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Effect of Weak Vaccination on the Outbreak of the Babesiosis-Bovine Epidemic Model

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Abstract. The mathematical model of bovine babesiosis with vaccination helps researchers understand disease spread, assess vaccine effectiveness, and make predictions to guide the development of vaccination strategies that protect cattle. This model uses a number known as the basic reproduction number $\mathcal{R}_0$ to predict the spread of a disease in herds of cattle. The model predicts that the illness will eventually spread if $\mathcal{R}_0 < 1$. The Babesiosis bovine disease is predicted to persist if $\mathcal{R}_0 > 1$. Knowing this makes it easier to create immunization plans that work. To combat babesiosis, a serious disease that is spread by ticks to cattle, a vaccination is desperately needed. Some further discussion on the optimal amount of bovine vaccination is discussed. Some numerical simulations are utilized to validate the theoretical results.

2020 Mathematics Subject Classifications: 92D30, 34D20, 37N25

Key Words and Phrases: Babesiosis, diseases, vaccination, basic reproduction number

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1. Introduction

The tick-borne hemoprotozoans Babesia bovis, Babesia bigemina, and Babesia divergent commonly cause significant morbidity and mortality of livestock in many parts of the world. Although existing vaccines provide protection, and they have significant drawbacks. For this reason, significant research is being carried out to improve vaccination strategies, especially in countries where large numbers of cattle are at risk. Here, we provide a comprehensive overview of live vaccines currently in use and as the current status of investigational vaccine trials. Additionally, there are relevant areas of research that could help create new live or non-living vaccines, notably the parasitic antigens involved in the invasion of the host cell and in the interactions between pathogens and ticks, as well as immune protection against infections. The range of potential vaccine candidates is constantly being expanded through the exploration of available parasite genomes, furthermore, the recent creation of a Babesia transfection technique can have a major impact on vaccine development. However, vaccine research against Babesia is hampered by the complexity and high costs of vaccination trials, and by far severely, methodically antigen testing as well as candidates' evaluation of formulations for various immunomodulators using in-depth immunomodulators and antigen delivery technologies [1–6].

Recently in [7], Jerzak et al. proposed that currently attenuated Babesia vaccinations, anti-Babesia medications, and tick management are used in a multifaceted strategy to prevent bovine babesiosis. Vaccines that have been attenuated show promise in reducing neurological symptoms and limiting the disease's transmission by ticks. Adult animals are vulnerable to acute sickness, however, calves less than a year old are immunized against babesiosis in endemic countries. Erythrocytes infected with B. bovis are used in a formula to make live, attenuated vaccines. Usually, the cells were taken from splenectomy-treated infected calves. During the acute phase of the reaction, following infection with a particular parasite strain, the blood of pathogen-free animals is drawn from their jugular vein. The possibility of the vaccine strain regaining virulence and contamination by blood-borne pathogens are two disadvantages of live attenuated blood-based vaccinations that are highlighted by Mazuz et al. Furthermore, it is difficult to maintain the vaccine's efficacy in tropical, endemic areas because of the cold chain requirement. The ability to cultivate Babesia parasites outside of the host (in vitro) has opened up new avenues for vaccine development [7].
A live B. bovines inoculation offers immunity for a minimum of four years; for B. bigemina, this duration may be shortened. Studies indicate that the degree of protection (acquired immunity) correlates with the severity of a previous infection (antigenic stimulation) rather than the presence of an ongoing infection [8]. About the future of vaccine development to stop the spread of babesiosis global effort is in progress. Scientists are carefully studying each phase of the B. bovis life cycle in order to determine which targets will work best for a vaccine [7].

Since the susceptible-infected-recovered (SIR) model developed by McKendrick and

Kermack [9] is the most important model for understanding the disease dynamics of epidemics in the simplest formulation to describe how epidemic spreads on a population. Aranda et al. (2012) in [10] used a system of ordinary differential equations to present a mathematical model for bovine babesiosis. To improve the formulation in [10], Friedman and Yacobo. (2014) in [11] reformulated it using partial differential equations to illustrate the transmission of bovine babesiosis in both bovine and ticks. This reformulation incorporated diffusion parameters within each of the temporal equations. Based on the work of [10], Carvalho et al. (2015) in [12] incorporated the fractional Caputo derivative into the model, replacing the ordinary derivative. This approach, utilizing comparison theory for partial order systems, allowed them to demonstrate the global stability of the disease-free equilibrium point. Moreover, the model presented in [10] provided a fundamental basis for later research examining other factors. For example, [13] examined a two-stage strategy (approach) in the livestock domain, while [13] examined the effects of seasonal variations. Pourbashash H. (2018) used the multi-stage modified Sinc approach in [14] to explore the dynamic features of the [10] model. Using discrete temporal models, Hu Z. et al. enhanced numerical simulations in [14]. Analogously, Aranda et al. (2017) investigated a discrete-time adaptation of the [10] model, producing dynamics similar to those seen in [10]. Mezouaghi and Belhamiti (2019) created a spatiotemporal mathematical model to predict the transmission of bovine babesiosis in cattle herds, taking into account the role of tick populations. A mathematical model that considers the role of tick populations in spatiotemporal prediction of bovine babesiosis transmission in cattle herds was developed by Mezouaghi and Belhamiti (2019) [15]. Aqeel et al. (2020), in their study models and analyzes bovine babesiosis disease using fractional calculus. A system describing the disease and tick populations in a fractional order framework is solved using the Caputo and Atangana-Baleanu-Caputo (ABC) fractional derivatives [16]. For more reading in this context, we mention a few works [17, 18].

In this work, we present a model of a bovine babesiosis epidemic with vaccination, and we represent it using ordinary differential equations to study the dynamic behavior of bovine babesiosis and monitor the effect of vaccination on this behavior, We can investigate the efficacy of vaccination in stopping the spread of bovine babesiosis in cattle by integrating it into the model SIR. We may then assess the effect of vaccination on the disease by simulating and analyzing different vaccination scenarios using the model's results. Therefore, for this study, it is important to asses whether the vaccination for babesiosis-bovine infection is important, and efficient to contain the epidemic.

The structure of this paper is as follows. In Section 2, we model bovine babesiosis with first-order nonlinear differential equations. In Section 3, we show that the model presented in Section 2 is well posed by verifying the existence and uniqueness of the solution. In Section 4, we identify the basic reproduction number $\mathcal{R}_0$ using the next generation matrix method. In Section 5, We show the conditions for the existence of equilibrium points. In Section 6, we study the local and global stability of equilibrium points. In Section 7, We show the extent of the effect of vaccination on reducing the spread of bovine babesiosis.

In Section 8, we present a numerical simulation of the results obtained from studying this model.

