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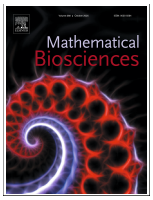

Original research article

Do "soft" interventions matter more than vaccination? Rabies as an example

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ABSTRACT

Interventions such as vaccinations, treatment *et cetera* are usually the gold standard of disease control, as measured by reducing the reproduction number below unity. However, in practice, few diseases are reduced below this eradication threshold and instead persist despite active intervention campaigns. We propose an epidemic model of rabies with a saturated incidence rate that represents "soft" interventions such as public-awareness campaigns, animal curfews, fences etc. We prove local and global stability results based on the reproduction number. However, numerical simulations suggest that eradication is unlikely to occur using current practices. We thus investigate the effect of altering the saturated incidence term using "soft" interventions and show that near-eradication can be achieved even when the reproduction number exceeds unity. Soft interventions such as public-awareness campaigns, reducing contacts, animal curfews and fences can have a greater effect on eradicating rabies than current vaccination programs.

1. Introduction

Disease modelling usually focuses on "hard" interventions, such as vaccines, drugs, insecticide spraying, etc. Mathematical models often pay less attention to "soft" interventions, such as public-awareness campaigns, education, health promotion etc. Generally, this is because the former are easier to quantify [1]. However, the latter are also a key plank of disease management [2]. Recent epidemics such as Ebola in West Africa and COVID-19 have reinforced the importance of developing infectious-disease models that better integrate social and behavioural dynamics and theories [3]. Here we use modelling to compare the effects of vaccination with "soft" interventions for rabies virus (RABV) in dogs and humans.

Rabies

For nearly 4000 years, the rabies virus has been one of the most important global health threats [4,5], as it is an ancient deadly infectious disease that affects both canines and humans once symptoms develop [4,5]. It is one of the lethal zoonotic illnesses caused by a neurotropic virus of the genus *lyssavirus*, which belongs to the *Rhabdoviridae* family [4–8]. It is also one of the vaccine-preventable viral infections that affect both warm-blooded animals and humans [9]. RABV is transmitted to a susceptible host mainly via the bite of an infected host, due to viral-loaded saliva or scratches [4,7–9]. Most rabies cases in humans and dogs have an incubation period of 20 days to 3 months; however, it can range from less than a week to over a year. This variation depends on the individual's age, the location of exposure in relation to brain, intensity of exposure and the species of the animal involved [9,10]. During this period, it infects the host's central nervous system and causes gradual and lethal inflammation of the brain and spinal cord (encephalomyelitis) [4,7,9]. The inflammation will eventually lead to seizures, respiratory and circulating failure, paralysis or coma, personality changes and death. Signs of apprehension or nervousness, irritability, sudden anorexia, hyperexcitability and aggressiveness are practically certain if the disease is not treated immediately [9,11,12]. Only 14 people have been documented to have survived rabies after symptoms appear [13].

A vast spectrum of mammals functions as reservoirs or carriers for RABV, including dogs, cats, coyotes, gray foxes, raccoons, skunks and bats [14–17]. Dogs are the primary reservoir for more than 90 % of rabies infections in the human population, mostly in Africa and Asia [5–9]. Despite the fact that rabies is preventable, thanks to effective

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vaccines, the morbidity and mortality rates of this disease are still high due to limited resources, high costs of vaccination and medical treatment, cultural hostility and lack of strategic coordination [9,10]. Rabies is one of the neglected tropical diseases that kills approximately 59,000 people each year in more than 150 countries, with up to 95 % of cases occurring in Africa and Asia, 40 % of whom are children under the age of 15 [4,6–10].

The rabies vaccine, invented by Louis Pasteur, was the second vaccine developed, after smallpox [18]. There are two types of rabies vaccines: pre-exposure prophylaxis (PrEP) and post-exposure prophylaxis (PEP). The PrEP vaccine is given before exposure occurs, whereas PEP is given right after exposure but before symptoms begin [19]. According to the World Health Organization (WHO) [9], rabies affects approximately 80 % of the poor and unprotected populations who live in secluded rural areas, and children are the major victims of this disease. This is mainly due to lack of access to proper medication or treatment, lack of awareness and negligence towards the disease. The WHO and its partners have launched a global campaign to achieve zero human deaths from dog-mediated rabies by 2030 [20–22]. The campaign will focus on improving awareness of RABV, enhancing access to PEP for the poor and vulnerable populations who live in remote rural areas, mass dog vaccination, rabies surveillance with veterinary services, expanding oral vaccination of wildlife, which can reduce rabies infection in reservoir populations, dog registration and education campaigns to the communities [5,9,20,21].

A number of mathematical models have been introduced to gain some insights into the effectiveness of rabies-control measures such as dog vaccination, the interactions and movements among different subpopulations of animals and rabies reaction-diffusion analysis in order to examine the spread of rabies in geographic regions and how different factors such as population density, habitat fragmentation and wildlife reservoirs affect the disease transmission. Multi-host zoonotic models can be used to understand how different host species contribute to the transmission of rabies and how interventions targeting one species can affect transmission in other species. Multi-patch models describe the spread of rabies across different geographic locations and illustrate how movement between patches can affect transmission. Seasonal models of rabies can demonstrate how factors such as host behaviour and climate variability can influence transmission patterns. For instance, to assess the effectiveness of vaccination in controlling the spreading of rabies, Asamoah *et al.* [23] developed an SEIR (Susceptible-Exposed-Infected-Recovered) model to study rabies transmission in both dog and human populations in order to identify the most effective strategies to control the disease spread. They discovered that the recruitment rate, loss of immunity and transmission rate of the dog population have the greatest effect on R_0. In addition, they observed that, by reducing the additional death rate of dogs and implementing PrEP and PEP vaccination in both dog and human populations, we could effectively control the spread of rabies. Nevertheless, there will be a high prevalence of rabies in the human population if there is no intervention for the dog population and only PEP and PrEP are considered for the human population. They found that the best way to reduce infection rates is by implementing prophylaxis in both dog and human populations. If there are limited funds to control the rabies outbreak, prioritizing the vaccine-control strategy for dogs is the key to disease eradication.

Laager *et al.* [24] developed a combination of field data and an SEIV (Susceptible-Exposed-Infected-Vaccinated) metapopulation model to stimulate rabies transmission in the dog population in N'Djamena, Chad. The model accounted for factors such as the movement of dogs between populations, vaccination coverage and the effectiveness of vaccination. This model was fit to the weekly rabies incidence data for four years and a sensitivity analysis conducted in order to evaluate the effect of underreporting on the transmission rates. The results of the study showed that vaccination campaigns targeting specific dog populations are more effective in reducing the incidence of rabies than campaigns that target the entire dog population. They also found that increasing vaccination coverage was crucial for reducing the incidence of the disease and that even small increases in vaccination coverage could have a significant impact on the spread of rabies. The model was able to predict the incidence of rabies in the dog population over time and provide insights into the dynamics of the disease transmission. The study highlights the importance of targeted vaccination campaigns in controlling the spread of dog rabies, especially in high-risk areas. Hailemicheal *et al.* [25] constructed an epidemic model to examine transmission between stray dogs and domestic dog populations. They applied vaccination and culling as the control strategies in their model. They assumed that rabies could be transmitted from stray dogs to domestic dogs, but not vice versa. They discovered that the transmission rates of stray dogs and the annual stray dog birth rate were the most sensitive parameters. Based on their simulation results, the most effective method to control the spread of rabies is a combination of vaccination and culling of infected dogs. Additionally, the yearly birth rate of dogs has a significant impact on the frequency of rabies cases.

In addition, some studies have been carried out to investigate the effect of seasonal or periodic variation in the occurrence of rabies [26–28]. Zhang *et al.* [26] examined the spread of rabies in both dog and human populations by considering periodic transmission rates. They discovered that to prevent the spread of human rabies in China, several measures are required: raising awareness about the disease; decreasing the birth rate of dogs; improving measures to prevent children from being bitten by dogs, particularly during the summer; and increasing the vaccination rate of dogs. Moreover, providing prompt medical treatment after dog bites is particularly important. Ruan *et al.* [27] examined the effects of seasonality, diffusion and dog movement in spreading the disease. They found that there are more human rabies cases in the summer and autumn seasons; hence, more efforts and control strategies are needed in the summer months to reduce the prevalence. In addition, by controlling the movement of exposed and infected dogs, the transmission of rabies can be reduced. Huang *et al.* [28] employed a multi-host zoonotic model to study the spreading dynamics of rabies among dogs, Chinese ferret-badgers (CFBs) and human populations. This model was applied to human rabies data reported in Zhejiang Province from 2004 to 2017. They found that the transmission rate between CFB and dog populations, the number of infected dogs and the vaccination rate of dogs are the most influential parameters in controlling the disease transmission. They suggested that control is favoured by enhancing rabies awareness, increasing the vaccination rate of dogs, preventing bites from CFBs and reducing contact between CFBs and dogs.

In order to achieve the WHO goal to end human deaths from dog-mediated rabies by 2030 [9,21,22], mathematical modelling can provide us with some insights about the disease-transmission dynamics, cost-effectiveness in controlling the disease, estimation of the probable outbreak duration and size, and assess the impact of control measures in curbing disease transmission [29–32]. We employ a mathematical model with a saturated incidence rate to study the transmission dynamics of rabies in both dog and human populations. We aim to identify under which conditions disease eradication is likely to happen and why the disease remains in an endemic state. To be more prepared and strengthen rabies-outbreak management, we identify the conditions of disease persistence. Moreover, sensitivity analysis of the model will be performed to find out which parameters have the greatest influence in controlling the disease.

Incidence functions

Analysis of disease persistence or eradication tends to focus only on stability of the equilibria, usually the disease-free equilibrium. While determining conditions for eradication — especially global stability — is useful, it is not the only factor involved in managing an endemic disease, something many modelling papers overlook. Here, we model rabies using an incidence function, which describes the long-term dynamics of

the disease but plays no role in the stability of the disease-free equilibrium.

We consider the inhibition effect; i.e., the "psychological" effect and behavioural change of the susceptible population when the number of infected dogs is increasing. Susceptible individuals may change their behaviour when the number of infected individuals increases, such as avoiding contacts. The rate of infection will slow down if many individuals are unable to be infected, since finding true susceptibles may be difficult. We can describe this mathematically by changing the transmission function.

The choice of an appropriate incidence function in a mathematical model holds significant importance, as it determines the dynamics of the epidemic [33,34]. The three most commonly employed incidence functions in deterministic mathematical models are the bilinear incidence rate, the standard incidence rate and the saturated incidence rate. Each of these functions has its implications for the generation of new infection cases. The bilinear incidence rate, known as mass-action incidence, is given by βSI, where β represents the transmission rate, S represents susceptible individuals and I denotes infected individuals [33]. Mass-action incidence is considered density-dependent, which means that the rate of contact per infective is proportional to the density of the infectious host population. It is suitable for modelling communicable diseases like influenza, but it may not be suitable for sexually transmitted diseases. This is because it would imply that the number of susceptible individuals contacting infectives is unbounded, which is not a plausible assumption. In the case of sexually transmitted diseases, it has been suggested that the standard incidence rate could be a better approximation [33,34].

The standard incidence rate, $\frac{\beta SI}{N}$, where N is the total population size, assumes a constant number of contacts per infective in unit time, which is applicable to dynamics of disease transmission in large populations. It provides a more realistic representation of how new cases of infection occur and allows for a better understanding of the spread of diseases in human populations. In reality, the probability of infection per contact is likely influenced by the number of infective individuals present, as a higher number of infective individuals can increase the overall infection risk [33,34]. However, the standard-incidence rate may encounter some difficulties and challenges when it is used to illustrate the proportion of effective contacts between susceptible and infectious populations, which may reach a saturated level due to various factors such as overcrowding of infected populations or implementation of protective measures by the susceptible individual [33,34].

If a population is crowded or saturated with infectives, then saturated incidence is a better option [35,36]. The saturation incidence rate $\frac{\beta SI}{1+\alpha I}$ [33] tends to a saturated level, $\frac{\beta}{\alpha}$, when I is sufficiently large, where βSI measures the infection force of the disease, α measures the inhibitory effect and $\frac{1}{1+\alpha I}$ represents the measure of psychological or inhibitory effect from the behaviour change of susceptible individuals when the number of infected individuals increases or due to a crowding effect. This may occur due to humans avoiding contacts in high-endemic situations or dog-control methods such as fences, animal curfews, etc. The net effect is to slow the rate of transmission as the infected population gets large. The parameter α is our proxy for soft interventions. If $\alpha = 0$, the saturated-incidence rate becomes a bilinear incidence rate [42]. Capasso and Serio [43] stated that the bilinear incidence rate might be suitable for a small number of infected individuals but is unrealistic for a large infected population. This type of incidence function may occur when a population has achieved herd immunity against a certain disease, meaning that the majority of people in the population have been exposed to the disease and are now resistant to it [33]. Even though this incidence rate is more challenging to cope with, it encompasses both behavioural changes and effects of crowding on the contact rate [34]. Saturated incidence is not the only way to model "soft" interventions when it comes to infectious diseases; a number of alternatives have been proposed, such as representing the crowding effect using a probability density function [37], using adaptive dynamics to model learning [38] or agent-based models to describe social processes [39].

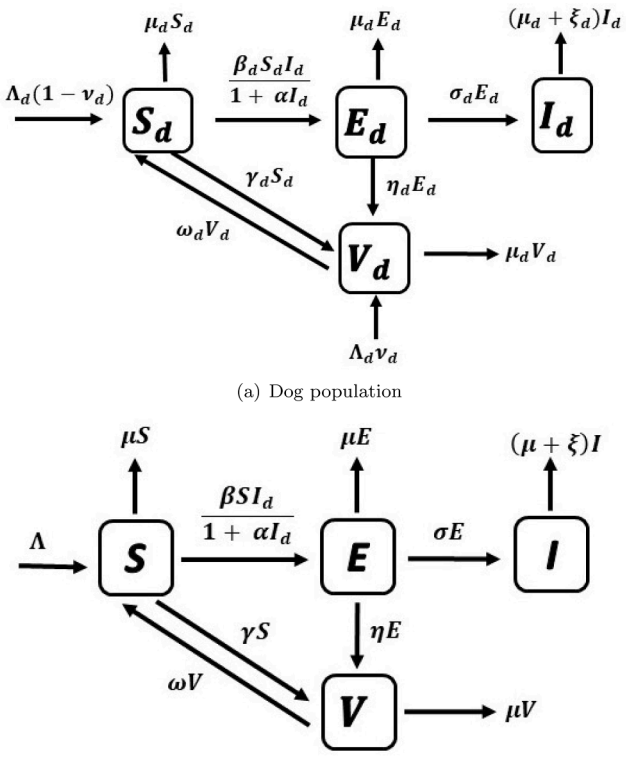

Fig. 1. The flow chart of model (2.1).

