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Impact of dogs with deltamethrin-impregnated collars on prevalence of visceral leishmaniasis

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ABSTRACT

Leishmaniasis is a vector borne zoonosis which is classified as a neglected tropical disease. Among the three most common forms of the disease, Visceral Leishmaniasis (VL) is the most threatening to human health, causing 20,000 to 30,000 deaths worldwide each year. Areas where VL is mostly endemic have unprotected dogs in community and houses. The "presence of dogs usually increases VL risk for humans since dogs are the principal reservoir host for the parasite of the disease. Based on this fact, most earlier studies consider culling dogs as a control measure for the spread of VL. A more recent control measure has been the use of deltamethrin-impregnated dog collars (*DIDC*s) to protect both humans and dogs by putting *DIDC*s on dogs neck. The presence of dogs helps to grow the sandfly population faster by offering a more suitable blood-meal source. On the other hand, the presence of *DIDC*s on dogs helps to reduce sandfly population by the lethality of deltamethrin insecticide. This study brings an ecological perspective to this public health concern, aiming to understand the impact of an additional host (here, protected dogs) on disease risk to a primary host (here, humans). To answer this question, we compare two different settings: a community without dogs, and a community with dogs protected with *DIDC*. Our analysis shows the presence of protected dogs can reduce VL infection risk in humans. However, this disease risk reduction depends on dogs' tolerance for sandfly bites.

1. Introduction

The Leishmaniases are a group of diseases caused by the protozoa parasite *Leishmania* (Global Health Observatory (GHO), 2019 and World Health Organization; Centers for Disease Control and Prevention, 2018), which is transmitted by the bites of female sandflies (Centers for Disease Control and Prevention, 2018; World Health Organization, 2019). Over 20 Leishmania species known to be infective to humans are transmitted by the bite of infected female phlebotomine sandflies. Leishmaniasis is classified as a neglected tropical disease (NTD). It is found in parts of the tropics, subtropics, and southern Europe (Centers for Disease Control and Prevention, 2018). However, the disease mainly affects poor people in Africa, Asia and Latin America (Global Health Observatory (GHO), 2019 and World Health Organization). There are three main types of leishmaniasis among which visceral, often known as Kala-azar, is the most serious form of the disease

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(Global Health Observatory (GHO), 2019 and World Health Organization). Regarding visceral leishmaniasis, more than 90% of all cases occur in just the six countries of India, Bangladesh, Sudan, South Sudan, Brazil, and Ethiopia (Alvar et al., 2012).

Out of 200 countries and territories reporting to the World Health Organization (WHO), 77 countries are endemic for visceral leishmaniasis in 2017. In 2016, over 90% of global VL cases were reported from seven countries: Brazil, Ethiopia, India, Kenya, Somalia, South Sudan and Sudan. As of October 2018, 50 VL-endemic countries have reported 2017 data to the WHO Global Leishmaniasis programme (Global Health Observatory (GHO), 2019 and World Health Organization). Annually, 700,000 to 1 million new cases and 20,000 to 30,000 deaths occur (World Health Organization, 2018). Currently, this is one of the major public health concerns. Even though human VL is spread by female sandflies, dogs are the main reservoir of the VL parasite. Thus, dogs presence in a community normally increases human VL incidence. In the last couple of decades, many clinical and mathematical studies have been conducted aiming to understand the dynamics of Zoonotic VL (ZVL). In these studies, researchers tried to find ways of controlling VL prevalence. Different strategies for controlling the incidence of VL have been considered, most notably culling dogs and putting insecticide-impregnated dog collars on dogs.

In 2002, Gavgani et al. clinically studied two possible strategies– early diagnosis and treatment, and use of deltamethrin-impregnated dog collars (*DIDC*). The presence of deltamethrin insecticide on dog collars helps to kill a portion of sandlies, and eventually reduces the disease transmission. Outcomes of the study showed that use of *DIDC* is helpful in reducing human infection, and this strategy can replace the controversial dog culling program (Mazloumi Gavgani, Hodjati, Mohite, & Davies, 2002). In addition to clinical studies, several mathematical models have also been used to study transmission and control strategies for ZVL. In 2010, ELmojtaba et al. used a modified SIR model to study the dynamics of leishmaniasis in Sudan (ELmojtaba, Mugisha, & Hashim, 2010). Their study found the human treatment rate to be the key parameter in disease control since they considered humans as competent hosts. However, human treatment needs to be accompanied by control of the vector, and reservoir populations to eradicate the disease from trhe community. They suggest to maintain a distance between the hosts (humans and dogs); a similar suggestion is claimed in a later study by Zahid and Kribs (Zahid & Kribs, 2019). Later, Ribas, Zaher, Shimozako, and Massad (2013) proposed and analyzed a deterministic mathematical model in order to compare different control strategies. They showed that using *DIDC* is better at reducing human infections (Ribas et al., 2013).

In 2016, Zhao et al. studied ZVL transmission using an SEIR deterministic model (Zhao, Kuang, Wu, Ben-Arieh, & Ramalho-Ortigao, 2016). In their model they incorporated hospitalization of infected humans, migration for the sandfly population, and infection-related death for sandflies. Also, they assumed that the contact rates between sandflies and hosts (dogs and humans) are independent of vector population, biting exposed dogs cannot infect sandflies, and immunity for both of the hosts, humans and dogs, is permanent. Later, they compared three different control strategies– vaccination of dogs, use of insecticide at vector breeding sites, and personal protection. Their analysis found controlling the sandfly population to be the most effective control measure.

The next year, in 2017, Shimozako et al. used the SEIR deterministic model also to study ZVL disease dynamics. They included a delay term instead of using latent compartment for the sandfly population (Shimozako, Wu, & Massad, 2017). In this study, they estimated the basic reproduction number R_0 and analyzed the stability and sensitivity of the system, and finally made some recommendation regarding control strategies. Unlike Zhao's work, they assumed that sandflies can be infected from biting exposed dogs, and immunity gained by both of the host populations is temporary. Interestingly, they assumed that exposed dogs and humans become susceptible to VL when recovery precedes the appearance of symptoms. Their work also assumed that the rate at which vectors bite ""hosts is independent of host density. Outcomes of their study showed that control strategy for ZVL should be focused on sandflies and infected dogs. However, considering the ethical concerns regarding culling dogs they recommend to prioritize the control of sandfly population.

All the research related to ZVL has mainly addressed public health concerns where researchers study different control strategies. In these studies, we always find the presence of human and dog populations as hosts for sandflies, the vector of the disease. This host richness (host diversity) leads us to think about the dilution effect (reduction in disease risk resulting from species diversity). The effect of the presence of an additional host is not straightforward. It can increase, or decrease disease risk depending on varieties of factors. In 2010, Johnson and Thieltges showed that host diversity helps in reducing human infections depending on the relative abundance of additional host(s) relative to the focal host (Johnson & Thieltges, 2010). In 2014, Miller and Huppert (2014) proved that species diversity in host population can amplify or can dilute disease prevalence depending on vectors' preference of host (Miller & Amit, 2014). Recently in 2019, Zahid and Kribs established that the presence of an additional host in domestic settings can help to reduce disease prevalence in humans if the distance between two host populations remains within a certain range (Zahid & Kribs, 2019). These works challenge the established idea that biodiversity always helps to reduce disease risk. It is always interesting to observe how the presence of other hosts, in addition to humans, influences the dynamics of vector-borne diseases, impacts disease risk and human health.

