Environment considerations on the spread of rabies among African wild dogs (Lycaon pictus) with control measures

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Abstract

Wildlife conservation is crucial in preventing endangered species from becoming extinct, and disease outbreaks is one of the biggest threats. Rabies is one of the devastating viruses that can almost be impossible to control once an outbreak occurs. There is no effective treatment or cure of rabies once symptoms show. In this work, we propose euthanasia of infected Lycaon pictus dogs as a control strategy to reduce the spread of the virus. We propose a simple deterministic SEI-type rabies model that takes into account the contaminated environment and the euthanasia of infectious dogs to better understand the transmission and progression dynamics of the disease, as well as to seek to predict the effectiveness of this control strategy. Qualitative analysis and numerical simulations are provided to support our findings.

1 Introduction

There are numerous effective conservation measures that contribute to the on-going preservation and protection of wildlife. Disease outbreaks are one of the biggest threats especially to endangered animals, [1]. In this regard, wildlife conservation is crucial in preventing endangered species from becoming extinct. Among the world's most endangered carnivores is the African wild dog - the Lycaon pictus, [2, 3]. In general, most social animals such as the Lycaon pictus are more vulnerable to diseases such as rabies owing to their close proximity.

The study of animal viruses is of importance and a lot of these viruses result in diseases that are economically devastating. This is particularly important in cases where the wildlife-human-domestic population connections are nearly impossible to ignore. These diseases include the Ebola hemorrhagic virus, West Nile virus encephalitis, rabies, etc. The research has critical contribution to the better understanding of disease progression and transmission dynamics: their replication, evolution, molecular biology as well as interaction with the host. Such research helps in controlling the virus from spreading uncontrollably in case of outbreaks. Some animals can be treated after getting infected but unfortunately there are some diseases with no cure, and may be a danger to all wildlife leading to the extinction of some species.

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Rabies is a severe zoonotic disease often causing a fatal outcome. It is caused by representative of a virus in the genus *Lyssavirus*, family *Rhabdoviridae*. A family of Rhabdoviruses can replicate in vertebrates and invertebrates as well as in vitro in certain cold-blooded tissues. It is a virus that infects the Central Nervous System (CNS), referred to as *Encephalomyelitis*, and consequently, reaches the brain. Ebola virus is also one of the zoonotic viruses originating in wildlife [4], and its emergence is associated with environment, [5]. In [1], the authors proposed the hypothesis that most emerging pathogens originate in wildlife and spillover into human hosts due to several ecological, demographic and socio-economic changes.

Rabies virus is primarily transmitted from a vector through a bite or by saliva touching an open wound of a susceptible animal. Although all mammals are prone to getting the virus infection, only certain domestic and wildlife species are the main reservoirs of rabies. Transmission can also be a result of predation [6]. In continents such as Africa and Asia, dogs (including stray domestic dogs) are the major vectors of the rabies virus and usually come in contact with wild animals such as yellow mongooses [7], foxes, jackals [8], skunks, bats, coyotes [9], wolves [10], etc. Geographic expansion has also been identified as one of the explanations for emergence of rabies in wild dogs in the Serengeti [2]. Several basic reproduction ratios, $\mathcal{R}_0$, values have been reported in the literature, e.g. [1.8,2.1] for hyenas in the Serengeti [11], [1.0,2.0] in domestic dogs from around the word [12], etc. This supports the indemnity of rabies, in what is referred to as the environment in the study.

To prevent, control or respond to outbreak casualties, intervention strategies have to be introduced. These strategies include both control (e.g., vaccination) and treatment of wild animal diseases as well as rehabilitation and rescuing of either infected or susceptible animals: which is often referred to as wildlife medicine. These implemented endeavours are to either help in conserving threatened species or to boost an animals welfare. The use of vaccination as one of the application of wildlife medicine is employed to prevent threatened free-living animals species from getting diseases or viruses resulting to unfavored outcomes such as extinction or ecosystem distractionss, [13]. Different ethical perspectives regarding human interventions for the welfare of wild animals is still a continuous subject matter, [14]. It is evident that without any conservation involvement a multitude of wild animals are prone to contracting a virus or disease, consequently, becoming extinct. According to nature reserves or wildlife preserves, ideally, they would not interfere with the ecosystem. However, if necessary interventions are not executed then there could be a great loss of wildlife. Due to disease outbreaks, wildlife preserves have been involved in projects that develop nanochips that sense or detect infections and any abnormalities in animals' circulatory system. Such technology also helps in deciding whether or not to intervene and what type of intervention strategy is necessary.

There were two reported outbreaks of rabies in the Madikwe Game Reserve (South Africa) resulting in the reduction of a pack of 23 to just 3 [15, 16]. Other reports came from the western boundary of Kruger National Park [17], and in most of those cases it is suspected that domestic dog populations served as reservoirs of rabies outbreaks. The work [18] reports

some of the recent interventions and management mitigations recommendations linked to the most recent outbreak in the Limpopo-Lipadi Private Game Reserve in Botswana.

Once an outbreak occurs, some human intervention measures may potentially be harmful, as a result, the decision to interfere should not be based solely on welfare basis. Generally, there are more or less three modes of conduct: no intervention, vaccination or humanly put to death.

(a) No intervention: If an animal can recover with no treatment necessary.

(b) Vaccination: This strategy is aimed at reducing or eliminating virus shedding.

(c) Euthanasia: If there is no possibility that an animal will recover and/or may be a danger to other surrounding animals.

Several mathematical models for rabies transmission in animals or between humans and animals can be found in the literature, see for example [19–23], to name a few. However, to the best of our knowledge, non of the existing models consider, in the transmission dynamics, the contribution of other rabies hosts, outside the African wild dogs, and the intervention strategy such as euthanasia of infected dogs. The proposed work investigates the dynamics and control of rabies in (wild dogs) Lycaon pictus, and the effects of spillover from domestic stray dogs or other canines. In a study of rabies outbreak in the Madikwe game reserve, the authors in [15] proposed euthanasia as a way of lowering the contacts between infected and susceptible dogs as an alternative to isolating the susceptible wild dogs on the grounds that this could cause unnecessary stress on the animals and interfere with the monitoring process. In this work, we will test this hypothesis by developing a deterministic model of rabies transmission in African wild dogs in a controlled environment such as a game reserve. In particular, we propose the culling of infectious dogs as an intervention to a disease outbreak.

The structure of this work is as follows: we begin in Section 2 where the mathematical model is formulated and we also outline the modeling assumptions. In Section 3 we study the qualitative analysis of the model in terms of the stability/instability of equilibria and bifurcation. A similar analysis is also presented in Section 4 where we ignore the environmental contributions. A sensitivity analysis based on the threshold parameter, the basic reproduction number, is presented in Section 5. In Section 6, we provide a discussion in terms of the proposed control measures. Numerical simulations to support our theoretical results are also provided throughout the work.

2 Mathematical model formulation

To formulate a feasible model, the following key considerations are highlighted. First, in addition to the direct dog-to-dog contacts, we wish to address the contribution of the environment in the transmission process. In the current context, environment refers to other rabies hosts other than the wild dogs (Lycaon pictus), without explicitly taking into account the specific traits of each host. Our motivation is as follows:

(a) Due to human geographic expansion, people are now settling in areas previously reserved for wildlife. In such cases, there are potential contacts between stray dogs and wild animals.