2. Mathematical model

We consider both dog-to-dog transmission and dog-to-human transmission. Both species can be vaccinated, but the vaccine also wanes. Once the vaccine wanes, there is no protection. Some dogs can be vaccinated at birth. There is no natural immunity against rabies in either species.

We propose a deterministic model with a saturated incidence rate to examine the transmission dynamics of rabies in both dog and human populations. Both the dog and human populations are classified into four subclasses: susceptible, exposed, vaccinated and infected, with the dog population denoted by $S_d(t)$, $E_d(t)$, $V_d(t)$ and $I_d(t)$, and the human population denoted by $S(t)$, $E(t)$, $V(t)$ and $I(t)$, at time t, respectively. Thus, our proposed rabies mathematical model is governed by a set of nonlinear ordinary differential equations defined as follows:

$$
\begin{aligned}
S_d'(t) &= \Lambda_d(1-\nu_d) + \omega_d V_d - \frac{\beta_d S_d I_d}{1+\alpha I_d} - (\mu_d+\gamma_d)S_d \\
E_d'(t) &= \frac{\beta_d S_d I_d}{1+\alpha I_d} - (\sigma_d+\eta_d+\mu_d)E_d \\
V_d'(t) &= \Lambda_d \nu_d + \gamma_d S_d + \eta_d E_d - (\mu_d+\omega_d)V_d \\
I_d'(t) &= \sigma_d E_d - (\mu_d+\xi_d)I_d \\
S'(t) &= \Lambda + \omega V - \frac{\beta S I_d}{1+\alpha I_d} - (\mu+\gamma)S \\
E'(t) &= \frac{\beta S I_d}{1+\alpha I_d} - (\sigma+\eta+\mu)E
\end{aligned}
$$

Table 1
Description of associated parameters in model (2.1), and the values for numerical simulations of the disease-free equilibrium (E_1) and the endemic equilibrium (E_*).

Parameter	Description	Unit	Parameter value for E_1 (Source)	Parameter value for E_* (Source)
Λ_d	Recruitment rate of dogs	$\frac{\text{individuals}}{\text{year}}$	325 ([40])	325 ([40])
β_d	Disease transmission rate from an infected to a susceptible dog	year^{-1}	5.2×10^{-4} (Assumed)	0.00092 (Assumed)
μ_d	Natural death rate of dogs	year^{-1}	0.0833 ([41])	0.0833 ([41])
σ_d	The progression rate from exposure to an infected dog	year^{-1}	6 ([27])	2.8 ([27])
ξ_d	Disease-related death rate of dogs	year^{-1}	1 ([27])	1 ([27])
ν_d	Fraction of newly recruited vaccinated dogs	unitless	0.9 (Assumed)	0.5 (Assumed)
ω_d	The rate at which vaccinated dogs lose vaccine-based immunity	year^{-1}	0.5 ([28])	1 ([28])
γ_d	The vaccinated rate of susceptible dogs	year^{-1}	0.5 ([44])	0.7 ([44])
η_d	The vaccination rate of exposed dogs	year^{-1}	0.5 ([27])	0.09 ([27])
Λ	Recruitment rate of humans	$\frac{\text{individuals}}{\text{year}}$	411 (Assumed)	411 (Assumed)
β	Rate at which humans contract rabies	year^{-1}	3.8×10^{-10} (Assumed)	1.29×10^{-3} (Assumed)
μ	Natural death rate of humans	year^{-1}	0.0137 ([45])	0.0137 ([45])
σ	The progression rate from exposed to infected humans	year^{-1}	6 ([27])	2.5 ([27])
ξ	Disease-related death rate of humans	year^{-1}	1 ([27])	1 ([27])
α	Inhibition effect	individuals^{-1}	(Varied)	(Varied)
ω	The rate at which vaccinated humans lose immunity	year^{-1}	1 ([27])	1 ([27])
γ	The vaccination rate of susceptible humans	year^{-1}	0.54 ([27])	0.54 ([27])
η	The vaccination rate of exposed humans	year^{-1}	0.328 ([28])	0.9 ([44])

$$\begin{aligned} V'(t) &= \gamma S + \eta E - (\mu+\omega)V \\ I'(t) &= \sigma E - (\mu+\xi)I, \end{aligned} \tag{2.1}$$

where $0 < \nu_d < 1$. The total population of dogs and humans at time t is given by $N_d(t) = S_d(t) + E_d(t) + V_d(t) + I_d(t)$ and $N(t) = S(t) + E(t) + V(t) + I(t)$, respectively. The flow chart of model (2.1) is shown in Fig. 1. The descriptions of associated parameters are listed in Table 1.

There are several assumptions in our model:

(a) Dogs are the only source of transmission in this study.
(b) The recruitment rates of susceptible dogs and humans are constant.
(c) Only a fraction ν_d of newly recruited dogs are vaccinated.
(d) Both dog and human populations will become susceptible whenever immunity wanes.
(e) The inhibitory effect, α, for dog and human populations are similar.

3. Theoretical analysis

3.1. Invariant region

First, we would like to identify the domain wherein the solutions of model (2.1) are both biologically and mathematically relevant: that is, by determining the region Ω where model (2.1) remains positively invariant and attracting for all $t \geq 0$. Particularly, all the solutions of model (2.1) are bounded and remain in Ω for sufficiently large t.

Lemma 1. *The set* $\Omega \equiv \left\{(S_d, E_d, V_d, I_d, S, E, V, I) \in \mathbb{R}^8_+ \mid \ 0 < S_d + E_d + V_d + I_d \leq \frac{\Lambda_d}{\mu_d} \text{ and } 0 < S + E + V + I \leq \frac{\Lambda}{\mu}\right\}$ *is a positively invariant and attracting region for model (2.1).*

Proof. Let $N_d(t) = S_d(t) + E_d(t) + V_d(t) + I_d(t)$ and $N(t) = S(t) + E(t) + V(t) + I(t)$ be the total populations of dogs and humans, respectively. Then we obtain

$$N_d'(t) = S_d'(t) + E_d'(t) + V_d'(t) + I_d'(t) \leq \Lambda_d - \mu_d N_d(t). \tag{3.2}$$

Using an integrating factor, we have

$$\begin{aligned} \int_0^t \frac{d}{da}\left(N_d e^{\mu_d a}\right) da &\leq \int_0^t \left(\Lambda_d e^{\mu_d a}\right) da \\ N_d(t) &\leq \left[N_d(0) - \frac{\Lambda_d}{\mu_d}\right] e^{-\mu_d t} + \frac{\Lambda_d}{\mu_d}. \end{aligned} \tag{3.3}$$

From (3.3), we obtain $N_d(t) \leq \frac{\Lambda_d}{\mu_d}$ if $N_d(0) \leq \frac{\Lambda_d}{\mu_d}$.

Next, to show that Ω is an attracting region, if we have $N_d(t) > \frac{\Lambda_d}{\mu_d}$, then

$$N_d'(t) \leq \Lambda_d - \mu_d N_d(t) < 0.$$

We deduce that the total population of dogs is bounded by $\frac{\Lambda_d}{\mu_d}$. By applying a similar approach, we find that the total human population is bounded by $\frac{\Lambda}{\mu}$; i.e., $N \leq \frac{\Lambda}{\mu}$. Hence the solution of model (2.1) with arbitrary initial conditions will either remain in or approach Ω as $t \to \infty$. This shows that the ω-limit sets of model (2.1) are contained in Ω. □

3.2. Stability analysis

Here, we perform a standard local stability analysis. We will prove global stability of the DFE when $R_0 < 1$ and some additional conditions apply. We will prove global stability of the endemic equilibrium when $R_0 > 1$ and some additional conditions apply. It follows that R_0 is a threshold of eradication. Is this enough to control rabies?

Two equilibria exist in model (2.1): the disease-free equilibrium (DFE) and the endemic equilibrium (EE). The DFE of model (2.1) is $E_1 = (S_{d0}, E_{d0}, V_{d0}, I_{d0}, S_0, E_0, V_0, I_0)$, where

$$S_{d0} = \frac{\Lambda_d[\mu_d(1-\nu_d)+\omega_d]}{\mu_d(\mu_d+\gamma_d+\omega_d)}, \quad V_{d0} = \frac{\Lambda_d(\nu_d\mu_d+\gamma_d)}{\mu_d(\mu_d+\gamma_d+\omega_d)},$$

$$S_0 = \frac{\Lambda(\mu+\omega)}{\mu(\mu+\gamma+\omega)} \quad V_0 = \frac{\Lambda\gamma}{\mu(\mu+\gamma+\omega)},$$

$$E_{d0} = I_{d0} = E_0 = I_0 = 0,$$

whereas the EE of model (2.1) is $E_* = (S_d^*, E_d^*, V_d^*, I_d^*, S^*, E^*, V^*, I^*)$, where

$$S_d^* = \frac{(\mu_d+\xi_d)(\sigma_d+\eta_d+\mu_d)(1+\alpha I_d^*)}{(\beta_d)\sigma_d}, \quad E_d^* = \frac{(\mu_d+\xi_d)I_d^*}{\sigma_d},$$

$$V_d^* = \frac{\Lambda_d\nu_d\sigma_d\beta_d + (\mu_d+\xi_d)[\gamma_d(\sigma_d+\eta_d+\mu_d)(1+\alpha I_d^*)+\beta_d\eta_d I_d^*]}{\beta_d\sigma_d(\mu_d+\omega_d)},$$

$$I_d^* = \frac{\Lambda_d\sigma_d\beta_d[\mu_d(1-\nu_d)+\omega_d] - \mu_d(\mu_d+\xi_d)(\mu_d+\gamma_d+\omega_d)(\sigma_d+\eta_d+\mu_d)}{(\mu_d+\xi_d)\{\mu_d(\sigma_d+\eta_d+\mu_d)[\beta_d+\alpha(\mu_d+\gamma_d+\omega_d)]+\beta_d\omega_d(\sigma_d+\mu_d)\}},$$

$$S^* = \frac{(\mu+\xi)(\sigma+\eta+\mu)(1+\alpha I_d^*)I^*}{\sigma\beta I_d^*}, \quad E^* = \frac{(\mu+\xi)I^*}{\sigma},$$

$$V^* = \frac{(\mu+\xi)[\gamma(\sigma+\eta+\mu)(1+\alpha I_d^*)+\eta\beta I^*]I^*}{\sigma\beta(\mu+\omega)I_d^*}$$

$$I^* = \frac{\Lambda\sigma\beta(\mu+\omega)I_d^*}{(\mu+\xi)\{\beta\omega(\sigma+\mu)I_d^*+\mu(\sigma+\eta+\mu)[\beta I_d^*+(\mu+\gamma+\omega)(1+\alpha I_d^*)]\}}.$$

By applying the next-generation matrix approach [46,47], the basic reproduction number of model (2.1) is given as follows:

$$R_0 = \frac{\Lambda_d\sigma_d\beta_d[\mu_d(1-\nu_d)+\omega_d]}{\mu_d(\mu_d+\xi_d)(\mu_d+\gamma_d+\omega_d)(\sigma_d+\eta_d+\mu_d)}. \tag{3.4}$$

Theorem 2. *An endemic equilibrium of model (2.1), E_*, exists if and only if $R_0 > 1$.*

Proof. Assume $R_0 > 1$. To prove the existence of E_*, we need to show S_d^*, E_d^*, V_d^*, S^*, E^*, V^* and $I^* > 0$. Since $R_0 > 1$ implies $\Lambda_d\sigma_d\beta_d[\mu_d(1-\nu_d)+\omega_d] > \mu_d(\mu_d+\xi_d)(\mu_d+\gamma_d+\omega_d)(\sigma_d+\eta_d+\mu_d)$, we obtain

$$I_d^* = \frac{\Lambda_d\sigma_d\beta_d[\mu_d(1-\nu_d)+\omega_d]-\mu_d(\mu_d+\xi_d)(\mu_d+\gamma_d+\omega_d)(\sigma_d+\eta_d+\mu_d)}{(\mu_d+\xi_d)\{\mu_d(\sigma_d+\eta_d+\mu_d)[\beta_d+\alpha(\mu_d+\gamma_d+\omega_d)]+\beta_d\omega_d(\sigma_d+\mu_d)\}} > 0,$$

where all associated parameters are positive. Since $I_d^* > 0$, this implies S_d^*, E_d^*, V_d^*, S^*, E^*, V^* and $I^* > 0$.

Assume E_* exists; i.e., S_d^*, E_d^*, V_d^*, I_d^*, S^*, E^*, V^* and $I^* > 0$. Since $I_d^* > 0$, it follows that S_d^*, E_d^*, V_d^*, S^*, E^*, V^* and $I^* > 0$, we get

$$\frac{\Lambda_d\sigma_d\beta_d[\mu_d(1-\nu_d)+\omega_d]}{\mu_d(\mu_d+\xi_d)(\mu_d+\gamma_d+\omega_d)(\sigma_d+\eta_d+\mu_d)} > 1 \equiv R_0 > 1.$$

□

Next, we investigate the local stability of both disease-free and endemic equilibria of model (2.1) using a linearization approach.