This study shifts the research question from a public health viewpoint to an ecological one. As dogs are the main reservoir for the parasite, the usual presence of dogs in a domestic, or in a community setting makes VL transmission faster ensuring more suitable blood-meal source for its vector sandflies. Hence, this paper aims to identify the impact of the presence of protected dogs (protected by putting deltamethrin-impregnated dog collars) as an additional host on the prevalence of VL in humans.

The presence of protected dogs in a community has two contradictory effects. It ensures a better blood-meal source for vector population since biting a dog is much easier for sandflies than biting a human. Thus, dog presence in a community helps sandfly population to grow faster. Moreover, dog presence increases the proportion of infected sandflies (since dogs are the main reservoir of the parasite) which eventually increases human infection risk. On the contrary, use of *DIDC*s on dogs as topical insecticide reduces the sandfly population by the lethality effect, which can result in fewer cases of VL. The net result of these two contrary effects may reduce or enhance the risk of human prevalence. The goal of this study is to understand and examine this net effect, and eventually to understand if the presence of protected dogs has any dilution effect on human risk of VL infections. To answer this question, here we consider two different settings: a setting with protected dogs, and a setting with no dogs. To analyze and understand these two different settings, in this study we use an SEIRS deterministic model.

2. Model development

The SEIRS model we use here to understand the dynamics of VL incorporates three different populations: two hosts– dogs and humans, and the vector – sandflies. In our model, we do not consider the PKDL phase of leishmaniasis, because our research question is to identify the effect of insecticide dog collars on the number of human cases, which is independent of the eect of the PKDL class. We neither consider hospitalization of sick humans, nor any migration for the vectors. Based on results of an early clinical study (Alvar et al., 2000), we include disease transmission from symptomatic infected humans to vectors. This inclusion makes this model different from the models formulated by Zhao et al. (Zhao et al., 2016), and by Shimozako et al. (Shimozako et al., 2017).

Like the models proposed by (Ribas et al., 2013; Shimozako et al., 2017), we also assume hosts' immunity temporary. Similar to their model, we assume both of the hosts may acquire natural immunity directly from the exposed state. In contrast to (Ribas et al., 2013; Shimozako et al., 2017), however, we assume that exposed hosts cannot become susceptible without having any immunity. In our model, we have $\mu'_D = \mu_D + \alpha_D$ and $\mu'_H = \mu_H + \alpha_H$ where α_D and α_H represent VL-induced death rates for dogs and humans respectively. The presence of deltamethrin on the collars causes additional deaths at the rate α_S (migration of sandflies due to presence of *DIDC*s, if any, can be included in α_S) for vectors. So, the vectors are leaving at a rate μ'_S where $\mu'_S = \mu_S + \alpha_S$.

The most important factor which makes our model distinct from others' models is the encounter (biting) rate between hosts and vectors, which incorporates the notion of host irritability. The maximum number of bites per unit time a dog can tolerate is not the same as the maximum number of bites per unit time a human can tolerate. This issue of host-density

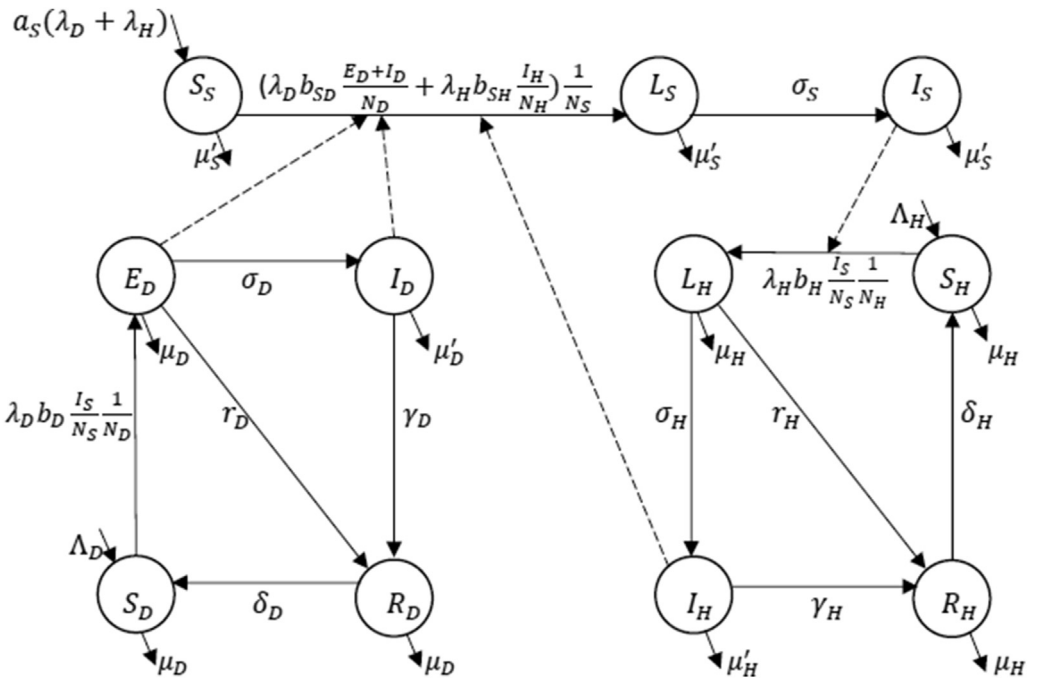


Fig. 1. Population flow among the compartments.

Table 1
Model variables with definition.

Variable	Definition
S_D, S_H	Susceptible dogs, Susceptible humans
L_H, L_S	Latent humans, Latent sandflies
E_D	Exposed dogs
I_D, I_H	Infected dogs, Infected humans
S_S	Susceptible sandflies
I_S	Infected vectors
R_D, R_H	Recovered (Temporary) Dogs, Recovered (Temporary) humans

dependent encounter rate is addressed by Blayneh et al., in 2010 (Blayneh & Gumel, 2010) while modeling dynamics of West Nile Virus. However, the contact rate they used is independent of host population size. But, the sandfly biting rate is limited both by the sandfly's preferred feeding rate and by host irritability (unlike mosquitos, which are limited primarily by availability of breeding sites). So, we consider the encounter rate between sandflies and dogs $\lambda_D = \frac{c_D N_D}{c_D N_D + c_H N_H} \min(c_S N_S, c_D N_D + c_H N_H)$, and the encounter rate between sandflies and humans $\lambda_H = \frac{c_H N_H}{c_D N_D + c_H N_H} \min(c_S N_S, c_D N_D + c_H N_H)$ where c_D and c_H represent the number of bites a dog, and a human can tolerate per unit time, c_S represents the number of bites a single sandfly desires to make per unit time, and N's represent population sizes. This inclusion of the host population dependent biting rate makes our model distinct from other earlier proposed models. However, not all the bites (encounters) can transmit the disease and so we multiply the total number of encounters by b_D (or b_H or b_{SH} or b_{SD}) (Table 2) which represents the proportion of bites (between 0 and 1) that result new infections to dogs (or to humans or to sandflies). Finally, we have our model which is shown in Fig. 1 and described by system (1). All the model variables are summarized in Table 1.

