}{dt} &= \alpha_h I_h - \left(\mu_h + \rho_h\right) T_h \\
\frac{dR_h}{dt} &= \rho_h T_h - \left(\mu_h + \delta_h\right) R_h \\
\frac{dS_d}{dt} &= \Lambda_d - \beta_{dd} S_d I_d\left(t-\tau\right) e^{-\mu_d \tau} - \left(\mu_d + \kappa_d\right) S_d + c_d V_d \\
\frac{dI_d}{dt} &= \beta_{dd} S_d I_d\left(t-\tau\right) e^{-\mu_d \tau} - \left(\mu_d + m_d\right) I_d \\
\frac{dV_d}{dt} &= \kappa_d S_d - \left(c_d + \mu_d\right) V_d
\end{aligned}
\tag{1}
$$

with initial data,

$$S_h, I_h, V_h, T_h, R_h, S_d, I_d, V_d = \Psi_1, \Psi_2, \Psi_3, \Psi_4, \Psi_5, \Psi_6, \Psi_7, \Psi_8 \text{ for } t \in [-\tau, 0], \qquad (2)$$

where:

$\left(\Psi_1, \Psi_2, \Psi_3, \Psi_4, \Psi_5, \Psi_6, \Psi_7, \Psi_8\right) \in \mathbb{C}^+ : \mathbb{C}^+\left([-\tau, 0], \mathbb{R}_+^8\right)$

is the space of continuous function on the interval $[-\tau, 0]$, equipped with maximum norm, for $t \in [-\tau, 0]$.

2.2 Basic Properties of the Model

The basic dynamical features of the model equations (1) will now be explored. For the model to be epidemiologically meaningful, it is important to prove that all variables are non-negative for all time, and a bounded solution exists. The model equations (1) is divided into two regions: $\Omega_h = [S_h, I_h, V_h, T_h, R_h] \in \mathbb{R}_+^5$ and $\Omega_d = [S_d, I_d, V_d] \in \mathbb{R}_+^3$, thus

$\Omega = \Omega_h \times \Omega_d \in \mathbb{R}_+^8$

Theorem 1: The solution $\left(S_h, I_h, V_h, T_h, R_h, S_d, I_d, V_d\right)$ of model equations (1) is positive for all time $t > 0$ and bounded in Ω, given the initial data in (2).

Proof: Suppose all the initial data in (2) are positive. Consider the first Equation in (1) with $\Lambda_h > 0$, we have

$\frac{dS_h}{dt} = \left[\beta_{dh} I_d (t-\tau) e^{-\mu_d \tau} - (\mu_h + \upsilon_h)\right] S_h \geq 0,$ so that

$$S_h(t) \geq S_h(0) \exp\left(-\int_0^t \left[\beta_{dh} I_d (u-\tau) e^{-\mu_d \tau} - (\mu_h + \upsilon_h)\right] du\right) \qquad (3)$$

Hence, $S_h(t) > 0$. Similarly, from the sixth Eq. in (1), with $\Lambda_d > 0$, we have

$\frac{dS_d}{dt} = \left[\beta_{dd} I_d (t-\tau) e^{-\mu_d \tau} - (\mu_d + \kappa_d)\right] S_d \geq 0$ so that

$$S_d(t) \geq S_d(0) \exp\left(-\int_0^t \left[\beta_{dd} I_d (u-\tau) e^{-\mu_d \tau} - (\mu_d + \kappa_d)\right] du\right) \qquad (4)$$

Hence, $S_d(t) > 0$. Likewise, it is provable that $V_h(t) > 0,\ T_h(t) > 0,\ R_h(t) > 0,\ V_d(t) > 0\ \forall t > 0$.

For the other variables $(I_h(t) \text{ and } I_d(t))$, we used the method of contradiction as used in Fan *et al* (2010) (noting that $I_h(\theta) > 0 \text{ and } I_d(\theta) > 0$ for any $\theta \in [-\tau, 0]$). Suppose there exists a time $t_1 > 0$ such that $I_h(t_1) = 0$, $I_d(t_1) = 0$ and $S_h(t) > 0$, $I_h(t) > 0$, $V_h(t) > 0$, $S_d(t) > 0$, $I_d(t) > 0$, $V_d(t) > 0$ for all $t \in [0, t_1)$ and $\frac{dI_h(t_1)}{dt} \leq 0$, $\frac{dI_d(t_1)}{dt} \leq 0$ it follows from the second equation in (1)

$$\frac{dI_h(t_1)}{dt} = \beta_{dh} S_h(t_1) I_d(t_1 - \tau) e^{-\mu_d \tau} > 0 \qquad (5)$$

a contradiction. Hence $I_h(t) > 0$ for any finite time $t \geq 0$. Similarly, from the seventh equation in (1)

$$\frac{dI_d(t_1)}{dt} = \beta_{dd} S_d(t_1) I_d(t_1 - \tau) e^{-\mu_d \tau} > 0 \qquad (6)$$

Which also contradicts our earlier assumption. Hence, $I_d(t) > 0$ for any finite time $t \geq 0$. This proves the first part of Theorem 1.