Theorem 3. *The DFE, E_1, of model (2.1) achieves local asymptotic stability if $R_0 < 1$.*

Proof. The Jacobian matrix of model (2.1) at E_1 is defined as follows:

$$J(E_1) = \begin{bmatrix} -(\mu_d+\gamma_d) & 0 & \omega_d & -\beta_d S_{d0} & 0 & 0 & 0 & 0 \\ 0 & -(\sigma_d+\eta_d+\mu_d) & 0 & \beta_d S_{d0} & 0 & 0 & 0 & 0 \\ \gamma_d & \eta_d & -(\mu_d+\omega_d) & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_d & 0 & -(\mu_d+\xi_d) & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\beta S_0 & -(\mu+\gamma) & 0 & \omega & 0 \\ 0 & 0 & 0 & \beta S_0 & 0 & -(\sigma+\eta+\mu) & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma & \eta & -(\mu+\omega) & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma & 0 & -(\mu+\xi) \end{bmatrix}.$$

Let λ be the eigenvalue and I be the 8×8 identity matrix. The characteristic equation, $|J(E_1)-\lambda I| = 0$, is defined as follows:

$$(\mu+\xi+\lambda)(\sigma+\mu+\lambda)(\mu_d+\lambda)(\mu_d+\omega_d+\gamma_d+\lambda)(\mu+\lambda)\times (\mu+\omega+\gamma+\lambda)\big[(\sigma_d+\eta_d+\mu_d+\lambda)(\mu_d+\xi_d+\lambda)-\sigma_d\beta_d S_{d0}\big] = 0. \tag{3.5}$$

Since all associated parameters are positive, the nontrivial eigenvalues of (3.5) satisfy

$$\lambda^2 + (\sigma_d+\eta_d+2\mu_d+\xi_d)\lambda + \big[(\mu_d+\xi_d)(\sigma_d+\eta_d+\mu_d)-\sigma_d\beta_d S_{d0}\big] = 0.$$

Denote $A = 1$, $B = \sigma_d+\eta_d+2\mu_d+\xi_d$ and $C = (\mu_d+\xi_d)(\sigma_d+\eta_d+\mu_d)-\sigma_d\beta_d S_{d0}$. By applying the quadratic formula,

$$B^2-4AC = B^2+4(\mu_d+\xi_d)(\sigma_d+\eta_d+\mu_d)(R_0-1) < B^2 \quad \text{if} \quad R_0 < 1.$$

The lower bound is given by

$$\begin{aligned} B^2-4AC &= \big[\sigma_d+\eta_d+2\mu_d+\xi_d\big]^2-4\big[(\mu_d+\xi_d)(\sigma_d+\eta_d+\mu_d) \\ &\quad -\sigma_d\beta_d S_{d0}\big] \\ &= (\sigma_d+\eta_d+\mu_d)^2-2(\mu_d+\xi_d)(\sigma_d+\eta_d+\mu_d)+(\mu_d+\xi_d)^2 \\ &\quad +4\sigma_d\beta_d S_{d0} \\ &> (\sigma_d+\eta_d)^2, \quad \text{since} \quad S_{d0} > 0. \end{aligned}$$

It follows that the roots are real. Since $\sqrt{B^2-4AC} < B$, we get $\lambda_+ = \frac{-B+\sqrt{B^2-4AC}}{2A} < 0$. Next,

$$\sqrt{B^2-4AC} > \sigma_d+\eta_d.$$

Thus, $\lambda_- = \frac{-B-\sqrt{B^2-4AC}}{2A} < -(\mu_d+\xi_d/2) < 0$.

Since all eigenvalues of model (2.1) are negative whenever $R_0 < 1$, E_1 achieves local asymptotic stability. □

We now address the local stability of endemic equilibrium, E_*, of model (2.1).

Theorem 4. *The endemic equilibrium, E_*, of model (2.1) achieves local asymptotic stability if $R_0 > 1$.*

Proof. By employing a similar approach as in Theorem 3, the characteristic equation of model (2.1) at E_* is given as follows:

$$(\mu+\xi+\lambda)\big(\lambda^3+a_1\lambda^2+a_2\lambda+a_3\big)\big(\lambda^4+a_4\lambda^3+a_5\lambda^2+a_6\lambda+a_7\big) = 0, \tag{3.6}$$

where λ denotes the eigenvalue of model (2.1) and

$$a_1 = \frac{\beta I_d^*}{1+\alpha I_d^*}+\sigma+\eta+3\mu+\gamma+\omega,$$

$$a_2 = \mu(\sigma+\eta+\mu)+(\sigma+\eta+2\mu)\left(\frac{\beta I_d^*}{1+\alpha I_d^*}+\mu+\gamma\right) + \omega\left(\frac{\beta I_d^*}{1+\alpha I_d^*}+\sigma+\eta+2\mu\right),$$

$$a_3 = \mu(\sigma+\eta+\mu)\left(\frac{\beta I_d^*}{1+\alpha I_d^*}+\mu+\gamma+\omega\right)+\omega(\sigma+\mu)\frac{\beta I_d^*}{1+\alpha I_d^*},$$

$$a_4 = \frac{\beta_d I_d^*}{1+\alpha I_d^*}+4\mu_d+\gamma_d+\omega_d+\sigma_d+\eta_d+\xi_d,$$

$$a_5 = \frac{\omega_d\beta_d I_d^*}{1+\alpha I_d^*}+\mu_d(2\mu_d+\xi_d+\sigma_d+\eta_d) + \left(\frac{\beta_d I_d^*}{1+\alpha I_d^*}+\mu_d+\gamma_d+\omega_d\right)(3\mu_d+\xi_d+\sigma_d+\eta_d) + (\mu_d+\xi_d)(\sigma_d+\eta_d+\mu_d)\frac{\alpha I_d^*}{1+\alpha I_d^*},$$

$$a_6 = \frac{\beta_d I_d^*}{1+\alpha I_d^*}\Big[\omega_d(2\mu_d+\xi_d+\sigma_d)+\mu_d(\mu_d+\xi_d)+(2\mu_d+\xi_d)(\sigma_d+\eta_d+\mu_d)\Big] + \mu_d(\mu_d+\gamma_d+\omega_d)(2\mu_d+\xi_d+\sigma_d+\eta_d)+(\mu_d+\xi_d)(\sigma_d+\eta_d+\mu_d) (2\mu_d+\gamma_d+\omega_d)\frac{\alpha I_d^*}{1+\alpha I_d^*} \quad \text{and}$$

$$a_7 = (\mu_d+\xi_d)\left\{\frac{\beta_d I_d^*}{1+\alpha I_d^*}\Big[\mu_d(\sigma_d+\eta_d+\mu_d)+\omega_d(\sigma_d+\mu_d)\Big] + \mu_d(\mu_d+\gamma_d+\omega_d)(\sigma_d+\eta_d+\mu_d)\frac{\alpha I_d^*}{1+\alpha I_d^*}\right\}.$$

We employ the Routh–Hurwitz Criterion [48–50] to find the nontrivial eigenvalues of (3.6). For $\lambda^3+a_1\lambda^2+a_2\lambda+a_3 = 0$, we find that $a_1, a_2, a_3 > 0$ if $R_0 > 1$, and

$$\begin{aligned} a_1a_2-a_3 &= \frac{\beta I_d^*}{1+\alpha I_d^*}\left[(\sigma+\eta+2\mu)\left(\frac{\beta I_d^*}{1+\alpha I_d^*}+\mu+\gamma\right)+\frac{\omega\beta I_d^*}{1+\alpha I_d^*}+\mu+\gamma\right] \\ &\quad +(\sigma+\eta+3\mu+\gamma+\omega)\left[(\sigma+\eta+2\mu)\left(\frac{\beta I_d^*}{1+\alpha I_d^*}+\mu+\gamma\right)\right. \\ &\quad \left.+\omega\left(\frac{\beta I_d^*}{1+\alpha I_d^*}+\sigma+\eta+2\mu\right)\right] \\ &\quad +\mu(\sigma+\eta+\mu)(\sigma+\eta+2\mu) \\ &> 0 \quad \text{if} \quad R_0 > 1. \end{aligned}$$

Hence, by the Routh–Hurwitz Criterion, the eigenvalues of $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$ are negative or have negative real parts.

Next, we would like to solve $\lambda^4 + a_4\lambda^3 + a_5\lambda^2 + a_6\lambda + a_7 = 0$ for λ. By applying a similar approach as above, we find that $a_4, a_5, a_6, a_7 > 0$ and $a_6(a_4a_5 - a_6) - a_4^2a_7 > 0$ if $R_0 > 1$. See Appendix 1 for more details. Thus, all eigenvalues are negative or have negative real parts. Therefore, E_* achieves local asymptotic stability if $R_0 > 1$. □

The global stability of the disease-free and endemic equilibria is investigated using different approaches, under some additional restrictions. We prove the global stability of the DFE, E_1, by applying the comparison principle [51] and the theory of asymptotic autonomous systems [52], whereas we prove the global stability of the EE, E_*, by using the geometric approach proposed by Li and Muldowney [53].

Theorem 5. *E_1 of model (2.1) is globally asymptotically stable if $R_0 < 1, \eta_d \leqslant \gamma_d$ and $\eta \leqslant \gamma$.*

Proof. First, we consider the dog-only population of model (2.1); i.e., the first four equations of model (2.1), since it is independent of the human-only variables of model (2.1). From model (2.1), the third equation is given as follows if $\eta_d \leqslant \gamma_d$:

$$\frac{dV_d}{dt} \leq \Lambda_d\nu_d + \gamma_d(S_d + E_d) - (\mu_d + \omega_d)V_d. \tag{3.7}$$

Since $S_d + E_d + V_d + I_d \to \dfrac{\Lambda_d}{\mu_d}$ as $t \to \infty$, Eq. (3.7) is an asymptotically autonomous differential equation with the limit equation as follows:

$$\begin{aligned}\frac{dV_d}{dt} &\leq \Lambda_d\nu_d + \gamma_d\left(\frac{\Lambda_d}{\mu_d} - I_d - V_d\right) - (\mu_d + \omega_d)V_d\\ &\leq \frac{\Lambda_d(\mu_d\nu_d + \gamma_d)}{\mu_d} - (\mu_d + \omega_d + \gamma_d)V_d \end{aligned}\tag{3.8}$$

since $I_d \geq 0$ and all associated parameters are positive. By using an integrating factor, we obtain

$$\begin{aligned}&\frac{d}{dt}\left[V_d e^{(\mu_d+\omega_d+\gamma_d)t}\right] \leq \frac{\Lambda_d(\mu_d\nu_d + \gamma_d)}{\mu_d} e^{(\mu_d+\omega_d+\gamma_d)t}\\ &V_d e^{(\mu_d+\omega_d+\gamma_d)t} \leq V_d(0) + \frac{\Lambda_d(\mu_d\nu_d + \gamma_d)}{\mu_d(\mu_d + \omega_d + \gamma_d)}\left[e^{(\mu_d+\omega_d+\gamma_d)t} - 1\right]\\ &V_d(t) \leq V_{d0} + V_d(0)e^{-(\mu_d+\omega_d+\gamma_d)t}, \quad \text{where}\\ &V_{d0} = \frac{\Lambda_d(\mu_d\nu_d + \gamma_d)}{\mu_d(\mu_d + \omega_d + \gamma_d)}. \end{aligned}\tag{3.9}$$

Let $\varepsilon = \frac{(\mu_d+\gamma_d)\varepsilon_1}{\omega_d}$. For every $\varepsilon > 0$, there exists a $t_1 > 0$ such that $V_d \leqslant V_{d0} + \varepsilon$ for all $t > t_1$. Then, for all $t > t_1$,

$$\begin{aligned}\frac{dS_d}{dt} &= \Lambda_d(1 - \nu_d) + \omega_d V_d - \frac{\beta_d S_d I_d}{1 + \alpha I_d} - (\mu_d + \gamma_d)S_d\\ &\leq \Lambda_d(1 - \nu_d) + \omega_d(V_{d0} + \varepsilon) - (\mu_d + \gamma_d)S_d \quad \text{since} \quad V_d \leqslant V_{d0} + \varepsilon,\\ &\qquad \text{and all associated variables and parameters are positive}\\ &= \frac{\Lambda_d[\mu_d(1 - \nu_d) + \omega_d] - \mu_d\omega_d S_{d_0} + \mu_d\omega_d\varepsilon}{\mu_d} - (\mu_d + \gamma_d)S_d,\\ &\qquad \text{where} \quad S_{d0} = \frac{\Lambda_d[\mu_d(1 - \nu_d) + \omega_d]}{\mu_d(\mu_d + \omega_d + \gamma_d)}. \end{aligned}\tag{3.10}$$

By using an integrating factor,

$$\begin{aligned}&\frac{d}{dt}\left[S_d e^{(\mu_4+\gamma_d)t}\right] \leq \frac{\Lambda_d[\mu_d(1 - \nu_d) + \omega_d] - \mu_d\omega_d S_{d_0} + \mu_d\omega_d\varepsilon}{\mu_d} e^{(\mu_d+\gamma_d)t}\\ &S_d(t)e^{(\mu_d+\gamma_d)t} \leq S_d(0) + \frac{(\mu_d + \gamma_d)S_{d0} + \omega_d\varepsilon}{\mu_d + \gamma_d}\left[e^{(\mu_d+\gamma_d)t} - 1\right]. \end{aligned}$$

Let $\varepsilon_1 = \frac{\omega_d\varepsilon}{\mu_d+\gamma_d}$. For every $\varepsilon_1 > 0$, there exists a $t_2 > 0$ such that $S_d \leqslant S_{d_0} + \varepsilon_1$ for all $t > t_2 > t_1$. Thus, for all $t > t_2 > t_1$, the basic reproduction number of model (2.1) is defined as

$$\begin{aligned}R_0 &= \frac{\sigma_d\beta_d S_d}{(\mu_d + \xi_d)(\sigma_d + \eta_d + \mu_d)} \leq \frac{\sigma_d\beta_d(S_{d_0} + \varepsilon_1)}{(\mu_d + \xi_d)(\sigma_d + \eta_d + \mu_d)}\\ &= R_0 + \frac{\sigma_d\beta_d\varepsilon_1}{(\mu_d + \xi_d)(\sigma_d + \eta_d + \mu_d)}. \end{aligned}$$

Next, we consider the following dog-only system

$$\begin{aligned}\frac{dE_d}{dt} &\leqslant \beta_d(S_{d0} + \varepsilon_1)I_d - (\sigma_d + \eta_d + \mu_d)E_d,\\ \frac{dI_d}{dt} &= \sigma_d E_d - (\mu_d + \xi_d)I_d. \end{aligned}\tag{3.11}$$

The corresponding linear system of (3.11) is given as follows:

$$\begin{aligned}\frac{d\hat{E}_d}{dt} &= \beta_d(S_{d0} + \varepsilon_1)\hat{I}_d - (\sigma_d + \mu_d + \mu_d)\hat{E}_d,\\ \frac{d\hat{I}_d}{dt} &= \sigma_d\hat{E}_d - (\mu_d + \xi_d)\hat{I}_d. \end{aligned}\tag{3.12}$$

Let $\hat{\lambda}$ denote the eigenvalue, with the characteristic equation of (3.12) defined as

$$\hat{A}\hat{\lambda}^2 + \hat{B}\hat{\lambda} + \hat{C} = 0,$$

where $\hat{A} = 1$, $\hat{B} = (\sigma_d + \eta_d + \mu_d) + (\mu_d + \xi_d)$ and $\hat{C} = (\mu_d + \xi_d)(\sigma_d + \eta_d + \mu_d) - \sigma_d\beta_d(S_{d0} + \varepsilon_1)$.

