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Models of Bovine Babesiosis Including Juvenile Cattle

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Abstract Bovine Babesiosis in cattle is caused by the transmission of protozoa of *Babesia* spp. by ticks as vectors. Juvenile cattle (<9 months of age) have resistance to Bovine Babesiosis, rarely show symptoms, and acquire immunity upon recovery. Susceptibility to the disease varies between breeds of cattle. Models of the dynamics of Bovine Babesiosis transmitted by the cattle tick that include these factors are formulated as systems of ordinary differential equations. Basic reproduction numbers are calculated, and it is proved that if these numbers are below the threshold value of one, then Bovine Babesiosis dies out. However, above the threshold number of one, the disease may approach an endemic state. In this case, control measures are suggested by determining target reproduction numbers. The percentage of a particular population (for example, the adult bovine population) needed to be controlled to eradicate the disease is evaluated numerically using Columbia data from the literature.

Keywords Bovine Babesiosis · Global stability · Disease control strategy · Target reproduction number

Mathematics Subject Classification 92D30 · 34D23

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1 Introduction

Ticks are vectors for multiple diseases throughout the animal world, from Lyme disease in humans to Bovine Babesiosis in cattle. The pathogens they transmit range from various viruses, for instance Omsk hemorrhagic fever virus (Charrel et al. 2004), to bacteria such as *Borrelia burgdoferi*, the causative agent of Lyme disease, and finally to protozoa, such as *Babesia bigemina*, *Babesia bovis* (also known as *Babesia argentina*), and *Babesia divergens*, all causative agents of Bovine Babesiosis. These *Babesia* spp. are transmitted by the ixodid ticks. More specifically, *B. bigemina* and *B. bovis* are transmitted by *Boophilus* (now *Rhipicephalus*) *microplus* (known as the cattle tick), which is a single host tick (Randolf 2004), while *B. divergens*' main vector is *Ixodes ricinus*, which is not a single host tick (Randolf 2004). *B. bigemina* is found in Africa, Asia, North, Central and South America, Southern Europe, and Australia; *B. bovis* is found in much the same places as *B. bigemina*, but is less present in Africa; and finally, *B. divergens* is localized to central Europe (Bock et al. 2004). Bovine Babesiosis is endemic in areas of the above regions. We focus here on Bovine Babesiosis caused by *B. bigemina* and *B. bovis*.

The symptoms of Bovine Babesiosis include increased respiratory rate, depression, weakness, fever, haemoglobinuria, jaundice, with more severe symptoms including tremors, muscle wasting, coma, and even death (Bock et al. 2004). The economic implications of Bovine Babesiosis are massive. Most of the 1–2 billion cattle worldwide are currently exposed to Bovine Babesiosis caused by one or more of the protozoa mentioned above (Schnittger et al. 2012; Uilenberg 1995), and the economic impact of Bovine Babesiosis includes lowered meat and milk production, abortions, lowered fertility in bulls, control measure costs, and a general impact on the global cattle trade industry (Bock et al. 2004). However, the USA managed to save an estimated 3 billion dollars per year by completing a cattle tick eradication in the early 1900s (Schnittger et al. 2012). Recently, it has been noted that the USA is not immune to Bovine Babesiosis, as outbreaks of Bovine Babesiosis still do occur (Guerrero et al. 2007). We assume that other hosts are not epidemiologically relevant to the dynamics of Bovine Babesiosis in cattle caused by both *B. bigemina* and *B. bovis*, although Ramos et al. (2010) and Holman et al. (2011) report that white-tailed deer in Texas, USA, and Northern Mexico may be alternate hosts.

The life cycles of *B. bigemina* and *B. bovis* are very similar. For transmission of *Babesia* spp. from tick to vertebrate host, sporozoites are produced by the ticks' salivary glands, which are then transmitted to the vertebrate host, while the tick is attached and feeding (Bock et al. 2004). The merozoites then proceed to complete a process similar to binary fission (within the vertebrate host erythrocytes), and this results in doubling the number of merozoites. Transmission from vertebrate host to tick occurs during the feeding of the tick, when the tick engorges itself on infected blood. It is important to state that the protozoa spreads through the ticks' hemolyph and organs as vermicules. These can infect the tick ovaries and be responsible for transovarial vertical transmission of *Babesia* spp. These vermicules infect the salivary glands and eventually develop into the sporozoites discussed above. This development only occurs during tick attachment to the vertebrate host; for *B. bigemina*, this begins

9 days after initial tick attachment, and for *B. bovis*, this begins 2–3 days after initial tick attachment (Bock et al. 2004).