2. Mathematical model

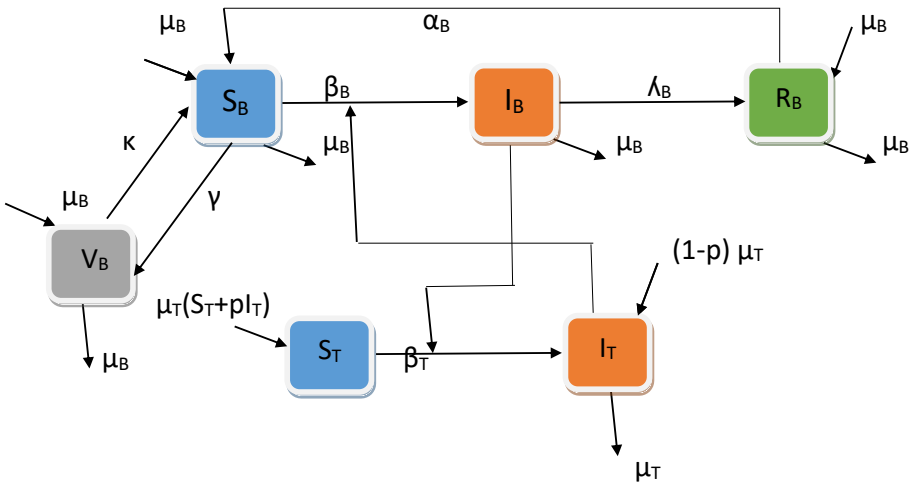

Figure 1: The transmission chart.

The dynamic spread of Babesiosis disease between bovine and tick populations with vaccination is represented by the following system of nonlinear first-order differential equations:

$$\begin{cases} \bar{S_B}'(t) &= \mu_B \bar{S_B}(t) + (\mu_B + \alpha_B)\bar{R_B}(t) - (\gamma + \mu_B)\bar{S_B}(t) - \beta_B \bar{S_B}(t)\frac{\bar{I_T}(t)}{N_B} + \kappa \bar{V_B}(t), \\ \bar{V_B}'(t) &= \mu_B \bar{V_B}(t) + \gamma \bar{S_B}(t) - (\kappa + \mu_B)\bar{V_B}(t) \\ \bar{I_B}'(t) &= \mu_B \bar{I_B}(t) + \beta_B \bar{S_B}(t)\frac{\bar{I_T}(t)}{N_B} - (\mu_B + \lambda_B)\bar{I_B}(t) \\ \bar{R_B}'(t) &= \lambda_B \bar{I_B}(t) - (\mu_B + \alpha_B)\bar{R_B}(t) \\ \bar{S_T}'(t) &= \mu_T(\bar{S_T}(t) + p\bar{I_T}(t)) - \beta_T \bar{S_T}(t)\frac{\bar{I_B}(t)}{N_T} - \mu_T \bar{S_T}(t) \\ \bar{I_T}'(t) &= \beta_T \bar{S_T}(t)\frac{\bar{I_B}(t)}{N_T} + \mu_T(1-p)\bar{I_T}(t) - \mu_T \bar{I_T}(t) \end{cases} \tag{1}$$

with B is the infection rate. $\beta_B = \beta_{TB}B$, such that β_{TB} is the probability that a bovine may get infected after being bitten by an infectious tick. $\beta_T = \beta_{BT}B$, such that β_{BT} is the probability that a tick may become infected after biting an infectious bovine. λ_B is the rate of recovery of infected bovines. α_B The rate of return of controlled bovine to the risk of infection. μ_B represents the birth rate of bovines which is thought to be equivalent to the mortality rate. β_T is the infection rate of susceptible tick. p is the probability that a susceptible tick was born from an infected one. γ is the vaccination rate. κ The rate of return of vaccinated bovine to a susceptible bovine after vaccination. μ_B represents the birth rate of bovines which is thought to be equal to the mortality rate. In our mathematical model for babesiosis in bovine and ticks, bovine population is divided into four compartments: susceptible bovine S_B, infected bovine I_B, controlled bovine R_B, and vaccinated bovine V_B. Similarly, ticks are classified into susceptible ticks $S_T$and infected ticks I_T, where a susceptible bovine can become infected with babesiosis at a rate β_B, this is the result of infected tick bites. Also, the infected bovine can recover with treatment at a rate λ_B to become controlled bovine. However, after treatment, they remain susceptible to contracting the disease again. With the development of studies regarding babesiosis, which led to the development of vaccination mechanisms and methods, a susceptible bovine can be vaccinated at a vaccination rate γ, After vaccination, a vaccinated bovine can return to the risk of infection at a rate κ. For susceptible ticks, ticks become infected as a result of their bites on infected bovine, and this occurs at a rate β_T.

After simplification of the system (1), we get

$$\begin{cases} \bar{S_B}'(t) &= (\mu_B+\alpha_B)\bar{R_B}(t) - \gamma\bar{S_B}(t) - \beta_B\bar{S_B}(t)\frac{\bar{I_T}(t)}{N_B} + \kappa\bar{V_B}(t) \\ \bar{V_B}'(t) &= \gamma\bar{S_B}(t) - \kappa\bar{V_B}(t) \\ \bar{I_B}'(t) &= \beta_B\bar{S_B}(t)\frac{\bar{I_T}(t)}{N_B} - \lambda_B\bar{I_B}(t) \\ \bar{R_B}'(t) &= \lambda_B\bar{I_B}(t) - (\mu_B+\alpha_B)\bar{R_B}(t) \\ \bar{S_T}'(t) &= \mu_T p\bar{I_T} - \beta_T\bar{S_T}(t)\frac{\bar{I_B}(t)}{N_T} \\ \bar{I_T}'(t) &= \beta_T\bar{S_T}(t)\frac{\bar{I_B}(t)}{N_T} - \mu_T p\bar{I_T}(t). \end{cases} \tag{2}$$

3. Preliminaries and Existence and uniqueness of the solution

In this part, we demonstrate some preliminary results regarding the well-posedness of the model (2).

Theorem 1. *Let $(S_B(t), V_B(t), I_B(t), R_B(t), S_T(t), I_T(t))$ be the solution of (2), hence, $S_B(t) > 0,\ V_B(t) > 0,\ I_B(t)(t) > 0,\ R_B(t) > 0,\ S_T(t) > 0,\ I_T(t) > 0$ for all finite $t \geq 0$. Moreover, the following set*

$\Omega = \{(S_B, V_B, I_B, R_B, S_T, I_T), S_B \geq 0, V_B \geq 0, I_B \geq 0, R_B \geq 0, S_T \geq 0, I_T \geq 0, S_B{+}V_B{+}I_B{+}R_B \leq N_B, S_T{+}I_T$

is a positively invariant set.

