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A discrete epidemic model for bovine Babesiosis disease and tick populations

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Abstract: In this paper, we provide and study a discrete model for the transmission of Babesiosis disease in bovine and tick populations. This model supposes a discretization of the continuous-time model developed by us previously. The results, here obtained by discrete methods as opposed to continuous ones, show that similar conclusions can be obtained for the discrete model subject to the assumption of some parametric constraints which were not necessary in the continuous case. We prove that these parametric constraints are not artificial and, in fact, they can be deduced from the biological significance of the model. Finally, some numerical simulations are given to validate the model and verify our theoretical study.

Keywords: Discrete-time epidemic models; Local and global stability; Jury criterion; Lyapunov methods

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1 Introduction

Bovine babesiosis is the most important arthropod-borne disease of cattle worldwide. It provokes morbidity and even mortality of cattle after a tick-borne, parasitic infection. Ticks infected due to the ingestion of parasites in the blood of infected cattle are the most relevant transmission agent of such a disease. The permanence of the infection depends on the probability of the vertical transmission in ticks population as suggested in [1] for dengue.

The most common varieties of babesiosis are *Babesia bovis* and *Babesia bigemina* which can be found throughout the majority of tropical regions. In fact, as reported in the literature [2] both varieties became endemic in the south of USA and affected to the associated industry very seriously.

Control strategies based on vaccination and antiparasitic treatments have been performed [3]. But, due to residues and other problems, some vaccines and drugs have been eliminated from these strategies [4, 5].

All these features make this disease interesting to be modeled mathematically in order to know its dynamical behavior. Actually, to know its behavior could lead to the design of new control strategies.

In the literature, one can find few deterministic mathematical models of bovine Babesiosis. In [6], we introduced a model based on ordinary differential equations for bovine Babesiosis caused by *Babesia bigemina* and *Babesia bovis* for the first time. After that, in view of this previous one, a model of partial differential equations for *Babesia bovis* was formulated by Friedman and Yakubu [7]. Besides, based on our work in [6], Carvalho et al. [8] presented a new version of our classical model changing the ordinary derivative by fractional Caputo derivate. Likewise, in [9], authors study a fractional-order scheme model on the disease. Our continuous model in [6] has also served as a basis to set out and study similar models, including other factors such as a two-stage in the cattle class in [10] or the effect of seasonal changes in [11]. Moreover, in [12] a study of the dynamic behavior of our model is performed using a multistage modified sinc method which is a computational algorithm for approximating solutions of the classical system in a sequence of (time) intervals.

In the theory of epidemics, there are two fundamental types of mathematical models: continuous-time models described by differential equations and discrete-time models described by difference equations. As said in [13], discretization of continuous-time models is an interesting and prominent trend. Following this idea, in the present work, we deal with a discrete-time version of the classical model for bovine Babesiosis proposed in [6]. In fact, discretization of classical continuous models could help to check the skill in the selection of the factors to the design of the model. Note that factors providing very different dy-

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namics could be not adequate to be considered in such a design.

Discrete versions of models can be obtained by direct formulation of the evolution with difference equations, as we do in this work, or using discretization schemes of continuous models as for example the Euler scheme [14]. In the last years, the study of discrete-time (epidemic) models has increased (see for instance ([15] or[16]) due to different considerations (see [17]). First of all, statistical data on epidemics are collected in discrete time. Hence, to describe epidemics using such discrete models seems to be convenient. In addition, more accurate numerical simulation results are maybe obtained using discrete-time models, although the dynamic behaviors of such discrete models are often more complex [16]. Furthermore, numerical simulations of continuous-time models are obtained by discretizing these models. All these considerations justify the interest and appropriateness of the present work.

In this work, the discrete system is set out by considering the same parameters as in the design of the continuous case. After a simplification of the system, the model is reduced to three difference equations. For such a discrete-time model some necessary parametric constraints, which are fundamental for the rest of the results in the paper, are demonstrated. In fact, these parametric constraints are not artificial and they can be deduced from the biological significance of the model. This shows that additional care should be taken when dealing with discrete-time systems.

After setting the constraints, we calculate the equilibria of the system and the basic reproduction number R_0. This number should be regarded as the expected number of new infections from one infected individual in a fully susceptible population during its infectious period, and its value provides a insight in the designing control interventions for infectious diseases [18–20]. In fact, it is a key concept in epidemiology as can be seen in [21–23]. Finally, we demonstrate that the appearance of the endemic equilibrium depends on the value of the threshold parameter R_0 and that such a parameter determines the local and global stability of the disease-free and the endemic equilibria. Actually, it is shown that the system undergoes a transcritical bifurcation when passing through R_0.

This study represents a first attempt to model the dynamics of bovine Babesiosis by a discrete model. Due to that, it must be compared to the continuous model. Since the obtained results on the dynamics are similar, we are able to confirm the absence of contradictions between both versions. Furthermore, it opens a way to stimulate the modelization by using other discretization schemes and the comparison of the corresponding results in order to inform the debate on such new formulations.

The document is organized as follows. In Section 2, the discrete-time mathematical model is established considering the same influencing parameters as in the continuous case and some necessary conditions which will become fundamental for the next sections are proved. Section 3 is devoted to analyzing the existence and stability of equilibria, once the threshold value R_0 is provided. In particular, conditions for local and global stability of the equilibria that allow us to explain the dynamics of the disease are demonstrated. As a consequence, a scheme of the bifurcation of the system is also shown. In section 4, some numerical simulations, which corroborate the previous theoretical study, are provided. Finally, in Section 5, we provide an interpretation of the results in relation to the previously published work and spell out the major conclusions and open research directions on the significance of such conclusions.

2 Mathematical model

In this section, we establish a discrete-time model for the dynamics of the evolution of the Babesiosis disease in bovine and tick populations, considering the same influencing parameters as in the continuous-time model proposed in [6].