An interesting and possibly unique fact about Bovine Babesiosis is that juvenile cattle (<9 months of age) have an innate resistance to Bovine Babesiosis (Bock et al. 2004). Once infected, they rarely show clinical symptoms, and once recovered, they acquire natural immunity. Zintl et al. (2005) state that the level of parasitaemia in the bloodstream of these juvenile cattle is lower than in adult cattle.

The European cattle breed, the *Bos taurus* cattle, seems to be more susceptible to infection by both *B. bovis* and *B. bigemina* than the Asian cattle breed, the *Bos indicus* cattle. Bock et al. (1997, 1999b) both report lower mean parasitaemia scores of *B. bigemina* in *B. taurus* cattle, and Bock et al. (1997) also report lower mean parasitaemia scores of *B. bovis* in *B. taurus* cattle. Moreover, Piper et al. (2008) state that compared with the *B. taurus* cattle, the *B. indicus* cattle have a higher resistance to actual tick attachment, which implies that less ticks feed on *B. indicus* individuals, and it follows that the *B. indicus* breed is more resistant to Bovine Babesiosis than the *B. taurus* breed.

There currently exist few deterministic mathematical models of Bovine Babesiosis. Aranda et al. (2012) introduced a mathematical model based on ordinary differential equations (ODEs) for Bovine Babesiosis caused by *B. bigemina* and *B. bovis*; Friedman and Yakubu (2014) formulated a mathematical model based on partial differential equations for *B. bovis*, of which the model by Aranda et al. (2012) is a special case. Both of these models separate cattle solely based upon their status of infection by Bovine Babesiosis.

An ODE model of Bovine Babesiosis caused by *B. bigemina* and *B. bovis* transmitted by the cattle tick is first introduced in Sect. 2, which is similar to the model in Aranda et al. (2012) but with a juvenile cattle class (i.e., the juvenile cattle are separated from the adult cattle). By interpreting vertical transmission in ticks in two different ways for the next generation matrix, we calculate two basic reproduction numbers $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$, which are threshold parameters. These are then used in the discussion of equilibria of this model. Numerical simulations using the data from Colombia introduced by Aranda et al. (2012) are performed for this model. We then proceed to a local sensitivity analysis to determine the relative importance of each parameter with respect to the threshold parameters, and we also determine an array of target reproduction numbers [as in Shuai et al. (2013)] in order to evaluate the effectiveness of various control methods for the disease. In Sect. 3, we introduce an ODE model for Bovine Babesiosis, once again caused by *B. bigemina* and *B. bovis*, that separates cattle based on breed. We calculate the threshold parameters associated with this model, discuss the equilibria and associated stability of this model, and conclude this section with target reproduction numbers. In Sect. 4, we combine both previous models to attain a more realistic model for control purposes. While $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$ are calculated and the equilibria of this model are discussed, a strong emphasis is placed on the target reproduction numbers that are associated with this model, in order to more realistically evaluate various possible control measures. We conclude with a summary and comments in Sect. 5.

2 Model with Juveniles (Model J)

2.1 Formulation of Model J

We first introduce the parameters and the assumptions that are used in this model and in subsequent ones. The birth rate of cattle is denoted by b_B, and the death rate of cattle is denoted by d_B. For all of our models, we assume that $b_B = d_B$ for simplification purposes. The parameter β_{BT} denotes the rate of infection of susceptible cattle by successful transmission of the parasites during feedings of infected ticks, i.e., the biting rate of ticks times the probability of transmission from tick to cattle. The disease transmission dynamics are assumed to be frequency dependent. In utero transmission of Bovine Babesiosis is known to have been observed, namely by Yeruham et al. (2003), but they first state that vertical transmission is most likely rare and accidental and seems to require actual physiological change to the cattle, so vertical transmission in cattle is unlikely and improbable (Hall et al. 1968; Spickler et al. 2010). Thus, in our models, we assume that there is no vertical transmission in cattle. It is important to note that Aranda et al. (2012) assumed the opposite in their model of Bovine Babesiosis, i.e., 100 % vertical transmission. Next, the bovine population naturally recovers from Bovine Babesiosis at rate $\tau_B > 0$; thus, $\frac{1}{\tau_B}$ is the average infectious time. Note that Friedman and Yakubu (2014) take τ_B as the treatment rate of infected cattle. Recovered cattle become susceptible to contract Bovine Babesiosis again at rate $\alpha_B \geq 0$; thus, if $\alpha_B > 0$, then $\frac{1}{\alpha_B}$ is the average period of immunity. For Model J, we assume all cattle have exactly the same susceptibility to infection of Bovine Babesiosis. However, it is important to note that it is known in the literature that the *B. indicus* breed of cattle is more highly resistant than the *B. taurus* breed to Bovine Babesiosis caused by *B. bigemina* and *B. bovis* (Bock et al. 1999a). This is addressed in subsequent models in Sects. 3 and 4 in order to more accurately determine valid control methods for Bovine Babesiosis. Our last general assumption with regard to cattle is that Bovine Babesiosis has no latent period of infection in cattle [it is known that post-recovery cattle remain carriers yet are asymptomatic, lasting months in the case of *B. bovis* and years for *B. bigemina* (Bock et al. 2004)].