(b) In natural habitats, other than dogs there are several additional rabies hosts capable of transmitting the rabies virus. These include the yellow mongooses, foxes, skunks, bats and coyotes.

Secondly, as a control measure, we propose culling of infectious wild dogs. Clinical symptoms of rabies in wild dogs include swollen head and neck, restlessness, apparent hydrophobia, discoordination, stiff gait, listlessness, diarrhoea and progressive ataxia, [4]. These signs can be used for the targeted intervention. Furthermore, we have the following assumptions:

(a) We assume that reproduction only occurs in the susceptible class. In particular, in the absence of the disease, the population obeys the logistic growth.

(b) We are investigating African wild dogs (Lycaon pictus) in a habitat that might be a nature reserve or wildlife preserve, and assuming that the life span of a dog in its rabid stage is very short.

(c) All infected dogs will die, i.e., no recovery after being infected.

The dog population is divided into three disjoint compartments: the density (dogs per 100 square meters) of dogs susceptible to rabies virus, $s(t)$, the density of exposed dogs (infected but not yet infectious) are placed in the $e(t)$ class, and the density of rabid dogs are in the compartment $i(t)$. Moreover, we include the possibility of dogs getting infected through contaminated environment, where the virus density is denoted by $p(t)$. Some of the hosts contributing to the contaminated environment are generally inaccessible for parenteral vaccination. In the current study, contaminated environment refers to un-owned stray dogs and other wildlife carnivores [24]. The flow diagram of the transmission and progression dynamics of rabies disease among African wild dogs is given below in Fig. 1.

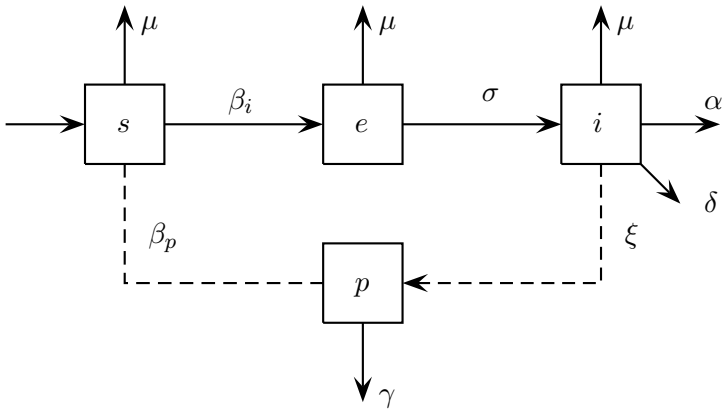


Figure 1: Compartmental diagram describing the interaction between the density of susceptible dogs $s(t)$, the density of exposed dogs $e(t)$, the density of infectious rabid dogs $i(t)$, and the environment $p(t)$.

The corresponding system of differential equations are

$$\begin{aligned}
\frac{ds}{dt} &= rs\,(1 - s/k) - \lambda(i)s - \beta_p sp, \\
\frac{de}{dt} &= \lambda(i)s + \beta_p sp - (\sigma + \mu)e, \\
\frac{di}{dt} &= \sigma e - (\alpha + \delta + \mu)i, \\
\frac{dp}{dt} &= \xi i - \gamma p,
\end{aligned} \tag{1}$$

where

$$\eta(t) = s(t) + e(t) + i(t),$$

is the total population of wild dogs at time $t \geq 0$. The susceptible dogs may acquire the virus through being in contact with infected dogs at the rate β_i, or by contact with infected environment at the rate β_p. In the formulation, $\lambda(i)$ is the force of infection. For example, if $\lambda(i) = \dfrac{i\beta_i}{1 + \nu i}$ we have the Holling type II incidence and $\lambda(i) = i\beta_i$ the bilinear incidence when $\nu = 0$ which is a non-negative saturation factor that measures the effect of rabid wild dogs losing their senses and moving out of the pack. In addition, r is the intrinsic growth rate of susceptible wild dogs, k is the carrying capacity of the dogs, σ is the rate at which infected dogs become infectious, μ is the natural death rate for the dog population, and α is the mortality due to the disease. In this model we assume there is some human intervention, i.e., rabid dogs are removed at a rate δ. We denote the shedding rate of infectious dogs to the environment by ξ and γ to denote the natural decay rate of the rabies virus in the environment. The parameters ξ and γ also takes into account the way infected carcasses are disposed, [4].

A remark regarding the choice of parameters in Table 1 is required. These are the baseline parameters used in the numerical simulations, including the sensitivity analysis in Section 5. Some of them are based on the values reported in the literature, [25, 21, 22, 26, 27], and some of them are estimated based on the available knowledge on the virus. Despite so much literature on rabies in wild dogs, parameter estimation remains a critical issue. The global population of African wild dogs (Lycaon pictus) has significantly declined over the last 2 decades with current estimates of 6600 individuals, including approximately 1400 mature individuals, [27]. In spite of the decline in numbers, wild dogs are currently found in many parts of Africa. The time between births is usually 12 - 14 months with an average of 10 pups and maximum age of 11 years, [26]. Pup mortality is likely to be very high. Based on the literature on jackals [22], we assume the life span of wild dogs to be in the range $2 - 5$ years. In the period 1998 to 2017, the population density in Kruger National Park ranged between 1.5 - 2.0 dogs per 100 km^2, [27]. The average for Serengeti (Kenya) and Hwange (Zimbabwe) is 1.5 adult wild dogs per 100 km^2, [26]. Based on this information, here we take $k = 10$. The reported per capita rate for the average viral incubation period has been reported to be 13 days ($\sigma \approx 28\ year^{-1}$) in Europen foxes [21] and 20 days ($\sigma \approx 18\ year^{-1}$)

in jackals [22]. Without much information for African wild dogs, we estimate the incubation period to be 13 days. The life expectancy of rabid dogs is very short. In [25] they reported average of 5 days for Europen foxes, i.e., $\alpha = 73\ year^{-1}$. The same value was reported by [22] for jackals. In Table 1 we summarise the description of the parameters for model (1).

Clinical symptoms observed in infected wild dogs are similar, with minor variations. In a study by [3], they observed that sick individuals would maintain normal behavior including participating in hunts, but sometimes wander off by themselves but rejoin the group within a few hours. This is reflected in the paper by the choice of the Holling type II contact function. The model does not explicitly follow the behavior of each canid as these are all considered under the *environment*. We are only interested in their contribution to the spread of the virus. Different modelling approaches have been adopted in the literature to describe how the behaviour of infected dogs influence the transmission of the virus. In [20, 21], a spatial model was proposed, where a diffusion term was included in the rabid dogs compartment to simulate the random motion of infected dogs. In the current formulation, we assume this behavioural change can be included by considering a general nonlinear incidence function $\lambda(i)$. Throughout, we assume $\lambda(i)$ is a twice differentiable function satisfying the following conditions:

$$\lambda(i) \geq 0, \quad \lambda'(i) \geq 0, \quad \lambda''(i) \leq 0, \ \text{ for all } \ i \geq 0. \tag{2}$$

In addition, $\lambda(0) = 0$. The above assumptions express the idea that, as the contact rate increases, the number of new infections increases, [28]. We note that the conditions in (2) satisfies the mass action formulation, moreover, we also wish to investigate the effects of behavioural changes in the transmission process. In particular, by the use of Holling type II functional response - which also satisfies the monotonicity conditions in (2).