According to the notation in [6], we denote the bovine population by $N_B(t)$ while the tick population is denoted by $N_T(t)$. Bovines are split into three subpopulations, namely, susceptible $\bar{S}_B(t)$; infected $\bar{I}_B(t)$; and controlled $\bar{C}_B(t)$, i.e., treated against Babesiosis. On the other hand, ticks are naturally divided only into two subpopulations, specifically, susceptible $\bar{S}_T(t)$ and infected $\bar{I}_T(t)$. The birth and death rates are considered equal in each population, being denoted by μ_B for the bovine population and μ_T for the tick population.

For our purposes, susceptible bovines can become infected due to an effective transmission caused by a bite of an infected tick at a rate β_B. Similarly, susceptible ticks can become infected when biting an infected bovine at a rate β_T. To complete the model, we denote by λ_B the fraction of infected bovines which are controlled, while α_B denotes the fraction of controlled ones which return to the susceptible ones. Finally, p represents the probability that a susceptible tick is born from an infected one. We assume an homogeneous-mixing for disease dynamics, that is, all the populations have same rates of disease-causing contacts.

To build our discrete-time epidemic model, we assume that population in the $(t+1)-th$ generation is a function of the $t-th$ generation with $t \in \mathbb{N}$. Under this assumptions,

we obtain a discrete model described by the following system of difference equations:

$$\begin{cases} \bar{S}_B(t+1) = \bar{S}_B(t) + (\mu_B + \alpha_B)\,\bar{C}_B(t) - \beta_B \bar{S}_B(t)\dfrac{\bar{I}_T(t)}{N_T(t)}, \\ \bar{I}_B(t+1) = \bar{I}_B(t) + \beta_B \bar{S}_B(t)\dfrac{\bar{I}_T(t)}{N_T(t)} - \lambda_B \bar{I}_B(t), \\ \bar{C}_B(t+1) = \bar{C}_B(t) + \lambda_B \bar{I}_B(t) - (\mu_B + \alpha_B)\,\bar{C}_B(t), \\ \bar{S}_T(t+1) = \bar{S}_T(t) + \mu_T p \bar{I}_T - \beta_T \bar{S}_T(t)\dfrac{\bar{I}_B(t)}{N_B(t)}, \\ \bar{I}_T(t+1) = \bar{I}_T(t) + \beta_T \bar{S}_T(t)\dfrac{\bar{I}_B(t)}{N_B(t)} - \mu_T p \bar{I}_T(t), \end{cases} \tag{1}$$

We shall suppose that the bovine and tick populations are constant. That is, $N_B(t+1) = N_B(t)$ and $N_T(t+1) = N_T(t)$. Besides, we shall assume that all the parameters are positive, since this is biologically logical.

For the system (1) above, we use the following proportions

$$S_B(t) = \frac{\bar{S}_B(t)}{N_B(t)}, I_B(t) = \frac{\bar{I}_B(t)}{N_B(t)}, C_B(t) = \frac{\bar{C}_B(t)}{N_B(t)},$$
$$S_T(t) = \frac{\bar{S}_T(t)}{N_T(t)}, I_T(t) = \frac{\bar{I}_T(t)}{N_T(t)},$$

and the following equalities $C_B(t) = 1 - S_B(t) - I_B(t)$ and $S_T(t) = 1 - I_T(t)$ to obtain the next system of nonlinear difference equations:

$$\begin{cases} S_B(t+1) = S_B(t) + (\mu_B + \alpha_B) \\ \cdot \big(1 - S_B(t) - I_B(t)\big) - \beta_B I_T(t) S_B(t), \\ I_B(t+1) = I_B(t) + \beta_B S_B(t) I_T(t) - \lambda_B I_B(t), \\ I_T(t+1) = I_T(t) + \beta_T \big(1 - I_T(t)\big) I_B(t) - \mu_T p I_T(t). \end{cases} \tag{2}$$

Lemma 2.1. *System (2) is epidemiologically meaningful if and only if*

$$1 - (\mu_B + \alpha_B) \geq 0.$$

Proof. Observe that, at any time t, the controlled bovine population $C_B(t)$ cannot be less than zero, that is, $C_B(t) \geq 0$. Then, when passing from time t to time $t+1$, as formulated in the third equation of (1), a fraction $\mu_B C_B(t)$ of this population dies a natural death while a fraction $\alpha_B C_B(t)$ of the controlled bovine return to susceptible state. Therefore, it is impossible that the sum of these two amounts exceed the value of the initial stock, i.e.,

$$C_B(t) - (\mu_B C_B(t) + \alpha_B C_B(t)) = [1 - (\mu_B + \alpha_B)] C_B(t) \geq 0,$$
$$\forall C_B(t) \geq 0.$$

From the inequality above, we can deduce that

$$1 - (\mu_B + \alpha_B) \geq 0, \quad \forall C_B(t) > 0,$$

which is also valid for $C_B(t) = 0$, since $1 - (\mu_B + \alpha_B)$ does not depend on $C_B(t)$. □

Remark 2.2. Proceeding in a similar way as in the proof of Lemma 2.1, from the rest of the equations of system (1), it can be also deduced

$$1 - \beta_B \geq 0, \quad 1 - \lambda_B \geq 0, \quad 1 - \beta_T \geq 0, \quad 1 - \mu_T p \geq 0.$$

Nevertheless, it is not necessary to proceed in such a way. Actually, since all the parameters involved in such inequalities represent rates, fractions of a normalized population or probability (see [6]) in order to have a epidemiologically meaningful model, all of them are less than or equal 1 and such inequalities hold automatically.

Proposition 2.3. *The region*

$$\Omega = \Big\{(S_B, I_B, I_T) \in \mathbb{R}^3_+ : 0 \leq S_B + I_B \leq 1, 0 \leq I_T \leq 1\Big\}$$

is a positive invariant set for system (2).