The following applies to the cattle in Model J and to the cattle in the model in Sect. 4. According to Bock et al. (2004), juvenile cattle (below 9 months of age) have an innate immune response to Bovine Babesiosis, and they rarely show symptoms if they are infected by the disease. Here, we assume that all of the calves that are infected remain asymptomatic and that calves that are susceptible or asymptomatic transfer to the adult population at a rate $m_{BJ} > 0$. Thus, $\frac{1}{m_{BJ}}$ is the average time that we consider a calf to be a 'juvenile.' We assume no vertical transmission in cattle, and we assume that the infected (asymptomatic) juvenile bovine are still a source of infection for the ticks but that their rate of transmission is reduced by a factor ϵ, where $0 < \epsilon < 1$, since juvenile cattle have a lower parasite count in their blood (Zintl et al. 2005). Also, S_{BJ} denotes the susceptible juvenile bovine population, A_{BJ} denotes the asymptomatic infectious juvenile bovine population, $N_{BJ} = S_{BJ} + A_{BJ}$ denotes the total juvenile bovine population, S_{BA} denotes the susceptible adult bovine population, I_{BA} denotes the infectious adult bovine population, R_{BA} denotes the naturally recovered adult bovine population, and N_{BA} denotes the total adult bovine population.

Table 1 Definitions of parameters used in Model J

Parameter	Definition
b_B	Bovine birth rate
d_B	Bovine death rate
b_T	Tick birth rate
d_T	Tick death rate
τ_B	Bovine natural recovery rate
α_B	Bovine loss of immunity rate
β_{BT}	Infectivity rate, tick to bovine
β_{TB}	Infectivity rate, bovine to tick
p	Probability of no vertical transmission in ticks
m_{BJ}	Maturation rate of juvenile cattle
$\epsilon\beta_{TB}$	Infectivity rate, juvenile bovine per tick

The cattle tick population, which is a single host tick, is split into two classes: Susceptibles (S_T) and Infectious (I_T), and N_T represents the total population of ticks, so $N_T = S_T + I_T$. Note that for *B. bigemina*, the tick retains the parasite as it goes through its entire life cycle; for *B. bovis*, the tick rids itself of the parasitic infection once it molts (Bock et al. 2004). The birth rate of ticks is denoted by b_T, and the death rate of ticks is denoted by d_T. Just as for b_B and d_B, we assume that $b_T = d_T$ for simplification purposes. The parameter β_{TB} denotes the rate of infection of susceptible ticks by successful transmission of the parasites during feedings of the susceptible ticks on infected adult cattle, i.e., β_{TB} ($\epsilon\beta_{TB}$) is the biting rate of ticks times the probability of transmission from adult (juvenile) cattle to tick. We also consider only female ticks as they feed on bovine blood (Bock et al. 2004), while the males only live a shorter time and are thought not to feed (Hilpertshauser et al. 2006). Vertical transmission in ticks occurs, by transovarial transmission (Bock et al. 2004). We denote this as $(1-p)$ where p represents the probability of no vertical transmission and $0 < p \leq 1$; we assume $p > 0$ for this model. We also assume that once a tick is infected with *B. bigemina* or *B. bovis*, it can never recover [this is completely true for *B. bigemina*, but the tick rids itself of *B. bovis* when it molts from a larval stage to a nymph stage (Bock et al. 2004)]. We assume that all ticks have the same susceptibility to infection with the protozoa responsible for Bovine Babesiosis. In addition, we do not distinguish the different life stages of the ticks, an assumption also made in Friedman and Yakubu (2014), Aranda et al. (2012) and Gaff and Gross (2007). However, Hoch et al. (2012) state that parasite transmission is mainly by adult ticks. As in other models cited, we also assume no diapause in ticks. A summary of the positive parameters used in Model J, with their definitions, is given in Table 1, where (except for p) all parameters have units $\frac{1}{\text{time}}$.