$$
\begin{aligned}
\frac{dS_D}{dt} &= \Lambda_D - \left(\lambda_D b_D \frac{I_S}{N_S}\frac{1}{N_D} + \mu_D\right) S_D + \delta_D R_D \\
\frac{dE_D}{dt} &= \lambda_D b_D \frac{I_S}{N_S}\frac{S_D}{N_D} - (\sigma_D + r_D + \mu_D) E_D \\
\frac{dI_D}{dt} &= \sigma_D E_D - (\gamma_D + \mu'_D) I_D \\
\frac{dR_D}{dt} &= r_D E_D + \gamma_D I_D - (\delta_D + \mu_D) R_D \\
\frac{dS_H}{dt} &= \Lambda_H - \left(\lambda_H b_H \frac{I_S}{N_S}\frac{1}{N_H} + \mu_H\right) S_H + \delta_H R_H \\
\frac{dL_H}{dt} &= \lambda_H b_H \frac{I_S}{N_S}\frac{S_H}{N_H} - (\sigma_H + r_H + \mu_H) L_H \\
\frac{dI_H}{dt} &= \sigma_H L_H - (\gamma_H + \mu'_H) I_H \\
\frac{dR_H}{dt} &= r_H L_H + \gamma_H I_H - (\delta_H + \mu_H) R_H \\
\frac{dS_S}{dt} &= a_S(\lambda_D + \lambda_H) - \left(\left(\lambda_D b_{SD}\frac{E_D + I_D}{N_D} + \lambda_H b_{SH}\frac{I_H}{N_H}\right)\frac{1}{N_S} + \mu'_S\right) S_S \\
\frac{dL_S}{dt} &= \left(\lambda_D b_{SD}\frac{E_D + I_D}{N_D} + \lambda_H b_{SH}\frac{I_H}{N_H}\right)\frac{S_S}{N_S} - (\sigma_S + \mu'_S) L_S \\
\frac{dI_S}{dt} &= \sigma_S L_S - \mu'_S I_S
\end{aligned}
\tag{1}
$$

where

$$
\begin{aligned}
N_D &= S_D + E_D + I_D + R_D \\
N_H &= S_H + L_H + I_H + R_H \\
N_S &= S_S + L_S + I_S
\end{aligned}
$$

$$\lambda_D = \frac{c_D N_D}{c_D N_D + c_H N_H} \quad \min(c_S N_S, c_D N_D + c_H N_H)$$

$$\lambda_H = \frac{c_H N_H}{c_D N_D + c_H N_H} \quad \min\,(c_S N_S, c_D N_D + c_H N_H)$$

$$\mu'_D = \mu_D + \alpha_D,\ \mu'_H = \mu_H + \alpha_H,\ \mu'_S = \mu_S + \alpha_S$$

3. Parameter estimation

In 2017 the World Bank listed 207,833,831 as total population and 13.918/year as the birth rate (crude) per 1000 people for Brazil (The World Bank, 2017). We use the total number of municipalities in Brazil, 5570 (Instituto Brasilerio de Geografia Estatistica (IBGE), 2017), to estimate the average population (37,313) in a single municipality of Brazil. Then using this average population and the birth rate we estimate the recruitment rate for humans to get $\Lambda_H = 1.42$ humans/day. To estimate the recruitment rate in the dog population, we use the results of a study performed in 2005–2008 in Vargem Grande, a neighborhood of the municipality of São Paulo, Brazil, with a population of 16,946 (GarciaMarcos et al., 2018). The study estimated 1337 and 1445 new dogs in 2006 and 2008 respectively, which gives a mean increase of 1391 dogs/year. Then we apply the ratio 2.20, of the population per municipality (estimated above) to the population of the study area of (GarciaMarcos et al., 2018), which estimates an increment of 3060 dogs/year in a municipality of average population. And, we get $\Lambda_D = \frac{3060}{365}$dogs/day $= 8.39$ dogs/day.

Female sandflies take 3–5 days after their emergence to take a blood-meal, and it takes 7.67 days (mean of 6 days, 8 days, and 9 days) from blood-meal to oviposition (laying of eggs) (Lawyer, Killick-Kendrick, Rowland, Rowton, & Volf, 2017). They usually take only one blood-meal until they lay eggs, and begin feeding again after oviposition (European Centre for Disease Prevention and Control, 2019). Therefore, each vector has a single bite in every 7.67 days, and thus we have $c_S = \frac{1}{7.67}$bite/vector-day $= 0.13$ bite/vector-day. Also, we have 10 (mean of 13, 8 and, 9) new female sandflies per egg batch (Lawyer et al., 2017) which gives us $a_S = 10$ sandflies/bite.

A study in 2010 found 12 sandflies infected when 81 sandflies were fed on people with active VL infection (Alvar et al., 2000), and this gives us $b_{SH} = \frac{12}{81} = 0.148$ infected sandfly/bite. Another study in 2013 showed that 35.8% of sandflies that fed on asymptomatic dogs, and 24.7% of sandflies that fed on symptomatic dogs got the infection (Laurenti et al., 2013). We take the mean (30.25%) of these two estimations to get $b_{SD} = 0.3025$ sandfly/bite. However, for b_H, b_D we take the estimations from (Trotz-Williams & Gradoni, 2003) as 0.5 infected human/bite, and 0.01 infected dog/bite respectively.

In 2017, life expectancy at birth in Brazil was 76 years (The World Bank, 2017), so we take its reciprocal to estimate the natural death rate for people in Brazil which gives $\mu_H = 3.6 \times 10^{-5}$/day. In Brazil, there are four common dog breeds, and their mean lifespan is 11.875 years (DOGELL, 2019). We take the reciprocal of the life span as death rate, and we get $\mu_D = 2.30 \times 10^{-4}$/day. To estimate the natural death rate of sandflies we take the reciprocal of their mean lifespan which is 2 weeks (howMedSandfly-Characteristics, 2019), and get $\mu_S = 0.0174$/day.