Proof. The previous conditions in Lemma 2.1 and Remark 2.2 can be used to prove that (2) is well posed. Effectively, suppose that $\big(S_B(t), I_B(t), I_T(t)\big)$ is in the region Ω at any initial time t.

First of all, looking at (1) from which these conditions come, one can easily check that $\bar{S}_B(t+1), \bar{I}_B(t+1), \bar{I}_T(t+1)$ in system (2) are greater than or equal to zero and consequently $S_B(t+1), I_B(t+1), I_T(t+1)$ are greater than or equal to zero. Thus, in particular, we have

$$0 \leq S_B(t+1) + I_B(t+1) \quad \text{and} \quad 0 \leq I_T(t+1).$$

Additionally, we have

$$\begin{aligned} S_B(t+1) + I_B(t+1) &= S_B(t) + I_B(t) + (\mu_B + \alpha_B) \\ &\quad \cdot \big(1 - S_B(t) - I_B(t)\big) - \lambda_B I_B(t) \\ &= [1 - (\mu_B + \alpha_B)](S_B(t) \\ &\quad + I_B(t)) + (\mu_B + \alpha_B) - \lambda_B I_B(t) \\ &\leq 1 - (\mu_B + \alpha_B) + (\mu_B + \alpha_B) = 1. \end{aligned}$$

Analogously, we have

$$\begin{aligned} I_T(t+1) &= I_T(t) + \beta_T \big(1 - I_T(t)\big) I_B(t) - \mu_T p I_T(t) \\ &\leq I_T(t) + \beta_T - \beta_T I_T(t) \\ &= (1 - \beta_T) I_T(t) + \beta_T \\ &\leq 1 - \beta_T + \beta_T = 1. \end{aligned}$$

Therefore, the region Ω is a positive invariant set for system (2). □

In such a context, we shall consider the region Ω as the state space of system (2).

3 Results on the existence and stability of equilibria

This section is devoted to studying the existence and stability of the equilibria of model (2). In this sense, we shall assume the following threshold parameter:

$$\mathcal{R}_0 = \frac{\beta_B \beta_T}{\lambda_B \mu_T p}.$$

The value of this parameter means that each infected bovine produces $\frac{\beta_T}{\mu_T p}$ new infected ticks over its expected infectious period, and each infected tick produces $\frac{\beta_B}{\lambda_B}$ new infected bovines over its expected infectious period.

Contrary to the continues-time case, the (parametric) positive constraints obtained in Lemma 2.1 and Remark 2.2 become fundamental for the proofs of the results in our discrete-time case. Although such results are similar to the continuous case, the proofs need to be performed by using different techniques corresponding to discrete dynamical systems.

Proposition 3.1. *System (2) has a disease-free equilibrium* $E_1^\star = (1, 0, 0)$ *for all the values of the (positive) parameters, while only if* $\mathcal{R}_0 > 1$, *there exists a unique endemic equilibrium* $E_2^\star = (S_{B_2}^\star, I_{B_2}^\star, I_{T_2}^\star)$ *in the region* Ω.

Proof. A fixed point $E^\star = (S_B^\star, I_B^\star, I_T^\star)$ of model (2) can be obtained by solving the equations below

$$\begin{cases} (\mu_B + \alpha_B)\left(1 - S_B(t) - I_B(t)\right) + \left(1 - \beta_B I_T(t)\right) S_B(t) = S_B(t), \\ \beta_B S_B(t) I_T(t) + (1 - \lambda_B) I_B(t) = I_B(t), \\ \beta_T \left(1 - I_T(t)\right) I_B(t) + (1 - \mu_T p) I_T(t) = I_T(t). \end{cases} \tag{3}$$

This is equivalent to solve the following system

$$\begin{cases} (\mu_B + \alpha_B)\left(1 - S_B(t) - I_B(t)\right) - \beta_B I_T(t) S_B(t) = 0, \\ \beta_B S_B(t) I_T(t) - \lambda_B I_B(t) = 0, \\ \beta_T \left(1 - I_T(t)\right) I_B(t) - \mu_T p I_T(t) = 0. \end{cases} \tag{4}$$

If $I_B(t) = 0$ and $I_T(t) = 0$, from the first equation of system (4), one can easily check that $S_B(t) = 1$ independently of the values of the parameters. Therefore the disease-free equilibrium $E_1^\star = (1, 0, 0)$ exists for any value of the parameters. This is epidemiologically meaningful since, when there is no infected individual, all the bovine population become susceptible.

Now, if we suppose that $I_B(t) > 0$, $I_T(t) > 0$ and consider the second and third equations of (4), we get

$$S_B(t) = \frac{\lambda_B I_B(t)}{\beta_B I_T(t)}, \; I_T(t) = \frac{\beta_T I_B(t)}{\beta_T I_B(t) + \mu_T p}. \tag{5}$$

Taking into account the above equalities, we can replace $S_B(t)$ and $I_T(t)$ into the first equation of (4) to obtain the following dependent function for the subpopulation of infected bovines:

$$\begin{aligned} F(I_B) = (\mu_B + \alpha_B)\left(1 - \frac{1}{\mathcal{R}_0}\right) \\ - \frac{(\mu_B + \alpha_B)\lambda_B}{\beta_B}\left(1 + \frac{\beta_B}{\lambda_B} + \frac{\beta_B}{\mu_B + \alpha_B}\right) I_B, \end{aligned} \tag{6}$$

where $F(I_B)$ is obviously continuous and strictly decreasing in $[0, 1]$.

If $\mathcal{R}_0 \le 1$, then

$$F(0) = (\mu_B + \alpha_B)\left(1 - \frac{1}{\mathcal{R}_0}\right) \le 0$$

and since F is strictly decreasing in $[0, 1]$, there is no $0 < I_B^\star < 1$ such that $F(I_B^\star) = 0$. Thus, the model (2) has only an equilibrium: the disease-free one.