The dynamics of Model J are formulated as follows:

$$\frac{\mathrm{d}S_{BJ}}{\mathrm{d}t} = b_B N_{BA} - m_{BJ} S_{BJ} - \beta_{BT} I_T \frac{S_{BJ}}{N_{BJ}}$$
$$\frac{\mathrm{d}A_{BJ}}{\mathrm{d}t} = \beta_{BT} I_T \frac{S_{BJ}}{N_{BJ}} - m_{BJ} A_{BJ}$$

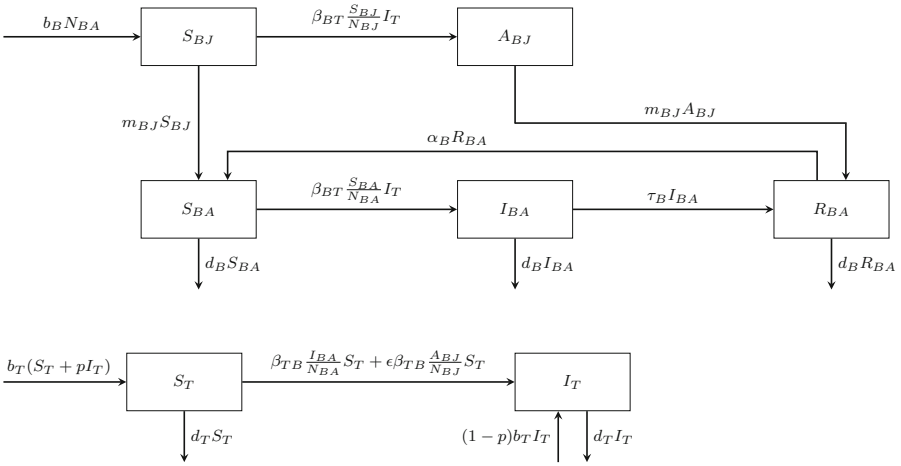

Fig. 1 Bovine and tick populations flowchart for Model J

$$
\begin{aligned}
\frac{\mathrm{d}S_{BA}}{\mathrm{d}t} &= m_{BJ}S_{BJ} + \alpha_B R_{BA} - \beta_{BT}\frac{S_{BA}}{N_{BA}}I_T - d_B S_{BA}\\
\frac{\mathrm{d}I_{BA}}{\mathrm{d}t} &= \beta_{BT}\frac{S_{BA}}{N_{BA}}I_T - (\tau_B + d_B)I_{BA}\\
\frac{\mathrm{d}R_{BA}}{\mathrm{d}t} &= \tau_B I_{BA} - (\alpha_B + d_B)R_{BA} + m_{BJ}A_{BJ}\\
\frac{\mathrm{d}S_T}{\mathrm{d}t} &= pb_T I_T + b_T S_T - \beta_{TB}S_T\frac{I_{BA}}{N_{BA}} - \epsilon\beta_{TB}S_T\frac{A_{BJ}}{N_{BJ}} - d_T S_T\\
\frac{\mathrm{d}I_T}{\mathrm{d}t} &= (1-p)b_T I_T + \beta_{TB}S_T\frac{I_{BA}}{N_{BA}} + \epsilon\beta_{TB}S_T\frac{A_{BJ}}{N_{BJ}} - d_T I_T
\end{aligned}
\tag{1}
$$

In order to visualize Model J, the flowchart depicted in Fig. 1 is useful. Equations (1) directly imply the next set of equations:

$$
\begin{aligned}
\frac{\mathrm{d}N_{BJ}}{\mathrm{d}t} &= b_B N_{BA} - m_{BJ}N_{BJ}\\
\frac{\mathrm{d}N_{BA}}{\mathrm{d}t} &= m_{BJ}N_{BJ} - d_B N_{BA}\\
\frac{\mathrm{d}N_T}{\mathrm{d}t} &= (b_T - d_T)N_T
\end{aligned}
\tag{2}
$$

Since $S_{BJ} = N_{BJ} - A_{BJ}$ and $R_{BA} = N_{BA} - S_{BA} - I_{BA}$, the model can be reduced to a 4-dimensional one as follows:

$$
\begin{aligned}
\frac{\mathrm{d}S_{BA}}{\mathrm{d}t} &= m_{BJ}(N_{BJ} - A_{BJ}) + \alpha_B(N_{BA} - S_{BA} - I_{BA}) - \beta_{BT}\frac{S_{BA}}{N_{BA}}I_T - d_B S_{BA}\\
\frac{\mathrm{d}A_{BJ}}{\mathrm{d}t} &= \beta_{BT}I_T\frac{(N_{BJ} - A_{BJ})}{N_{BJ}} - m_{BJ}A_{BJ}
\end{aligned}
$$

$$\begin{aligned}\frac{\mathrm{d}I_{BA}}{\mathrm{d}t} &= \beta_{BT}\frac{S_{BA}}{N_{BA}}I_T - (\tau_B + d_B)I_{BA} \\ \frac{\mathrm{d}I_T}{\mathrm{d}t} &= (1-p)b_T I_T + \beta_{TB}(N_T - I_T)\frac{I_{BA}}{N_{BA}} + \epsilon\beta_{TB}(N_T - I_T)\frac{A_{BJ}}{N_{BJ}} - d_T I_T\end{aligned} \tag{3}$$