A study of ZVL cases in Bihar, India, in 2013 showed that 154 patients died among a total of 3641 patients (JervisLloyd et al., 2017), and their average life span was 66.73 years. However, the life expectancy at birth in Bihar at that time period was 68.1 years (UNDP, 2011). Therefore, we take the difference of the reciprocals of these two life span and estimated lethality of ZVL as $\alpha_H = \left(\frac{1}{66.73} - \frac{1}{68.1}\right) \Big/ \text{years} = 3.015 \times 10^{-4}/\text{years} = 8.26 \times 10^{-7}$/day. Earlier studies show that average life-span of infected dogs is two years (Moreno and Alvar, 2002; Trotz-Williams & Gradoni, 2003) which gives us $\mu'_D = \mu_D + \alpha_D = \frac{1}{2 \times 365 \text{ days}} = 1.37 \times 10^{-3}$/day. This value, and already estimated value $\mu_D = 2.30 \times 10^{-4}$/day give us $\alpha_D = \mu'_D - \mu_D = 1.37 \times 10^{-3}/\text{day} - 2.30 \times 10^{-4} = 1.14 \times 10^{-3}$/day. To estimate the *DIDC* induced death rate (α_S) for sandflies, we study (Killick-Kendrick et al., 1997) where sandflies were exposed to dogs protected (with *DIDC*). From each individual experiment, we take the number of exposed sandflies, and the number of flies that died in 20 h duration from their exposure, and pooled data from all trials to get 5766 and 1245 as the totals of exposed sandflies, and dead sandflies respectively. However, our estimation of sandflies' natural death rate (μ_s=0.0714/day) estimates 333 natural deaths [1] $5766 \times \left(1 - e^{-\mu_s \frac{20}{24}\text{day}}\right) = 5766 \times \left(1 - e^{(-0.0714/\text{day}) \times \frac{20}{24}\text{day}}\right) = 333$ of sandflies in a span of 20 h. The remaining deaths of $1245 - 333 = 912$ sandflies are not attributable to natural mortality. These estimations, and sandflies' natural death rate 0.0714/day give us the estimation $\alpha_S = \left(\frac{912}{333}\right) \times \mu_S = 0.0714/\text{day} = 0.1955/\text{day}$.

A laboratory study in 2011 observed sandflies' incubation rate as 7–10 days (Stamper et al., 2011). So, here we take the reciprocal of the mean of 7 and 10 days to estimate the latent period as $\sigma_S = \frac{1}{8.5}$/day $= 0.117$/day. Another work in the same year estimated that individuals without symptomatic VL need on average about 146 days to develop LST-positivity after a PCR-positive finding (Stauch et al., 2011), and this leads us to estimate $\sigma_H = \frac{1}{146}$day $= 6.85 \times 10^{-3}$day. A review

Table 2
Summary of model parameters.

Par.	Definition	Value	Units	Reference
Λ_D	recruitment rate for dogs (by birth)	8.39	dogs/day	This study
Λ_H	recruitment rate for humans (by birth)	1.42	humans/day	This study
a_S	birth rate for sandflies	10	sandflies/bite	This study
b_D	infection to dogs from sandflies' bite	0.01	infected dog/bite	Trotz-Williams and Gradoni (2003)
b_H	infection to humans from sandflies' bite	0.5	infected human/bite	Trotz-Williams and Gradoni (2003)
b_{SD}	infection to sandflies from biting dogs	0.3025	infected sandfly/bite	This study
b_{SH}	infection to sandflies from biting humans	0.148	infected sandfly/bite	This study
c_D	inverse of dogs' irritability	45	bites/dog-day	This study
c_H	inverse of humans' irritability	15	bites/human-day	This study
c_S	bites a single sandfly disere	0.13	bite/sandfly-day	This study
σ_D	incubation rate	1.11×10^{-2}	1/day	This study
σ_H	incubation rate	6.85×10^{-3}	1/day	Stauch et al. (2011)
σ_S	incubation rate	0.117	1/day	This study
γ_D	recovery rate (dogs)	9.04×10^{-4}	1/day	Lanotte, Rioux, Perieres, and Vollhardt (1979)
γ_H	recovery rate (humans)	2.5×10^{-3}	1/day	Shimozako et al. (2017)
δ_D	Inverse of temporary recovery period (dogs)	2.74×10^{-3}	1/day	Burattini, Coutinho, Lopez, and Massad (1998)
δ_H	Inverse of temporary recovery period (humans)	5.48×10^{-4}	1/day	Burattini et al. (1998)
r_D	spontaneous recovery rate	1.1×10^{-2}	1/day	Badaro et al. (1986)
r_H	spontaneous recovery rate	8.22×10^{-3}	1/day	Lanotte et al. (1979)
μ_D	natural death rate	2.30×10^{-4}	1/day	This study
μ_H	natural death rate	3.6×10^{-5}	1/day	This study
μ_S	natural death rate	0.0714	1/day	This study
α_D	disease induced death rate for dogs	1.14×10^{-3}	1/day	This study
α_H	disease induced death rate for humans	8.26×10^{-7}	1/day	This study
α_S	*DIDC*induced death rate for sandflies	0.1995	1/day	This study

paper published in 2002 mentioned the incubation period for dogs as 2–4 months (Moreno and Alvar, 2002). We took reciprocal of the mean of this range of 2–4 months (3 months = 90 days), and estimate $\sigma_D = \frac{1}{90}$/day$= 1.11 \times 10^{-2}$/day.

All other parameter values are taken directly from earlier research studies.

4. Analysis

We begin with the general model where we incorporate three different populations – sandflies (the vector), and two host populations– dogs (protected with *DIDC*) and humans. Later, we consider a special case without dogs in the setting. Finally, we compare these two cases to understand the impact of the presence of protected dogs on the prevalence of VL infections in humans. The study actually aims to understand the effects of host diversity on disease transmission, and eventually on human health.

In the general model, the total number of desired bites for sandflies and the total number of bites that hosts can tolerate together may not be equal. Thus, in our analysis we consider two cases in terms of total number of possible encounters. In the first case, we assume the maximum total number of possible vector bites is less than the total possible number of bites that both hosts (humans and protected dogs) can tolerate together, that is when $c_S N_S < c_D N_D + c_H N_H$. The other scenario takes place when $c_S N_S > c_D N_D + c_H N_H$. For the initial case our calculation estimates the total vector population as $N_S(t) = N_S(0)e^{(a_S c_S - \mu_S')t}$(recall $\mu_S' = \mu_S + \alpha_S$) which shows that the vector population decreases with time and eventually dies out, if $a_S c_S < \mu_S'$. However, $N_S$increases with time under the condition $a_S c_S > \mu_S'$. If the vector population continues to grow then the base condition of the first case, that is $c_S N_S < c_D N_D + c_H N_H$, cannot be true after a certain time. Eventually, the relation between the maximum possible number of vector bites and the maximum host bite tolerance (inverse of hosts' irritability) turns into $c_S N_S > c_D N_D + c_H N_H$, which is the second case. These analyses give us two scenarios: either the vector population dies out, or the only possible case is $c_S N_S > c_D N_D + c_H N_H$ (second case). Since we are interested to understand the disease dynamics, from here our study will consider only the case of $c_S N_S > c_D N_D + c_H N_H$.