$$\begin{aligned}\hat{B}^2 - 4\hat{A}\hat{C} &= \hat{B}^2 + 4[\sigma_d\beta_d(S_{d0} + \varepsilon_1) - (\mu_d + \xi_d)(\sigma_d + \eta_d + \mu_d)]\\ &< \hat{B}^2 \quad \text{if} \quad R_0 + \frac{\sigma_d\beta_d\varepsilon_1}{(\mu_d + \xi_d)(\sigma_d + \eta_d + \mu_d)} < 1\\ \sqrt{\hat{B}^2 - 4\hat{A}\hat{C}} &< \hat{B}. \end{aligned}\tag{3.13}$$

By using the quadratic formula, we get

$$\hat{\lambda}_+ = \frac{-\hat{B} + \sqrt{\hat{B}^2 - 4\hat{A}\hat{C}}}{2\hat{A}} < 0.$$

In addition, we have

$$\begin{aligned}\hat{B}^2 - 4\hat{A}\hat{C} &= (\sigma_d + \eta_d - \xi_d)^2 + 4\sigma_d\beta_d\left(S_{d_0} + \varepsilon_1\right)\\ &> (\sigma_d + \eta_d - \xi_d)^2\\ \sqrt{\hat{B}^2 - 4\hat{A}\hat{C}} &> \sigma_d + \eta_d - \xi_d \end{aligned}\tag{3.14}$$

and

$$\hat{\lambda}_- = \frac{-\hat{B} - \sqrt{\hat{B}^2 - 4\hat{A}\hat{C}}}{2\hat{A}} < -(\sigma_d + \eta_d + \mu_d) < 0.$$

Thus, the general solution for (3.12) is

$$x(t) = c_1u_1e^{\hat{x}_1t} + c_2u_2e^{\hat{\lambda}-t},$$

where $x(t) = \left[\hat{E}_d(t), \hat{I}_d(t)\right]^\top$, c_1 and c_2 are arbitrary constants, and u_1 and u_2 are the corresponding eigenvectors of eigenvalues $\hat{\lambda}_+$ and $\hat{\lambda}_-$, respectively. Furthermore, $x(t) \to 0$ as $t \to \infty$. By applying the comparison principle [52], $E_d, I_d \to 0$ as $t \to \infty$. Consequently, by the theory of asymptotic autonomous systems [51], we obtain $V_d \to V_{d0}$ and $S_d \to S_{10}$ as $t \to \infty$ from (3.7) and (3.10), respectively.

We now consider the (humans only) final four equations of model (2.1). By looking at the equation

$$\frac{dV}{dt} = \gamma S + \eta E - (u + \omega)V,$$

we have

$$\frac{dV}{dt} \leq \gamma(S + E) - (\mu + \omega)V \quad \text{if} \quad \eta \leqslant \gamma. \tag{3.15}$$

Since $S + E + V + I \to \frac{\Lambda}{\mu}$ as $t \to \infty$, Eq. (3.15) is an asymptotically autonomous differential equation with the following limit equation:

$$\frac{dV}{dt} \leq \gamma\left(\frac{\Lambda}{\mu} - V - I\right) - (\mu + \omega)V \quad \text{if} \quad \eta \leqslant \gamma$$

$$\leq \frac{\Lambda\gamma}{\mu} - (\mu+\omega+\gamma), \tag{3.16}$$

since all the associated variables and parameters are positive. By using an integrating factor,

$$\frac{d}{dt}\left[Ve^{(\mu+\omega+\gamma)t}\right] \leq \frac{\Lambda\gamma}{\mu}e^{(\mu+\omega+\gamma)t}$$

$$V(t)e^{(\mu+\omega+\gamma)t} \leq V(0) + \frac{\Lambda\gamma}{\mu(\mu+\omega+\gamma)}\left[e^{(\mu+\omega+\gamma)t} - 1\right]$$

$$V(t) \leq V_0 + V(0)e^{-(\mu+\omega+\gamma)t}, \quad \text{where} \quad V_0 = \frac{\Lambda\gamma}{\mu(\mu+\omega+\gamma)}.$$

Let $\varepsilon_2 = \frac{(\mu+\gamma)\varepsilon_3}{\omega}$. For every $\varepsilon_2 > 0$, there exists a $t_3 > 0$ such that $V \leq V_0 + \varepsilon_2$ for all $t > t_3$. Thus, $\forall t > t_3$,

$$\frac{dS}{dt} = \Lambda + \omega V - \frac{\beta S I_d}{1+\alpha I_d} - (\mu+\gamma)S$$
$$\leq \Lambda + \omega\left(V_0 + \varepsilon_2\right) - (\mu+\gamma)S, \tag{3.17}$$

since $V \leq V_0 + \varepsilon_2$ and all associated variables and parameters are positive. Next, we have

$$\frac{d}{dt}\left[Se^{(\mu+\gamma)t}\right] \leqslant \left[\Lambda + \omega\left(\frac{\Lambda}{\mu} - S_0\right) + \omega\varepsilon_2\right]e^{(\mu+\gamma)t}, \quad \text{where}$$

$$S_0 = \frac{\Lambda(\mu+\omega)}{\mu(\mu+\omega+\gamma)}$$

$$S(t)e^{(\mu+\gamma)t} \leqslant S(0) + \frac{\Lambda(\mu+\omega) - \mu\omega S_0 + \mu\omega\varepsilon_2}{\mu(\mu+\gamma)}\left[e^{(\mu+\gamma)t} - 1\right]$$

$$S(t) \leqslant S_0 + \frac{\omega\varepsilon_2}{\mu+\gamma} + S(0)e^{-(\mu+\gamma)t}.$$

Let $\varepsilon_3 = \frac{\omega\varepsilon_2}{\mu+\gamma}$. For every $\varepsilon_3 > 0$, there exists a $t_4 > 0$ such that $S \leqslant S_0 + \varepsilon_3$ for all $t > t_4 > t_3$. By applying the comparison principle [52], $E, I \to 0$ as $t \to \infty$ since $I_d \to 0$ as $t \to \infty$. By applying the theory of asymptotic autonomous systems [51], from (3.16) and (3.17), we have $V \to V_0$ and $S \to S_0$ as $t \to \infty$, respectively. Therefore, E_1 is globally asymptotically stable if $R_0 < 1$, $\eta_d \leqslant \gamma_d$ and $\eta \leqslant \gamma$. □

Next, we would like to discuss the global stability of the EE, E_*, of model (2.1) by using the geometric approach proposed by Li and Muldowney [53]. This approach has been commonly applied to three- or four-dimensional systems [53–58]. However, we aim to expand its application to an eight-dimensional model (2.1). We briefly present some preliminaries on the geometric approach developed by Li and Muldowney [53] in proving global stability. These preliminaries are summarized from [53]. Otherwise, we will specify it.

Let $\tilde{\Omega}$ denote the interior of Ω and $E_* \in \tilde{\Omega}$ if $R_0 > 1$. Consider the autonomous ordinary differential equation

$$\frac{dx}{dt} = f(x), \tag{3.18}$$

where $x \mapsto f(x) \in \mathbb{R}^n$ is a C^1 function for x in an open set $D \subset \mathbb{R}^n$. Let x^*, x_0 and $x(t, x_0)$ respectively denote an equilibrium point, initial point and solution of (3.18) such that $x(0, x_0) = x_0$ is satisfied.

Assume that the following hypotheses hold:

(H1) D is simply connected;
(H2) There exists a compact absorbing set $\tilde{D} \subset D$;
(H3) Eq. (3.18) has a unique equilibrium x^* in D.

$P(x)$ is a $\binom{n}{2} \times \binom{n}{2}$ C^1 matrix-valued function defined on a domain D. $P^{-1}(x)$ exists and is continuous on a subset $\tilde{D} \subset D$.

The Lozinskiĭ measure $\tilde{\sigma}(B)$ of a matrix B with respect to a vector norm $\|\cdot\|$ in $\mathbb{R}^{\binom{n}{2}}$ is defined as:

$$\tilde{\sigma}(B) = \lim_{h\to 0^+} \frac{\|I + hB\| - 1}{h},$$

where I is the identity matrix and matrix B is given by

$$B = P_f P^{-1} + PJ^{[2]}P^{-1}.$$

Here P_f is the matrix obtained by taking the derivative of each entry of P along the direction of a vector field f and $J^{[2]}$ represents the second additive compound matrix associated with the Jacobian matrix $J(x)$. This measure is used to quantify the growth rate of the norm of $I + hB$ as $h \to 0^+$.

The quantity q is defined as:

$$q = \lim_{t\to\infty} \sup_{x_0\in\tilde{D}} \frac{1}{t}\int_0^t \tilde{\sigma}(B(x(0,x_0)))\,d\theta,$$

where $x(0, x_0)$ represents the solution of a dynamical system starting from an initial condition x_0. This quantity captures the asymptotic supremum of the average Lozinskiĭ measure of B along trajectories.

Lemma 6 ([53]). *Assume that hypotheses (H1)–(H3) hold and (3.18) satisfies a Bendixson criterion that is robust under C^1 local perturbations of f at all non-equilibrium non-wandering points of (3.18). Then x^* is globally asymptotically stable with respect to D provided it is stable.*

Theorem 7. *The endemic equilibrium, E_*, of model (2.1) is globally asymptotically stable in the interior if $R_0 > 1$, $\sigma_d + \eta_d \geq \gamma_d$, $\sigma + \eta \geq \gamma$, $\gamma_d > \max\left\{\frac{\Lambda_d\beta_d}{\mu_d} - (2\mu_d + \sigma_d + \eta_d), \quad \sigma_d - (2\mu_d + \xi_d + \omega_d)\right\}$ and $\gamma > \max\left\{-(2\mu+\sigma+\eta), \quad \sigma - (2\mu+\xi+\omega)\right\}$.*

Proof. $\Omega = \left\{(S_d, E_d, V_d, I_d, S, E, V, I) \in \mathbb{R}^8_+ \mid \quad 0 < S_d + E_d + V_d + I_d \leq \frac{\Lambda_d}{\mu_d} \text{ and } 0 < S + E + V + I \leq \frac{\Lambda}{\mu}\right\}$ is simply connected in $\mathbb{R}^8_+$. By Theorems 2 and 4, E_* is a unique endemic equilibrium of model (2.1) that exists in $\tilde{\Omega}$, the interior of Ω, and it is locally asymptotically stable if $R_0 > 1$. However, the disease-free equilibrium, E_1, is unstable whenever $R_0 > 1$. The uniform persistence of model (2.1) and the boundedness of Ω imply the existence of a compact absorbing set $\tilde{\Omega}$ in Ω [24,53,59]. Hence model (2.1) satisfies the assumptions (H1)–(H3).

Since $S_d + E_d + V_d + I_d \to \frac{\Lambda_d}{\mu_d}$ as $t \to \infty$, the dog-only population of model (2.1) is a three-dimensional asymptotically autonomous differential system with limit system

$$S_d'(t) = \Lambda_d(1-\nu_d) + \omega_d\left(\frac{\Lambda_d}{\mu_d} - S_d - E_d - I_d\right) - \frac{\beta_d S_d I_d}{1+\alpha I_d} - (\mu_d+\gamma_d)S_d,$$

$$E_d'(t) = \frac{\beta_d S_d I_d}{1+\alpha I_d} - (\sigma_d+\eta_d+\mu_d)E_d,$$

$$I_d'(t) = \sigma_d E_d - (\mu_d+\xi_d)I_d. \tag{3.19}$$

The Jacobian matrix of (3.19) is

$$\hat{J} = \begin{bmatrix} -\left(\frac{\beta_d I_d}{1+\alpha I_d} + \mu_d + \gamma_d + \omega_d\right) & -\omega_d & -\left(\frac{\beta_d S_d}{(1+\alpha I_d)^2} + \omega_d\right) \\ \frac{\beta_d I_d}{1+\alpha I_d} & -(\sigma_d+\eta_d+\mu_d) & \frac{\beta_d S_d}{(1+\alpha I_d)^2} \\ 0 & \sigma_d & -(\mu_d+\xi_d) \end{bmatrix},$$

whereas the second additive compound matrix [53–55] of model (3.19) is defined as

$$\hat{J}^{[2]} = \begin{bmatrix} \hat{J}_{11}+\hat{J}_{22} & \hat{J}_{23} & -\hat{J}_{13} \\ \hat{J}_{32} & \hat{J}_{11}+\hat{J}_{33} & \hat{J}_{12} \\ -\hat{J}_{31} & \hat{J}_{21} & \hat{J}_{22}+\hat{J}_{33} \end{bmatrix},$$

where

$$\hat{J}_{11} + \hat{J}_{22} = -\left[\frac{\beta_d I_d}{1+\alpha I_d} + (\mu_d+\gamma_d+\omega_d) + (\sigma_d+\eta_d+\mu_d)\right],$$

$$\hat{J}_{23} = \frac{\beta_d S_d}{(1+\alpha I_d)^2},$$

$$-\hat{J}_{13} = \frac{\beta_d S_d}{(1+\alpha I_d)^2} + \omega_d, \hat{J}_{32} = \sigma_d,$$

$$\hat{J}_{11} + \hat{J}_{33} = -\left[\frac{\beta_d I_d}{1+\alpha I_d} + (\mu_d + \gamma_d + \omega_d) + \left(\mu_d + \xi_d\right)\right], \hat{J}_{12} = -\omega_d$$

$$-\hat{J}_{31} = 0, \quad \hat{J}_{21} = \frac{\beta_d I_d}{1+\alpha I_d}\hat{J}_{22} + \hat{J}_{33} = -\left[\left(\sigma_d + \eta_d + \mu_d\right) + \left(\mu_d + \xi_d\right)\right].$$

Let $P = \operatorname{diag}\left\{\frac{I_d}{E_d}, \frac{I_d}{E_d}, \frac{I_d}{E_d}\right\}$. Then $P^{-1} = \operatorname{diag}\left\{\frac{E_d}{I_d}, \frac{E_d}{I_d}, \frac{E_d}{I_d}\right\}$, $P_f = \operatorname{diag}\left\{\frac{I_d}{E_d}\left(\frac{I'_d}{I_d} - \frac{E'_d}{E_d}\right), \frac{I_d}{E_d}\left(\frac{I'_d}{I_d} - \frac{E'_d}{E_d}\right), \frac{I_d}{E_d}\left(\frac{I'_d}{I_d} - \frac{E'_d}{E_d}\right)\right\}$, and $P_f P^{-1} = \operatorname{diag}\left\{\left(\frac{I'_d}{I_d} - \frac{E'_d}{E_d}\right), \left(\frac{I'_d}{I_d} - \frac{E'_d}{E_d}\right), \left(\frac{I'_d}{I_d} - \frac{E'_d}{E_d}\right)\right\}$.