The system (3) cab ce expressed as follows

$$\frac{dX(t)}{dt} = f(X(t)),$$

$$f(X) = \begin{pmatrix} (\mu_B + \alpha_B)\bar{R_B}(t) - \gamma\bar{S_B}(t) - \beta_B\bar{S_B}(t)\frac{\bar{I_T}(t)}{N_B} + \kappa\bar{V_B}(t), \\ \gamma\bar{S_B}(t) - \kappa\bar{V_B}(t) \\ \beta_B\bar{S_B}(t)\frac{\bar{I_T}(t)}{N_B} - \lambda_B\bar{I_B}(t) \\ \lambda_B\bar{I_B}(t) - (\mu_B + \alpha_B)\bar{R_B}(t) \\ \mu_T p\bar{I_T} - \beta_T\bar{S_T}(t)\frac{\bar{I_B}(t)}{N_T} \\ \beta_T\bar{S_T}(t)\frac{\bar{I_B}(t)}{N_T} - \mu_T p\bar{I_T}(t) \end{pmatrix},$$

with $X(t) = (\bar{S_B}(t), \bar{V_B}(t), \bar{I_B}(t), \bar{R_B}(t), \bar{S_T}(t), \bar{I_T}(t))$. Observe that f_i belongs to class C^1. So it is locally Lipschitz, then, using the Cauchy Lipschitz theorem we deduce the existence and uniqueness of the solution on the maximal interval $[0, T[$.

Lemma 1. *Assume that $\Omega \subset \mathbb{R} \times \mathbb{C}^n$ an open set, $f_i \in \mathbb{C}(\Omega, \mathbb{R}), i = 1, 2, 3...n$. if $f_i|_{x_i=0}$, $X_t \in \mathbb{C}^n_{0+} \geq 0$, $X_t = (x_{1t}, x_{2t}, x_{3t}...x_{nt})^T$, $i = 1, 2, 3...n$ hence $\mathbb{C}^n_{0+}$ is the invariant set of the following system:*

$$\frac{dx_i(t)}{dt} = f_i(t, X_t), t > \sigma, i = 1, 2, 3...n$$

Theorem 2. *Let $(\bar{S_B}(t), \bar{V_B}(t), \bar{I_B}(t), \bar{I_T}(t))$ the solution of (3), then, $\bar{S_B}(t) \geq 0$, $\bar{V_B}(t) \geq 0$, $\bar{I_B}(t) \geq 0$, $\bar{R_B}(t) \geq 0$, $\bar{S_T}(t) \geq 0$, $\bar{I_T}(t) \geq 0$, for all finite $t \geq 0$.*

Proof.

$$\begin{array}{lcl} \frac{d\bar{S_B}}{dt}|_{\bar{S_B}=0} & = & (\mu_B + \alpha_B)\bar{R_B}(t)) + \kappa\bar{V_B}(t) \geq 0 \\ \frac{d\bar{V_B}}{dt}|_{\bar{V_B}=0} & = & \gamma\bar{S_B}(t) \geq 0 \\ \frac{d\bar{I_B}}{dt}|_{\bar{I_B}=0} & = & \beta_B\bar{S_B}(t)\bar{I_T}(t) \geq 0 \\ \frac{d\bar{R_B}}{dt}|_{\bar{R_B}=0} & = & \lambda_B\bar{I_B}(t) \geq 0 \\ \frac{d\bar{S_T}}{dt}|_{\bar{S_T}=0} & = & \mu_T p\bar{I_T}(t) \geq 0 \\ \frac{d\bar{I_T}}{dt}|_{\bar{I_T}=0} & = & \beta_T\bar{I_B}(t) \geq 0 \end{array}$$

Now, concentrating on the second part of the demonstration. Adding the first four equations, one can get:

$$N'_B(t) = \bar{S_B}'(t) + \bar{V_B}'(t) + \bar{I_B}'(t) + \bar{R_B}'(t) = 0$$

and, then, the category of bovine is constant. Similarly, by summing the last two equations, we get that:

$$N'_T(t) = \bar{S_T}'(t) + \bar{I_T}'(t) = 0$$

and therefore, the tick population is constant. We obtain that Ω is a positive invariant domain. Then for $t = +\infty$

Taking into account the proportions:

$$S_B = \frac{\bar{S_B}}{N_B}, V_B = \frac{\bar{V_B}}{N_B}, I_B = \frac{\bar{I_B}}{N_B}, R_B = \frac{\bar{R_B}}{N_B}, S_T = \frac{\bar{S_T}}{N_T}, I_T = \frac{\bar{I_T}}{N_T}$$

From the system (2), and according to the relation $R_B = 1 - S_B - V_B - I_B$, $S_T = 1 - I_T$, we get the following normalized system

$$\begin{cases} S'_B(t) &= (\mu_B + \alpha_B)(1 - S_B(t) - V_B(t) - I_B(t)) - \gamma S_B(t) - \beta_B S_B(t) I_T(t) + \kappa V_B(t), \\ V'_B(t) &= \gamma S_B(t) - \kappa V_B(t), \\ I'_B(t) &= \beta_B S_B(t) I_T(t) - \lambda_B I_B(t), \\ I'_T(t) &= \beta_T (1 - I_T(t)) I_B(t) - \mu_T p I_T(t). \end{cases} \tag{3}$$

4. Basic reproduction number

In this section, we identify the basic reproduction number by the next generation matrix method. It is evident that, (3) has a disease free equilibrium $E_0 = (S_{B0}, V_{B0}, 0, 0)$, with:

$$S_{B0} = \frac{(\mu_B + \alpha_B)\kappa}{(\mu_B + \alpha_B)\kappa + \gamma}, V_{B0} = \frac{\gamma S_{B0}}{\kappa}.$$

Letting $Y = (I_B, I_T)$. We introduce the problem as follows which corresponds to the infected categories for (5)

$$Y' = \mathcal{F}(Y) + \mathcal{V}(Y)$$

such that $\mathcal{F}$ is the rate of appearance of new cases of infections,

$$\mathcal{F} = \begin{pmatrix} \beta_B S_B I_T \\ 0 \end{pmatrix},$$

with $\mathcal{V} = \mathcal{V}^- + \mathcal{V}^+$, where $\mathcal{V}^+$ represents the rate of infected bovines who come from other compartments to compartment i, and $\mathcal{V}^-$ represents the rate of infected who leave compartment i,

$$\mathcal{V} = \begin{pmatrix} -\lambda_B I_B(t) \\ \beta_T (1 - I_T(t)) I_B(t) - \mu_T p I_T(t) \end{pmatrix}.$$

The matrix $F\ V^{-1}$ is called the next generation operator, where

$$F := D\mathcal{F} = \begin{pmatrix} 0 & \beta_B S_{B0} \\ 0 & 0 \end{pmatrix},$$

$$V = D\mathcal{V} = \begin{pmatrix} -\lambda_B & 0 \\ \beta_T & -\mu_T p \end{pmatrix},$$

with $D\mathcal{F}$ and $D\mathcal{V}$ is the Jacobian matrices of $\mathcal{F}$ and $\mathcal{F}$ are evaluated at the disease free equilibrium. As a result, we give the form of the next generation matrix as follows

$$-FV^{-1} = \begin{pmatrix} \frac{\beta_B \beta_T S_{B0}}{\lambda_B \mu_T p} & \frac{\beta_B (S_{B0}}{\mu_T p} \\ 0 & 0 \end{pmatrix}.$$

By definition, the spectral radius of the new generation matrix $-FV^{-1}$ is the basic reproduction number. Thus, the basic reproduction number $\mathcal{R}_0$=$\rho(-FV^{-1})$. Consequently, we have

$$\mathcal{R}_0 = \frac{\beta_B \beta_T S_{B0}}{\lambda_B \mu_T p}.$$

5. Equilibria

In this section, we prove the existence of the equilibrium points. Depending on the previous section. The system (3) has a unique disease-free equilibrium $E_0 = (S_{B0}, V_{B0}, 0, 0)$, with

$$S_{B0} = \frac{(\mu_B + \alpha_B)\kappa}{(\mu_B + \alpha_B)(\kappa + \gamma)}, \quad V_{B0} = \frac{\gamma S_{B0}}{\kappa}.$$

Next, we study the existence of the endemic equilibrium. That is the positive equilibrium.