To show the boundedness of solution, from the model equation in (1), adding the human sub-populations at time t, we have

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - m_h I_h$$
$$\le \Lambda_h - \mu_h N_h \tag{7}$$

Using the standard comparison theorem (Lakshmikantham *et al*., 1989), we can have that

$$N_h(t) \le N_h(0)e^{-\mu_h t} - \frac{\Lambda_h}{\mu_h}\left(e^{-\mu_h t} - 1\right) \tag{8}$$

as $t \to \infty$, the value of $N_h \to \frac{\Lambda_h}{\mu_h}$. This shows that N_h is bounded above by $\frac{\Lambda_h}{\mu_h}$ as the value of t goes to infinity. Therefore, $N_h = S_h + I_h + V_h + T_h + R_h \ge 0$. That is $N_h(t) \ge 0$, which means $0 \le N_h$. Therefore

$$0 \le N_h(t) \le \frac{\Lambda_h}{\mu_h}. \tag{9}$$

Therefore, the feasible solution for the human population in the model (1) is in the region

$$\Omega_h = \left[\left(S_h, I_h, V_h, T_h, R_h\right) \in \mathbb{R}_+^5 : 0 \le N_h(t) \le \frac{\Lambda_h}{\mu_h}\right]. \tag{10}$$

Similarly, from the model equation in (1), adding dog sub-populations at time t, we have

$$\frac{dN_h}{dt} = \Lambda_d - \mu_d N_d - m_d I_d \tag{11}$$
$$\le \Lambda_d - \mu_d N_d$$

Using the standard comparison theorem (Lakshmikantham *et al*., 1989), we can show that

$$N_d(t) \le N_d(0)e^{-\mu_d t} - \frac{\Lambda_d}{\mu_d}\left(e^{-\mu_d t} - 1\right) \tag{12}$$

as $t \to \infty$, the value of $N_d \to \frac{\Lambda_d}{\mu_d}$. This shows that N_d is bounded above by $\frac{\Lambda_d}{\mu_d}$ as the value of t goes to infinity. Therefore, $N_d = S_d + I_d + V_d \ge 0$. That is $N_d(t) \ge 0$, which means $0 \le N_d$. Therefore

$$0 \le N_d(t) \le \frac{\Lambda_d}{\mu_d}. \tag{13}$$

Therefore, the feasible solution for the human population in the model (1) is in the region

$$\Omega_d = \left[\left(S_d, I_d, V_d\right) \in \mathbb{R}_+^3 : 0 \le N_d(t) \le \frac{\Lambda_d}{\mu_d}\right]. \tag{14}$$

Thus,

$$\Omega = \Omega_h \times \Omega_d = \left[\left(S_h(t), I_h(t), V_h(t), T_h(t), R_h(t)\right) \le \frac{\Lambda_h}{\mu_h}; \left(S_d(t), I_d(t), V_d(t)\right) \le \frac{\Lambda_d}{\mu_d}\right] \tag{15}$$

Therefore, the model equations in (1) are mathematically well-posed and epidemiologically meaningful.

3 Model Analysis

In this section, we shall examine the equilibrium solutions, obtain the basic reproduction number, conduct the stability analysis of the RABV free equilibrium (RFE), RABV endemic equilibrium (REE), the local and global stability of the RABV free equilibrium.

At equilibrium, we set $I_d(t) = I_d(t-\tau) = I_d^*,\ S_h(t) = S_h^*,\ I_h(t) = I_h^*,\ V_h(t) = V_h^*,\ T_h(t) = T_h^*,\ R_h(t) = R_h^*,$ $S_d(t) = S_d^*,\ V_d(t) = V_d^*$ and set the right-hand side of the system (1) to zero. Thus,

$$\begin{aligned}
\Lambda_h-\beta_{dh}S_h^*I_d^*e^{-\mu_d\tau}-\left(\mu_h+\upsilon_h\right)S_h^*+d_hV_h^*+\delta_hR_h^*&=0\\
\beta_{dh}S_h^*I_d^*e^{-\mu_d\tau}-\left(\mu_h+m_h+\alpha_h\right)I_h^*&=0\\
\upsilon_hS_h^*-\left(d_h+\mu_h\right)V_h^*&=0\\
\alpha_hI_h^*-\left(\mu_h+\rho_h\right)T_h^*&=0\\
\rho_hT_h^*-\left(\mu_h+\delta_h\right)R_h^*&=0\\
\Lambda_d-\beta_{dd}S_d^*I_d^*e^{-\mu_d\tau}-\left(\mu_d+\kappa_d\right)S_d^*+c_dV_d^*&=0\\
\beta_{dd}S_d^*I_d^*e^{-\mu_d\tau}-\left(\mu_d+m_d\right)I_d^*&=0\\
\kappa_dS_d^*-\left(c_d+\mu_d\right)V_d^*&=0
\end{aligned}\tag{16}$$