Now, if $\mathcal{R}_0 > 1$, then we have

$$F(0) = (\mu_B + \alpha_B)\left(1 - \frac{1}{\mathcal{R}_0}\right) > 0,$$

while

$$\begin{aligned} F(1) &= (\mu_B + \alpha_B)\left(1 - \frac{1}{\mathcal{R}_0}\right) \\ &\quad - \frac{(\mu_B + \alpha_B)\lambda_B}{\beta_B}\left(1 + \frac{\beta_B}{\lambda_B} + \frac{\beta_B}{\mu_B + \alpha_B}\right) \\ &< (\mu_B + \alpha_B) - (\mu_B + \alpha_B)\frac{\lambda_B}{\beta_B}\left(1 + \frac{\beta_B}{\lambda_B} + \frac{\beta_B}{\mu_B + \alpha_B}\right) \\ &= (\mu_B + \alpha_B)\left[1 - \left(1 + \frac{\lambda_B}{\beta_B} + \frac{\lambda_B}{\mu_B + \alpha_B}\right)\right] \\ &= (\mu_B + \alpha_B)\left[-\left(\frac{\lambda_B}{\beta_B} + \frac{\lambda_B}{\mu_B + \alpha_B}\right)\right] < 0 \end{aligned}$$

and, since F is continuous and strictly decreasing in $[0, 1]$, there exists a unique $0 < I_B^\star < 1$ such that $F(I_B^\star) = 0$. Therefore, model (2) has a unique endemic equilibrium $E_2^\star = \left(S_{B_2}^\star, I_{B_2}^\star, I_{T_2}^\star\right)$. In fact, one can check that

$$I_{B_2}^\star = \frac{(\mu_B + \alpha_B)(\beta_B \beta_T - \lambda_B \mu_T p)}{\beta_T \alpha_B (\beta_B + \lambda_B) + \mu_B \beta_T \lambda_B + \beta_T \beta_B (\lambda_B + \mu_B)}.$$

Substituting $I_{B_2}^\star$ in the second equation of (5), we obtain

$$I_{T_2}^\star = \frac{(\mu_B + \alpha_B)(\beta_B \beta_T - \lambda_B \mu_T p)}{\beta_T \beta_B (\alpha_B + \mu_B) + \beta_B \mu_T p (\alpha_B + \lambda_B + \mu_B)}.$$

and, replacing $I_{B_2}^\star$ and $I_{T_2}^\star$ in the first equation of (5), we have

$$S_{B_2}^\star = \frac{\beta_T \lambda_B (\alpha_B + \mu_B) + \lambda_B \mu_T p (\alpha_B + \lambda_B + \mu_B)}{\beta_T \alpha_B (\beta_B + \lambda_B) + \beta_T \lambda_B \mu_B + \beta_T \beta_B (\lambda_B + \mu_B)}.$$

$\left(S^{\star}_{B_2}, I^{\star}_{B_2}, I^{\star}_{T_2}\right)$ is in the interior of Ω, as is demonstrated below. To do such a demonstration, we must verify that

$$0 < S^{\star}_{B_2} + I^{\star}_{B_2} < 1 \quad \text{and} \quad 0 < I^{\star}_{T_2} < 1.$$

First of all, observe that, since $R_0 > 1$, we have that $\lambda_B\mu_T p - \beta_T\beta_B > 0$. Besides, all the parameters involved in the expressions of $S^{\star}_{B_2}, I^{\star}_{B_2}, I^{\star}_{T_2}$ are greater than zero. Hence, both conditions allow us to prove that $S^{\star}_{B_2}, I^{\star}_{B_2}, I^{\star}_{T_2} > 0$. In particular, we also have that $S^{\star}_{B_2} + I^{\star}_{B_2} > 0$.

Secondly, observe that

$$S^{\star}_{B_2} + I^{\star}_{B_2} = \frac{\beta_T\lambda_B\left(\alpha_B+\mu_B\right) + \lambda_B^2\mu_T p + \beta_B\beta_T\left(\mu_B+\alpha_B\right)}{\beta_T\alpha_B\left(\beta_B+\lambda_B\right) + \mu_B\beta_T\lambda_B + \beta_T\beta_B\left(\lambda_B+\mu_B\right)} < 1,$$

if and only if

$$\beta_T\lambda_B\left(\alpha_B+\mu_B\right) + \lambda_B^2\mu_T p + \beta_B\beta_T\left(\mu_B+\alpha_B\right) < \beta_T\alpha_B\left(\beta_B+\lambda_B\right) + \mu_B\beta_T\lambda_B + \beta_T\beta_B\left(\lambda_B+\mu_B\right),$$

Canceling all the common terms, the inequality above becomes

$$\lambda_B^2\mu_T p < \lambda_B\beta_T\beta_B.$$

But, this last inequality is equivalent to $\lambda_B\mu_T p - \beta_T\beta_B < 0$, which holds since $R_0 > 1$.

Finally, note that $I^{\star}_{T_2} < 1$ if and only if

$$\left(\mu_B+\alpha_B\right)\left(\beta_B\beta_T - \lambda_B\mu_T p\right) < \beta_T\beta_B\left(\alpha_B+\mu_B\right) + \beta_B\mu_T p\left(\alpha_B+\lambda_B+\mu_B\right)$$

Again, canceling some common terms, the inequality above becomes the following equivalent one

$$\left(\mu_B+\alpha_B\right)\left(-\lambda_B\mu_T p\right) < \beta_B\mu_T p\left(\alpha_B+\lambda_B+\mu_B\right),$$

which is true since its left hand side is less than zero, while its right hand side is greater than zero.