The matrix $B = P_f P^{-1} + PJ^{[2]}P^{-1}$ can be written in the following form:

$$B = \begin{pmatrix} b_{1,1} & \frac{\beta_d S_d}{(1+\alpha I_d)^2} & \beta_d S_d (1+\alpha I_d)^2 + \omega_d \\ \sigma_d & b_{2,2} & -\omega_d \\ 0 & \frac{\beta_d I_d}{1+\alpha I_d} & b_{3,3} \end{pmatrix},$$

where $b_{1,1} = \left(\frac{I'_d}{I_d} - \frac{E'_d}{E_d}\right) - \left[\hat{J}_{11} + \hat{J}_{22}\right]$,

$b_{2,2} = \left(\frac{I'_d}{I_d} - \frac{E'_d}{E_d}\right) - \left[\hat{J}_{11} + \hat{J}_{33}\right]$ and

$b_{3,3} = \left(\frac{I'_d}{I_d} - \frac{E'_d}{E_d}\right) - \left[\hat{J}_{22} + \hat{J}_{33}\right]$.

Let $z = (z_1, z_2, z_3)$ be the vector in $\mathbb{R}^3 \cong \mathbb{R}^{\binom{n}{2}}$. We choose a norm in $\mathbb{R}^3$ to be

$$|(z_1, z_2, z_3)| = \max\left\{|z_1|, |z_2| + |z_3|\right\}.$$

Let $\tilde{\sigma}(B)$ be the Lozinskiĭ measure with respect to this norm. Then, by applying the logarithmic norm method [54], we have

$$\tilde{\sigma}(B) \le \max\{g_1, g_2\},$$

where

$$g_1 = \tilde{\sigma}_1(b_{1,1}) + |b_{1,2}|, \quad g_2 = \tilde{\sigma}_1(b_{2,2}) + |b_{2,1}|,$$

with $|b_{1,2}|, |b_{2,1}|$ the matrix norms with respect to the l_1 vector norm. More specifically,

$$\tilde{\sigma}_1(b_{1,1}) = \frac{I'_d}{I_d} - \frac{E'_d}{E_d} - \left[\hat{J}_{11} + \hat{J}_{22}\right],$$

$$|b_{1,2}| = \max\left\{\frac{\beta_d S_d}{(1+\alpha I_d)^2}, \left(\frac{\beta_d S_d}{(1+\alpha I_d)^2} + \omega_d\right)\right\}$$

$$= \frac{\beta_d S_d}{(1+\alpha I_d)^2} + \omega_d,$$

$$|b_{2,1}| = \sigma_d.$$

$$\tilde{\sigma}_1(b_{2,2}) = \max\left\{\frac{I'_d}{I_d} - \frac{E'_d}{E_d} - \left[(\mu_d + \gamma_d + \omega_d) + \left(\mu_d + \xi_d\right)\right], \frac{I'_d}{I_d} - \frac{E'_d}{E_d} - \left[\left(\sigma_d + \eta_d + \mu_d\right) + \left(\mu_d + \xi_d\right)\right] - \omega_d\right\}$$

$$= \frac{I'_d}{I_d} - \frac{E'_d}{E_d} - \left(2\mu_d + \xi_d + \omega_d\right) + \max\{-\gamma_d, -(\sigma_d + \eta_d)\}.$$

Therefore, we have

$$g_1 = \tilde{\sigma}_1(b_{1,1}) + |b_{1,2}|$$

$$= \frac{I'_d}{I_d} - \frac{E'_d}{E_d} - \left[\frac{\beta_d I_d}{1+\alpha I_d} + (\mu_d + \gamma_d) + \left(\sigma_d + \eta_d + \mu_d\right)\right] + \frac{\beta_d S_d}{(1+\alpha I_d)^2}$$

$$\le \frac{I'_d}{I_d} - \frac{E'_d}{E_d} - \left(2\mu_d + \sigma_d + \eta_d + \gamma_d\right) + \frac{\Lambda_d \beta_d}{\mu_d},$$

where $1 + \alpha I_d \ge 1, S_d \le N_d \le \frac{\Lambda_d}{\mu_d}$ and all associated parameters and variables are positive.

$$g_2 = \tilde{\sigma}_1(b_{2,2}) + |b_{2,1}|$$

$$= \sigma_d + \frac{I'_d}{I_d} - \frac{E'_d}{E_d} - \left(2\mu_d + \xi_d + \omega_d\right) + \max\{-\gamma_d, -(\sigma_d + \eta_d)\} + \sigma_d$$

$$= \frac{I'_d}{I_d} - \frac{E'_d}{E_d} - \left(2\mu_d + \xi_d + \omega_d + \gamma_d\right) + \sigma_d \quad \text{if} \quad \sigma_d + \eta_d \ge \gamma_d.$$

Let $\tilde{a} = \min\left\{2\mu_d + \sigma_d + \eta_d + \gamma_d - \frac{\Lambda_d \beta_d}{\mu_d}, \quad 2\mu_d + \xi_d + \omega_d + \gamma_d - \sigma_d\right\}$. Suppose

$$\gamma_d > \max\left\{\frac{\Lambda_d \beta_d}{\mu_d} - (2\mu_d + \sigma_d + \eta_d), \quad \sigma_d - (2\mu_d + \xi_d + \omega_d)\right\}.$$

Thus we have $\tilde{a} > 0$. Then

$$g_1 \le \frac{I'_d}{I_d} - \frac{E'_d}{E_d} - \tilde{a},$$

$$g_2 \le \frac{I'_d}{I_d} - \frac{E'_d}{E_d} - \tilde{a} \quad \text{if} \quad \sigma_d + \eta_d \ge \gamma_d.$$

Along each solution $\left(S_d(t), E_d(t), I_d(t)\right)$ of model (2.1) with arbitrary initial condition $x_0 = \left(S_d(0), E_d(0), I_d(0)\right)$ in $\tilde{\Omega}$, we have

$$\frac{1}{t}\int_0^t g_1\, d\theta, \quad \frac{1}{t}\int_0^t g_2\, d\theta \le -\tilde{a} + \frac{1}{t}\left\{\ln\left[\frac{I_d(t)}{I_d(0)}\right] - \ln\left[\frac{E_d(t)}{E_d(0)}\right]\right\}.$$

Hence

$$\frac{1}{t}\int_0^t \tilde{\sigma}(B)\, d\theta \le \max\left\{-\tilde{a} + \frac{1}{t}\left(\ln\left[\frac{I_d(t)}{I_d(0)}\right] - \ln\left[\frac{E_d(t)}{E_d(0)}\right]\right)\right\}.$$

Therefore,

$$\limsup_{t\to\infty} \sup_{x_0 \in \tilde{\Omega}} \frac{1}{t}\int_0^t \tilde{\sigma}(B)\, d\theta \le -\tilde{a} < 0 \quad \text{since} \quad \tilde{a} > 0.$$

Since $\left(S_d, E_d, I_d, V_d\right) \to \left(S_d^*, E_d^*, I_d^*, V_d^*\right)$ and $S + E + I + V \to \frac{\Lambda}{\mu}$ as $t \to \infty$, the humans-only model (final four equations of model (2.1)) is a three-dimensional asymptotically autonomous differential system with limit system

$$S'(t) = \Lambda + \omega\left(\frac{\Lambda}{\mu} - S - E - I\right) - \frac{\beta S I_d}{1+\alpha I_d} - (\mu + \gamma)S,$$

$$E'(t) = \frac{\beta S I_d}{1+\alpha I_d} - (\sigma + \eta + \mu)E,$$

$$I'(t) = \sigma E - (\mu + \xi)I. \tag{3.20}$$

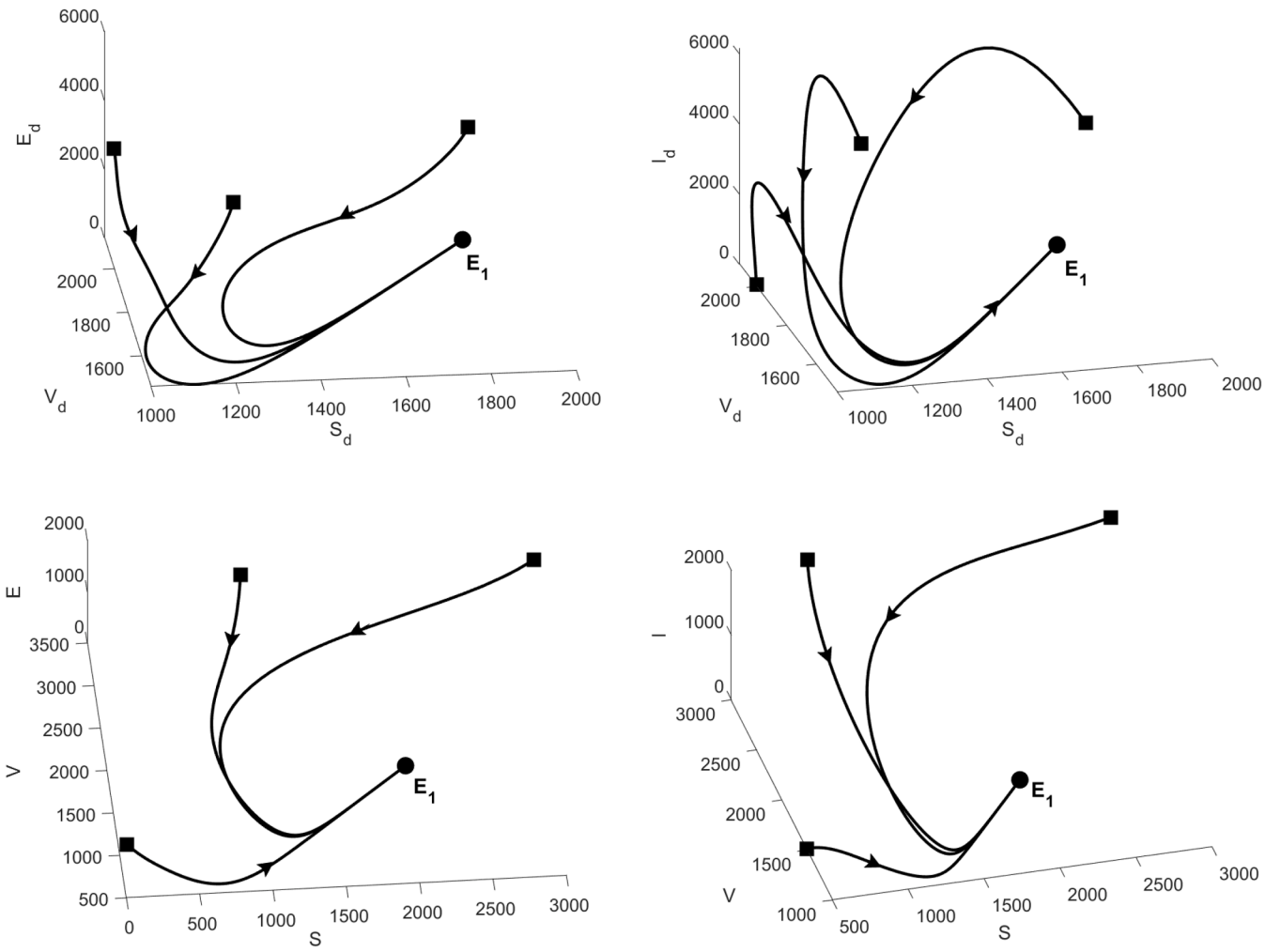

Fig. 2. All trajectories converge to the disease-free equilibrium (E_1) whenever $R_0 < 1$, $\eta_d \leq \gamma_d$ and $\eta \leq \gamma$.

The Jacobian matrix of (3.20) is given as follows:

$$J_h = \begin{bmatrix} -\left(\dfrac{\beta I_d^*}{1+\alpha {I_d}^*} + \omega + \mu + \gamma\right) & -\omega & -\omega \\ \dfrac{\beta I_d^*}{1+\alpha I_d^*} & -(\sigma+\eta+\mu) & 0 \\ 0 & \sigma & -(\mu+\xi) \end{bmatrix}.$$

The second additive compound matrix of J_h is defined as

$$J_h^{[2]} = \begin{bmatrix} -\left[\frac{\beta I_d^*}{1+\alpha I_d^*} + (2\mu+\omega+\gamma+\sigma+\eta)\right] & 0 & \omega \\ \sigma & -\left[\frac{\beta I_d^*}{1+\alpha I_d^*} + (\omega+2\mu+\gamma+\xi)\right] & -\omega \\ 0 & \frac{\beta I_d^*}{1+\alpha I_d^*} & -(2\mu+\xi+\sigma+\eta) \end{bmatrix}.$$

Let

$$\hat{P} = \text{diag}\left\{\frac{I}{E}, \frac{I}{E}, \frac{1}{E}\right\},$$

$$\hat{P}_f = \text{diag}\left\{\frac{I}{E}\left(\frac{I'}{I} - \frac{E'}{E}\right), \frac{I}{E}\left(\frac{I'}{I} - \frac{E'}{E}\right), \frac{I}{E}\left(\frac{I'}{I} - \frac{E'}{E}\right)\right\},$$

$$\hat{P}^{-1} = \text{diag}\left\{\frac{E}{I}, \frac{E}{I}, \frac{E}{I}\right\}$$

$$\hat{P}_f \hat{P}^{-1} = \text{diag}\left\{\frac{I'}{I} - \frac{E'}{E}, \frac{I'}{I} - \frac{E'}{E}, \frac{I'}{I} - \frac{E'}{E}\right\},$$

where $\hat{P}_f$ is the matrix obtained by taking the derivative of each entry of $\hat{P}$ along the direction of a vector field f.