3.1 Rabies Virus-Free Equilibrium

This is achieved if there is no RABV infection in the community, which means that the number of infected dogs infected humans, treated humans and recovered humans are zero, that is ($I_d^*=I_h^*=T_h^*=R_h^*=0$). The RABV free equilibrium of model equations (1) is obtained and is given by (17)

$$\begin{aligned}
E_0^*&\equiv\left(S_h^0,I_h^0,V_h^0,T_h^0,R_h^0,S_d^0,I_d^0,V_d^0\right)\\
&=\left(\frac{\Lambda_h(d_h+\mu_h)}{\mu_h(d_h+\mu_h+\upsilon_h)},0,\frac{\Lambda_h\upsilon_h}{\mu_h(d_h+\mu_h+\upsilon_h)},0,0,\frac{\Lambda_d(c_d+\mu_d)}{\mu_d(c_d+\mu_d+\kappa_d)},0,\frac{\Lambda_d\kappa_d}{\mu_d(c_d+\mu_d+\kappa_d)}\right)
\end{aligned}\tag{17}$$

3.2 Basic Control Reproduction Number

The basic reproduction number denoted by R_c is defined as the expected number of secondary cases produced by introducing one infected in a completely susceptible population. The basic reproduction depends mainly on the definition of the infected and uninfected compartments. We determine R_c using the next-generation matrix approach (Diekmann and Heesterbeek, 2000). The R_c is $\rho\left(FV^{-1}\right)$, where ρ is the spectral radius,

$$F=\begin{bmatrix}0 & \dfrac{\Lambda_h\beta_{dh}(d_h+\mu_h)e^{-\mu_h\tau}}{\mu_h(d_h+\upsilon_h+\mu_h)}\\ 0 & \dfrac{\Lambda_d\beta_{dd}(c_d+\mu_d)e^{-\mu_d\tau}}{\mu_d(c_d+\kappa_d+\mu_d)}\end{bmatrix}\quad\text{and}\quad V=\begin{bmatrix}\mu_h+m_h+\alpha_h & 0\\ 0 & \mu_d+m_d\end{bmatrix}$$

The inverse of V denoted by V^{-1} is given as

$$V^{-1}=\begin{bmatrix}\dfrac{1}{\mu_h+m_h+\alpha_h} & 0\\ 0 & \dfrac{1}{\mu_d+m_d}\end{bmatrix}$$

Thus, the basic control reproduction number for RABV model is given by:

$$R_c=\frac{\Lambda_d\beta_{dd}(c_d+\mu_d)e^{-\mu_d\tau}}{\mu_d(c_d+\kappa_d+\mu_d)(\mu_d+m_d)}\tag{18}$$

3.3 Rabies-Virus Endemic Equilibrium

The computation of the endemic equilibria is very important for any epidemiological model because its provides information about the endemicity and for how long a time it will exist. Therefore, we obtain an expression for the endemic equilibrium of RABV model (1) and denote it by E_0^{**}, and

$E_0^{**} \equiv \left(S_h^{**}, I_h^{**}, V_h^{**}, T_h^{**}, R_h^{**}, S_d^{**}, I_d^{**}, V_d^{**}\right)$

where:

$$\left.\begin{aligned}
S_h^{**} &= \frac{\Lambda_h + \delta_h R_h^{**} + d_h V_h^{**}}{\beta_{dd} I_d^{**} e^{-\mu_d \tau} + (\mu_h + \upsilon_h)} \\
I_h^{**} &= \frac{\beta_{dd} S_h^{**} I_d^{**} e^{-\mu_d \tau}}{\mu_h + m_d + \alpha_h} \\
V_h^{**} &= \frac{\upsilon_h S_h^{**}}{d_h + \mu_h} \\
T_h^{**} &= \frac{\alpha_h I_h^{**}}{\mu_h + \rho_h} \\
R_h^{**} &= \frac{\rho_h T_h^{**} I_h^{**}}{(\mu_h + \rho_h)(\mu_h + \delta_h)} \\
S_d^{**} &= \frac{(\mu_d + m_d) e^{\mu_d \tau}}{\beta_{dd}} \\
I_d^{**} &= \frac{\mu_d (\mu_d + m_d)(c_d + \mu_d + \kappa_d) e^{\mu_d \tau}}{\beta_{dd} (c_d + \mu_d)(\mu_d + m_d)} \left(R_c - 1\right) \\
V_d^{**} &= \frac{\kappa_d (\mu_d + m_d) e^{\mu_d \tau}}{\beta_{dd} (c_d + \mu_d)}
\end{aligned}\right\} \tag{19}$$

Which exists when $R_c > 1$.