□

Now, we are going to analyze the local stability of the disease-free fixed point. In order to do that, we consider the Jacobian matrix related to system (2) given by

$$J\left(S^{\star}_B, I^{\star}_B, I^{\star}_T\right) = \begin{pmatrix} 1-\left(\mu_B+\alpha_B\right)-\beta_B I^{\star}_T & -\left(\mu_B+\alpha_B\right) & -\beta_B S^{\star}_B \\ \beta_B I^{\star}_T & 1-\lambda_B & \beta_B S^{\star}_B \\ 0 & \beta_T\left(1-I^{\star}_T\right) & 1-\mu_T p-\beta_T I^{\star}_B \end{pmatrix} \tag{7}$$

As in the continuous case, the demonstration takes into account the eigenvalues of this Jacobian matrix evaluated at the equilibria. Nevertheless, the method we apply here is different from the one employed for the continuous-time case in [6]. In this case, if all the eigenvalues of $J(E^{\star})$ have magnitude less than one, then the equilibrium $E^{\star}$ is locally asymptotically stable, i.e., all solutions of system (2) sufficiently close to the equilibrium point approach it. Actually, we will prove that the conditions of the Jury-Criterion (see [14] or [24]) are satisfied by the equilibria.

Theorem 3.2. *The disease-free equilibrium $E^{\star}_1$ of (2) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. The characteristic polynomial at the disease-free equilibrium $E^{\star}_1 = (1, 0, 0)$ is

$$p(\gamma) = (1-\mu_B-\alpha_B-\gamma)\left(\gamma^2 + a_1\gamma + a_2\right),$$

where $a_1 = \mu_T p + \lambda_B - 2$ and $a_2 = 1 + \mu_T p\lambda_B - \beta_T\beta_B - \mu_T p - \lambda_B$.

The first root of the polynomial is $\gamma_1 = 1-(\mu_B+\alpha_B)$. By Lemma (2.1), we have that $|1-(\mu_B+\alpha_B)| = 1-(\mu_B+\alpha_B)$ and since μ_B, α_B are greater than zero,

$$|\gamma_1| = |1-(\mu_B+\alpha_B)| = 1-(\mu_B+\alpha_B) < 1.$$

Now, to analyze if the other two roots satisfy $|\gamma_{2,3}| < 1$, we need to check the Jury conditions.

The characteristic polynomial of the Jacobian matrix $J(E^{\star}_1)$ can be rewritten as

$$p(\gamma) = (1-\mu_B-\alpha_B-\gamma)\left(\gamma^2 - tr(\hat{J})\gamma + det(\hat{J})\right),$$

where $\hat{J}$ is the submatrix 2×2 of $J\left(E^{\star}_1\right)$ given by,

$$\hat{J}(1,0,0) = \begin{pmatrix} 1-\lambda_B & \beta_B \\ \beta_T & 1-\mu_T p \end{pmatrix}. \tag{8}$$

In this context, $\mathrm{Tr}(\hat{J}) = (1-\lambda_B) + (1-\mu_T p)$ and $\det(\hat{J}) = (1-\lambda_B)(1-\mu_T p) - \beta_T\beta_B$. The (simplified) Jury criterion (see [24]) states that the eigenvalues of $\hat{J}$ have magnitude less than one if and only if $\left|\mathrm{Tr}(\hat{J})\right| < \det(\hat{J}) + 1 < 2$.

Note that, the inequality $\det(\hat{J}) + 1 < 2$ is equivalent to $\det(\hat{J}) < 1$. This is the one we are going to prove next. Since $1-\lambda_B \le 1$ and $1-\mu_T p \le 1$, we also have that $(1-\lambda_B)(1-\mu_T p) \le 1$ and, taking into account that $\beta_T, \beta_B > 0$, we have that

$$\det(\hat{J}) = (1-\lambda_B)(1-\mu_T p) - \beta_T\beta_B < 1$$

is always satisfied.

On the other hand, observe that in this case $\mathrm{Tr}(\hat{J}) \ge 0$, since $1-\lambda_B \ge 0$ and $1-\mu_T p \ge 0$. Hence, $\left|\mathrm{Tr}(\hat{J})\right| = \mathrm{Tr}(\hat{J})$ and we must demonstrate that

$$(1-\lambda_B) + (1-\mu_T p) < 1 + (1-\lambda_B)(1-\mu_T p) - \beta_T\beta_B$$

which is equivalent to

$$2 - \lambda_B - \mu_T p < 2 - \lambda_B - \mu_T p + \lambda_B \mu_T p - \beta_T \beta_B.$$

After removing all the common terms in both sides , the inequality $\mathrm{Tr}(\hat{J}) < 1 + \det(\hat{J})$ becomes

$$0 < \mu_T p \lambda_B - \beta_T \beta_B$$

which is true if and only if $\mathcal{R}_0 < 1$. □

In the next theorem, we prove the global stability of the disease-free equilibrium when $\mathcal{R}_0 \leq 1$, using the LaSalle Invariance Principle for discrete-time systems given in [25].

Theorem 3.3. *The disease-free equilibrium E_1^* of system (2) is globally asymptotically stable if $\mathcal{R}_0 \leq 1$.*

Proof. First of all, we relocate our disease-free equilibrium to the origin of coordinates. That is, we perform the change of coordinates

$$X_B(t) = 1 - S_B(t)$$

in the system (2) and it becomes

$$\begin{cases} X_B(t+1) = \left[1 - (\mu_B + \alpha_B) - \beta_B I_T(t)\right] X_B(t) \\ +(\mu_B + \alpha_B) I_B(t) + \beta_B I_T(t), \\ I_B(t+1) = \beta_B \left(1 - X_B(t)\right) I_T(t) + (1 - \lambda_B) I_B(t), \\ I_T(t+1) = \beta_T \left(1 - I_T(t)\right) I_B(t) + (1 - \mu_T p) I_T(t). \end{cases} \tag{9}$$

We consider the following Lyapunov function $V : \Omega \to \mathbb{R}_+$ defined by

$$V\left(X_B(t), I_B(t), I_T(t)\right) = \beta_T I_B(t) + \lambda_B I_T(t),$$

where $V \in C(\Omega, \mathbb{R}_+)$, such that $V(0,0,0) = 0$ and $V\left(X_B(t), I_B(t), I_T(t)\right) \geq 0$ in $\Omega - (0,0,0)$.