The matrix $\hat{B} = \hat{P}_f \hat{P}^{-1} + \hat{P} J_h^{[2]} \hat{P}^{-1}$ can be written in the following form:

$$\hat{B} = \begin{pmatrix} \hat{b}_{1,1} & 0 & \omega \\ \sigma & \hat{b}_{2,2} & -\omega \\ 0 & \dfrac{\beta I_d^*}{1+\alpha I_d^*} & \hat{b}_{3,3} \end{pmatrix},$$

where

$$\hat{b}_{1,1} = \frac{I'}{I} - \frac{E'}{E} - \left[\frac{\beta I_d^*}{1+\alpha I_d^*} + (2\mu+\omega++\gamma+\sigma+\eta)\right],$$

$$\hat{b}_{2,2} = \frac{I'}{I} - \frac{E'}{E} - \left[\frac{\beta I_d^*}{1+\alpha I_d^*} + (2\mu+\gamma+\omega+\xi)\right]$$

$$\hat{b}_{3,3} = \frac{I'}{I} - \frac{E'}{E} - (2\mu+\xi+\sigma+\eta).$$

By using the same approach as in the dog population, we obtain

$$\hat{\sigma}_1(\hat{b}_{11}) = \frac{I'}{I} - \frac{E'}{E} - \left[\frac{\beta I_d^*}{1+\alpha I_d^*} + (2\mu+\omega++\gamma+\sigma+\eta)\right]$$

$$\left|\hat{b}_{12}\right| = \omega$$

$$\hat{g}_1 = \hat{\sigma}_1(\hat{b}_{11}) + \left|\hat{b}_{12}\right|$$

$$\hat{g}_1 = \frac{I'}{I} - \frac{E'}{E} - \left[\frac{\beta I_d^*}{1+\alpha I_d^*} + (2\mu+\omega++\gamma+\sigma+\eta)\right] + \omega$$

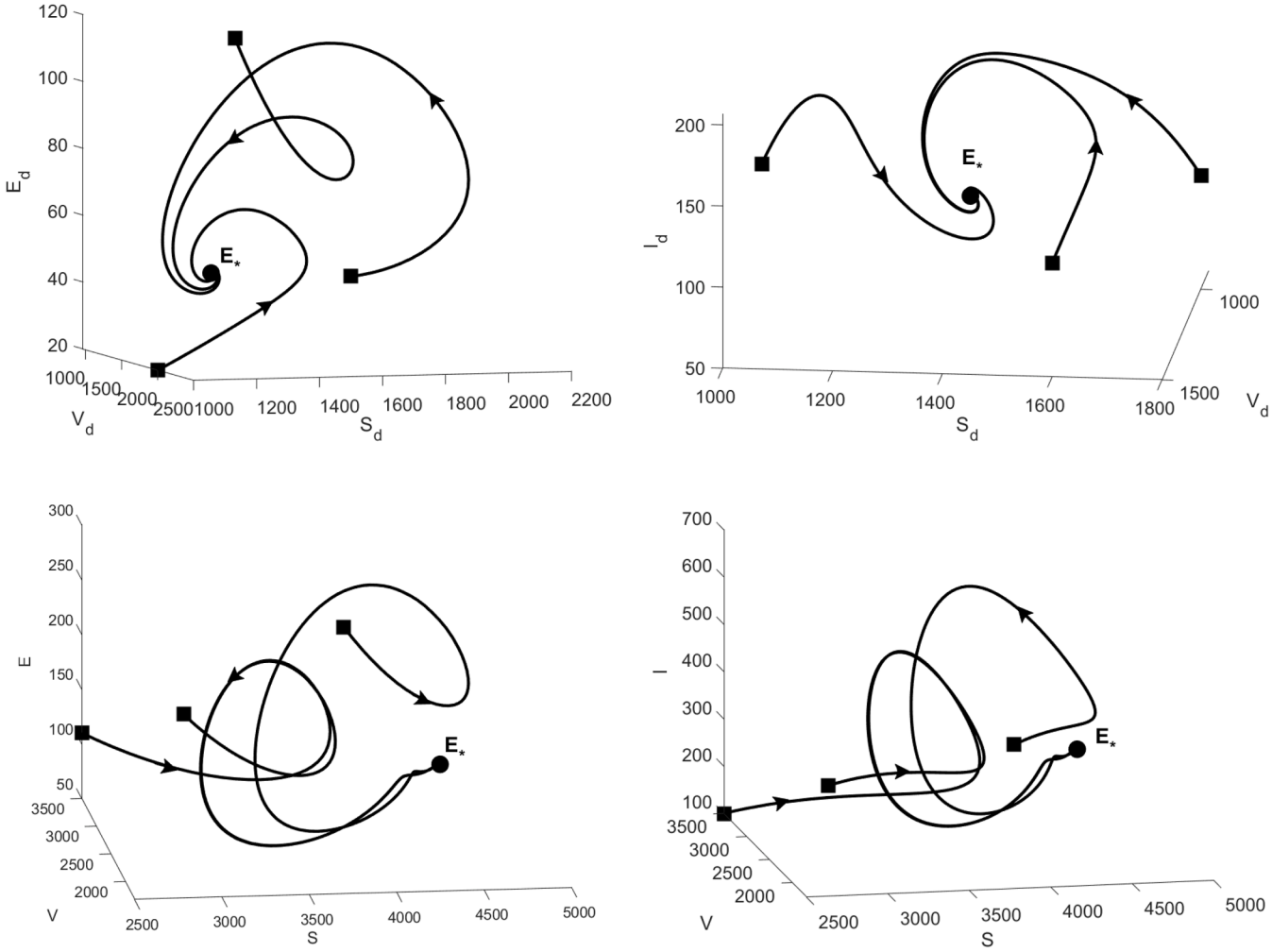

Fig. 3. All trajectories converge to the endemic equilibrium (E_*) whenever $R_0 > 1$ and all conditions as in Theorem 7 are satisfied.

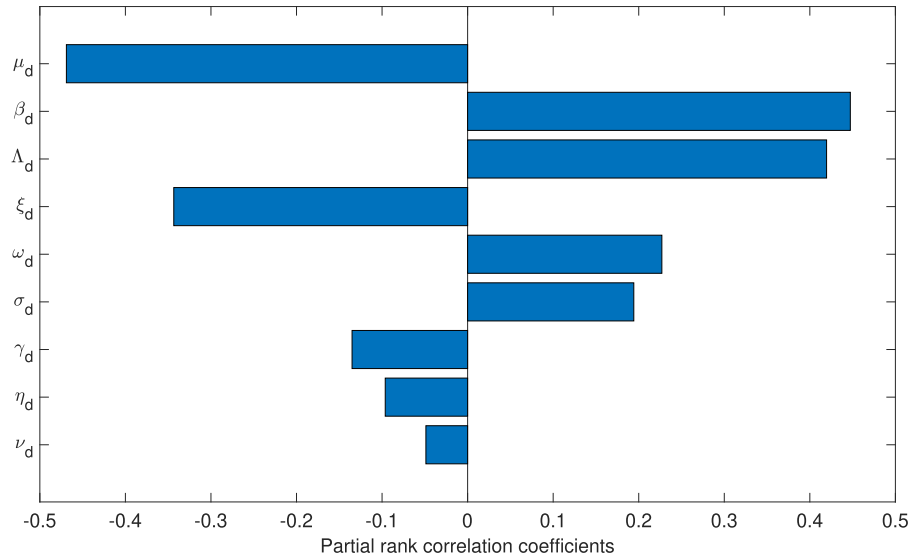

Fig. 4. Tornado plot illustrating the partial rank correlation coefficients (PRCCs) with R_0 as the output variable. The three parameters with the greatest impact on the reproduction number are the birth, death and transmission rates of dogs.

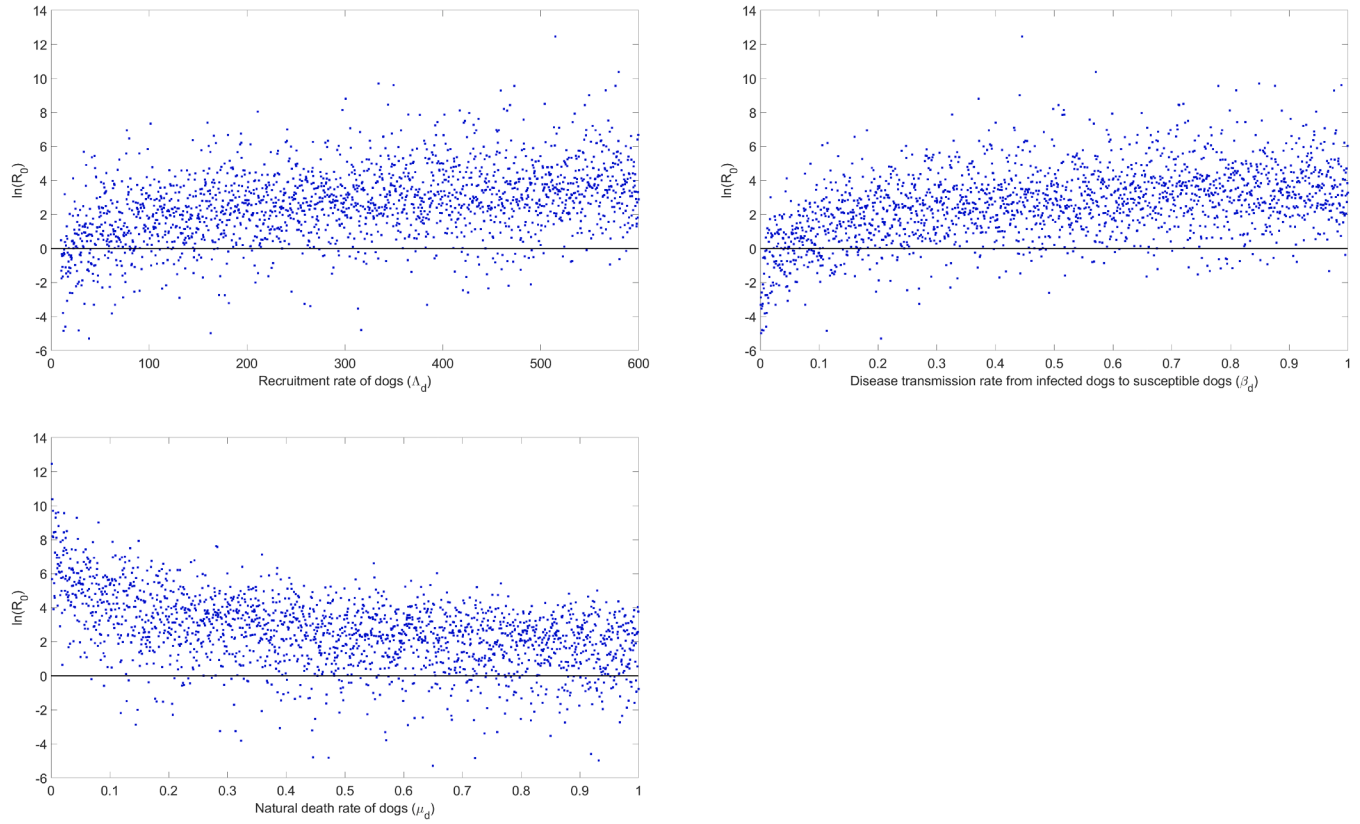

Fig. 5. Monte Carlo simulations illustrating sensitivity of R_0 to the three parameters (Λ_d, β_d and μ_d) that have the greatest influence on the disease.

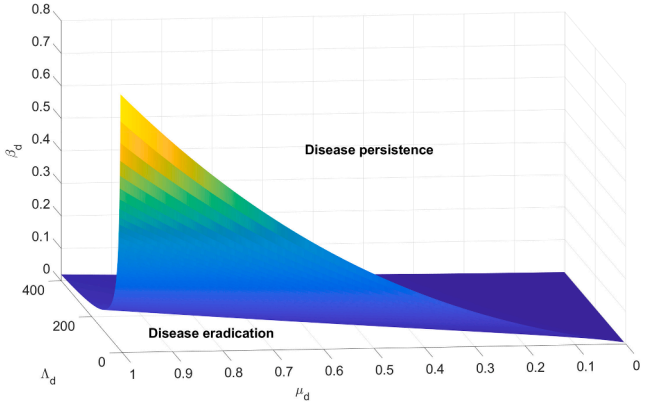

Fig. 6. The surface $R_0 = 1$ and the influence of the three most influential parameters (Λ_d, β_d and μ_d). Disease eradication is possible for parameter values below the surface, whereas the disease persists for parameter values above the surface.

$$\leq \frac{I'}{I} - \frac{E'}{E} - [2\mu + \sigma + \gamma + \eta],$$

since all associated parameters are positive. Next, we have

$$\left|\hat{b}_{21}\right| = \sigma$$

$$\hat{\sigma}_1(\hat{b}_{22}) = \max\left\{ \frac{I'}{I} - \frac{E'}{E} - (2\mu + \gamma + \omega + \xi), \right.$$
$$\left. \frac{I'}{I} - \frac{E'}{E} - (2\mu + \xi + \sigma + \eta) - \omega \right\}$$
$$= \frac{I'}{I} - \frac{E'}{E} - (2\mu + \xi + \omega) + \max\{-\gamma, -(\sigma + \eta)\}$$

$$\hat{g}_2 = \left|\hat{b}_{21}\right| + \hat{\sigma}_1(\hat{b}_{22})$$

$$\hat{g}_2 = \frac{I'}{I} - \frac{E'}{E} - (2\mu + \xi + \omega + \gamma) + \sigma \quad \text{if} \sigma + \eta \geqslant \gamma.$$

Let $\hat{a} = \min\left\{2\mu + \sigma + \gamma + \eta, \quad 2\mu + \xi + \omega + \gamma - \sigma\right\}$. Suppose $\gamma > \max\left\{-(2\mu + \sigma + \eta), \quad \sigma - (2\mu + \xi + \omega)\right\}$. Thus we have $\hat{a} > 0$. Then

$$\hat{g}_1 \leqslant \frac{I'}{I} - \frac{E'}{E} - \hat{a},$$

$$\hat{g}_2 = \frac{I'}{I} - \frac{E'}{E} - \hat{a} \quad \text{if} \quad \sigma + \eta \geq \gamma.$$

Along each solution $(S(t), E(t), I(t))$ of model (3.20) with arbitrary initial condition $\hat{x}_0 = \left(S(0), E(0), I(0)\right)$ in $\tilde{\Omega}$, we have

$$\frac{1}{t}\int_0^t g_1\, d\theta, \quad \frac{1}{t}\int_0^t g_2\, d\theta \leq -\tilde{a} + \frac{1}{t}\left\{ \ln\left[\frac{I(t)}{I(0)}\right] - \ln\left[\frac{E(t)}{E(0)}\right] \right\}.$$