For simplification purposes, we assume that $b_T = d_T$ and that $b_B = d_B$; thus, N_T and $N_{BJ} + N_{BA}$ are constant. Letting $N_{BJ} + N_{BA} = N$, a constant, (2) shows that N_{BJ} and N_{BA} each approach a unique positive equilibrium, namely $N_{BJ} \to \frac{b_B N}{b_B + m_{BJ}}$, $N_{BA} \to \frac{m_{BJ} N}{b_B + m_{BJ}}$. In order to focus on the disease dynamics, we thus assume that both adult and juvenile bovine populations have reached their equilibrium; namely, N_{BJ} and N_{BA} are constants with

$$m_{BJ} N_{BJ} = b_B N_{BA} \tag{4}$$

It is easy to show that the model is well posed within the feasible region $\Gamma = \{(S_{BA}, A_{BJ}, I_{BA}, I_T) \in \mathbb{R}^4_+ \mid 0 \le S_{BA} + I_{BA} \le N_{BA}, 0 \le A_{BJ} \le N_{BJ}, 0 \le I_T \le N_T\}$, which is positively invariant with respect to (3).

2.2 Calculation of Basic Reproduction Numbers

The method used to calculate the basic reproduction number $\mathcal{R}_0$ is the next generation matrix method; see Diekmann and Heesterbeek (2000) and van den Driessche and Watmough (2002).

From (3), it is obvious that at $S_{BA} = N_{BA}$ and $A_{BJ} = I_{BA} = I_T = 0$, the system is at equilibrium. Denoting this point as P_0, the disease-free equilibrium (DFE), consider the Jacobian matrix for the infected classes A_{BJ}, I_{BA}, I_T about P_0:

$$J(P_0) = \begin{bmatrix} -m_{BJ} & 0 & \beta_{BT} \\ 0 & -(\tau_B + d_B) & \beta_{BT} \\ \epsilon\beta_{TB}\frac{N_T}{N_{BJ}} & \beta_{TB}\frac{N_T}{N_{BA}} & (1-p)b_T - d_T \end{bmatrix}$$

Thus, writing $J(P_0) = F_1 - V_1$, it follows that

$$F_1 = \begin{bmatrix} 0 & 0 & \beta_{BT} \\ 0 & 0 & \beta_{BT} \\ \epsilon\beta_{TB}\frac{N_T}{N_{BJ}} & \beta_{TB}\frac{N_T}{N_{BA}} & (1-p)b_T \end{bmatrix} \text{ and } V_1 = \begin{bmatrix} m_{BJ} & 0 & 0 \\ 0 & (\tau_B + d_B) & 0 \\ 0 & 0 & d_T \end{bmatrix}$$

So the next generation matrix is

$$F_1 V_1^{-1} = \begin{bmatrix} 0 & 0 & \frac{\beta_{BT}}{d_T} \\ 0 & 0 & \frac{\beta_{BT}}{d_T} \\ \frac{\epsilon\beta_{TB}}{m_{BJ}}\frac{N_T}{N_{BJ}} & \frac{\beta_{TB}}{\tau_B + d_B}\frac{N_T}{N_{BA}} & \frac{(1-p)b_T}{d_T} \end{bmatrix} \tag{5}$$

Now, it is assumed that $b_T = d_T$ and $b_B = d_B$ so that

$$\mathcal{R}_0 = \rho\left(\begin{bmatrix} 0 & 0 & \frac{\beta_{BT}}{b_T} \\ 0 & 0 & \frac{\beta_{BT}}{b_T} \\ \frac{\epsilon\beta_{TB}}{m_{BJ}}\frac{N_T}{N_{BJ}} & \frac{\beta_{TB}}{\tau_B+b_B}\frac{N_T}{N_{BA}} & (1-p) \end{bmatrix}\right) \tag{6}$$

where ρ denotes the spectral radius. Thus, $\mathcal{R}_0$ can be calculated explicitly through the characteristic polynomial, giving

$$\mathcal{R}_0 = \frac{1}{2}(1-p) + \sqrt{\frac{\beta_{BT}\beta_{TB}N_T}{b_T}\left(\frac{\epsilon}{m_{BJ}N_{BJ}} + \frac{1}{N_{BA}(\tau_B+b_B)}\right) + \frac{1}{4}(1-p)^2} \tag{7}$$