Then, the difference

$$\begin{aligned} \Delta V(X_B, I_B, I_T) = {} & V\left(X_B(t+1), I_B(t+1), I_T(t+1)\right) \\ & - V\left(X_B(t), I_B(t), I_T(t)\right) \end{aligned}$$

is given by

$$\begin{aligned} & \beta_T \left[\beta_B \left(1 - X_B(t)\right) I_T(t) + (1 - \lambda_B) I_B(t)\right] \\ & + \lambda_B \left[\beta_T \left(1 - I_T(t)\right) I_B(t) + (1 - \mu_T p) I_T(t)\right] \\ & - \beta_T I_B(t) - \lambda_B I_T(t). \end{aligned}$$

Simplifying and grouping terms, we have

$$\begin{aligned} & \Delta V\left(X_B(t), I_B(t), I_T(t)\right) = \\ & \left[(\beta_T \beta_B - \lambda_B \mu_T p) - \beta_T \beta_B X_B(t) - \lambda_B \beta_T I_B(t)\right] I_T(t). \end{aligned} \tag{10}$$

At this point, we shall prove that, if $\mathcal{R}_0 \leq 1$, this last expression is non-positive for every $\left(X_B(t), I_B(t), I_T(t)\right)$ in Ω.

As by hypotheses $\mathcal{R}_0 \leq 1$, equivalently, we have that

$$(\beta_T \beta_B - \lambda_B \mu_T p) \leq 0.$$

Additionally, as all the parameters are greater than zero and $X_B(t), I_B(t), I_T(t)$ are non-negative in Ω, we can deduce that

$$\Delta V\left(X_B(t), I_B(t), I_T(t)\right) \leq 0, \qquad \text{for } \mathcal{R}_0 \leq 1.$$

Now, we need to obtain the maximal positively invariant set G^* contained in the subset $G \subset \Omega$ given by

$$G = \{(X_B, I_B, I_T) \in \Omega : \quad \Delta V(X_B, I_B, I_T) = 0\}.$$

We shall distinguish two cases, depending on the values of the threshold parameter R_0:

- $\underline{\mathcal{R}_0 < 1}$: In this case, expression (10) equals zero if and only if $I_T(t) = 0$. The system (9) in such points becomes

$$\begin{cases} X_B(t+1) = \left[1 - (\mu_B + \alpha_B)\right] X_B(t) + (\mu_B + \alpha_B) I_B(t), \\ I_B(t+1) = (1 - \lambda_B) I_B(t), \\ I_T(t+1) = \beta_T I_B(t). \end{cases} \tag{11}$$

Observe that, for every initial state of the form $\left(X_B(t), I_B(t), 0\right)$, with $I_B(t) > 0$, the following state in its orbit verifies that $I_T(t+1) = \beta_T I_B(t) > 0$. Thus, no orbit of a point of the form $\left(X_B(t), I_B(t), 0\right)$, with $I_B(t) > 0$ is contained in such a set of points $G \subset \Omega$ for which $\Delta V(X_B, I_B, I_T) = 0$. Nevertheless, if we avoid this problem considering only the points of G for which $I_B(t) = 0$, one can easily check that, for any point in such a subset, the iteration of the system (9) reduces to

$$\begin{cases} X_B(t+1) = \left[1 - (\mu_B + \alpha_B)\right] X_B(t), \\ I_B(t+1) = 0, \\ I_T(t+1) = 0. \end{cases} \tag{12}$$

That is, the orbit of any initial state in the subset of G given by $I_B(t) = 0, I_T(t) = 0$ remains in such a subset, i.e., the largest positively invariant set contained in G is

$$G^* = \{(X_B, I_B, I_T) \in \Omega : \ I_B = I_T = 0\}.$$

Moreover, note that, since $0 \leq \left[1 - (\mu_B + \alpha_B)\right] < 1$, the (disease-free) equilibrium $(0,0,0)$ is G^*–globally asymptotically stable. At this point, since all the orbits of the system remain in Ω, all of them are bounded. Therefore, applying the LaSalle Invariance Principle for discrete dynamical systems given in Theorem 3.3 of [25], we can conclude that the disease-free equilibrium is globally asymptotically stable in Ω.

- $\mathcal{R}_0 = 1$: In this case, expression (10) equals zero if and only if $I_T(t) = 0$ or $X_B(t) = I_B(t) = 0$. Therefore, the set $G \subset \Omega$ for which $\Delta V(X_B, I_B, I_T) = 0$ is the following:

$$G = \{(X_B, I_B, I_T) \in \Omega : I_T = 0\} \cup \{(X_B, I_B, I_T) \in \Omega : X_B I_B = 0\}.$$

Observe that for any initial point of the form $(0, 0, I_T(t))$ with $I_T(t) > 0$, the system (9) becomes

$$\begin{cases} X_B(t+1) = \beta_B I_T(t), \\ I_B(t+1) = \beta_B I_T(t), \\ I_T(t+1) = (1 - \mu_T p) I_T(t). \end{cases} \tag{13}$$

This proves that no orbit of an initial state in the subset $\{(X_B, I_B, I_T) \in \Omega : X_B = I_B = 0\}$ with $I_T(t) > 0$ remains in such a subset of G. In fact, only the orbit originated by $(0, 0, 0)$ remains in this subset.