3.3 Stability of Rabies Virus-Free Equilibrium

Let $F(t)$ represents the state variables, $\left(S_h(t), I_h(t), V_h(t), T_h(t), R_h(t), S_d(t), I_d(t), V_d(t)\right)^T$, of the model equations (1). Thus, the linearized form of the model equations (1) around the RFE, E_0^* is given by;

$$\frac{dF}{dt} = Q_0 F(t) + Q_1 F(t - \tau) \tag{20}$$

where Q_0 is the Jacobian for $F(t)$, and Q_1 is the Jacobian for $F(t-\tau)$, with

$$Q_0 = \begin{bmatrix}
-p_1 & 0 & d_h & 0 & \delta_h & 0 & 0 & 0 \\
p_2 & -p_3 & 0 & 0 & 0 & 0 & 0 & 0 \\
\upsilon_h & 0 & -p_4 & 0 & 0 & 0 & 0 & 0 \\
0 & \alpha_h & 0 & -p_5 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \rho_h & -p_6 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & -p_7 & 0 & c_d \\
0 & 0 & 0 & 0 & 0 & p_8 & -p_9 & 0 \\
0 & 0 & 0 & 0 & 0 & \kappa_d & 0 & -p_{10}
\end{bmatrix}$$

$$\begin{aligned}&p_1=(\mu_h+\upsilon_h+\beta_{dh}I_d^*e^{-\mu_d\tau}),\quad p_2=\beta_{dh}I_d^*e^{-\mu_d\tau},\quad p_3=(\mu_h+m_h+\alpha_h),\quad p_4=(d_h+\mu_h),\\&p_5=(\mu_h+\rho_h),\quad p_6=(\mu_h+\delta_h),\quad p_7=(\mu_d+\kappa_d+\beta_{dd}I_d^*e^{-\mu_d\tau}),\quad p_8=\beta_{dd}I_d^*e^{-\mu_d\tau},\\&p_9=(\mu_d+m_d),\quad p_{10}=(c_d+\mu_d)\end{aligned}$$

and

$$Q_1=\begin{bmatrix}0&0&0&0&0&0&-\beta_{dh}S_he^{-\mu_d\tau}&0\\0&0&0&0&0&0&\beta_{dh}S_he^{-\mu_d\tau}&0\\0&0&0&0&0&0&0&0\\0&0&0&0&0&0&0&0\\0&0&0&0&0&0&0&0\\0&0&0&0&0&0&-\beta_{dd}S_de^{-\mu_d\tau}&0\\0&0&0&0&0&0&\beta_{dd}S_de^{-\mu_d\tau}&0\\0&0&0&0&0&0&0&0\end{bmatrix}$$

We seek a solution for the system (1) of the form

$$F(t)=e^{\lambda t}\mathbf{z} \tag{21}$$

Where $\mathbf{z}$ is a constant matrix and λ an eigenvalue, substituting equation (21) into (20) and simplifying, we have

$$\left(Q_0+Q_1e^{-\lambda\tau}\right)\mathbf{z}e^{\lambda t}=\mathbf{0} \tag{22}$$

Therefore, for the nonzero solution, the transcendental equation at any equilibrium point is given by

$$B(\lambda)=\left|\lambda I-Q_0-Q_1e^{-\lambda\tau}\right|=0 \tag{23}$$

where I is an 8×8 identity matrix.

3.3.1 Local Stability of Rabies Virus-Free Equilibrium

Theorem 2. If $R_c<1$, the Rabies free equilibrium E_0^* is absolutely stable for all delay $\tau\geq 0$ and unstable if $R_c>1$.