System (1) is closed by specifying the following initial conditions

$$s(0) = s_0 \geq 0, \ e(0) = e_0 \geq 0, \ i(0) = i_0 \geq 0, \ p(0) = p_0 \geq 0. \tag{3}$$

3 Qualitative analysis

In this section we consider model (1), for a general force of infection function $\lambda(i)$ satisfying the hypotheses outlined in (2). We start with the well-posedness result which we state as follows.

Theorem 1. *Assume $\lambda(i)$ satisfies the conditions in (2). System (1) defines a dynamical system on the biologically feasible region*

$$\Omega = \left\{(s, e, i, p) \in \mathbb{R}^4_+ : s + e + i \leq \eta^\dagger, p \leq p^\dagger\right\}$$

where $\eta^\dagger = \dfrac{k(r+1)}{\omega}$ *and* $p^\dagger = \dfrac{\xi k(r+1)}{\gamma\omega}$ *with* $\omega = \min\{1, \mu\}$.

Table 1: Description and values of the parameters of the model.

Parameter	Baseline Value	Source	Description
μ	0.5	[26]	average per capita death rate, [1/year]
a	1.0	[22]	average per capita birth rate
r	$a-\mu$	calculated	net population growth rate
α	73	[22]	the mortality due to the disease and $1/\alpha$ average life expectancy of a rabid dog
σ	13	[25]	$1/\sigma$ average viral incubation period
δ	10	estimate	culling rate
β_i	79	[25]	contact rate between rabid dog and susceptible dog
β_p	2	estimate	contact rate between susceptible dog and infected environment
ξ	2.5	estimate	shedding rate of infectious dogs
γ	8	estimate	decay rate of virus in the environment
k	10	estimate	wild dog carrying capacity

Proof. We show that for any initial data satisfying (3), the system possesses for all $t \geq 0$, a unique solution which lies in Ω.

First we show that Ω is a positively invariant set. In particular, no trajectories leave Ω by crossing one of its faces. On the contrary, let us assume there exists $t_1 > 0$ such that $s(t_1) = 0$ and $s'(t_1) < 0$ with $s(t) > 0$, $e(t) > 0$, $i(t) > 0$ $p(t) > 0$ for all $t \in (0, t_1)$. The first equation in (1) gives $\dfrac{ds}{dt}(t_1) = 0$, which is a contradiction. Furthermore, $\dfrac{de}{dt}\Big|_{e=0} > 0$, $\dfrac{di}{dt}\Big|_{i=1} > 0$ and $\dfrac{dp}{dt}\Big|_{p=0} > 0$ within Ω, then by Proposition 2.1 of [29], Ω is positively invariant.

In the second step we use the prior estimates below together with the fact that the right hand of the system is a locally Lipschitz function. It follows from the first equation of the system (1) that $s'(t) \leq rs(t)\,(1 - s/k)$, which implies that $\limsup_{t\to\infty} s(t) \leq k$. Then for sufficiently large t, we have

$$\begin{aligned}\frac{d(s(t)+e(t)+i(t))}{dt} &= rs(1-s/k) - \mu e - (\mu+\delta+\alpha)i \\ &= rs(t) - \frac{r}{k}s^2(t) + s(t) - s(t) - \mu e - (\mu+\delta+\alpha)i(t) \\ &\leq (r+1)s(t) - \omega(s(t)+e(t)+i(t)).\end{aligned}$$

Hence, we have $\limsup_{t\to\infty} \eta(t) \leq \dfrac{k(r+1)}{\omega} = \eta^{\dagger}$. The above results show that the solution $(s(t), e(t), i(t))$ is nonnegative and uniformly bounded for any positive initial values. Consequently, $i(t) \leq \eta^{\dagger}$. Substituting this in the fourth equation of system (1) gives

$$\frac{dp}{dt} \leq \xi\eta^{\dagger} - \gamma p.$$

Application of the Gronwall Inequality, [30], yields,

$$\begin{aligned} p(t) &\leq \frac{\xi\eta^{\dagger}}{\gamma}\left(1-e^{-\gamma t}\right)+p_0 e^{-\gamma t} \\ &= \frac{\xi\eta^{\dagger}}{\gamma}+\left(p(0)-\frac{\xi\eta^{\dagger}}{\gamma}\right)e^{-\gamma t}, \end{aligned}$$

from which

$$p(t) \leq p^{\dagger}, \quad \text{whenever} \quad p(0) \leq p^{\dagger}.$$

Combining the above two steps we conclude that (1) defines a dynamical system on the biologically feasible region Ω. □

3.1 Computation of $\mathcal{R}_0$

Setting the right hand side of system (1) to zero, one can verify that $E_{\text{DFE}} = (k, 0, 0, 0)$ is a a disease-free equilibrium of the system. As is the case with compartmental models, we use the next generation matrix approach to find the basic reproduction number to be used in the stability analysis of the equilibria. Here the basic reproduction number, denoted by $\mathcal{R}_0$ gives a number of secondary cases one infectious dog will produce in a population consisting of only susceptible dogs in its infectious stage. The sources of infection are the two compartments, i and p.

We consider the equations where the infection progress in the form

$$\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x),$$

where $x = (e, i, p)^t$, and

$$\mathcal{F}(x) = \begin{pmatrix} s(\lambda(i) + p\beta_p) \\ 0 \\ 0 \end{pmatrix}, \quad \mathcal{V}(x) = \begin{pmatrix} (\sigma+\mu)e \\ (\alpha+\delta+\mu)i - \sigma e \\ \gamma p - \xi i \end{pmatrix}.$$

Then

$$J_{\mathcal{F}}(x) = \begin{pmatrix} 0 & s\lambda'(i) & s\beta_p \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad J_{\mathcal{V}}(x) = \begin{pmatrix} \sigma+\mu & 0 & 0 \\ -\sigma & \alpha+\delta+\mu & 0 \\ 0 & -\xi & \gamma \end{pmatrix},$$

such that

$$F = J_{\mathcal{F}}(E_{\text{DFE}}) = \begin{pmatrix} 0 & k\lambda'(0) & k\beta_p \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V = J_{\mathcal{V}}(E_{\text{DFE}}) = \begin{pmatrix} \sigma+\mu & 0 & 0 \\ -\sigma & \alpha+\delta+\mu & 0 \\ 0 & -\xi & \gamma \end{pmatrix}.$$

The next generation matrix is given by the spectral radius of FV^{-1} from which we deduce

that

$$\begin{aligned}\mathcal{R}_0 &= \frac{\sigma k\lambda'(0)}{(\sigma+\mu)(\mu+\delta+\alpha)} + \frac{\sigma\beta_p k\xi}{\gamma(\sigma+\mu)(\mu+\delta+\alpha)} \\ &= \frac{\sigma k\lambda'(0)}{\phi\psi} + \frac{\sigma\beta_p k\xi}{\gamma\phi\psi},\end{aligned} \tag{4}$$

with $\phi = \sigma+\mu$ and $\psi = \mu+\delta+\alpha$. Notice that we have $\mathcal{R}_0 = \mathcal{R}_{0,d}+\mathcal{R}_{0,p}$ where $\mathcal{R}_{0,d} = \dfrac{\sigma k\lambda'(0)}{\phi\psi}$ is the contribution of the dog-to-dog contacts and $\mathcal{R}_{0,p} = \dfrac{\sigma\beta_p k\xi}{\gamma\phi\psi}$ is the contribution of the dog-environment-dog contacts.