Since the adult and juvenile populations are assumed constant and satisfy (4), the above can be rewritten as

$$\mathcal{R}_0 = \frac{1}{2}(1-p) + \sqrt{\frac{\beta_{BT}\beta_{TB}N_T}{N_{BA}b_T}\left(\frac{\epsilon}{b_B} + \frac{1}{\tau_B+b_B}\right) + \frac{1}{4}(1-p)^2} \tag{8}$$

Alternatively, vertical transmission in ticks can be taken as transfer. Writing $J(P_0) = F_2 - V_2$ with the vertical transmission in V_2 gives

$$F_2 = \begin{bmatrix} 0 & 0 & \beta_{BT} \\ 0 & 0 & \beta_{BT} \\ \epsilon\beta_{TB}\frac{N_T}{N_{BJ}} & \beta_{TB}\frac{N_T}{N_{BA}} & 0 \end{bmatrix} \text{ and } V_2 = \begin{bmatrix} m_{BJ} & 0 & 0 \\ 0 & (\tau_B+b_B) & 0 \\ 0 & 0 & pb_T \end{bmatrix},$$

where $b_B = d_B$ and $b_T = d_T$ have been used to simplify. It follows that the next generation matrix is

$$F_2V_2^{-1} = \begin{bmatrix} 0 & 0 & \frac{\beta_{BT}}{pb_T} \\ 0 & 0 & \frac{\beta_{BT}}{pb_T} \\ \epsilon\frac{\beta_{TB}}{m_{BJ}}\frac{N_T}{N_{BJ}} & \frac{\beta_{TB}}{\tau_B+b_B}\frac{N_T}{N_{BA}} & 0 \end{bmatrix} \tag{9}$$

From the characteristic polynomial of $F_2V_2^{-1}$ and by (4)

$$\rho(F_2V_2^{-1}) = \widetilde{\mathcal{R}}_0 = \sqrt{\frac{\beta_{BT}\beta_{TB}N_T}{pb_TN_{BA}}\left(\frac{\epsilon}{b_B} + \frac{1}{\tau_B+b_B}\right)} \tag{10}$$

The following general result can be used to order $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$. Inequalities on matrices are entrywise. If $A = sId - B$ where $B \geq 0$, Id is the identity matrix, and $s > \rho(B)$, then A is a nonsingular M-matrix, and $A^{-1} \geq 0$; see Chapter 6 in Berman and Plemmons (1994).

Theorem 1 *For $i = 1, 2$, let F_i be a nonnegative matrix with $F_1 \geq F_2$ and V_i be a nonsingular M-matrix, where $F_1 - V_1 = F_2 - V_2$. Assume $F_i V_i^{-1}$ is irreducible. If $0 < \rho(F_1 V_1^{-1}) < 1$, then $\rho(F_2 V_2^{-1}) \leq \rho(F_1 V_1^{-1})$, whereas if $\rho(F_1 V_1^{-1}) > 1$, then $\rho(F_2 V_2^{-1}) \geq \rho(F_1 V_1^{-1})$. In addition $\rho(F_1 V_1^{-1}) = 1$ if and only if $\rho(F_2 V_2^{-1}) = 1$.*

Proof Assume that $0 < \rho(F_1 V_1^{-1}) < 1$, and assume to the contrary that $\rho(F_2 V_2^{-1}) > \rho(F_1 V_1^{-1})$. Consider $z F_1 V_1^{-1} = z\rho(F_1 V_1^{-1})$, where z is a positive vector since $F_1 V_1^{-1}$ is irreducible and nonnegative. Then,

$$\begin{aligned}
& z\left(F_1 V_1^{-1} - \rho(F_1 V_1^{-1}) Id\right) = 0 \\
& \Rightarrow z\left(\frac{1}{\rho(F_1 V_1^{-1})} F_1 - V_1\right) = 0 \\
& \Rightarrow z\left(\frac{1}{\rho(F_1 V_1^{-1})}(F_2 + F_1 - F_2) - V_2 - V_1 + V_2\right) = 0 \\
& \Rightarrow z\left(\left(\frac{1}{\rho(F_1 V_1^{-1})} F_2 - V_2\right) + \frac{1}{\rho(F_1 V_1^{-1})}(F_1 - F_2) + (V_2 - V_1)\right) = 0
\end{aligned}$$