To find equilibrium points for our model we set all the equations of our system equal to zero and solve them for state variables. After performing some basic arithmetic we get the disease free equilibrium (*DFE*) as $S_D = \frac{\Lambda_D}{\mu_D}, E_D = 0, I_D = 0, R_D = 0, S_H = \frac{\Lambda_H}{\mu_H}, L_H = 0, I_H = 0, R_H = 0, S_S = \frac{a_S}{\mu_S'}\left(c_D\frac{\Lambda_D}{\mu_D} + c_H\frac{\Lambda_H}{\mu_H}\right), I_S = 0, L_S = 0$. Then we use the next generation method (Diekmann, Heesterbeek, & Metz, 1990) to get the basic reproduction number (*BRN*),

$$R_0 = \frac{2^{\frac{1}{3}}}{3}\frac{Q_{12}}{\left(Q_3+\sqrt{Q_3^2-\frac{4}{27}Q_{12}^3}\right)^{\frac{1}{3}}}+\frac{1}{2^{\frac{1}{3}}}\left(Q_3+\sqrt{Q_3^2-\frac{4}{27}Q_{12}^3}\right)^{\frac{1}{3}} \tag{2}$$

where

$$Q_{12} = \frac{\Lambda_D}{\mu_D}\frac{b_D c_D}{a_S K_3}\frac{b_{SD}c_D}{K_4 K_6}\left(\frac{\gamma_D+\mu_D+\alpha_D}{\sigma_D}\right)+\frac{\Lambda_H}{\mu_H}\frac{b_H c_H}{a_S K_3}\frac{b_{SH}c_H}{K_5 K_6}, \text{ and } Q_3 = \frac{\Lambda_D}{\mu_D}\frac{b_D c_D}{a_S K_3}\frac{b_{SD}c_D}{K_4 K_6}.$$

here Q_{12}accounts for transmission between infected vectors and infected hosts, while Q_3accounts for transmission between infected vectors and exposed dogs. The terms are not simply added to form R_0because the cycles overlap as exposed dogs become infected dogs later. However, when $Q_{12}=0, R_0=Q_3^{\frac{1}{3}}$, and when $Q_3=0, R_0=\sqrt{Q_{12}}$(see Appendix 1). In other words, in the absence of one of the two transmission cycles, R_0measures the transmission efficiency of the other cycle. In general $R_0 \le \sqrt{Q_{12}}+Q_3^{\frac{1}{3}}$. The $K_i$are given below.

Later, we obtain the following quadratic equation (in I_H) for endemic equilibrium (see Appendix 2):

$$AI_H^2+BI_H+C=0 \tag{3}$$

where

$$A = \left(\frac{b_H c_H}{b_D c_D}\frac{K_4}{K_5}K_2-K_1\right)\left[b_{SH}c_H\left(K_2+\frac{K_5K_6}{b_H c_H}\right)-a_S\frac{K_5K_6}{b_H c_H}c_H\frac{\alpha_H}{\mu_H}\right],$$

$$B = -\left(\frac{\Lambda_D}{\mu_D}c_D b_{SD}K_7+\frac{\Lambda_H}{\mu_H}b_{SH}c_H\frac{b_H c_H}{b_D c_D}\frac{K_4}{K_5}\right)\left(K_2+\frac{K_5K_6}{b_H c_H}\right)-\left(\frac{b_H c_H}{b_D c_D}\frac{K_4}{K_5}K_2-K_1\right)\left(\frac{\Lambda_H}{\mu_H}b_{SH}c_H-a_S K_3\frac{k_5K_6}{b_H c_H}\right)$$
$$+\frac{\Lambda_H}{\mu_H}a_S\frac{K_4K_6}{b_D c_D}c_H\frac{\alpha_H}{\mu_H}+\frac{\Lambda_D}{\mu_D}a_S\frac{K_5K_6}{b_H c_H}c_D\frac{\alpha_D}{\mu_D},$$

$$C = \frac{\Lambda_H}{\mu_H}\left(\frac{\Lambda_H}{\mu_H}b_{SH}c_H\frac{b_H c_H}{b_D c_D}\frac{K_4}{K_5}+\frac{\Lambda_D}{\mu_D}c_D b_{SD}K_7-a_S K_3\frac{K_4K_6}{b_D c_D}\right),$$

with

$$K_1 = \frac{\sigma_D+r_D+\mu_D}{\mu_D}\frac{\gamma_D+\mu_D'}{\sigma_D}-\frac{\delta_D}{\mu_D}\left(\frac{r_D}{\delta_D+\mu_D}\frac{\gamma_D+\mu_D'}{\sigma_D}+\frac{\gamma_D}{\delta_D+\mu_D}\right)$$

$$K_2 = \frac{\sigma_H+r_H+\mu_H}{\mu_H}\frac{\gamma_H+\mu_H'}{\sigma_H}-\frac{\delta_H}{\mu_H}\left(\frac{r_H}{\delta_H+\mu_H}\frac{\gamma_H+\mu_H'}{\sigma_H}+\frac{\gamma_H}{\delta_H+\mu_H}\right)$$

$$K_3 = c_D\frac{\Lambda_D}{\mu_D}+c_H\frac{\Lambda_H}{\mu_H}$$

$$K_4 = (\sigma_D+r_D+\mu_D)\frac{\gamma_D+\mu_D'}{\sigma_D}$$

$$K_5 = (\sigma_H+r_H+\mu_H)\frac{\gamma_H+\mu_H'}{\sigma_H}$$

$$K_6 = \frac{\sigma_S+\mu_S'}{\sigma_S}$$

$$K_7 = \frac{\gamma_D+\mu_D'}{\sigma_D}+1$$

where $K_1, K_2 > 0$.

To understand the behavior of disease dynamics, the threshold value $BRN(R_0)$and endemic equilibrium (EE) need to be understood, and interpreted properly. Our analysis shows $R_0>1$if and only if $C>0$(see Appendix 3). For endemic equilibrium, we cannot establish any specific condition to ensure the positivity of EE since its analytic expression obtained from equation (3), is very complex (recall expressions of coefficients $A, B$and C). Consequently, we perform numerical explorations to understand the behavior of our dynamical system, and finally conclude that the model has a unique positive endemic equilibrium whenever $R_0>1$(when a second solution set exists, the other solution set is non-positive). It does not appear possible for A, B, C all to be positive together. To check the stability of our EE, we evaluate the Jacobian matrix of our dynamical system,

and perform numerical explorations (see Appendix 3 for further detail), which indicate that the endemic equilibrium is unconditionally stable.

Now, we consider the case of having no dogs in the scene, a special case (setting all variables and parameters related to dogs to zero) of our original model. Our analysis for this special case finds the *DFE* as $S_H = \frac{\Lambda_H}{\mu_H}, L_H = 0, I_H = 0, R_H = 0, S_S = \frac{a_S}{\mu_S}$, $L_S = 0, I_S = 0$, and *BRN*as $R_0 = \sqrt{\frac{b_{SH} b_H c_H}{a_S K_5 K_6}}$ where $K_6' = \frac{\sigma_S + \mu_S}{\sigma_S}$ (derived from $K_6$by setting $\alpha_S = 0$). In this case the endemic equilibrium (3) simplifies to a linear equation which thus has at most a single solution, in which we get the expression for the infected human population as

$$I_H^* = \frac{\Lambda_H}{\mu_H}\left(\frac{b_{SH} b_H c_H - K_5 K_6' a_S}{b_{SH}(b_H c_H K_2 + K_5 K_6') - a_S K_5 K_6' \frac{\alpha_H}{\mu_H}}\right)$$

which can be expressed in terms of R_0 as

$$I_H^* = \frac{\Lambda_H}{\mu_H}\left(\frac{R_0^2 - 1}{K_2 R_0^2 + \frac{b_{SH}}{a_S} - \frac{\alpha_H}{\mu_H}}\right).$$

Since some algebra shows that $K_2 > \alpha_H/\mu_H$, the unique endemic equilibrium exists precisely when $R_0 > 1$.