Proof: Substituting the Rabies free equilibrium E_0^* into equation (23), we obtain the transcendental equation as

$$B(\lambda)=(\lambda+t_1)(\lambda+t_2)(\lambda+t_3)(\lambda+t_4)(\lambda+t_5)(\lambda+t_6)(\lambda+t_7)(B_1\lambda)=0 \tag{24}$$

where:

$$B_1(\lambda)=\lambda+\left(\mu_d+m_d\right)-\beta_{dd}S_d^0e^{-\tau(\lambda+\mu_d)} \tag{25}$$

Clearly, equation (24) has seven negative roots

$$\begin{aligned}&\lambda_1=t_1=-(\mu_h+\upsilon_h),\ \lambda_2=t_2=-(\mu_h+m_h+\alpha_h),\ \lambda_3=t_3=-(\mu_h+\rho_h),\ \lambda_4=t_4=-(\mu_h+\delta_h),\\&\lambda_5=t_5=-(\mu_d+\kappa_d),\ \lambda_6=t_6=-(c_d+\mu_d+\kappa_d),\ \lambda_7=t_7=-(d_h+\mu_h+\upsilon_h)\end{aligned}$$

Therefore, the stability of E_0^* can now be obtained by the distribution of roots for $B_1(\lambda)=0$. Now,

$$\begin{aligned}B_1(0)&=(\mu_d+m_d)-\beta_{dd}S_d^0e^{-\mu_d\tau}\\&=(\mu_d+m_d)-\frac{\Lambda_d\beta_{dd}(c_d+\mu_d)e^{-\mu_d\tau}}{\mu_d(c_d+\mu_d+\kappa_d)}\\&=(\mu_d+m_d)\left(1-\frac{\Lambda_d\beta_{dd}(c_d+\mu_d)e^{-\mu_d\tau}}{\mu_d(c_d+\mu_d+\kappa_d)(\mu_d+m_d)}\right)\\&=(\mu_d+m_d)\left(1-R_c\right)\end{aligned}$$

if $R_c > 1$, then $B_1(\lambda) < 0$. and

$$B_1(+\infty) = +\infty + (\mu_d + m_d) - 0$$
$$= +\infty$$

Hence, $B_1(\lambda)$ has at least one positive root. Therefore, E_0^* is unstable. This proves the last part of theorem 2.

When $\tau = 0$.

$$\begin{aligned} B_1(\lambda) \Rightarrow \lambda &= \beta_{dd} S_d^0 - (\mu_d + m_d) \\ &= \frac{\Lambda_d \beta_{dd}(c_d + \mu_d)}{\mu_d(c_d + \mu_d + \kappa_d)} - (\mu_d + m_d) \\ &= \left(R_c - 1\right)\left(\mu_d + m_d\right) e^{\mu_d \tau} \\ &< 0 \end{aligned}$$

if $R_c < 1$. Hence, the root of (25) has a negative real part.

When $\tau > 0$, for a stability switch to necessarily occurs, there must be a root $\lambda = \pm i y_1$, for some $y_1 \in \mathbb{R}^+$. We assume that $\lambda = i y_1$ in equation (25) to get

$$B_1(iy_1) = iy_1 + \left(\mu_d + m_d\right) - \beta_{dd} S_d^0 e^{-\mu_d \tau}\left(\cos y_1 \tau - i \sin y_1 \tau\right) = 0 \qquad (26)$$

Separating real and imaginary parts of (26) and further simplification yields

$$y_1^2 = \left(\beta_{dd} S_d^0 e^{-\mu_d \tau}\right)^2 - \left(\mu_d + m_d\right)^2$$

hence,

$$y_1 = \sqrt{\left(\frac{\Lambda_d \beta_{dd}(c_d + \mu_d) e^{-\mu_d \tau}}{\mu_d(c_d + \mu_d + \kappa_d)} + \left(\mu_d + m_d\right)\right)\left(\mu_d + m_d\right)\left(R_c - 1\right)} \qquad (27)$$

if $R_c < 1$, there is no such $y_1 \in \mathbb{R}^+$, , which shows that equation (25) has no purely imaginary root. This implies that all roots of $B_1(\lambda)$ must have negative real parts (no stability switch). Therefore, the Rabies-free equilibrium E_0^* is absolutely stable when $R_c < 1$ for all delay $\tau \geq 0$.

3.3.2 Global Stability of Rabies Virus-Free Equilibrium

Theorem 3. The Rabies-free equilibrium E_0^* is globally asymptotically stable for delay $\tau \geq 0$ If $R_c < 1$.