But $\frac{1}{\rho(F_1 V_1^{-1})} > 1$ and $\frac{1}{\rho(F_2 V_2^{-1})} < \frac{1}{\rho(F_1 V_1^{-1})}$, so the left side above is greater than

$$\begin{aligned}
& z\left(\left(\frac{1}{\rho(F_2 V_2^{-1})} F_2 - V_2\right) + (F_1 - F_2) + (V_2 - V_1)\right) \\
& = z\left(\frac{1}{\rho(F_2 V_2^{-1})} F_2 - V_2\right)
\end{aligned}$$

This means that

$$z F_2 V_2^{-1} < z\rho(F_2 V_2^{-1})$$

It follows that $\rho(F_2 V_2^{-1}) < \rho(F_2 V_2^{-1})$ by Theorem 1.11 (Collatz' Theorem) in Chapter 2 of Berman and Plemmons (1994), and this establishes a contradiction. Therefore, if $0 < \rho(F_1 V_1^{-1}) < 1$, then $\rho(F_1 V_1^{-1}) \geq \rho(F_2 V_2^{-1})$. A similar argument can be made for the case where $\rho(F_1 V_1^{-1}) > 1$. If $\rho(F_1 V_1^{-1}) = 1$, then $z(F_1 - V_1) = 0$, which implies that $z(F_2 - V_2) = 0$ giving $\rho(F_2 V_2^{-1}) = 1$, and conversely. □

A corollary to this theorem, pertaining specifically to Model J also follows.

Corollary 1 *Consider Model J, with $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$ given by (8) and (10), respectively. If $\mathcal{R}_0 < 1$, then $\widetilde{\mathcal{R}}_0 \leq \mathcal{R}_0$. If $\mathcal{R}_0 > 1$, then $\widetilde{\mathcal{R}}_0 \geq \mathcal{R}_0$, and $\mathcal{R}_0 = 1$ if and only if $\widetilde{\mathcal{R}}_0 = 1$.*

2.3 Equilibria and Stability of Model J

The following two theorems address the local stability as well as the global stability of the DFE $P_0 = (N_{BA}, 0, 0, 0)$. The theorem that addresses local stability of the DFE immediately follows from Theorem 2 in van den Driessche and Watmough (2002) since Model J satisfies assumptions A1–A4 of that theorem. The results below are stated in terms of $\mathcal{R}_0$, but by Corollary 1, they apply also with $\widetilde{\mathcal{R}}_0$.

Theorem 2 *For Model J, if $\mathcal{R}_0 < 1$, then the DFE P_0 is locally asymptotically stable, and if $\mathcal{R}_0 > 1$, then it is unstable.*

Theorem 3 *For Model J, if $\mathcal{R}_0 \leq 1$, then the DFE P_0 is globally asymptotically stable, i.e., Bovine Babesiosis dies out in the population.*

Proof The matrix-theoretic method in Shuai and van den Driessche (2013) can be applied to construct a Lyapunov function to prove the global stability of P_0. Let $x = (A_{BJ}, I_{BA}, I_T)^T$ denote the infected classes. It can be verified that $x' \leq J(P_0)x = (F_2 - V_2)x$ in Γ. Following Theorem 2.1 in Shuai and van den Driessche (2013),

$$L = w^T V_2^{-1} x \tag{11}$$

is a Lyapunov function for Model J, where w^T is the left Perron eigenvector of $V_2^{-1}F_2$, which is irreducible. Specifically,

$$w^T = \left(\epsilon\sqrt{M\frac{N_{BA}}{N_{BJ}}}, \sqrt{M\frac{N_{BJ}}{N_{BA}}}, 1\right) \quad \text{with}$$

$$M = \frac{m_{BJ}N_T\beta_{BT}(\tau_B + b_B)}{pb_T\beta_{TB}(\epsilon\tau_B N_{BA} + \epsilon b_B N_{BA} + m_{BJ}N_{BJ})},$$

and

$$w^T V_2^{-1} F_2 = \rho(V_2^{-1}F_2)w^T = \widetilde{\mathcal{R}}_0 w^T.$$

Hence, $L' = w^T V_2^{-1} x' \leq w^T V_2^{-1}(F_2 - V_2)x = (\widetilde{\mathcal{R}}_0 - 1)w^T x \leq 0$ provided $\mathcal{R}_0 \leq 1$ (thus, $\widetilde{\mathcal{R}}_0 \leq 1$ by Corollary 1). It can be verified that the only invariant set in Γ where $L' = 0$ is the singleton $\{P_0\}$. By LaSalle's invariance principle [see, e.g., LaSalle (1976)], P_0 is globally asymptotically stable. □

As in Theorem 2.2 of Shuai and van den Driessche (2013), L in (11) can be used to establish the following result.