Based on our parameter estimation, we calculate R_0 and I_H^* for all three possible cases: community with dogs protected with *DIDC*, community without dogs, and community with dogs having no protective measure. We estimate $I_H^* = 3023, 3329$, and 4677 in a population size of 39,445, respectively, for these three cases. These estimates indicate that the presence of *DIDC*-protected dogs is better than having no dogs, which in turn is better than having unprotected dogs (in terms of human infections of leishmaniasis). The R_0 values for these same cases are 1.47, 3.51, and 2.05 respectively. These results provide fewer human infections with a higher R_0 value for the no dog case compared to the case of unprotected dogs. Humans are spreading infections faster in the case of no dog; however, the contribution of the dog population to new infections is missing in this case. Thus, the case of no dog in the community produces fewer infections even though the R_0 value is higher compared to the case of unprotected dogs. The expression for R_0 in (2) helps us to understand this apparently unusual result better. One of Q_{12}'s two terms, and all of Q_3, have to do with dogs. Removing them will reduce R_0, especially since sandfly biting rate is asymptotically host-dependent. Fig. 2 shows how the order of R_0 values changes for a certain parameter (c_H) range, to match the I_H^* ordering.

A local sensitivity analysis of the endemic prevalence of leishmaniasis (I_H^*), for the case of protected (with *DIDC*) dogs' presence, was performed for all of our model parameters (Fig. 3). Among the top 6 parameters with higher normalized sensitivity indices, γ_H, r_H, and δ_H are well known (Burattini et al., 1998; Lanotte et al., 1979; Shimozako et al., 2017). Among the remaining three, Λ_H, and μ_H are location-specific. The remaining of the top 6 influential parameters is a_S, which is estimated using documented data (Lawyer et al., 2017). Of the parameters with normalized sensitivity indices greater than $\frac{1}{4}$, the two most of interest to this study are c_D and α_S.

Now we vary the values of c_D and α_S together to observe the contribution of dogs' irritability and the efficacy of *DIDC* simultaneously in reducing human infections. Our analysis shows that the number of human infections increases with dogs' tolerance for bites (c_D), because this allows vectors' easy access to bite dogs which helps the sandfly population to grow. It also increases the proportion of infected sandflies, because the probability of infection to sandflies from biting dogs is higher than the probability of new infection from biting humans. The number of human infections increases also for extremely low dog

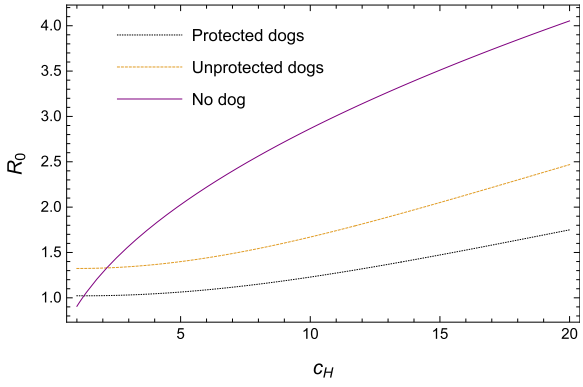


Fig. 2. R_0 values change their order while a parameter (c_H) varies.

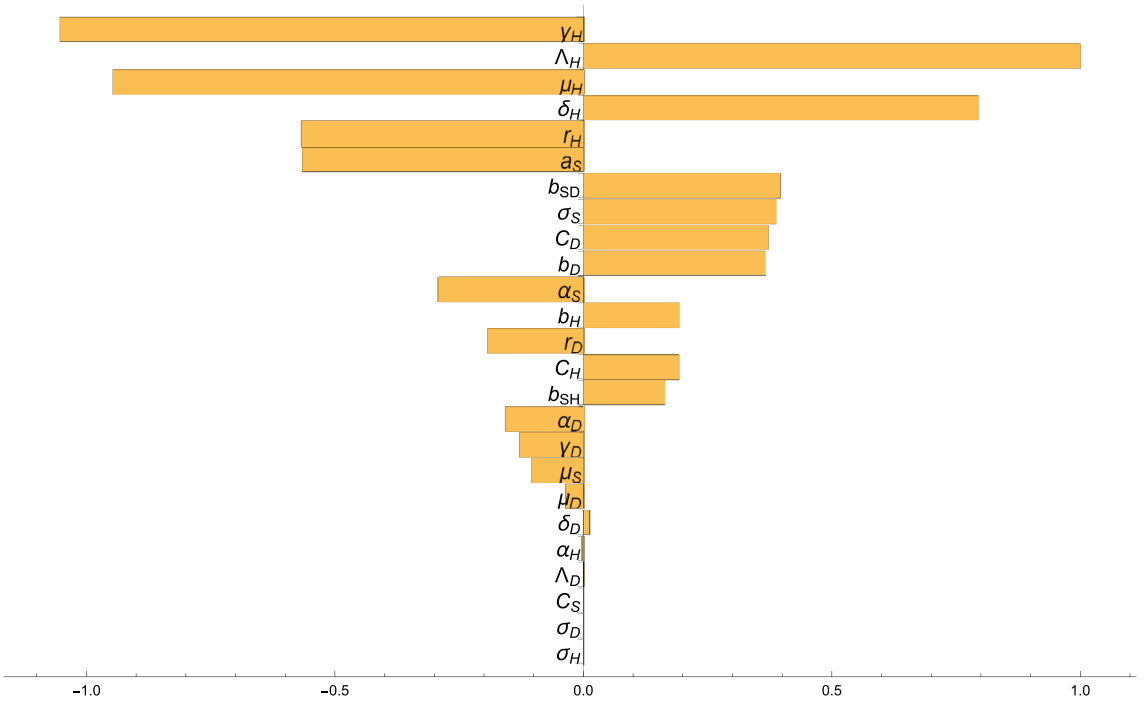
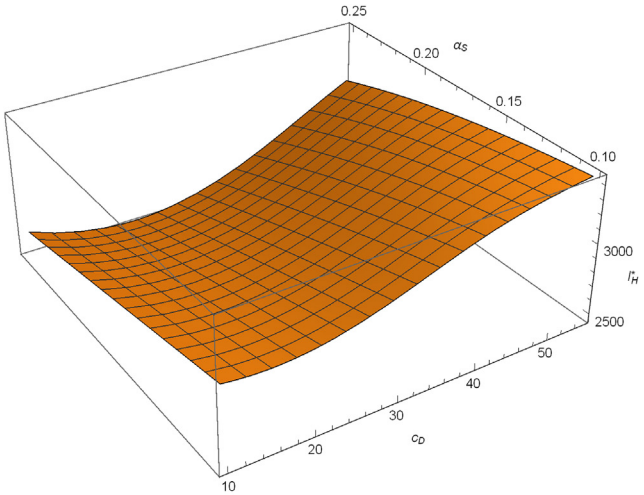
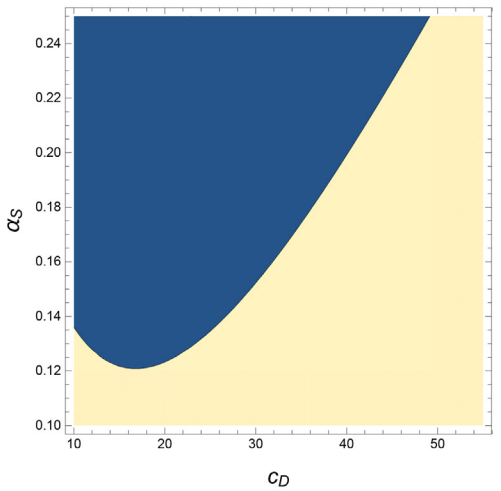