Lemma 2. *If $\mathcal{R}_0 > 1$, then the system (3) has a unique positive equilibrium $E_2^* = (S_B^*, V_B^*, I_B^*, I_T^*)$, that is the endemic equilibrium.*

The endemic equilibrium satisfies the system;

$$\begin{cases} 0 &= (\mu_B + \alpha_B)(1 - S_B^* - V_B^* - I_B^*) - \gamma S_B^* - \beta_B S_B^* I_T^* + \kappa V_B^*, \\ 0 &= \gamma S_B^* - \kappa V_B^* \\ 0 &= \beta_B S_B^* I_T^* - \lambda_B I_B^* \\ 0 &= \beta_T (1 - I_T^*) I_B^* - \mu_T p I_T^* \end{cases} \tag{4}$$

Using similar calculations of the disease free equilibrium and Solving the second equation, we have

$$V_B' = 0$$

equivalent to

$$\gamma S_B^* - \kappa V_B^* = 0$$

This leads to

$$V_B^* = \frac{\gamma S_B^*}{\kappa}. \tag{5}$$

Replacing the second and the third equations in the system (3), we get

$$S_B' + I_B' = 0$$

So,

$$(\mu_B + \alpha_B)(1 - S_B^* - \frac{\gamma S_B^*}{\kappa} - I_B^*) - \lambda_B I_B^* = 0$$

Hence,

$$S_B^* = \frac{[\mu_B + \alpha_B - (\mu_B + \alpha_B + \lambda_B) I_B^*]\kappa}{(\mu_B + \alpha_B)(\kappa + \gamma)} \tag{6}$$

which is biologically relevant if and only if
$(\mathbf{H_1}):\quad \frac{\mu_B+\alpha_B}{\mu_B+\alpha_B+\lambda_B} > I_B^*$.
We have $I_B' = 0$. Then $\beta_T(1-I_T^*)I_B^* - \mu_T p I_T^* = 0$. Therefore,

$$I_T^* = \frac{\beta_T I_B^*}{\beta_T I_B^* + \mu_T p} \tag{7}$$

substituting (6) and (7) into the third equation of (4), we get $I_B' = 0$ is equivalent to, $\beta_B \frac{[\mu_B+\alpha_B-(\mu_B+\alpha_B+\lambda_B)I_B^*]\kappa}{(\mu_B+\alpha_B)(\kappa+\gamma)} \frac{\beta_T I_B^*}{\beta_T I_B^* + \mu_T p} - \lambda_B I_B^* = 0$. Hence,

$$\frac{I_B^*(C_1 I_B^* + C_2)}{I(\alpha_B\gamma\beta_T+\alpha_B\kappa\beta_T+\gamma\mu_B\beta_T+\kappa\mu_B\beta_T)+\alpha_B\gamma p\mu_T+\alpha_B\kappa p\mu_T+\gamma\mu_B p\mu_T+\kappa\mu_B p\mu_T} = 0.$$

Since the denominator is strictly positive and differs from 0 and $I_B^* \neq 0$ then

$$I_B^*(C_1 I_B^* + C_2) = 0, \tag{8}$$

with

$$\begin{aligned} C_1 &= -(\alpha_B\beta_B\kappa\beta_T + \alpha_B\gamma\lambda_B\beta_T + \alpha_B\kappa\lambda_B\beta_T + \beta_B^\kappa\lambda_B\beta_T + \beta_B\kappa\mu_B\beta_T + \gamma\lambda_B\mu_B\beta_T + \kappa\lambda_B\mu_B\beta_T), \\ C_2 &= \lambda_B p\mu_T(\mu_B+\alpha_B)(\kappa+\gamma)(\mathcal{R}_0 - 1), \end{aligned}$$

with

$$\mathcal{R}_0 = \frac{\beta_B\kappa\beta_T(\alpha_B+\mu_B)}{\lambda_B p\mu_T(\mu_B+\alpha_B)(\kappa+\gamma)}.$$

Since $C_1 < 0$ and $C_2 > 0$, we conclude that (8) always has a unique positive root I_B^*, where

$$I_B^* = \frac{\lambda_B p\mu_T(\mu_B+\alpha_B)(\kappa+\gamma)(\mathcal{R}_0-1)}{(\alpha_B\beta_B\kappa\beta_T + \alpha_B\gamma\lambda_B\beta_T + \alpha_B\kappa\lambda_B\beta_T + \beta_B^\kappa\lambda_B\beta_T + \beta_B\kappa\mu_B\beta_T + \gamma\lambda_B\mu_B\beta_T + \kappa\lambda_B\mu_B\beta_T)}. \tag{9}$$

Now, it remains to show that I_B^* defined by (9) satisfies $(\mathbf{H_1})$. We let X be a positive constant determined by the inequality left-hand side in $(\mathbf{H_1})$, and the second degree polynomial L defined by the left hand side of the equation (8) which is a concave function that satisfies $H(0) = H(I_B^*) = 0$. We need only to show that $L(X) < 0$. Therefore, we have

$$\begin{aligned} L(X) &= -(\alpha_B\beta_B\kappa\beta_T + \alpha_B\gamma\lambda_B\beta_T + \alpha_B\kappa\lambda_B\beta_T + \beta_B^\kappa\lambda_B\beta_T + \beta_B\kappa\mu_B\beta_T + \gamma\lambda_B\mu_B\beta_T + \kappa\lambda_B\mu_B\beta_T) \\ &\quad (\tfrac{\mu_B+\alpha_B}{\mu_B+\alpha_B+\lambda_B})^2\lambda_B p\mu_T(\mu_B+\alpha_B)(\kappa+\gamma)(\mathcal{R}_0-1)(\tfrac{\mu_B+\alpha_B}{\mu_B+\alpha_B+\lambda_B}) \\ &= -I_B^*(\alpha_B+\mu_B)^2(\tfrac{\gamma+\kappa}{(\alpha_B+\lambda_B+\mu_B)^2})(\alpha_B\beta_T + \mu_B\beta_T + \alpha_B p\mu_T + \lambda_B p\mu_T + \mu_B p\mu_T) \\ &< 0. \end{aligned}$$

Therefore, I_B^* satisfies $(\mathbf{H_1})$.