Proof: In this part, we shall construct a Lyapunov function to prove the global stability of RVFE. Define a Lyapunov function as follows:

$$L = \mu_d I_d(t) \qquad (28)$$

Let (28) be a Lyapunov function. It is clear that $L > 0$ for all $I_d(t) > 0$ and $L = 0$ only if $I_d(t) = 0$. To show that $\dot{L} < 0$, take the time derivative of equation (28) to get

$$\frac{dL}{dt} = \mu_d \frac{dI_d}{dt} \qquad (29)$$

Substitute the value of $\frac{dI_d}{dt}$ from equation (1), we get

$$
\begin{aligned}
\frac{dL}{dt} &= \mu_d\left[\beta_{dd}S_d I_d(t-\tau)e^{-\mu_d\tau} - \left(\mu_d + m_d\right)I_d(t)\right] \\
&\le \mu_d\left[\beta_{dd}S_d^0 e^{-\mu_d\tau} - \left(\mu_d + m_d\right)\right]I_d\left(t-\tau\right) \\
&\le \mu_d\left(\mu_d + m_d\right)\left[R_c - 1\right]I_d\left(t-\tau\right) \\
&< 0
\end{aligned}
$$

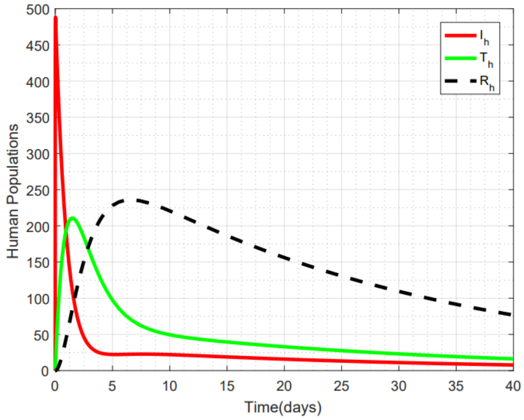
when $R_c < 1$ with equality only at E_0^*. Therefore, L is a Lyapunov function. If $R_c < 1$, there exist the only Rabies free equilibrium E_0^* which is globally asymptotically stable in Ω for all delay $\tau \ge 0$.

4 Numerical Simulations

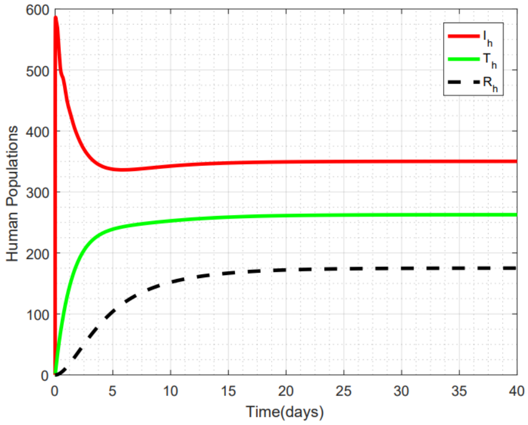
Numerical simulations were carried out using the MATLAB DDE23 function to illustrate the behavior of our system for different values of the model parameters. Some parameters have been obtained from literature,while others have been estimated. The parameter values used for the numerical simulation are shown in Table 2. In addition, $S_h(0) = 70, V_h(0) = 30, I_h(0) = T_h(0) = R_h(0) = 0,\ S_d(0) = 40,\ V_d(0) = 20$ and $I_d(0) = 0$.

Table 2 Parameter values used in the numerical simulations for the model equation (1).

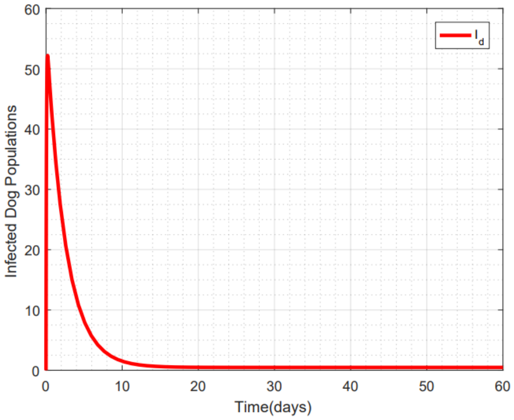
Parameters	Description	Value	Reference
Λ_h	Human annual birth	100	S. Abdulmajid (2021)
Λ_d	Dog annual crop of newborn puppies	20	S. Abdulmajid (2021)
μ_h	Natural death rates of Humans	0.2	Zhang *et al*., (2011)
μ_h	Natural death rates of Dogs	0.2	Zhang *et al*., (2011)
m_h	Human death rate due to RABV	1	Zhang *et al*., (2011)
m_d	Dog death rate due to RABV	1	Zhang *et al*., (2011)
υ_h	Human Vaccination rate	0.4	Asamoah (2017)
κ_d	Dog Vaccination rate	0.2	S. Abdulmajid (2021)
β_{dh}	Dog-Human transmission rate	0.3	Chapwanya (2016)
β_{dd}	Dog-Dog transmission rate	0.5	Chapwanya (2016)
d_h	Vaccine failure rate in Humans	0.004	Assumed
c_d	Vaccination failure rate in Dogs	0.001	Assumed
τ	Time lag between latency and infected stage	$1/6$	Zhang *et al*., (2011)
α_h	Treatment rate	0.6	Zhiting *et al*., (2011)
ρ_h	Recovery rate	0.02	Zhiting *et al*., (2012)
δ_h	Rate at which recovered humans becomes susceptible	0.01	Zhang *et al*., (2012)