Remark 1. *It is clear that increasing the culling rate δ, increasing the decay rate γ of the rabies virus in the environment as well as decreasing the shedding rate ξ will decrease the value of $\mathcal{R}_0$. Decreasing γ is not easy in general. This will involve the proper disposal of carcasses in an environment full of carnivores.*

Following [31], we have the following result

Theorem 2. *When $\mathcal{R}_0 < 1$, the disease-free-equilibrium E_{DFE}, is locally asymptotically stable, while unstable when $\mathcal{R}_0 > 1$.*

We can improve on the previous result and show that the disease-free-equilibrium is globally asymptotically stable when $\mathcal{R}_0 < 1$. Following the work of [32], we rewrite the system by splitting the compartments into $x = (s)$, vector representing of all compartments of the non-infected or non-transmitting dogs (i.e., susceptible) and the vector $y = (e, i, p)^t$, representing the state of all compartments of infected by the virus, i.e., exposed dogs, infectious dogs and the environment. We denote $x = (x, y)$ the state of the system. That is

$$\begin{aligned}\frac{dx}{dt} &= f(x, y), \\ \frac{dy}{dt} &= g(x, y),\end{aligned}$$

such that $g(x, 0) = 0$. Let us now consider the system

$$\frac{dx}{dt} = f(x, 0) = rs(1 - s/k). \tag{5}$$

This is a well known logistic equation for which $x^* = k$ is a globally asymptotically stable equilibrium. Now consider

$$g(x, y) = \begin{pmatrix} \lambda(i)s + \beta_p sp - \phi e \\ \sigma e - \psi i \\ \xi i - \gamma p \end{pmatrix},$$

such that the Jacobian is

$$J_g(x,y) = \begin{pmatrix} -\phi & \lambda'(i)s & \beta_p s \\ \sigma & -\psi & 0 \\ 0 & \xi & -\gamma \end{pmatrix},$$

and, the Jacobian at disease-free-equilibrium, is

$$J_g(x^*,0) = \begin{pmatrix} -\phi & \lambda'(0)k & \beta_p k \\ \sigma & -\psi & 0 \\ 0 & \xi & -\gamma \end{pmatrix}. \tag{6}$$

Clearly $J_g(x^*,0)$ is an M-matrix and Ω is the feasible region. Then we rewrite $g(x,y)$ is the following way

$$g(x,y) = \begin{pmatrix} -\phi & \lambda'(0)k & \beta_p k \\ \sigma & -\psi & 0 \\ 0 & \xi & -\gamma \end{pmatrix} y - \begin{pmatrix} k\lambda'(0)i - \lambda(i)s + \beta_p p(k-s) \\ 0 \\ 0 \end{pmatrix}. \tag{7}$$

We know $k \geq s$, thus, it remains to show that $k\lambda'(0)i - \lambda(i)s \geq 0$. In fact, we have

$$k\lambda'(0)i - \lambda(i)s = ik\left(\lambda'(0) - \frac{\lambda(i)}{i}\frac{s}{k}\right) \geq ik\left(\lambda'(0) - \lambda'(i)\frac{s}{k}\right) = ik\lambda'(0)\left(1 - \frac{\lambda'(i)}{\lambda'(0)}\frac{s}{k}\right) \geq 0,$$

where we have used the assumption that $\lambda''(i) \leq 0$ from (2), i.e., $\lambda'(i)$ is a monotonically decreasing function, and the inequality $i\lambda'(i) \leq \lambda(i)$, for all i, from [33, Proposition 4.1]. We have the following result:

Theorem 3. *The disease-free equilibrium point E_{DFE} is globally asymptotically stable for $\mathcal{R}_0 < 1$ and unstable for $\mathcal{R}_0 > 1$.*

3.2 Existence of endemic equilibrium

The system always has a disease-free-equilibrium $E_{\text{DFE}} = (k,0,0,0)$ which exists for all parameter values. The other equilibria are solutions of the system,

$$\begin{aligned} &rs(1-s/k) - \lambda(i)s - \beta_p sp = 0, \\ &\lambda(i)s + \beta_p sp - \phi e = 0, \\ &\sigma e - \psi i = 0, \\ &\xi i - \gamma p = 0. \end{aligned} \tag{8}$$

Proposition 1. *Assume $\mathcal{R}_0 > 1$. There exists a unique equilibrium $E_{EE} = (s^*, e^*, i^*) \in \mathbb{R}^3_{>0}$.*

Proof. Recall the properties of $\lambda(i)$ as given in (2) with $\lambda(0) = 0$. If $s \neq 0$, equation $(8)_2$ gives

$$s^* = \frac{\phi e^*}{\lambda(i^*) + \beta_p p^*}.$$

On the other hand, $(8)_3$ and $(8)_4$ give

$$e^* = \frac{\psi}{\sigma} i^*, \quad \text{and} \quad p^* = \frac{\xi}{\gamma} i^*$$

respectively. Hence, using $(8)_1$, the endemic equilibrium is a solution to

$$H(i^*) := r\left(1 - \frac{\phi\psi i^*}{\sigma k(\lambda(i^*) + \beta_p \xi i^*/\gamma)}\right) - (\lambda(i^*) + \beta_p \xi i^*/\gamma) = 0. \tag{9}$$

Differentiating with respect to i^* we have

$$H'(i^*) = -\frac{r\phi\psi}{\sigma k}\left[\frac{\lambda(i^*) - i^*\lambda'(i^*)}{(\lambda(i^*) + \beta_p \xi i^*/\gamma)^2} - (\lambda'(i^*) + \beta_p \xi/\gamma)\right] \geq 0,$$

where we have used the hypotheses (2) and the fact that $\lambda(i^*) - i^*\lambda'(i^*) \geq 0$ as shown in [33, Proposition 4.1]. Clearly $H(i^*)$ is a monotonically decreasing function on $(0, +\infty)$. Furthermore,

$$\lim_{i^* \to 0^+} H(i^*) = r\left(1 - \frac{\phi\psi}{\sigma k(\lambda'(i^*) + \beta_p \xi/\gamma)}\right) = r\left(1 - \frac{1}{\mathcal{R}_0}\right),$$

which is positive for $\mathcal{R}_0 > 1$. Now we need to show that for some large i^*, $H(i^*) < 0$. From (2), there are two cases to consider: first case where $\lambda(i^*)$ is not bounded, and the second case where $\lambda(i^*)$ is bounded.

Assuming $\lambda(i^*)$ is not bounded, then there exists i_1^* such that $\lambda(i_1^*) + \frac{\beta_p \xi}{\gamma} i_1^* = r$, from which we have $H(i^*) < 0$ for all $i^* \geq i_1^*$.

In the second case, we assume $\lambda(i^*)$ is bounded, so that from (2), $\dfrac{i^*}{\lambda(i^*) + \beta_p \xi i^*/\gamma}$ is unbounded on $(0, +\infty)$. In this case, there exists $i_2^* > 0$ such that $\dfrac{i_2^*}{\lambda(i_2^*) + \beta_p \xi i_2^*/\gamma} = \dfrac{\sigma k}{\phi\psi}$ which gives $H(i^*) < 0$ for all $i_2^* \geq i^*$.