As a consequence, the system (3) always has a unique positive equilibrium $E_1^* = (S_B^*, V_B^*, I_B^*, I_T^*)$, with

$$E^* = (S_B^*, V_B^*, I_B^*, I_T^*),$$

where

$$S_B^* = \frac{[\mu_B+\alpha_B-(\mu_B+\alpha_B+\lambda_B)I_B^*]\kappa}{(\mu_B+\alpha_B)(\kappa+\gamma)}, \quad V_B^* = \frac{\gamma S_B^*}{\kappa},$$
$$I_B^* = \frac{\lambda_B p\mu_T(\mu_B+\alpha_B)(\kappa+\gamma)(\mathcal{R}_0-1)}{(\alpha_B\beta_B\kappa\beta_T+\alpha_B\gamma\lambda_B\beta_T+\alpha_B\kappa\lambda_B\beta_T+\beta_B^\kappa\lambda_B\beta_T+\beta_B\kappa\mu_B\beta_T+\gamma\lambda_B\mu_B\beta_T+\kappa\lambda_B\mu_B\beta_T)},$$
$$I_T^* = \frac{\beta_T I_B^*}{\beta_T I_B^*+\mu_T p}.$$

Depending on the previous calculations, we have the following results in the lemma as follows.

Lemma 3. *The system* (3)*, has always an endemic equilibrium.*

6. Stability analysis

In this section, we demonstrate the local and global stability of the equilibrium points. We put $E = (S_B, I_B, S_T, I_T)$ is an arbitrary equilibrium, hence, we give the Jacobian matrix of the system (5) at E as follows

$$J(S_B, V_B, I_B, I_T) = \begin{pmatrix} -(\mu_B+\alpha_B+\gamma)-\beta_B I_T & -(\mu_B+\alpha_B)+\kappa & -(\mu_B+\alpha_B) & -\beta_B S_B \\ \gamma & -\kappa & 0 & 0 \\ \beta_B I_T & 0 & -\lambda_B & \beta_B S_B \\ 0 & 0 & \beta_T(1-I_T) & -\beta_T I_B-\mu_T p \end{pmatrix}.$$

Firstly, we demonstrate the stability of the disease free equilibrium

Theorem 3. *Let $\mathcal{R}_0 < 1$, then The disease-free equilibrium E_0 is globally asymptotically stable and if $\mathcal{R}_0 > 1$, then E_0 is unstable.*

Proof. Firstly, we begin by the local stability study. We evaluate the Jacobian matrix at the disease-free equilibrium, we get

$$J_{E_0} = \begin{pmatrix} -(\mu_B+\alpha_B+\gamma) & -(\mu_B+\alpha_B)+\kappa & -(\mu_B+\alpha_B) & -\beta_B S_{B0} \\ \gamma & -\kappa & 0 & 0 \\ 0 & 0 & -\lambda_B & \beta_B S_{B0} \\ 0 & 0 & \beta_T & -\mu_T p \end{pmatrix}.$$

The corresponding characteristic equation is given by

$$F(\lambda) = ((-\lambda_B-\lambda)(-\mu_T p-\lambda)-\beta_T\beta_B S_{B0})[[-(\mu_B+\alpha_B+\gamma)-\lambda](\kappa-\lambda)-\gamma[(-(\mu_B+\alpha_B)+\kappa)]] = 0,$$

So, we get

$$F_1(\lambda) = (-\lambda_B-\lambda)(-\mu_T p-\lambda)-\beta_T\beta_B S_{B0} = 0,$$

or

$$F_2(\lambda) = [-(\mu_B+\alpha_B+\gamma)-\lambda](-\kappa-\lambda)-\gamma[(-(\mu_B+\alpha_B)+\kappa)] = 0,$$

consequently, the eigenvalues of J_{E_0} are solutions of both equation $F_1(\lambda)$ and $F_2(\lambda)$ with:

$$F_1(\lambda) = \lambda^2 + a_1\lambda + a_2,$$

with:

$$a_1 = \lambda_B + p\mu_T, \quad a_2 = (\lambda_B p\mu_T)(1 - \mathcal{R}_0).$$

Notice that $a_2 > 0$ if and only if $\mathcal{R}_0 < 1$. Therefore, the Routh-Hurwitz criterion is satisfied in $F_1(\lambda)$. And

$$F_2(\lambda) = \lambda^2 + b_1\lambda + b_2 = 0,$$

where

$$b_1 = \alpha_B + \gamma + \kappa + \mu_B > 0, \quad b_2 = (\alpha_B + \mu_B)(\gamma + \kappa) > 0.$$

Therefore, the Routh-Hurwitz criterion is satisfied in $F_2(\lambda)$. We deduce that if $\mathcal{R}_0 < 1$, then the disease-free equilibrium E_0 is locally asymptotically stable and if $\mathcal{R}_0 > 1$, then E_0 is unstable. To complete the demonstration of Theorem 3.1, we need to prove the global attraction for R_0 ¡ 1 utilising Lyapunov function. We introduce the Lyapunov function

$$L(t) = \beta_T I_B(t) + \lambda_B I_T(t).$$

We calculate $L'(t)$ along (3) in the following manner. we possing $R_0^\epsilon = \frac{\beta_B\beta_T}{\lambda_B\mu_T p}$. Clearly, since $\mathcal{R}_0 < 1$, there exists ϵ^* sufficiently small in such a way for $R_0^\epsilon = \frac{\beta_T\beta_B(S_{B0}+\epsilon)}{\lambda_B\mu_T p} < 1$ all $\epsilon < \epsilon^*$.

$$\begin{aligned}
L'(t) &= \beta_T\tfrac{dI_B(t)}{dt} + \lambda_B\tfrac{dI_T(t)}{dt} \\
&= \beta_T\Big(\beta_B S_B(t)I_T(t) - \lambda_B I_B(t)\Big) + \lambda_B\Big(\beta_T(1 - I_T(t))I_B(t) - \mu_T p I_T(t)\Big) \\
&\leq \beta_T\Big(\beta_B(S_{B0} + \epsilon)I_T(t) - \lambda_B I_B(t)\Big) + \lambda_B\Big(\beta_T(1 - I_T(t))I_B(t) - \mu_T p I_T(t)\Big) \\
&\leq \beta_T\beta_B(S_{B0} + \epsilon)I_T(t) - \lambda_B\beta_T I_B(t) + \lambda_B\beta_T I_B(t) - \lambda_B I_T(t)I_B(t) - \lambda_B\mu_T p I_T(t) \\
&\leq \lambda_B\mu_T p I_T(t)(R_0^\epsilon - 1) - \lambda_B I_T(t)I_B(t) \\
&\leq 0
\end{aligned}$$

$L'(t) = 0$ if $I_T(t) = 0$, we substitute it in the equations of (3), then we obtain that $S_B(t)$ goes to S_{B0}, $I_B(t)$ goes to 0 and $V_B(t)$ goes to V_{B0} as t tends to ∞. This means that E_0 is globally attractant from where E_0 is globally asymptotically stable in Ω.