Hence

$$\frac{1}{t}\int_0^t \tilde{\sigma}(B)\, d\theta \leq \max\left\{ -\tilde{a} + \frac{1}{t}\left(\ln\left[\frac{I(t)}{I(0)}\right] - \ln\left[\frac{E(t)}{E(0)}\right] \right) \right\},$$

and

$$\limsup_{t\to\infty} \sup_{\hat{x}_0 \in \tilde{\Omega}} \frac{1}{t}\int_0^t \tilde{\sigma}(B)\, d\theta \leq -\tilde{a} < 0 \quad \text{since} \quad \tilde{a} > 0.$$

Therefore, the endemic equilibrium of model (2.1) is globally asymptotically stable if $R_0 > 1$, $\sigma_d + \eta_d \geq \gamma_d$ and $\sigma + \eta \geq \gamma$. □

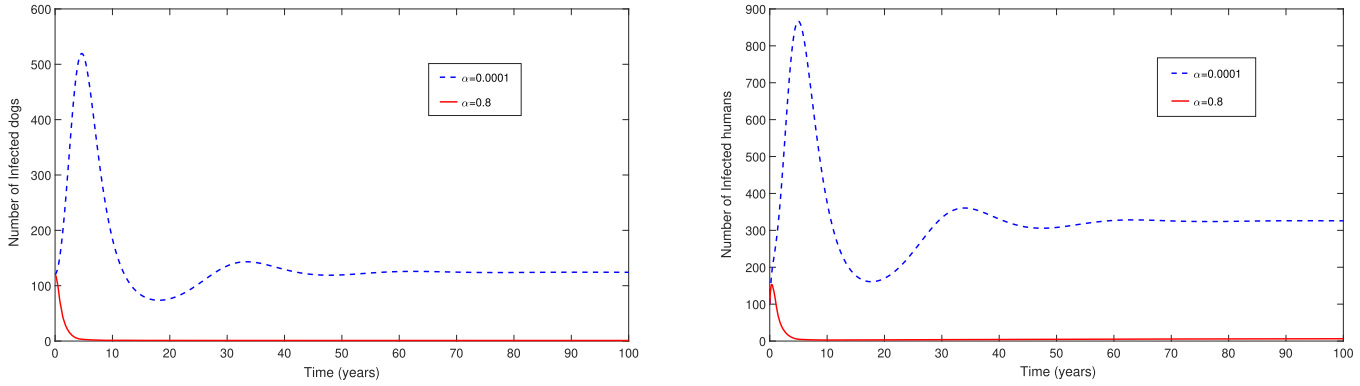

Fig. 7. The transmission dynamics of infected dogs and humans in model (2.1) with varying α values when $R_0 > 1$.

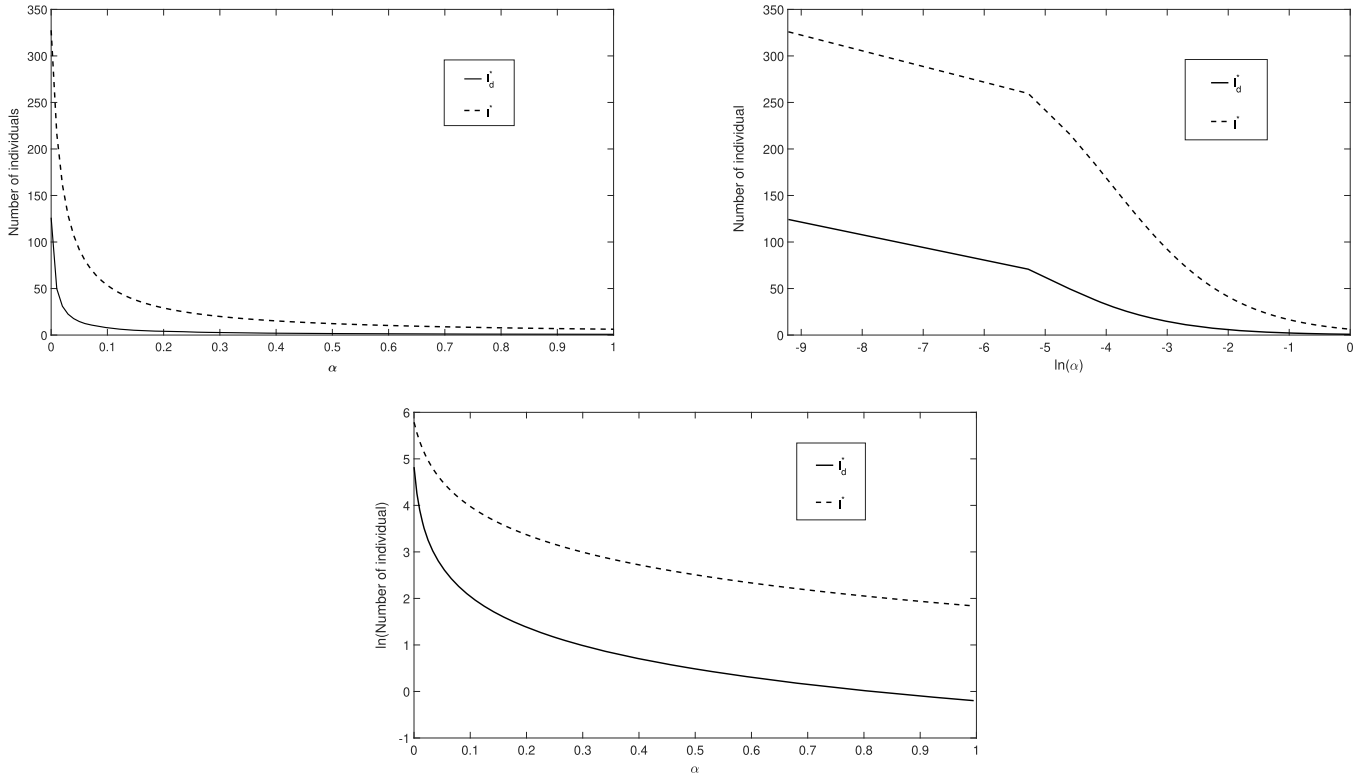

Fig. 8. The values of I_d^* and I^* of model (2.1) as α varies when $R_0 > 1$.

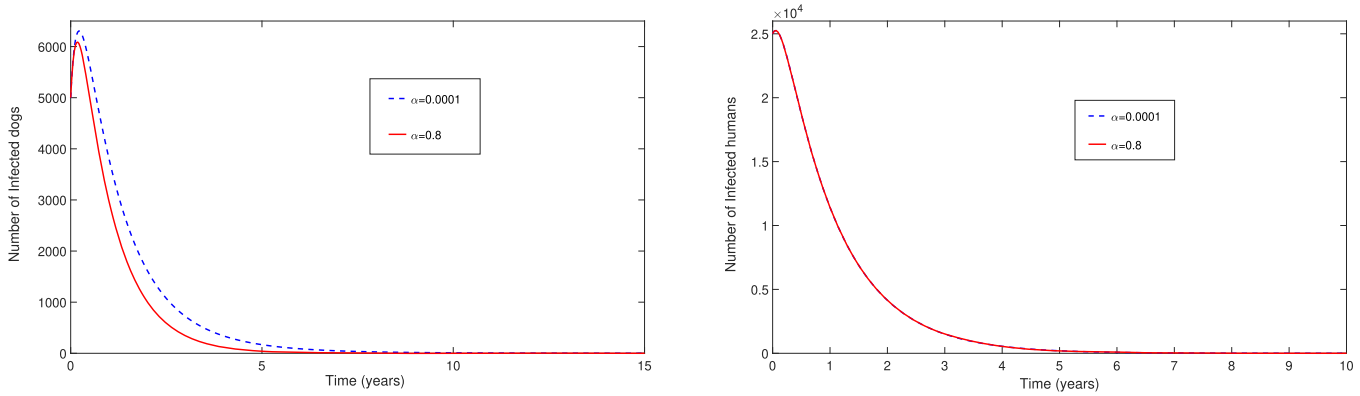

Fig. 9. The transmission dynamics of infected dogs and humans in model (2.1) with varying α values when $R_0 < 1$.

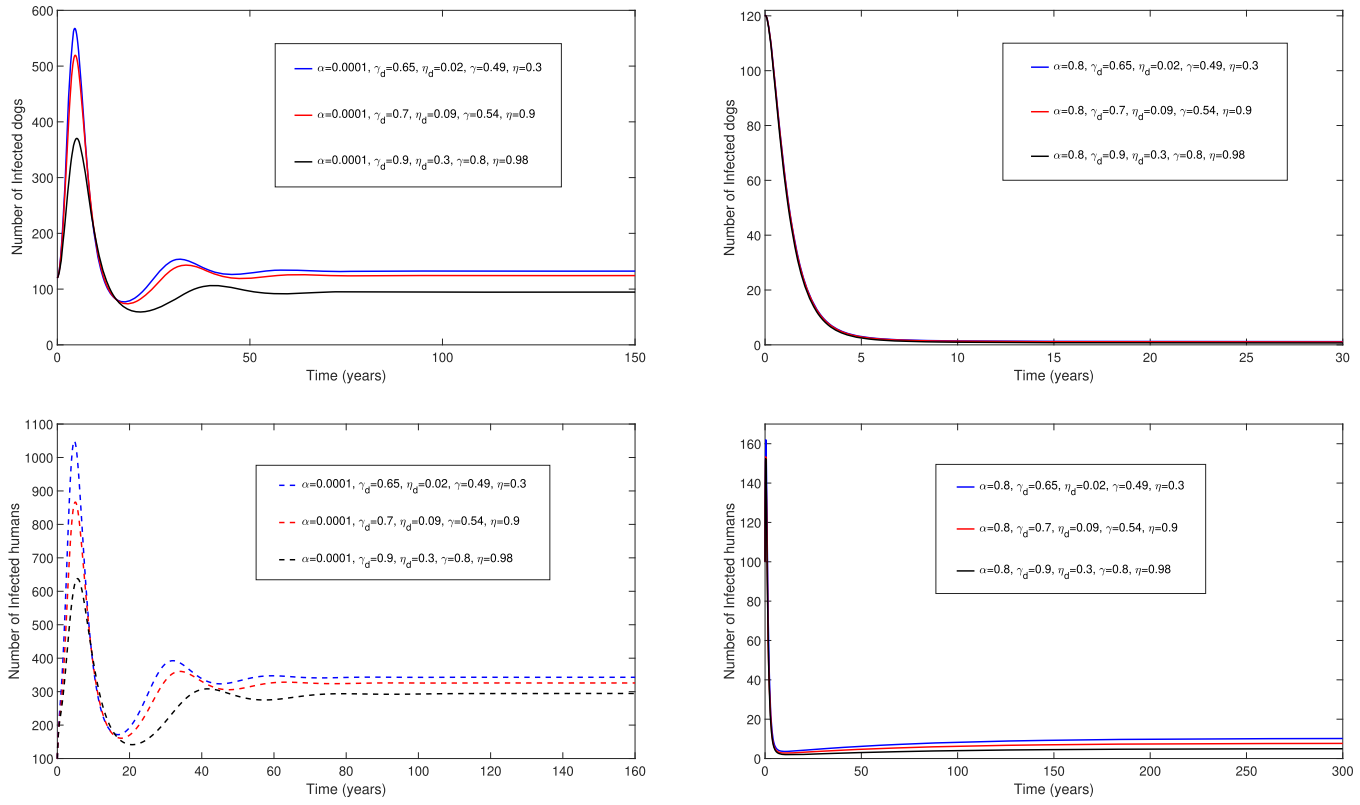

Fig. 10. Effect of vaccination with fixed α when $R_0 > 1$.

4. Numerical simulations

4.1. Stability of equilibria

We conduct numerical simulations of model (2.1) depicting the transmission dynamics of rabies in the dog and human populations in order to validate our results using the parameter values in Table 1. We represent the initial conditions using the symbol ■ and equilibrium points by ●. Parameters are given in Table 1.

From Fig. 2, we observe that all trajectories with arbitrary initial conditions are converging to E_1 as $t \to \infty$ whenever the conditions $R_0 < 1$, $\eta_d \leq \gamma_d$ and $\eta \leq \gamma$ are satisfied. Fig. 3 shows that all trajectories converge to E_* as $t \to \infty$ if $R_0 > 1$ and all conditions stated in Theorem 7 are satisfied.

4.2. Sensitivity analysis

We used Latin Hypercube Sampling (LHS) and partial rank correlation coefficients (PRCCs) to conduct a sensitivity analysis. LHS selects parameter values from a random grid without replacement, ensuring each row and column is used only once. Since there is limited or no empirical data available for the associated parameters of the basic reproduction number, R_0, but their feasible ranges are known, we default to assuming uniform distributions [60,61]. Suppose the range of each of the input variables x_i $(i = 1, 2, \ldots, k)$ is divided into n non-overlapping intervals of equal probability $\frac{1}{n}$. From each interval, one value is sampled uniformly at random. The sampled values for each variable are then independently shuffled to eliminate any correlation between variables. To ensure uniqueness, n distinct sample points are constructed by combining the shuffled values across all variables. (For more details, see [60–64].)

PRCCs are then used to assess the sensitivity of R_0 to each parameter by ranking their effects while holding all other parameters at median values. This approach has been widely applied in epidemiological modelling to determine the parameters that most significantly influence disease transmission and control [65–70].

In this study, we perform LHS with 2000 simulations per run to ensure conclusive results and allow us to observe distinct patterns. The ranges of the associated parameters that are used in performing the simulation are given in Table 2 unless otherwise stated.

Fig. 4 shows that there are three parameters out of nine that have the greatest effect in controlling disease transmission: Λ_d, β_d and μ_d. R_0 increases as Λ_d and β_d increase and decreases as μ_d increases. Fig. 5 illustrates the sensitivity analysis of R_0 to these three parameters. From this figure, it is more promising to control the spreading of rabies by reducing Λ_d and β_d values. However, we are unlikely to achieve disease extinction by increasing μ_d.

By setting $R_0 = 1$ and fixing all associated parameters at their sample values as stated in Table 2, we would like to further examine these three important parameters. The resulting surface is shown in Fig. 6. The combination of three parameters above the surface will lead to disease persistence, whereas the disease is likely to die off for parameter values below the surface. In addition, the R_0 value is directly proportional to Λ_d and β_d. Hence, if we could control the recruitment rate of dogs and the disease transmission rate from infected dogs to susceptible dogs, then $R_0 < 1$. However, such control is difficult to achieve in practice, and the actual R_0 values in the field may be higher than previously thought [71]. We thus turn to investigating other possibilities for control when $R_0 > 1$.