(a)



(b)



(c)

Figure (a) shows that when $R_c = 0.5047 < 1$ all trajectories of infective Humans converge to zero, which indicates the local stability for Rabies-free equilibrium E_0^* in human populations. Similarly, from Figure (b), using the values in Table 2 and changing the value of Λ_d to 15, we get $R_c = 7.5711 > 1$. When $R_c > 1$, the RABV-free equilibrium becomes unstable. Also, Figure (c) shows that when $R_c < 1$ all trajectories of infective

Dogs converge to zero, which also indicates the local stability for disease-free equilibrium E_0^* in dog populations.

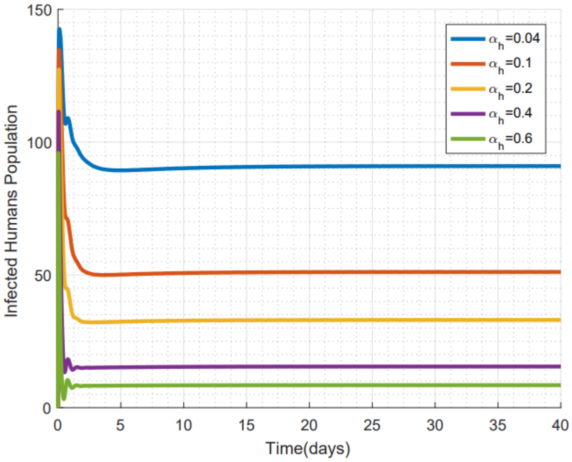


Figure (d): Simulations of the system (1) showing the effect of treatment on infected human population for varying values of α_h.

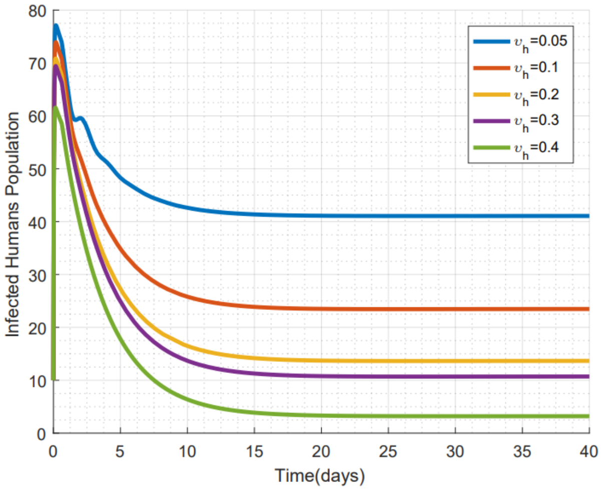


Figure (e): Simulations of the system (1) showing the effect of human Vaccination on infected human population for varying values of υ_h.

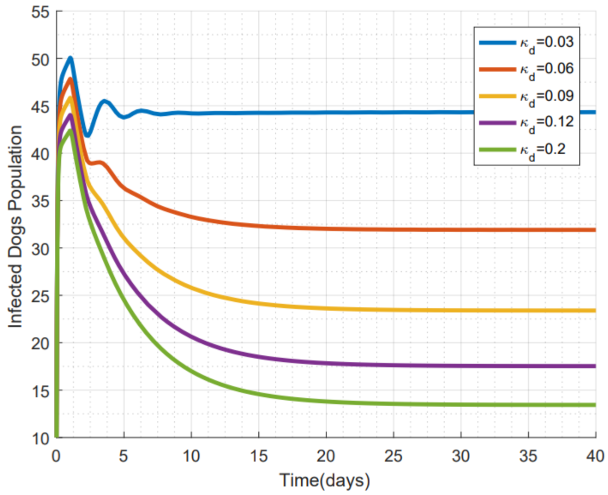


Figure (f): Simulations of the system (1) showing the effect of dog Vaccination on infected dogs population for varying values of κ_d .

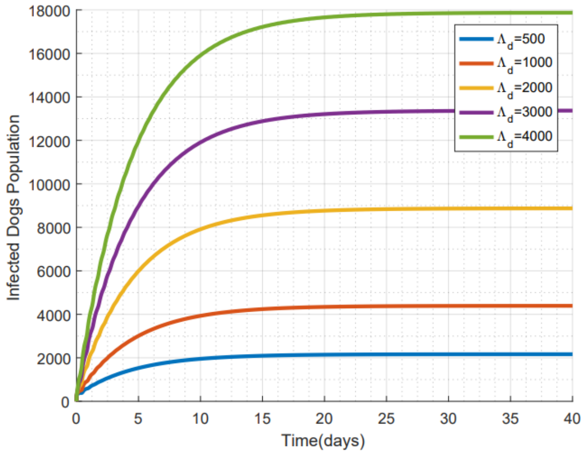


Figure (g): Simulations of the system (1) showing the effect of annual birth of newborn puppies on infected dogs population for varying values of Λ_d in the absence of any control measure.