For both cases, there is a unique positive equilibrium E_{EE} for $\mathcal{R}_0 > 1$. We also notice that there is no endemic equilibria for $\mathcal{R}_0 < 1$. □

3.3 Stability of endemic equilibrium

To study the local stability of the endemic equilibrium E_{EE}, we assume, for analytical tractebility, that the force of infection function takes the simplest form $\lambda(i) = i\beta_i$. Under this mass action formulation, the conditions in (2) are satisfied. We will proceed via numerical simulations to show that the stability result established here holds for any $\lambda(i)$ satisfying the

monotonicity conditions (2). The Jacobian matrix evaluated at E_{EE} is as follows,

$$J(E_{ee}) = \begin{pmatrix} -r\frac{s^*}{k} & 0 & -\beta_i s^* & -\beta_p s^* \\ \beta_i i^* + \beta_p p^* & -\phi & \beta_i s^* & \beta_p s^* \\ 0 & \sigma & -\psi & 0 \\ 0 & 0 & \xi & -\gamma \end{pmatrix} = \begin{pmatrix} -r\frac{s^*}{k} & 0 & -\beta_i s^* & -\beta_p s^* \\ r\left(1-\frac{s^*}{k}\right) & -\phi & \beta_i s^* & \beta_p s^* \\ 0 & \sigma & -\psi & 0 \\ 0 & 0 & \xi & -\gamma \end{pmatrix}.$$

In terms of the basic reproduction ratio, we have

$$J(E_{\text{EE}}) = \begin{pmatrix} -\frac{r}{\mathcal{R}_0} & 0 & -\frac{\beta_i k}{\mathcal{R}_0} & -\frac{\beta_p k}{\mathcal{R}_0} \\ \frac{r}{\mathcal{R}_0}(\mathcal{R}_0 - 1) & -\phi & \frac{\beta_i k}{\mathcal{R}_0} & \frac{\beta_p k}{\mathcal{R}_0} \\ 0 & \sigma & -\psi & 0 \\ 0 & 0 & \xi & -\gamma \end{pmatrix},$$

where

$$\mathcal{R}_0 = \frac{\sigma k \beta_i}{\phi\psi} + \frac{\sigma \beta_p k \xi}{\gamma \phi \psi}.$$

The first term represents transmission via dog-to-dog contacts, $\mathcal{R}_{0,d}$, and the second term denotes dog-to-environment contacts, $\mathcal{R}_{0,p}$. The trace is clearly negative and the characteristic equation of the Jacobian matrix is

$$\lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4 = 0, \tag{10}$$

where the coefficients of (10) are

$$\begin{aligned} b_1 &= \frac{r}{\mathcal{R}_0} + (\gamma + \phi + \psi), \\ b_2 &= \frac{r}{\mathcal{R}_0}(\gamma + \phi + \psi) + \gamma(\phi + \psi) + \frac{\phi\psi}{\mathcal{R}_0}\mathcal{R}_{0,p}, \\ b_3 &= \frac{r}{\mathcal{R}_0}\gamma(\phi + \psi) + \frac{r\phi\psi}{\mathcal{R}_0^2}\mathcal{R}_{0,p} + \frac{r\sigma\beta_i k}{\mathcal{R}_0^2}(\mathcal{R}_0 - 1), \\ b_4 &= \frac{r\gamma\phi\psi}{\mathcal{R}_0^2}(\mathcal{R}_0 - 1)(\gamma\beta_i + \xi\beta_p). \end{aligned}$$

All the coefficients b_i, are positive for $\mathcal{R}_0 > 1$. Secondly, the Routh-Hurwitz determinant is given by

$$
\begin{aligned}
\Delta_3 &= b_3b_2b_1 - b_3^2 - b_1^2b_4, \\
&= \frac{r_2}{\mathcal{R}_0^4}\left(r\phi\psi(\gamma+\phi+\psi)(\mathcal{R}_{0,p}+\mathcal{R}_{0,d}(\mathcal{R}_0-1)) - \sigma\beta_i k\phi\psi(\mathcal{R}_0-1)(\mathcal{R}_{0,p}+\mathcal{R}_{0,d}(\mathcal{R}_0-1))\right) \\
&+ \frac{r}{\mathcal{R}_0^3}\left(r^2\gamma(\phi+\psi)(\gamma+\phi+\psi) + r\phi\psi(\gamma+\phi+\psi)^2(\mathcal{R}_{0,p}+\mathcal{R}_{0,d}(\mathcal{R}_0-1))\right) \\
&+ \frac{r}{\mathcal{R}_0^3}\left((\phi\psi)^2(\gamma+\phi+\psi)(\mathcal{R}_0^{p2}+\mathcal{R}_{0,d}(\mathcal{R}_0-1)) - r\gamma\sigma\beta_i k(\mathcal{R}_0-1)(\phi+\psi)\right) \\
&+ \frac{r}{\mathcal{R}_0^2}\left[r\gamma(\phi+\psi)(\gamma+\phi+\psi)^2 + \gamma\phi\psi(\phi+\psi)(\gamma+\phi+\psi)\left(2\mathcal{R}_{0,p}+\mathcal{R}_{0,d}(\mathcal{R}_0-1)\right)\right] \\
&- \frac{r\gamma\phi\psi}{\mathcal{R}_0^2}(\mathcal{R}_0-1)(\gamma\beta_i+\xi\beta_p)(r+\gamma+\phi+\psi) + \frac{r\gamma^2}{\mathcal{R}_0}(\phi+\psi)^2(\gamma+\phi+\psi).
\end{aligned}
$$

We can see that the Routh-Hurwitz determinants can be negative and the stability follows from [34, Theorem 2]. All the conditions of the Theorem are satisfied and we only need to show that Δ_3 can be zero. In Fig. 2 we provide a sketch of Δ_3 as a function of δ to show that Δ_3 can be negative for some values of δ, i.e., $\Delta_3 = 0$ at some critical value δ_c. Hence E_{EE} can become unstable through a Hopf bifurcation leading to oscillatory solutions.

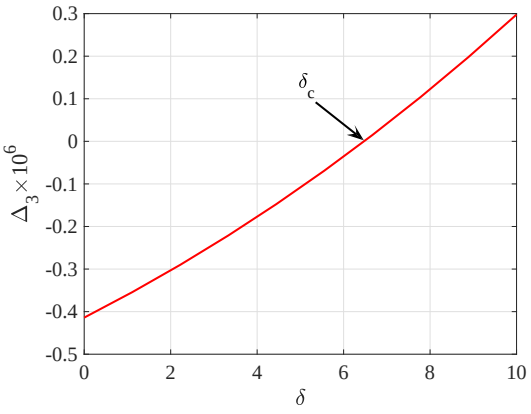
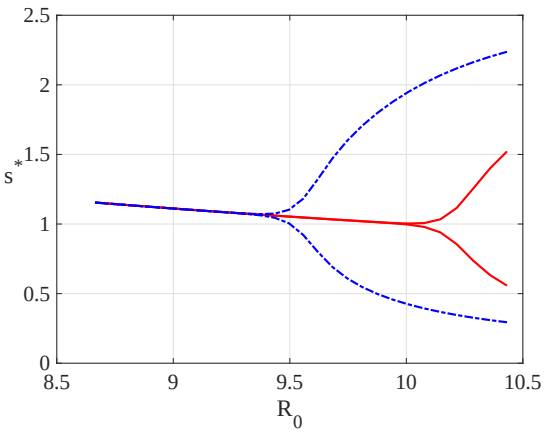


Figure 2: Plot of Δ_3 as a function of the culling rate δ. Clearly Δ_3 can be negative which can lead to the instability of E_{EE}.