4.3. Including incidence

It follows from Fig. 5 that we can only achieve disease control if the transmission rate is very small or the recruitment rate is tiny. Culling dogs does not even cross the threshold. A larger issue is that the majority of Monte Carlo simulations in Fig. 5 are above the threshold, suggesting that actually eradicating rabies is unlikely except with extreme control

Table 2
Sample values and ranges of associated parameters in performing sensitivity analysis of R_0.

Parameter	Value	Unit	Source	Range
Λ_d	325	$\frac{\text{individuals}}{\text{year}}$	[40]	[10, 600]
β_d	5.2×10^{-4}	year^{-1}	Estimation	[0.0001, 1]
μ_d	0.0833	year^{-1}	[41]	[0.001, 1]
σ_d	6	year^{-1}	[27]	[0.01, 15]
ξ_d	1	year^{-1}	[27]	[0.01, 10]
ν_d	0.9	unitless	Estimation	[0.01, 1]
ω_d	0.5	year^{-1}	[28]	[0.01, 10]
γ_d	0.5	year^{-1}	[44]	[0.01, 10]
η_d	0.09	year^{-1}	[27]	[0.001, 5]

measures. Furthermore, we know that the disease persists in reality, so eradication is unlikely. Instead, we examine disease management in the case of viral persistence.

A next-level approach is to examine the effect of changing α on the outcome. It should be noted that R_0 does not depend on the form of the incidence function or the parameter α. This is because R_0 is derived from a linearisation of the model around equilibria; nonlinear terms in the model, such as those describing saturated incidence, play no part in the calculation of R_0. The parameter α corresponds to either changing behaviour among susceptible humans as they avoid contacts due to rising infections or reducing dog contacts through targeted interventions.

Fig. 7 shows that changes in the inhibitory effect, α, significantly affect the number of infected dogs and humans when $R_0 > 1$. We can see that by increasing the α value, the saturated levels of model (2.1), $\frac{\beta_d}{\alpha}$ and $\frac{\beta}{\alpha}$, are decreasing; hence, the number of infected dogs and humans are converging to a smaller steady state as $t \to \infty$. This is illustrated in more detail in Fig. 8, showing the reduction in the endemic equilibrium as α increased. In Fig. 7, for $\alpha = 0.8$, we observe that $I_d \to 1.0201$ and $I \to 5.8277$ as $t \to \infty$. Furthermore, Fig. 8 shows that, for instance, when $\alpha = 0.7$, $I^* \to 8.8705$ and $I_d^* \to 1.1643$, while for $\alpha = 1$, $I^* \to 6.2602$ and $I_d^* \to 0.8173$. These values do not approach zero when $R_0 > 1$ and hence do not contradict Theorem 7 or Fig. 3. However, in practice, if the value of the endemic equilibrium can be sufficiently lowered, then it is functionally indistinguishable from eradication.

Conversely, when $R_0 < 1$, Fig. 9 shows that varying the α values only results in a small difference in the number of infected dogs and humans, slightly accelerating disease elimination. By fixing the value of α and varying the vaccination rates (γ_d, η_d, γ and η), Fig. 10 illustrates that the impact of vaccination becomes negligible when α is sufficiently large and $R_0 > 1$. However, when α is small, vaccination proves to be effective: higher vaccination rates can reduce both the peak and the final size of the number of infected dogs and humans. It follows that inhibiting infection due to crowding is critical, even when $R_0 > 1$. This can be achieved in dogs through surveillance and control in humans through education, awareness and prevention programs. The inhibitory effect thus plays a crucial role when rabies is endemic, with the potential to eliminate the disease even when vaccination cannot.

5. Discussion

We proposed a rabies mathematical model with saturated incidence rate, governed by a set of nonlinear ordinary differential equations describing dog and human populations. By applying the comparison principle [51] and the theory of asymptotic autonomous systems [52], we proved global stability of the disease-free equilibrium (E_1) whenever $R_0 < 1$ and under the additional parameter restrictions $\eta_d \leq \gamma_d$ and $\eta \leq \gamma$. A geometric approach [53] was used to prove that the endemic equilibrium (E_*) achieves global stability whenever $R_0 > 1$. A sensitivity analysis determined the three parameters that play the most significant effect in controlling the outbreak: Λ_d, β_d and μ_d. This suggests that increasing vaccination programmes, public-awareness campaigns and dog surveillance and monitoring will reduce the prevalence of the disease. Rabies in dogs has been eliminated in countries such as the United Kingdom, Austria, Australia and Belgium [72], largely through successful vaccination efforts [73]. However, global eradication has not yet been achieved, despite robust and active intervention and control programs in countries like Mexico and Nicaragua [72].

However, it should be noted from Figs. 5 and 6 that the vast majority of parameter combinations lead to disease persistence. We capped Fig. 6 at $\Lambda_d = 400$ for ease of illustration, but the graph is mostly flat at zero for the range of Λ_d values included in Table 2. It follows that disease eradication is unlikely in practice, unless the birth rate of dogs is significantly reduced. However, eradication is not the only goal of rabies control; reducing R_0 will improve the outcome, even if the value of R_0 in practice does not cross the threshold. Even with $R_0 > 1$, increasing the inhibitory effect has a noticeable impact on disease control. It follows that rabies can be controlled with soft interventions, such as public-awareness campaigns, reduced contacts, animal curfews and fences. These interventions can control rabies better than current vaccination programs; see Fig. 10. The importance of "soft" interventions — such as raising public or community awareness and enhancing educational campaigns — has been qualitatively shown to be more effective in combating rabies than the implementation of mass rabies vaccination in dogs in some locales [74–76].

Our model has several limitations, which should be acknowledged. We considered dogs to be the only primary source of rabies transmission in our model (2.1), and both dog and human populations will become susceptible if the loss of immunity occurs. Moreover, we assumed that the recruitment rates of dog and human populations were constant and that a constant fraction ν_d of newly recruited dogs is vaccinated. We did not include human-to-human transmission in our model; such transmission, via saliva or bite, is theoretically feasible, but it has never been proven [22]. Our sensitivity analysis is robust within the range of parameter values chosen, but we did not explore values outside of these (plausible) ranges, which could potentially lead to some outlier results.

For future work, we suggest integrating spatial and temporal dynamics (spatially, the disease can spread through different geographical regions or within communities; temporally, the disease can have seasonal fluctuations), incorporation of vaccination hesitancy (despite the effectiveness of vaccination programs, vaccine hesitancy can be a significant barrier to achieve high vaccination coverage in the populations) and/or environmental factors (environmental factors such as temperature, humidity and habitat fragmentation can influence the spread of the disease).

Despite the availability of a vaccine and dog-culling programs, rabies is a difficult disease to eradicate. Birth, death and transmission rates among dogs are driving factors, while increasing the inhibitory effect α (a measure of psychological or inhibitory effect from behaviour changes or crowding) can result in a decrease in infections. This may produce a counter-intuitive result: as rabies-control programs reduce the number of infected dogs, the crowding effect may lighten, hampering these efforts. It follows that intervention methods may require careful monitoring for confounding effects.

Although we can prove local and global stability results for the eradication threshold, this is not very useful in practice. Instead, including saturated incidence in our model shows that the soft interventions matter: they have a tangible effect on disease control, even when $R_0 > 1$. It follows that integrating soft interventions into mathematical models is a crucial element of disease management.

CRediT authorship contribution statement

Nyuk Sian Chong: Writing – original draft, Supervision, Project administration, Methodology, Conceptualization; **Thanisha Kaliapan:** Writing – original draft, Investigation, Formal analysis; **Can Chen:**

Visualization, Software, Investigation, Formal analysis; **Kok Choon Cheah:** Validation, Resources, Data curation; **Stacey R. Smith?:** Writing – review & editing, Project administration, Methodology, Funding acquisition, Conceptualization.

Data availability

No data was used for the research described in the article.

Declaration of competing interest

We declare that we have no conflicts of interest.

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Appendix A. Local stability of the endemic equilibrium

We find that $a_4, a_5, a_6, a_7 > 0$ if $R_0 > 1$. For $a_6(a_4a_5 - a_6) - a_4^2a_7$, we have

$$
\begin{aligned}
&a_6(a_4a_5 - a_6) - a_4^2a_7\\
&= \mu_d(\mu_d + \gamma_d + \omega_d)(2\mu_d + \xi_d + \sigma_d + \eta_d)(3\mu_d + \xi_d + \sigma_d + \eta_d)W_1\\
&\quad + \mu_d(2\mu_d + \xi_d + \sigma_d + \eta_d)\Bigg\{(\mu_d + \gamma_d + \omega_d)W_2 + (3\mu_d + \xi_d + \sigma_d + \eta_d)\\
&\qquad \Big[W_3 + (\mu_d + \xi_d)(\mu_d + \omega_d)\Big]\Bigg\}\frac{\beta_d I_d^*}{1 + \alpha I_d^*}\\
&\quad + (4\mu_d + \xi_d + \sigma_d + \eta_d + \gamma_d + \omega_d)\Big[(\mu_d + \gamma_d + \omega_d)W_4 + \gamma_d(\mu_d + \xi_d)\\
&\qquad (\sigma_d + \mu_d)(3\mu_d + \xi_d + \sigma_d + \eta_d)\Big]\frac{\beta_d I_d^*}{1 + \alpha I_d^*}\\
&\quad + (\mu_d + \xi_d)(\sigma_d + \eta_d + \mu_d)W_9\left(\frac{\beta_d I_d^*}{1 + \alpha I_d^*}\right)\left(\frac{\alpha I_d^*}{1 + \alpha I_d^*}\right)\\
&\quad + (\mu_d + \xi_d)(\sigma_d + \eta_d + \mu_d)\Big[W_3 + W_5 + (\mu_d + \xi_d)(2\mu_d + \omega_d) + (2\mu_d + \xi_d)\\
&\qquad (\mu_d + \gamma_d + \omega_d)\Big]\frac{\alpha I_d^*}{1 + \alpha I_d^*}\left(\frac{\beta_d I_d^*}{1 + \alpha I_d^*}\right)^2\\
&\quad + (2\mu_d + \gamma_d + \omega_d)(\mu_d + \xi_d)^2(\sigma_d + \eta_d + \mu_d)^2\\
&\qquad \left(\frac{\beta_d I_d^*}{1 + \alpha I_d^*} + 2\mu_d + \xi_d + \sigma_d + \eta_d\right)\left(\frac{\alpha I_d^*}{1 + \alpha I_d^*}\right)^2\\
&\quad + (\mu_d + \xi_d)(\sigma_d + \eta_d + \mu_d)(2\mu_d + \gamma_d + \omega_d)(2\mu_d + \xi_d + \sigma_d + \eta_d)\\
&\qquad W_6\left(\frac{\alpha I_d^*}{1 + \alpha I_d^*}\right)\\
&\quad + W_{10}\left(\frac{\beta_d I_d^*}{1 + \alpha I_d^*}\right)^2 + \Big[(\mu_d + \xi_d)W_8 + (3\mu_d + \xi_d + \sigma_d + \eta_d + \omega_d)W_3\Big]\\
&\qquad \left(\frac{\beta_d I_d^*}{1 + \alpha I_d^*}\right)^3 > 0 \quad \text{if } R_0 > 1,
\end{aligned}
$$

where

$$
\begin{aligned}
W_1 &= (\mu_d + \gamma_d + \omega_d)(4\mu_d + \xi_d + \sigma_d + \eta_d + \gamma_d + \omega_d)\\
&\quad + \mu_d(2\mu_d + \xi_d + \sigma_d + \eta_d),\\
W_2 &= (\mu_d + \gamma_d)(3\mu_d + \xi_d + \sigma_d + \eta_d) + (\mu_d + \xi_d)(3\mu_d + \xi_d + \gamma_d + \omega_d)\\
&\quad + \omega_d(\mu_d + \eta_d),\\
W_3 &= \omega_d(\sigma_d + \mu_d) + (2\mu_d + \xi_d)(\sigma_d + \eta_d + \mu_d),\\
W_4 &= \omega_d(\mu_d + \xi_d)(3\mu_d + \xi_d + \eta_d) + (3\mu_d + \xi_d + \sigma_d + \eta_d)\Big[(\mu_d + \omega_d)\\
&\quad (\sigma_d + \mu_d) + \mu_d(\mu_d + \xi_d + \eta_d)\Big] + \mu_d^2(\mu_d + \xi_d) + \mu_d(\sigma_d + \eta_d + \mu_d)\\
&\quad (2\mu_d + \sigma_d + \eta_d + \omega_d),\\
W_5 &= 2\mu_d^2 + (\sigma_d + \eta_d + \omega_d)(2\mu_d + \gamma_d + \omega_d),\\
W_6 &= (\mu_d + \gamma_d + \omega_d)(2\mu_d + \xi_d + \sigma_d + \eta_d + \gamma_d + \omega_d)\\
&\quad + \mu_d(3\mu_d + \xi_d + \sigma_d + \eta_d),\\
W_7 &= \mu_d\Big[(2\sigma_d + 2\eta_d + \mu_d + \omega_d)(2\mu_d + \sigma_d + \eta_d + \omega_d) + (\mu_d + \xi_d)\\
&\quad (2\mu_d + \omega_d)\Big] + \omega_d\Big[\sigma_d^2 + \xi_d\eta_d + (\mu_d + \eta_d + \omega_d)(\mu_d + \xi_d + \sigma_d)\Big]\\
&\quad + \xi_d(\sigma_d + \eta_d)(2\mu_d + \sigma_d + \eta_d),\\
W_8 &= \mu_d(2\mu_d + \xi_d + \omega_d) + \omega_d(2\mu_d + \xi_d + \eta_d + \omega_d),\\
W_9 &= (2\mu_d + \xi_d + \sigma_d + \eta_d)\Big[W_3 + \mu_d\gamma_d + (\mu_d + \omega_d)(2\mu_d + \xi_d)\Big]\\
&\quad + (2\mu_d + \gamma_d + \omega_d)W_2 + (4\mu_d + \xi_d + \sigma_d + \eta_d + \gamma_d + \omega_d)W_5 \quad \text{and}\\
W_{10} &= \mu_d(\mu_d + \gamma_d + \omega_d)(2\mu_d + \xi_d + \sigma_d + \eta_d)(3\mu_d + \xi_d + \sigma_d + \eta_d + \omega_d)\\
&\quad \Big[W_3 + (\mu_d + \xi_d)(\mu_d + \omega_d)\Big]W_2\\
&\quad + (4\mu_d + \xi_d + \sigma_d + \eta_d + \gamma_d + \omega_d)W_7.
\end{aligned}
$$

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