Fig. 3. Local sensitivity analysis of I_H^* for all model parameters.

tolerance, because it reduces the effects of protective collars by reducing the number of interactions between sandflies and dogs significantly. Also, humans get almost all the bites here which eventually increases human infection risk. Hence, the number of human cases of VL infections is not always proportional to dogs' tolerance to sandfly bites. Fig. 4 clearly explains the above discussion regarding the effect of dogs' tolerance for sandfly bites, where sub-figure (b) of 4 represents the human cases with respect to the dog tolerance and collar's efficiency. Our analysis also shows that human infections decrease with the population size of protected dogs. However, based on our parameter estimation, we find 58% of the dog population needs to be protected with *DIDC* to ensure the effectiveness of the presence of protected dogs in reducing human risk of VL infections.



(a) Human cases of VL with varied combination of efficacy of $DIDC$s and dogs' irritability (where irritability=$\frac{1}{c_D}$)



(b) Darker region represents lower infections and lighter region represents higher infections

Fig. 4. Effect of dogs' irritability, and *DIDC* efficacy on human infections of VL.

5. Discussion

The model used in this study gives a new insight to the study of visceral leishmaniasis transmission among human, dog, and sandfly populations. This happens as this study considers humans as a competent host (based on earlier research (Alvar et al., 2000)), in contrast to most other studies' assumption of treating humans as a dead-end host (assuming dogs as the only source of vector infections). The host-population-dependent biting rate for sandflies highlights the impact of host biodiversity more than other models used in the earlier studies of leishmanisis. Our results show the presence of dogs with *DIDC* (as topical insecticide) in a community produces fewer human infections compared to infections in the same community without dogs. It is also confirmed that a community without dogs is better in terms of VL infections in humans than a community containing unprotected dogs.

Zhao et al. (2016) develop and analyze an SEIR model assuming hosts' recovery from infections permanent. In our model, we consider recovery for hosts as temporary which leads us to use an SEIRS model. They incorporate sandfly migration in the model which we do not. Their analysis finds the condition $R_0 < 1$ insufficient for complete control of the disease since they observe the existence of backward bifurcation under the condition $R_c < R_0 < 1$. In our study, we find only one endemic equilibrium, which precludes any possibility of the existence of backward bifurcation. Identifying which feature of their model is responsible for the backward bifurcation is prevented by the fact that their study did not provide the definition of k_3 which is present in the definition of R_c. Their model does include disease-related death, but ours includes it also and does not appear to exhibit backward bifurcation. Even though our study does not focus on optimal control policy like Zhao et al., we study the impact of presence of protected (with *DIDC*) dogs, and find this presence helpful in producing fewer human infections.

Shimozako et al. (2017) assumed recovery for both hosts from VL infections is temporary, so they used an SEIRS model in studying ZVL transmission. We also use a modified SEIRS model even though we exclude their assumption that exposed hosts (dogs and humans both) can become susceptible without developing any immunity. We also do not adopt their assumption that latent, and clinically ill dogs have different probabilities of infecting sandflies. They find VL transmission completely dependent on the dog and sandfly populations. However, our study shows each of the three populations has contributions in the dynamics of VL transmission. Shimozako et al. (2017) suggests that control of VL transmission should be based on the sandfly population. Our analysis agrees with this suggestion, showing that presence of dogs with *DIDC* protection (which reduces sandfly population) reduces human cases of visceral leishmanisis.

Our study also draws on an ecological perspective to inform public health policy. In the literature review, we mentioned ecological studies (Johnson & Thieltges, 2010; Miller & Amit, 2014; Zahid & Kribs, 2019) in which the presence of additional hosts (known as host richness) may help in reducing the human infection risk depending on some factors, such as the abundance of additional host(s) relative to the focal host, vectors' preference of host for feeding, and distance between the primary host and additional host. This study also found the presence of an additional host (dogs protected with *DIDC*) helpful in reducing human risk of VL infections. However, this reduction is independent of all three of the factors which are identified in (Johnson & Thieltges, 2010; Miller & Amit, 2014; Zahid & Kribs, 2019). We find that dogs' presence in a community does not produce fewer human infections if dogs' irritability is very high, or extremely low even after *DIDC*s are ensured on them. This ecological change helps to protect human health only if dogs' irritability ranges somewhere in the intermediate level.

Our proposed model has a few limitations in its development. In our study, we assume all dogs in a community are protected by deltamethrin-impregnated collars which may not be possible in reality. Also, we do not incorporate sandfly migration in our model. However, this migration rate can be included in our *DIDC*-induced sandfly death rate (α_S). Inclusion of this migration may have some impact on our numerical results, and also on the range of dog irritability values which are helpful. However, our qualitative results will remain the same. We have also simplified the VL cycle in humans (for instance, omitting PKDL's role as a possible reservoir) in order to focus on the role played by *DIDC*-protected dogs. Addressing these limitations could produce better insights into visceral leishmaniasis dynamics. Our proposed model offers a better base than other models for studying control strategies for ZVL, and VL transmission since we incorporate some real, and very important issues, like human infectivity and the role of host irritability.

Declaration of competing interest

The authors declare no conflict of interest.