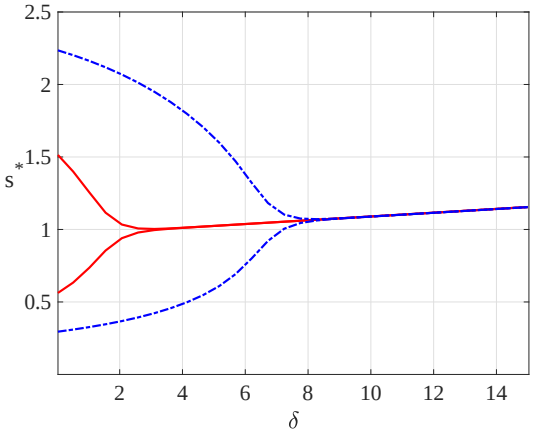
We further explore the existence of Hopf bifurcation by providing simulations of the culling rate, δ, versus the density of susceptible dogs. The simulations illustrating the theoretical results are provided in Fig. 3. We use the initial values of parameters as given in Table 1, and modify them as explained below, to get the different highlighted cases. In the figures, we choose the force of infection, $\lambda(i)$ such that

$$\lambda(i) = \frac{\beta_i i}{1+\nu i}.$$

Two cases are presented: first case with $\nu = 0$, such that the mass formulation discussed above holds; and $\nu = 0.1$ such that behavioural changes of infected dogs are taken into account. It is clear that sustained oscillations will still exist in the absence of behavioural effects and culling. However, increasing the two parameters will remove oscillatory solutions.



(a) Bifurcation with respect to $\mathcal{R}_0$.



(b) Bifurcation with respect to δ.

Figure 3: Bifurcation showing illustrating the maxima and minima of susceptible dogs. The parameters are chosen such that $\nu = 0$ for the broken curve and $\nu = 0.1$ for the solid curve.

4 Rabies-free environment

In this section, we assume that the environment is virus free, i.e., all contacts between the infected environment and the susceptible dogs does not result in any rabies transmission. Consequently, the model takes the following form

$$\begin{aligned} \frac{ds}{dt} &= rs\,(1 - s/k) - \lambda(i)s, \\ \frac{de}{dt} &= \lambda(i)s - (\sigma + \mu)e, \\ \frac{di}{dt} &= \sigma e - (\delta + \mu + \alpha)i, \end{aligned} \tag{11}$$

where all the parameters are defined as outlined before. The basic reproduction number for system (11) reduces to

$$\mathcal{R}_{0,d} = \frac{\sigma k \lambda'(0)}{\phi\psi}.$$

Proposition 2. *We have the following result:*

1. *The model (11) has a disease-free equilibrium* $P_{DFE} = (k, 0, 0)$.
2. *If* $\mathcal{R}_{0,d} \leq 1$, *there is no endemic equilibrium.*
3. *If* $\mathcal{R}_{0,d} > 1$, *there exists a unique endemic equilibrium* $P_{EE} = (s^*, e^*, i^*)$,

which is a solution to

$$r\left(1 - \frac{\phi\psi i^*}{\sigma k \lambda(i^*)}\right) - \lambda(i^*) = 0. \tag{12}$$

Following the qualitative analysis of model (1), from the previous section, we will state the following result without proof.

Theorem 4. *The disease-free equilibrium P_{DFE} is globally asymptotically stable for $\mathcal{R}_{0,d} < 1$ and unstable for $\mathcal{R}_{0,d} > 1$.*

To study the local stability of the endemic equilibrium P_{EE}, we consider the Jacobian evaluated at (s^*, e^*, i^*) as follows,

$$J(s^*, e^*, i^*) = \begin{pmatrix} -r\dfrac{s^*}{k} & 0 & -\lambda'(i^*)s^* \\ \lambda(i^*) & -\phi & \lambda'(i^*)s^* \\ 0 & \sigma & -\psi \end{pmatrix}.$$

The trace is negative. The characteristic equation for $J(P_{\text{EE}})$ is given by

$$\lambda^3 + n_1\lambda^2 + n_2\lambda + n_3 = 0, \tag{13}$$

where the coefficients are given by

$$\begin{aligned} n_1 &= (\psi + \phi) + r\frac{s^*}{k}, \\ n_2 &= (\phi\psi - \lambda'(i^*)s^*\sigma) + r\frac{s^*}{k}(\phi + \psi), \\ n_3 &= s^*\left(\frac{r}{k}\left[\phi\psi - s^*\lambda'(i^*)\sigma\right] + \lambda(i^*)\lambda'(i^*)\sigma\right). \end{aligned}$$

Proposition 3. *Assume $\mathcal{R}_{0,d} > 1$. Then all the roots of (13) have negative real part.*

Proof. Consider the coefficients of equation (13) above. To prove this result, it is sufficient to show that $\phi\psi - \lambda'(i^*)s^*\sigma \geq 0$. Bearing in mind the assumptions (2) and $\lambda(i^*) - i^*\lambda'(i^*) \geq 0$, we have

$$\phi\psi - \lambda'(i^*)s^*\sigma = \phi\psi - \lambda'(i^*)\sigma\frac{\psi\phi}{\lambda(i^*)\sigma}i^* = \frac{\phi\psi}{\lambda(i^*)}\left[\lambda(i^*) - i^*\lambda'(i^*)\right] \geq 0,$$

where we have used the equilibrium relationship $s^* = \dfrac{\phi\psi}{\lambda(i^*)\sigma}i^*$. Thus we conclude that all the roots of (13) have negative real part. □

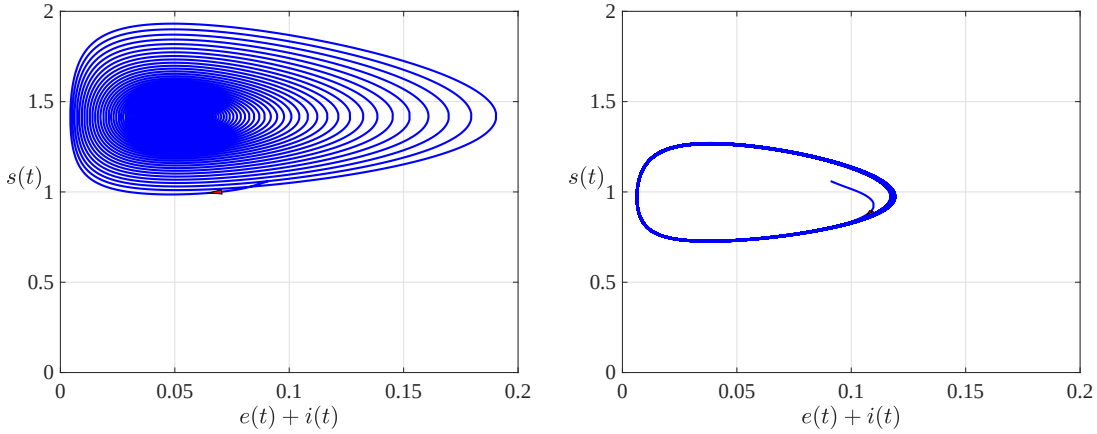
The first assumption of the Rough-Hurwitz is verified. Next we consider the Hurwitz determinant given by

$$\begin{aligned} \Delta_2(\delta) &= n_1n_2 - n_3 \\ &= \frac{\psi\phi}{\lambda(i^*)}\left(\psi + \phi + \frac{r\phi\psi}{k\sigma\lambda(i^*)}i^*\right)\left(\left[\lambda(i^*) - i^*\lambda'(i^*)\right] + \frac{r}{k\sigma}(\phi + \psi)i^*\right) \\ &- \frac{\psi\phi}{\lambda(i^*)}\left(\left[\lambda(i^*) - i^*\lambda'(i^*)\right] + \frac{r}{k\sigma}(\phi + \psi)i^*\right) \end{aligned}$$

We observe that $\Delta_2(\delta)$ can become negative which implies that $\Delta_2(\delta_c) = 0$ for some parameter value δ_c.