Now, we verify that the endemic equilibrium E^* is globally stable. The obtained results are highlighted in the Theorem as follows.

Theorem 4. *If $\mathcal{R}_0 > 1$ the unique endemic equilibrium is locally asymptotically stable.*

Proof. By evaluating the Jacobian matrix of system (3) at E^*, we find the following form

$$J_{E_2^*} = \begin{pmatrix} A & B & C & -D \\ \gamma & -\kappa & 0 & 0 \\ E & 0 & -\lambda_B & D \\ 0 & 0 & F & G \end{pmatrix},$$

with

$$\begin{array}{lll} A=-(\mu_B+\alpha_B+\gamma)-\beta_B I_T^*, & B=-(\mu_B+\alpha_B)+\kappa, & C=-(\mu_B+\alpha_B), \quad D=\beta_B S_B^* \\ E=\beta_B I_T^*, & F=\beta_T(1-I_T^*), & G=-\beta_T I_B^*-\mu_T p. \end{array}$$

The characteristic polynomial of $J_{E_2^*}$ is defined by

$$p(\lambda)=\lambda^4+a_3\lambda^3+a_2\lambda^2+a_1\lambda+a_0, \tag{10}$$

such that

$$\begin{aligned} a_3 &= \lambda_B-G-A+\kappa \\ a_2 &= AG-FD-CE-A\lambda_B-B\gamma-G\lambda_B-A\kappa-G\kappa+\lambda_B\kappa, \\ a_1 &= AFD+CGE-C\kappa E-F\kappa D+FDE+AG\lambda_B+BG\gamma+AG\kappa-Bb\gamma-A\lambda_B\kappa-G\lambda_B\kappa \\ a_0 &= BF\gamma D+AF\kappa D+CG\kappa E+F\kappa DE+BG\lambda_B\gamma+AG\lambda_B\kappa. \end{aligned}$$

According to Criterion of Routh-Hurwitz, we conclude the asymptotic local stability of E^* under the following conditions

$$(\mathbf{H_2}):\begin{cases} a_0,a_3>0 \\ a_3a_2-a_1>0 \\ a_3a_2a_1-a_1^2-a_3^2a_0>0 \end{cases}$$

Now, we show that $(\mathbf{H_2})$ is satisfied for $\mathcal{R}_0>1$. Notice that a_3 can be rewritten as

$$a_3 = \lambda_B+\beta_T I_B^*+\mu_T p+\mu_B+\alpha_B+\gamma+\beta_B I_T^*+\kappa>0$$

Next, we show that $a_0>0$.

$$\begin{aligned} a_0 &= BF\gamma D+AF\kappa D+CG\kappa E+F\kappa DE+BG\lambda_B\gamma+AG\lambda_B\kappa \\ &= (C+\kappa)F\gamma D+((C-\gamma)-E)F\kappa D+CG\kappa E+F\kappa DE+(C+\kappa)G\lambda_B\gamma+((C-\gamma)-E)G\lambda_B\kappa \\ &= CF\gamma D+CF\kappa D+CG\kappa E-G\lambda_B\kappa E+CG\lambda_B\gamma+CG\lambda_B\kappa \\ &> 0. \end{aligned}$$

Now, we turn our attention to the second line of $(\mathbf{H_2})$, we have

$$\begin{aligned} &a_3a_2-a_1= \\ &\big(\lambda_B-G-A+\kappa\big)\big(CE+FD-\lambda_B(A-\kappa)-G(\lambda_B-A+\kappa)-B\gamma-A\kappa\big) \\ &-\big(G(b(A-\kappa)-CE+B\gamma+A\kappa)-\lambda_B(B\gamma+A\kappa)-F(D(A-\kappa)-DE)+C\kappa E\big) \\ &=-C^2G+C^2\gamma+C^2\kappa+C_B^\lambda-C^2E-CG^2+2CG\gamma+2CG\kappa+2CG\lambda+2CGE-C\gamma^2-2C\gamma\kappa-2C\gamma\lambda_B \\ &-C\kappa^2-2C\kappa\lambda_B-2C\kappa E-C\lambda_B^2-C\lambda_BE+CE^2+G^2\gamma+G^2\kappa+G^2\lambda_B+G^2E-G\gamma^2-2G\gamma^2\kappa \\ &-2G\gamma\lambda_B-2G\gamma E-G\kappa^2-2G\kappa\lambda_B-2G\kappa E-G\lambda^2-2G\lambda_BE-GE^2-FDG+\gamma^2\lambda_B \\ &+2\gamma\kappa\lambda_B+\gamma\kappa E+\gamma\lambda_B^2+2\gamma\lambda_BE+\kappa^2\lambda_B+\kappa^2E+\kappa\lambda_B^2+2\kappa\lambda_BE+\kappa E^2+\lambda_B^2E+\lambda_BE^2+FD\lambda_B \\ &>0. \end{aligned}$$

Finally, we show the last inequality in $(\mathbf{H_2})$, that is, $(a_3a_2 - a_1)a_1 - a_3^2a_0 > 0$, we have