At this point, proceeding as in the previous case, the largest positively invariant set contained in G is

$$G^\star = \{(X_B, I_B, I_T) \in \Omega : I_B = I_T = 0\}.$$

Moreover, note that, since $0 \leq [1 - (\mu_B + \alpha_B)] < 1$, the (disease-free) equilibrium $(0, 0, 0)$ is $G^\star$–globally asymptotically stable, and, since all the orbits of the system remain in Ω, all of them are bounded.

Therefore, applying the LaSalle Invariance Principle for discrete dynamical systems again, we can conclude that the disease-free equilibrium is globally asymptotically stable in Ω, also in this case.

□

This last result is epidemiologically significant, because it indicates that if a small number of infective individuals is introduced in a susceptible population, then the disease vanishes.

For $\mathcal{R}_0 > 1$, the endemic equilibrium $E_2^\star$ is locally asymptotically stable, as shown by numerical simulations. Actually, it can be seen that $E_2^\star$ is globally asymptotically stable in $\Omega - \{(1, 0, 0)\}$. That is, every initial condition $(S_B(0), I_B(0), I_T(0)) \in \Omega$ produces a trajectory $(S_B(t), I_B(t), I_T(t)) \in \Omega$ which converges to the unique interior fixed point $E_2^\star$. However, $E_2^\star$ is not globally asymptotically stable in Ω, because the disease-free point is also in Ω.

Besides, when $\mathcal{R}_0 < 1$, the system has also the two equilibria, $E_1^\star$ asymptotically stable in Ω and $E_2^\star$ unstable, being $E_2^\star$ in the outside of Ω.

Moreover, when $\mathcal{R}_0 = 1$, $E_1^\star$ is the unique point of the system being a non-hyperbolic fixed point which is asymptotically stable in Ω.

Such issues allow us to infer the following corollary.

Corollary 3.4. *System (2) undergoes a transcritical bifurcation at the parameter value* $\mathcal{R}_0 = 1$.

4 Experimental procedures

In [6], we show through numerical simulations that when $\mathcal{R}_0 > 1$, the endemic equilibrium $E_2^\star$ is locally asymptotically stable. However, as the disease-free equilibrium $E_1^\star$ is in Ω, the endemic point is not globally asymptotically stable in this region. But, it can be observed (numerically) that it is in the region $\Omega - \{E_1^\star\}$. As in the continuous case, our numerical simulations in this work proved that it is still verified.

We consider the same parameter values as in the continuous-time model and the parameter constraints given by Lemma (2.1), with initial conditions $S_B = 0.3756$, $I_B = 0.5184$, $I_T = 0,6000$. In Figure 1 panel (a), we show that all the trajectories of S_B (blue curve), I_B (green curve) and I_T (red curve) converges to the disease-free equilibrium point $(1, 0, 0)$, if the reproduction number $\mathcal{R}_0 < 1$, as was demonstrated in Theorem 3.2. In this case, all the eigenvalues of its Jacobian matrix are less than 1, $(0.9987,\ 0.9735,\ 0.9992)$. Note that, if $\mathcal{R}_0 \leq 1$, $E_1^\star$ is still globally asymptotically stable, as shown in Figure 1, panel (b) in the state space.

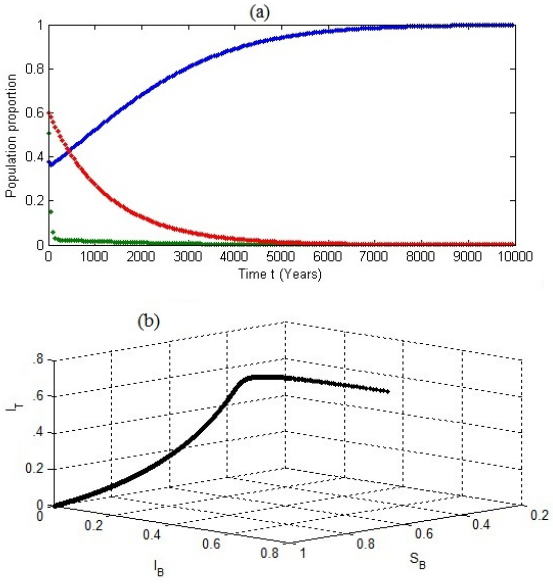


Figure 1: Parameter values: $\mu_B = 0.0002999$, $\mu_T = 0.001609$, $\alpha_B = 0.001$, $\beta_T = 0.00048$. (a) for $\mathcal{R}_0 = 0.0068$ with $\beta_B = 0.003$, $\lambda_B = 0.0265$ and $p = 0.5$. (b) for $\mathcal{R}_0 < 1$ with $\beta_B = 0.006$, $p = 0.1$ and $\lambda_B = 0.000265$

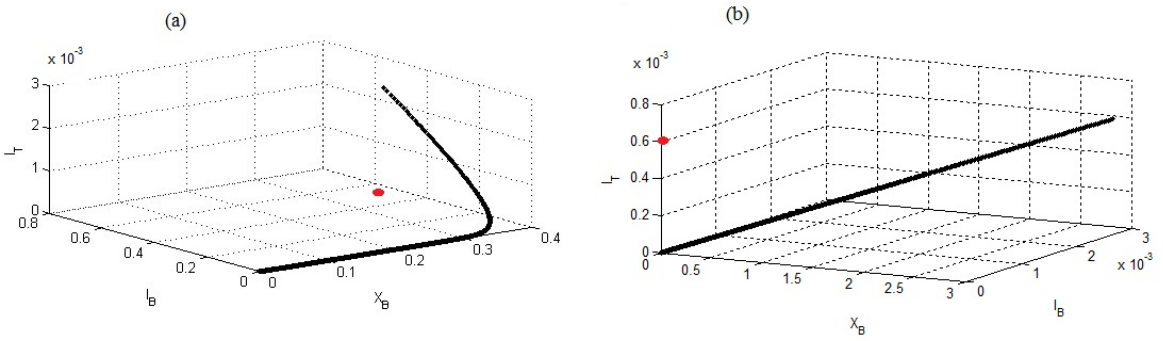


Figure 2: The red point is an initial value condition for $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 = 1$, panel (a) and (b) respectively