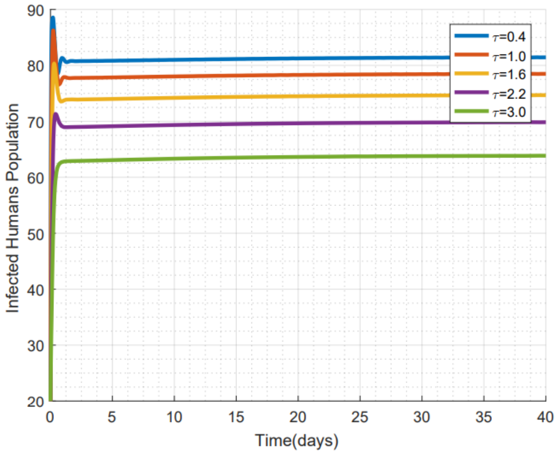


Figure (h): Simulations of the system (1) showing the effect of time delay as an incubation period on infected human population for varying values of τ in the absence of any control measure.

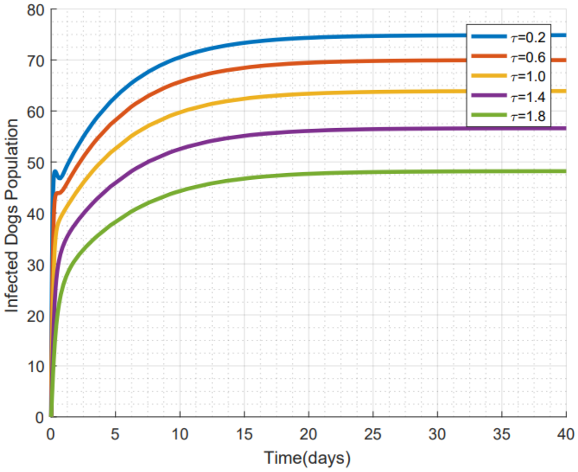


Figure (i): Simulations of the system (1) showing the effect of time delay as an incubation period on an infected dog population for varying values of τ in the absence of any control measure.

To explore the influence of control strategies on the number of infected humans and dogs, we varied the parameters of treatment, Vaccination, and annual dog birth, as shown in Figures (d), (e), (f) and (g). In Figure (d), it can be seen that increasing the rate of treatment of infected humans from $\alpha_h = 0.04$ to $\alpha_h = 0.6$ reduces the number of infected humans with RABV after attaining the highest value of 144 infected humans to about 7 infected humans.

Figure (e) shows that increasing the rate of Vaccination of the humans population from $\upsilon_h = 0.05$ to $\upsilon_h = 0.4$ decreases the rate of infected humans with RABV from 77 to less than 5, it can be further observed that an increase in human vaccine rate reduces the peak number of infected humans. Similarly, from Figure (f), it can be seen clearly that increasing the rate of pet Vaccination from $\kappa_d = 0.03$ to $\kappa_d = 0.2$ reduces the number of infected dogs from 50 infected dogs to less than 15 infected dogs. This shows that a combined Vaccination in human and dog populations will help eradicate the virus in both populations.

In Figure (g), we consider another effective control strategy for curtailing RABV by reducing the birth rate of new puppies annually using immunocontraception, as suggested by Carrol, *et al*., (2010). Thus, as Λ_d is reduced from 4000 to 500, the number of infected dogs reduces from 18000 to about 2000, which shows that with this strategy to eradicate the disease in both populations.

Lastly, we consider the effect of time delay as incubation period in both populations. From Figure (h), as the delay term, τ is increased from 0.4 to 3.0, the number of infected humans decreased. Similarly, from Figure (g), as the time delay term, τ is increased from 0.2 to 1.8 decreases the number of infected dogs with time. This suggests that if the incubation period can be elongated by any possible technique, the endemicity of Rabiescan be reduced.

4 Conclusions

In this research, the dynamic behavior of the Rabies model in co-habiting populations of humans and dogs incorporating three control strategies viz: Vaccination of dogs and humans coupled with annual birth of puppies, treatment and time delay as an incubation period to form a system of delay differential equations was formulated and analyzed. Basic properties of the model as par the theories of delay differential equations are established, and the model is well-posed mathematically and biologically.

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