$$
\begin{aligned}
&(a_3a_2 - a_1)a_1 - a_3^2a_0 = \\
&C^3FGD - C^3F\gamma D + C^3F\gamma - C^3F\lambda_B - C^3G^2(\gamma + \kappa + \lambda_B - E) + C^3G\gamma^2 + 2C^3G\gamma(\kappa + \lambda_B + E) \\
&+C^3G\kappa(\kappa + \lambda_B - E) + C^3G\lambda^2 - 2C^3G\lambda_BE + C^3GE^2 - C^3\gamma^2\lambda_B - 2C^3\gamma\kappa\lambda_B \\
&+C^3\gamma\kappa E - C^3\gamma\lambda_B^2 + C^3\gamma\lambda_BE - C^3\kappa^2\lambda_B + C^3\kappa^2E - C^3\kappa\lambda^2 + 2C^3\kappa\lambda_BE - C^3\kappa E^2 + C^2FG^2D \\
&-3C^2FG\gamma D + 2C^2FG\gamma - C^2FG\kappa D - 2C^2FG\lambda_BD - 4C^2FGDE + 2C^2F\gamma^2D \\
&-2C^2F\gamma^2 + C^2F\gamma^\kappa D + C^2F\gamma\kappa + 3C^2F\gamma\lambda_BD + 2C^2F\gamma\lambda_B + C^2F\gamma DE - 2C^2F\gamma E + C^2F\kappa_B^\lambda D \\
&-C^2F\kappa GE + C^2F\lambda^2D + 3C^2F\lambda_BDE - 3C^2FDE^2 - C^2G^3\gamma - C^2G^3\kappa - C^2G^3\lambda_B + C^2G^3E \\
&+2C^2G^2\gamma^2 + 4C^2G^2\gamma\kappa + 4C^2G^2\gamma\lambda_B + 2C^2G^2\kappa^2 + 4C^2G^2\kappa\lambda_B + 2C^2G^2\kappa E + 2C^2G^2\lambda_B^2 + C^2G^2\lambda_BE \\
&-2C^2G^2E^2 - C^2G\gamma^3 - 3C^2G\gamma^2\kappa - 4C^2G\gamma^2\lambda_B \\
&-C^2G\gamma^2E - 3C^2G\gamma\kappa^2 - 8C^2G\gamma\kappa\lambda_B - C^2G\kappa\gamma E \\
&-4C^2G\gamma\lambda_B^2 - C^2G\gamma\lambda_BE + C^2G\gamma E^2 - C^2G\kappa^3 - 4C^2G\kappa^2\lambda_B \\
&-2C^2G\kappa^2E - 4C^2G\kappa\lambda_B^2 - 2C^2G\kappa\lambda_BE - C^2G\lambda_B^3 - C^2G\lambda^2E + 3C^2G\lambda_BE^2 - C^2GE^3 \\
&+C^2\gamma^3\lambda_B + 3C^2\gamma^2\kappa\lambda_B - C^3\gamma^2\kappa E + 2C^2\gamma^2\lambda^2 + 3C^2\gamma\kappa^2\lambda_B \\
&-2C^2\gamma\kappa^2E + 4C^2\gamma\kappa\lambda_B^2 + C^2\gamma\kappa\lambda_BE + C^2\gamma\lambda_B^3 + C^2\gamma\lambda^2E \\
&+C^2\gamma\lambda_BE^2 + C^2\kappa^3\lambda_B - C^2\kappa^3E + 2C^2\kappa^2\lambda^2 + C^2\kappa^2\lambda_BE + C^2\kappa\lambda^{"} + C^2\kappa\lambda_B^2E \\
&+3C^2\kappa\lambda_BE^2 + C^2\kappa E^3 + CF^2GD^2 > 0
\end{aligned}
$$

7. Required animal health intervention

Our main purpose in this section is to ascertain the impact of the vaccination on the propagation of the contagious disease. The responsible rate for the vaccination force is γ. Note that this infection is perfect, where it is not possible that the vaccinated person to get infected. Hence of asymptotic dynamics to the value of $\mathcal{R}_0$, we investigate the sensitivity of $\mathcal{R}_0$ to γ using the derivation of $\mathcal{R}_0$ with respect to γ. By a simple computation, we get:

$$\frac{d\mathcal{R}_0}{d\gamma} = -\frac{\mathcal{R}_0\lambda_Bp\mu_T(\mu_B + \alpha_B)\beta_B\kappa\beta_T(\alpha_B + \mu_B)}{\lambda_Bp\mu_T(\mu_B + \alpha_B)(\kappa + \gamma)} < 0$$

Hence, γ has a negative impact on the value of $\mathcal{R}_0$. This means that the animal health interventions are needed, so we need to determine the minimal protection effort for reducing $\mathcal{R}_0$ below 1. It has been proved previously that $\mathcal{R}_0 = \frac{\beta_B\kappa\beta_T(\alpha_B+\mu_B)}{\lambda_Bp\mu_T(\mu_B+\alpha_B)(\kappa+\gamma)}$, which is needed to be reduced below 1. This means that

$$\mathcal{R}_0 = \frac{\beta_B\kappa\beta_T(\alpha_B+\mu_B)}{\lambda_Bp\mu_T(\mu_B+\alpha_B)(\kappa+\gamma)} < 1,$$

we get

$$\gamma > \gamma_{min} = \frac{\beta_B\kappa\beta_T(\alpha_B+\mu_B) - \lambda_Bp\mu_T(\mu_B+\alpha_B)\kappa}{\lambda_Bp\mu_T(\mu_B+\alpha_B)}.$$

8. Numerical simulation

In this section, we apply numerical simulations to our model and investigate the basic reproduction number and subpopulations.

8.1. Ecologic stability

$$S_B(0) = 0.6268 \ ; \ V_B(0) = 0.3134 \ ; \ I_B(0) = 0.0399 \ ; \ I_T(0) = 0.3743.$$

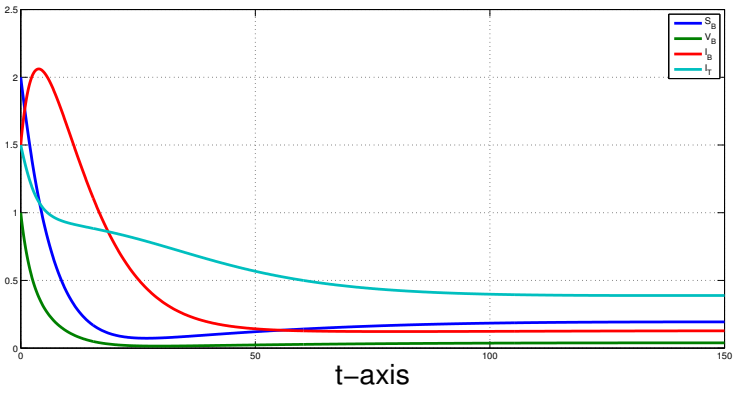

Figure 2: Ecologic stability when $\beta_T = 0.017,\ \beta_B = 0.015,\ \gamma = 0.1$ and $\mathcal{R}_0 = 1.7$.

The persistence of the diseas in Figure 2 where in this case we have $\mathcal{R}_0 > 1$.

8.2. Disease-free equilibrium

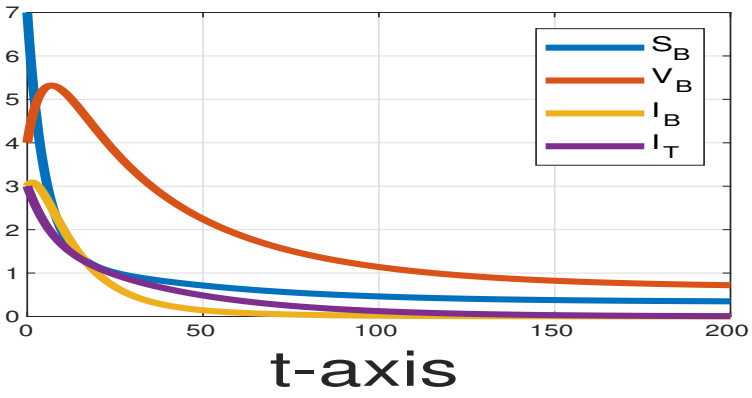

Figure 3: The behavior of the various subpopulations when $p = 0.3,\ \beta_B = 0.02,\ \gamma = 0.1$ and $\mathcal{R}_0 = 0.0444$.