Appendix 1. Simplification of R_0 when $Q_3 = 0$

$$\frac{2^{1/3}Q_{12}}{3\left(\sqrt{\frac{-4Q_{12}^3}{27}}\right)^{1/3}} + \frac{\left(\sqrt{\frac{-4Q_{12}^3}{27}}\right)^{1/3}}{2^{1/3}} = \frac{Q_{12}}{\sqrt{3}\left(-Q_{12}^3\right)^{1/6}} + \frac{\left(-Q_{12}^3\right)^{1/6}}{\sqrt{3}}$$

$$= \frac{Q_{12} + \left(-Q_{12}^3\right)^{1/3}}{\sqrt{3}\left(-Q_{12}^3\right)^{1/6}}$$

$$= \frac{Q_{12}\left(1 + (-1)^{1/3}\right)}{\sqrt{3}\sqrt{Q_{12}}(-1)^{1/6}}$$

$$= \frac{\sqrt{Q_{12}}\left(1 + \frac{1}{2} + i\frac{\sqrt{3}}{2}\right)}{\sqrt{3}\left(\frac{\sqrt{3}}{2} + \frac{i}{2}\right)}$$

$$= \frac{\sqrt{Q_{12}}\left(\frac{3}{2} + i\frac{\sqrt{3}}{2}\right)}{\left(\frac{3}{2} + i\frac{\sqrt{3}}{2}\right)}$$

$$= \sqrt{Q_{12}}$$

Appendix 2. Derivation of equation (3)

Using the value of λ_D in the 1st equation of the system (1) at steady state gives

$$S_D = \frac{\Lambda_D}{\mu_D} - \left[\left(\frac{\sigma_D + r_D + \mu_D}{\mu_D}\right)\left(\frac{\gamma_D + \mu'_D}{\sigma_D}\right)I_D - \frac{\delta_D}{\mu_D}\left(\frac{r_D}{\delta_D + \mu_D}\frac{\gamma_D + \mu'_D}{\sigma_D} + \frac{\gamma_D}{\delta_D + \mu_D}\right)\right] = \frac{\Lambda_D}{\mu_D} - K_1 I_D \tag{4}$$

At steady states, 3rd equation of system (1) gives

$$E_D = \frac{\gamma_D + \mu'_D}{\sigma_D} I_D \tag{5}$$

The value of λ_D, equation (7), equation (8), and the 2nd equation of system (1) at steady state gives

$$c_D b_D \frac{\Lambda_D}{\mu_D}\frac{I_S}{N_S} - K_1 c_D b_D \frac{I_S}{N_S} I_D - K_4 I_D = 0 \tag{6}$$

Similarly, 5th, 6th, and 7th equations of system (1) at steady states gives

$$c_H b_H \frac{\Lambda_H}{\mu_H}\frac{I_S}{N_S} - K_2 c_H b_H \frac{I_S}{N_S} I_H - K_5 I_H = 0 \tag{7}$$

The 9th equation of the system (1) at steady state give

$$L_S = \frac{\mu'_S}{\sigma_S} I_S \tag{8}$$

At steady state, the sum of 9th, and 10th equations of system (1), and equation (8) give

$$S_S = \frac{a_S(\lambda_D + \lambda_H)}{\mu'_S} - \frac{\sigma_S + \mu'_S}{\sigma_S} I_S \tag{9}$$

Now, equation (9), and the 10th equation of system (1) at steady state give

$$(c_D b_{SD} K_7 I_D + c_H b_{SH} I_H)\left(1 - K_6 \frac{I_S}{N_S}\right) - K_6 \mu'_S I_S = 0 \tag{10}$$

Now, equation (6), and equation (7) respectively give

$$\frac{I_S}{N_S} = \frac{K_4}{c_D b_D}\left(\frac{\Lambda_D}{\mu_D} - K_1 I_D\right)^{-1} I_D \tag{11}$$

$$\frac{I_S}{N_S} = \frac{K_5}{c_H b_H}\left(\frac{\Lambda_H}{\mu_H} - K_1 I_H\right)^{-1} I_H \tag{12}$$

Then, using equation (11), and equation (12) we get

$$I_D = \frac{\Lambda_D}{\mu_D}\left[\frac{b_H c_H}{b_D c_D}\frac{K_4}{K_5}(\Lambda_H \mu_H - K_2 I_H) + K_1 I_H\right]^{-1} I_H \tag{13}$$

Finally, substituting the value of $\frac{I_S}{N_S}$ from (12), and I_D from (13) in equation (10) we get

$$A I_H^2 + B I_H + C = 0$$

Appendix 3. Endemic equilibrium analysis

The expression for C can be written as

$$\begin{aligned}
\frac{C}{\frac{\Lambda_H}{\mu_H} c_D b_{SD} \frac{\Lambda_D}{\mu_D}} &= K_7 + \frac{\frac{\Lambda_H}{\mu_H}}{\frac{\Lambda_D}{\mu_D}} \frac{c_H b_{SH}}{c_D b_{SD}} \frac{c_H b_H}{c_D b_D} \frac{K_4}{K_5} - \frac{1}{Q_3} \\
\frac{C}{\frac{\Lambda_H}{\mu_H} c_D b_{SD} \frac{\Lambda_D}{\mu_D}} &= K_7 + \frac{\Lambda_H}{\mu_H} \frac{b_H c_H}{a_S K_3} \frac{b_{SH} c_H}{K_5 K_6} \frac{1}{Q_3} - \frac{1}{Q_3} \\
\frac{C Q_3}{\frac{\Lambda_H}{\mu_H} c_D b_{SD} \frac{\Lambda_D}{\mu_D}} &= K_7 Q_3 + \frac{\Lambda_H}{\mu_H} \frac{b_H c_H}{a_S K_3} \frac{b_{SH} c_H}{K_5 K_6} - 1 \\
&= Q_3\left(\frac{\gamma_D + \mu_D + \alpha_D}{\sigma_D} + 1\right) + \frac{\Lambda_H}{\mu_H} \frac{b_H c_H}{a_S K_3} \frac{b_{SH} c_H}{K_5 K_6} - 1 \\
&= Q_3\left(\frac{\gamma_D + \mu_D + \alpha_D}{\sigma_D}\right) + Q_3 + \frac{\Lambda_H}{\mu_H} \frac{b_H c_H}{a_S K_3} \frac{b_{SH} c_H}{K_5 K_6} - 1 \\
&= \left(\frac{\Lambda_D}{\mu_D} \frac{b_D c_D}{a_S K_3} \frac{b_{SD} c_D}{K_4 K_6}\left(\frac{\gamma_D + \mu_D + \alpha_D}{\sigma_D}\right) + \frac{\Lambda_H}{\mu_H} \frac{b_H c_H}{a_S K_3} \frac{b_{SH} c_H}{K_5 K_6}\right) + Q_3 - 1 \\
C \frac{Q_3}{\frac{\Lambda_H}{\mu_H} c_D b_{SD} \frac{\Lambda_D}{\mu_D}} &= Q_{12} + Q_3 - 1
\end{aligned} \tag{14}$$

Our analysis found R_0 as a increasing function in Q_{12}, and $R_0 = 1$ if and only if $Q_{12} + Q_3 = 1$. Since $\frac{Q_3}{\frac{\Lambda_H}{\mu_H} c_D b_{SD} \frac{\Lambda_D}{\mu_D}}$ is a positive quantity, then from (14) we have $R_0 > 1$ if and only if $C > 0$.

To explore the range of solutions to equation (3) numerically, we considered all parameters except δ_H and b_{SD} to be fixed at their default values, and then considered A and C as functions of δ_H and b_{SD} respectively. We found the intervals on which A and C were positive and negative, and explored each possible combination. We found no cases in which A, B, C are all positive.

Of the seven remaining combinations, only three ($A>0, B<0, C>0$; $A, B<0, C>0$; $A<0, B, C>0$) led to positive solutions. The Jacobian matrix for each of these three cases gives a set of eigenvalues with negative real part, which ensures that the endemic equilibrium is locally asymptotically stable.

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