Theorem 5. *Assume $\mathcal{R}_{0,d} > 1$, the endemic equilibrium P_{EE} can become unstable through a Hopf bifurcation leading to oscillatory solutions.*

The proof of this result follows [35, page 162]. We illustrate the results of Theorem 5 in Fig. 4. For $\mathcal{R}_{0,d} = 1.939$, the endemic equilibrium P_{EE} is locally asymptotically stable and for $\mathcal{R}_{0,d} = 10.21$, the equilibrium is unstable.



(a) Local stability for $\mathcal{R}_0 < \mathcal{R}_{0c}$. (b) Oscillaroty solution for $\mathcal{R}_0 > \mathcal{R}_{0c}$.

Figure 4: Existence of Hopf bifurcation for the reduced model.

The critical/threshold value, $\mathcal{R}_{0c}$, refers to the onset of Hopf bifurcation. From a mathematical point of view, it is important to identify this critical value as it highlights the dynamics when the disease may appear to disappear. In the next sections we discuss the results of the model in terms of the control measures.

5 Sensitivity analysis

We now perform a sensitivity analysis on the basic reproduction number, $\mathcal{R}_0$, in order to capture how the ratio responds to changes in the parameters, furthermore, gain understanding into the disease control strategy and the transmission dynamics described by model (1). The changes in or sensitivity of $\mathcal{R}_0$ with respect to a parameter q is mathematically given by

$$\varphi^q_{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial q}.$$

The concept of sensitivity only looks at local computation while all parameters, q included, are kept constant. That is, sensitivity does not consider the simultaneous variation of all parameters. Thus, we will make use of the percentage change in $\mathcal{R}_0$ with respect to the percentage change in the parameter q, referred to as elasticity. Particularly,

$$\varepsilon^q_{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial q}\frac{q}{\mathcal{R}_0}.$$

The elasticity of $\mathcal{R}_0$ with respect to q is negative if $\mathcal{R}_0$ is decreasing with respect to q, and positive if $\mathcal{R}_0$ is increasing with respect to q. The local sensitivity analysis on the basic

reproduction ratio $\mathcal{R}_0$ leads to the following

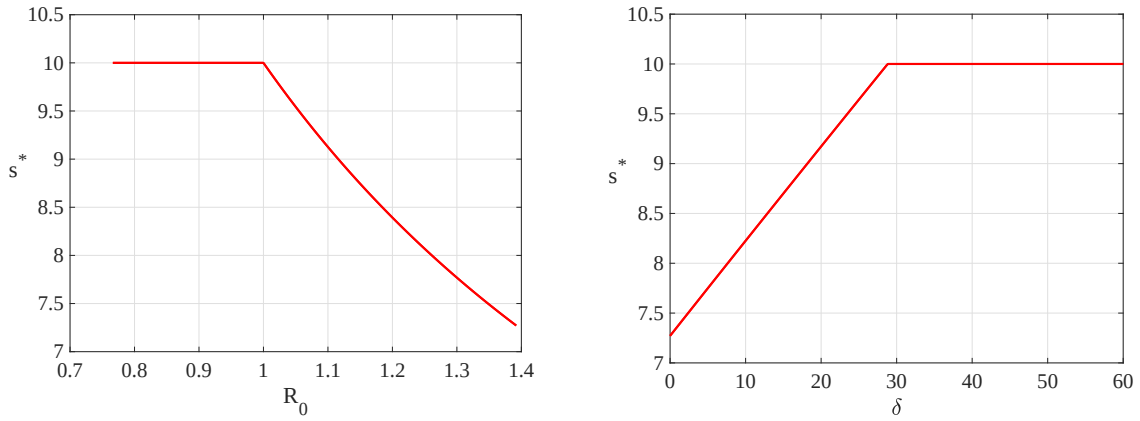
$$\varepsilon^{\sigma}_{\mathcal{R}_0} = \frac{\mu}{\sigma+\mu}, \qquad \varepsilon^{k}_{\mathcal{R}_0} = 1, \qquad \varepsilon^{\beta_p}_{\mathcal{R}_0} = \frac{\beta_p\xi}{\gamma\lambda'(0)+\beta_p\xi},$$
$$\varepsilon^{\xi}_{\mathcal{R}_0} = \frac{\beta_p\xi}{\gamma\lambda'(0)+\beta_p\xi}, \qquad \varepsilon^{\mu}_{\mathcal{R}_0} = -\mu\frac{(\mu+\delta+\alpha)+(\sigma+\mu)}{(\mu+\delta+\alpha)(\sigma+\mu)}, \qquad \varepsilon^{\delta}_{\mathcal{R}_0} = \frac{-\delta}{\mu+\delta+\alpha},$$
$$\varepsilon^{\alpha}_{\mathcal{R}_0} = \frac{-\alpha}{\mu+\delta+\alpha}, \qquad \varepsilon^{\gamma}_{\mathcal{R}_0} = \frac{-\sigma\beta_p k\xi}{\gamma\sigma k\lambda'(0)+\sigma\beta_p k\xi}, \qquad \varepsilon^{\beta_i}_{\mathcal{R}_0} = \frac{\beta_i\gamma}{\beta_i\gamma+\beta_p\xi}.$$

The analytic derivation given above can be numerically approximated. Thus, the result of such an analysis is shown below in Table 2.

Table 2: Sensitivity index of $\mathcal{R}_0$ with respect to each parameter.

Parameter	Sensitivity index
μ	-0.04375
α	-0.9799
σ	0.03704
ξ	0.007849
δ	-0.01342
β_i	0.9922
β_p	0.007849
k	1
γ	0.007849

We easily observe that different parameters have different extent of the effect on $\mathcal{R}_0$. For example, having that $\varepsilon^{\alpha}_{\mathcal{R}_0} = -0.9799$ means that 1% increase in α will result in 0.9799% decrease in $\mathcal{R}_0$. From Table 2 we see that an increase in $\sigma, \xi, \beta_p, \gamma, \beta_i$ and k will result to an increase in $\mathcal{R}_0$ with β_i and k having the most significant effect. On the other hand, an increase in μ, δ and α decreases $\mathcal{R}_0$.



(a) Bifurcation with respect to $\mathcal{R}_0$. (b) Bifurcation with respect to δ.