Figure 3 shows the behavior of the various cases of disease S_B, V_B, I_B, I_T for both categories of bovine and ticks indicative of the effect of parameters for $p = 0.3,\ \beta_B = 0.02,\ \gamma = 0.1$ and $\mathcal{R}_0 = 0.0444$. And we have the stability of the DFE.

8.3. Endemic point stability

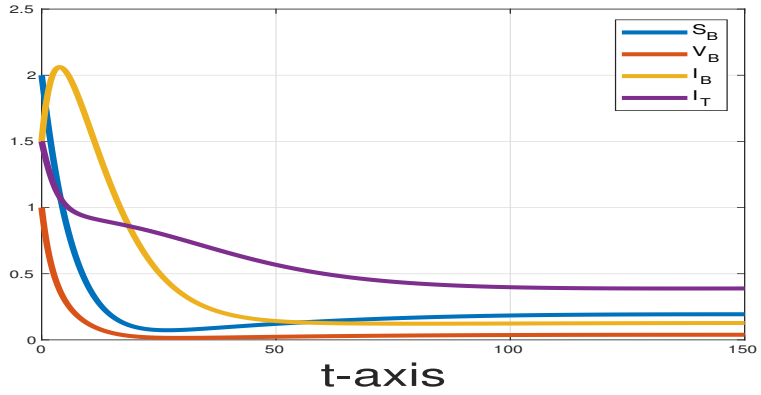

Figure 4: The behavior of the various subpopulations when $p = 0.3,\ \beta_B = 0.15,\ \gamma = 0.1$ and $\mathcal{R}_0 = 7.0833$.

Figure 4 shows the behaviour of the various cases of disease S_B, V_B, I_B, I_T for both categories of bovine and ticks indicative of the effect of parameters for $p = 0.3,\ \beta_B = 0.15,\ \gamma = 0.1$ and $\mathcal{R}_0 = 7.0833$. Therefore, the infection persists and the endemic equilibrium is locally asymptotically stable.

8.4. The sensitivity of the vaccination parameter

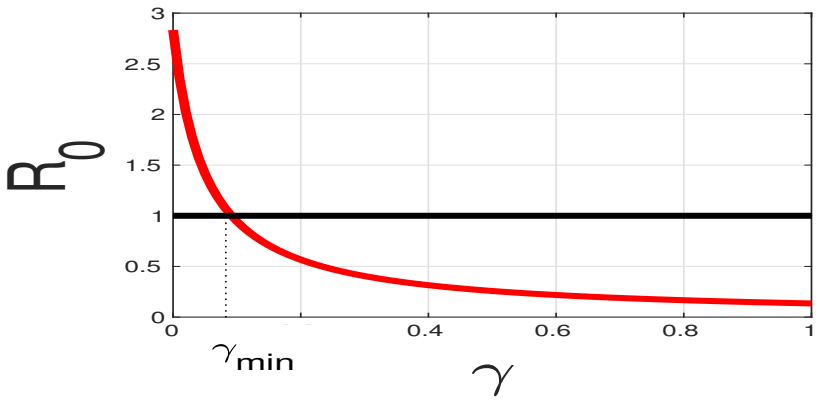

Figure 5: The dynamics of the basic reproduction number $\mathcal{R}_0 > 1$ are sensitive to the parameter of vaccination γ .

Figure 5 demonstrates the vaccination effect on the basic reproduction number. if $\gamma < \gamma_{min}$, we have $\mathcal{R}_0 <$ 1and then we get the stability of the endemic equilibrium. And

for $\gamma > \gamma_{min}$, we have $\mathcal{R}_0 < 1$ and then we get the stability of the disease free equilibrium. Therefore for controlling the epidemic it is necessary to apply a vaccination force larger than γ_{min}.

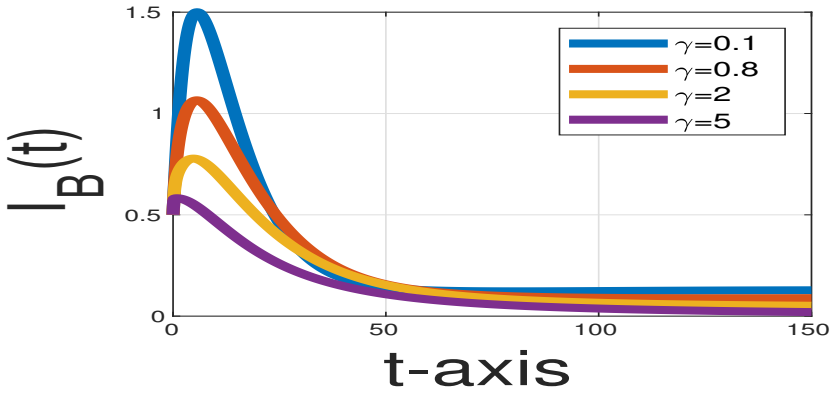

Figure 6: The behavior of the various subclasses is affected by the changes of the parameter γ.

Figure 6 shows the effect of the vaccination rate γ on containing the epidemic where for the values $\gamma = 0.1$, $\gamma = 0.8$, $\gamma = 2$ we have the persistence of the disease and for $\gamma = 5$ we have the extinction of the epidemic.

9. Conclusion

The ticks serve as both vectors and infectious agents for the protozoan hemiparasite Babesia. In this way, we have established a mathematical model with a constant population size to analyze the qualitative dynamics of the evolution of babesiosis-infected bovines. numerical simulations of the model that vary the parameters depict various scenarios of the disease's spread and the effect of vaccination on containing the Babesiosis bovine disease epidemic.
We have proven that the basic reproduction number $\mathcal{R}_0$ determines the extent of the spread of the disease and its relationship to the vaccination. Where if $\mathcal{R}_0 < 1$, then we find that the solution converges to the disease-free equilibrium point, that is the disease free equilibrium point is globally stable, and this is when the value of the vaccination parameter γ is large. However, if $\mathcal{R}_0 > 1$, then we find the solution converges to the endemic equilibrium point, then the endemic equilibrium point is locally stable, which is guaranteed for a lower value for the vaccination parameter γ. Which leads us to conclude that the vaccine contributes significantly to reducing the spread of the disease and recovering from it but not permanently, as he can return to the disease after recovery resulting from the vaccination. Also it is obtained that to contain the Babesiosis bovine epidemic it is required to achieve a vaccination rate γ that extends $\gamma_{min} = 0,1$, where this optimal value depends on the rate of losing immunity guaranteed by vaccination, which is on the

check of the existing vaccines. Therefore the vaccination of bovines can be very helpful in containing the Babesiosis bovine disease, but it will be more efficient if there is a more efficient vaccine to the infection, which is difficult to achieve due to the many mutations of the virus in nature.

In future works, we will investigate further complex features of the babesiosis bovine infection with spatiotemporal effect, and check the efficiency of the vaccination process in a spatially heterogeneous environment. We mention a few works in this context, [19–22].

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