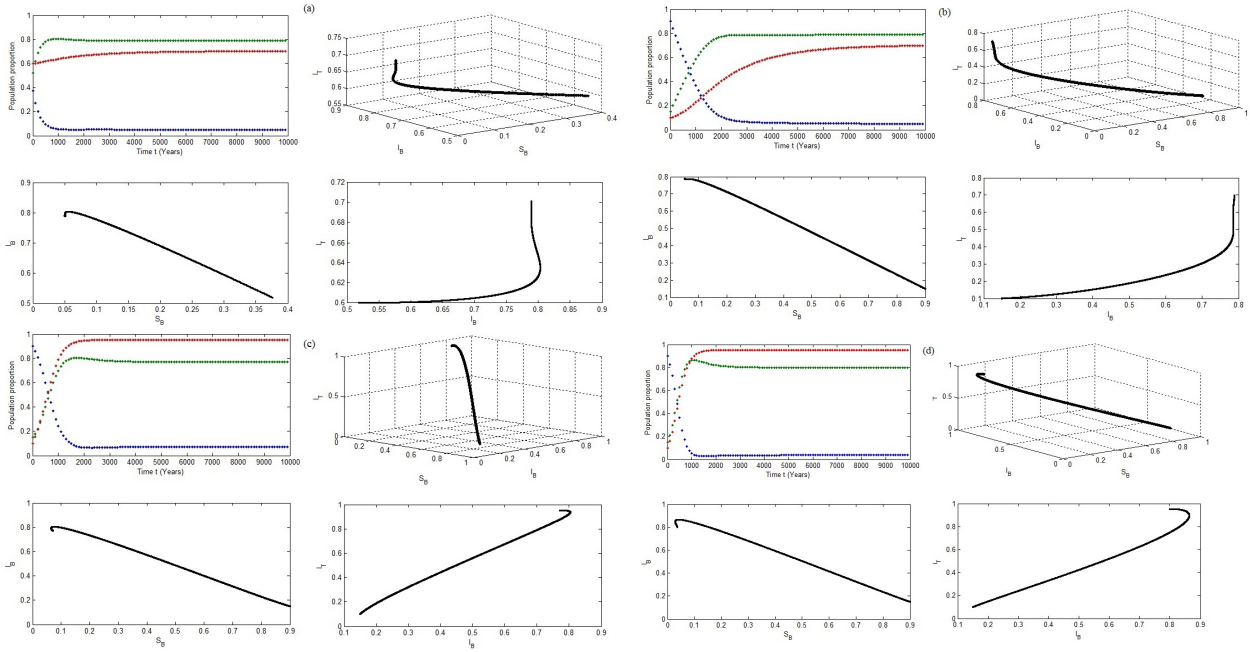


Figure 3: For panel (a) which initial condition $S_B = 0.3756$, $I_B = 0.5184$, $I_T = 0, 6000$ and panel (b), (c) and (d) which $S_B = 0.9$, $I_B = 0.15$, $I_T = 0, 1$

Table 1: $\mu_B = 0.0002999$, $\mu_T = 0.001609$, $\alpha_B = 0.001$, $p = 0.1$ and $\lambda_B = 0.000265$

Parameter	$\mathcal{R}_0$	Eigenvalues	$E^\star = (S_B^\star, I_B^\star . I_T^\star)$
$\beta_B = 0.006, \beta_T = 0.00048$	67, 5446	(0.9959, 0.9983, 0.995)	(0.0501, 0.7894, 0.7019)
$\beta_B = 0.006, \beta_T = 0.00048$	67, 5446	(0.9959, 0.9983, 0.995)	(0.0501, 0.7894, 0.7019)
$\beta_B = 0.003, \beta_T = 0.004$	281, 4358	(0.9980, 0.9977, 0.9967)	(0.0728, 0.7711, 0.9504)
$\beta_B = 0.006, \beta_B = 0.004$	562, 8716	(0.9959, 0.9983, 0.9967)	(0.0374, 0.7998, 0.9521)

In Figure 2, we can check that $\{(0, 0, 0)\}$ is the largest positively invariant set contained in Ω, therefore of disease-free equilibrium point is globally asymptotically stable as shown in Theorem 3.3.

The following scenarios show that $\mathcal{R}_0 > 1$ (see Table 1) when having high transmission of the disease in the bovine population, high vertical transmissibility in the ticks population and low control of infected cattle. In this context, we show that every trajectory converges to the endemic equilibrium $E_2^\star = (S_{B_2}^\star, I_{B_2}^\star . I_{T_2}^\star)$ except, of course, the one starting at $(0, 0, 0)$

5 Discussion

In the present work, we provide and study a discrete model for the transmission of Babesiosis disease in bovine and tick populations. It supposes a discretization of the continuous-time model developed by us previously which has served as a base for other works on this disease.

The results, here obtained by discrete methods as opposed to continuous ones, show that similar conclusions can be obtained for the discrete model subject to the assumption of some parametric constraints which were not necessary in the continuous case. We prove that these parametric constraints are not artificial and, in fact, they can be deduced from the biological significance of the model.

This study represents a first attempt to model the dynamics of bovine Babesiosis by a discrete model. Due to this novelty in the modelization, it must be compared to the continuous model. Since the obtained results on the dynamics are similar, we are able to confirm the absence of contradictions between both versions and the skill in the selection of the factors to design the model.

Furthermore, it opens a new research line by using other discretization schemes and the comparison of the corresponding results in order to debate on such new formulations.

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