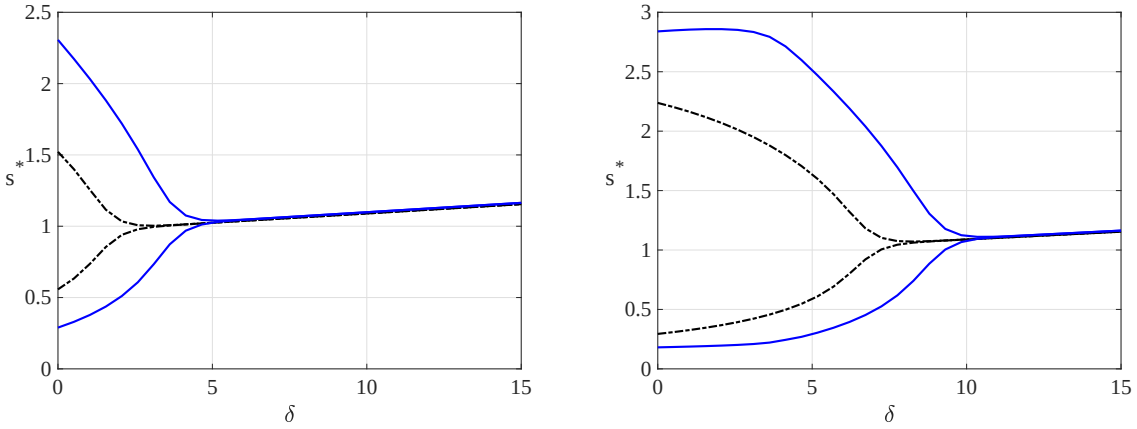
Figure 5: Bifurcation showing the effects of increasing the culling rate. Parameters are chosen as in Table 1 with $\beta_i = 10$.

From the sensitivity analysis, we know that $\mathcal{R}_0$ decreases when δ increases. As shown in Fig. 5, we observe that for small values of $\mathcal{R}_0$ or for $\mathcal{R}_0$ values less than one, the susceptible

population reach the carrying capacity, whereas, the number of susceptible dogs decreases as $\mathcal{R}_0$ increases. Moreover, from (b), as δ increases the susceptible population also increase to a carrying capacity which implies that the infected/infectious decrease.

6 Discussion

Wildlife intervention has always been a topical issue. However, faced with potential population extinction, vaccination or culling are some of the possible intervention strategies for wildlife, [4]. Other strategies include the prevention of domestic dogs from entering conservation areas, but this is not always possible due to many factors such as human geographical locations. The proposed model takes into account culling as a prevention strategy and environment as one of the reservoir for the virus.



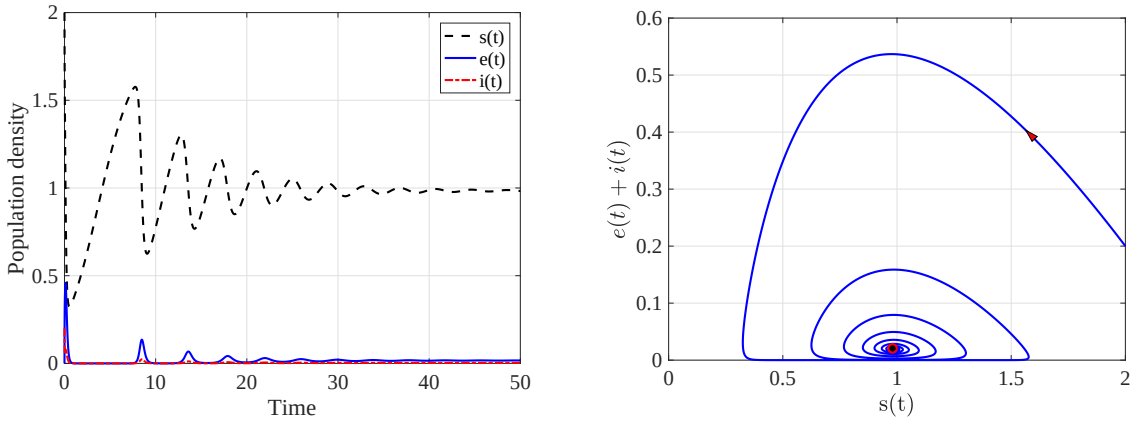
(a) $\nu = 0.1$. (b) $\nu = 0$.

Figure 6: Bifurcation diagram showing the maxima and minima of susceptible dogs for $\mathcal{R}_0 > \mathcal{R}_{0c}$. The parameters are chosen to simulate bifurcation curves with environmental contribution (broken curve) and without environmental contributions (solid curve).

The proposed model suggests culling can reduce the possible periodic behaviour of the system, see Fig. 3. This suggests that it is possible to stop culling prematurely as the disease may appear to disappear. Similar oscillations have been observed in rabies models for fox populations, see [25, 36]. However, the model presented here is different from these works. In particular, in addition to spatial considerations, their model assumed infected individuals contribute in the reproduction. No intervention strategies or environmental considerations are included in the model.

Earlier models, see for example [36], used spatial movement to take into account the behavioural changes in rabid dogs. The current model assumes a general transmission function satisfying conditions in (2). This includes the mass action incidence $\beta_i si$ and the saturation incidence $\beta_i \dfrac{i}{1+\nu i}$ as special cases. Numerical simulations in Fig. 6 suggest that more susceptible dogs are present when the mass action formulation is used as compared to the saturation incidence. Furthermore, the amplitude of the observed oscillations are less pronounced when saturating incidence is used.

The proposed work investigated the dynamics and control of rabies in (wild dogs) Lycaon pictus, and the effects of spillover from domestic stray dogs or other canines. The model does not explicitly follow the behavior of other canids (outside the Lycaon pictus) as these are all considered under the environment. We clearly see that neglecting the environmental contribution can lead to the underestimation of the severity of the disease. We remark that mathematical models are not there to replace field experts, but can be crucial in improving the knowledge on the biological system through testing certain hypothesis and designing appropriate experiments. It is important to note that these are only approximations of the reality and their predictability will always be subject to some uncertainty. Under the current reported parameter values, $\mathcal{R}_0$ remains in the interval $[1, 2]$ indicating the endemicity of the rabies virus among African wild dogs. See Fig. 7. In this case, the average population density is one wild dog per 100 km^2.



(a) Time series plot. (b) Phase plot.

Figure 7: Time series profiles, (a), and phase plot, (b) for the case $\mathcal{R}_0 = 1.828$.

7 Conclusions

In this paper, a simple SEI deterministic model for rabies epidemic was presented in which the population growth is logistic in the absence of the disease. The force of infection is given by a general nonlinear incidence function satisfying monotonicity conditions (2). We proved that the disease-free equilibrium is globally asymptotically stable for $\mathcal{R}_0 < 1$ and unstable for $\mathcal{R}_0 > 1$. The endemic equilibrium is locally asymptotically stable for $\mathcal{R}_0 < \mathcal{R}_{0c}$. In particular, for $\mathcal{R}_0 > 1$, the endemic equilibrium can become unstable through Hopf bifurcation leading to oscillatory solutions. The spread of the virus is controlled if $\mathcal{R}_0$ remains less than one. To conclude, while controlling the disposal of dead caucuses or controlling the movement of stray domestic animals can reduce the devastation of the disease, culling can be an effective tool to control the disease.

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