Theorem 4 *If $\mathcal{R}_0 > 1$, then Model J is uniformly persistent.*

Proof Let L be as defined in (11). When $S_{BJ} = N_{BJ}$, $S_{BA} = N_{BA}$, $S_T = N_T$, it follows that $L' = (\widetilde{\mathcal{R}}_0 - 1)w^T x > 0$ provided $\mathcal{R}_0 > 1$ and $x > 0$. Hence, by continuity, $L' > 0$ in a neighborhood of P_0 provided $\mathcal{R}_0 > 1$. Thus, solutions in the positive cone sufficiently close to P_0 move away from P_0, implying that P_0 is unstable and a repeller

in Γ. Since P_0 is the only equilibrium that lies on the boundary of Γ and is isolated, a uniform persistence result from Freedman et al. (1994) and a similar application as in Proposition 3.3 of Li et al. (1999) show that the instability of P_0 implies uniform persistence of Model J. □

Uniform persistence and uniform boundedness of solutions in the interior of Γ imply the existence of at least one endemic equilibrium (with all variables positive); see, for example, Theorem D.3 in Smith and Waltman (1995). The following result shows that there is exactly one endemic equilibrium.

Theorem 5 *For Model J, if* $\mathcal{R}_0 > 1$, *there exists a unique endemic equilibrium* $P^* = (S^*_{BA}, A^*_{BJ}, I^*_{BA}, I^*_T)$.

Proof From $\frac{\mathrm{d}S_{BA}}{\mathrm{d}t}$ when the system is at equilibrium,

$$0 = m_{BJ}(N_{BJ} - A^*_{BJ}) + \alpha_B(N_{BA} - S^*_{BA} - I^*_{BA}) - \beta_{BT}\frac{S^*_{BA}}{N_{BA}}I^*_T - b_B S^*_{BA} \tag{12}$$

By (3), the other equations for this 4-dimensional system can be rearranged to give

$$\begin{aligned} A^*_{BJ} &= \tfrac{N_{BJ}\beta_{BT}I^*_T}{\beta_{BT}I^*_T + b_B N_{BA}} \\ I^*_{BA} &= \tfrac{pb_T I^*_T - \epsilon\beta_{TB}(N_T - I^*_T)\frac{A^*_{BJ}}{N_{BJ}}}{\beta_{TB}(N_T - I^*_T)\frac{1}{N_{BA}}} \\ S^*_{BA} &= \tfrac{(\tau_B + b_B)N_{BA}I^*_{BA}}{\beta_{BT}I^*_T} \end{aligned} \tag{13}$$

After substituting A^*_{BJ}, I^*_{BA}, S^*_{BA} into (12) and some simplification, (12) becomes

$$\begin{aligned} 0 = {} & m_{BJ}b_B N_{BA}N_{BJ}\beta_{BT}N_{BJ}\beta_{TB}(N_T - I^*_T)I^*_T \\ & - \left(\frac{\beta_{BT}I^*_T}{N_{BA}} + b_B\right)N_{BA}(\tau_B + b_B)N_{BA}[N_{BJ}pb_T I^*_T(\beta_{BT}I^*_T + b_B N_{BA}) \\ & - \epsilon\beta_{TB}(N_T - I^*_T)N_{BJ}\beta_{BT}I^*_T] \\ & + \alpha_B(N_{BA}N_{BJ}\beta_{TB}\beta_{BT}(N_T - I^*_T)(\beta_{BT}I^*_T + b_B N_{BA})I^*_T \\ & - N_{BA}I^*_T[N_{BJ}pb_T(\beta_{BT}I^*_T + b_B N_{BA}) \\ & - \epsilon\beta_{TB}(N_T - I^*_T)N_{BJ}\beta_{BT}](N_{BA}(\tau_B + b_B) + \beta_{BT}I^*_T) \end{aligned}$$

Factoring I^*_T from the above results in an equation of the form

$$F(I^*_T) = AI^{*^2}_T + BI^*_T + C$$

It is clear that $A < 0$, and

$$\begin{aligned} C = {} & \Big(\beta_{TB}\beta_{BT}N_T - pb_T N_{BA}(\tau_B + b_B) \\ & + \epsilon\beta_{TB}\beta_{BT}N_T(\tau_B + b_B)\frac{1}{b_B}\Big)[\alpha_B N_{BA}N_{BJ}N_{BA}b_B + b_B N_{BA}b_B N_{BA}N_{BJ}] \end{aligned}$$

Table 2 Values of parameters used in Model J

Parameter	Value assigned in Aranda et al. (2012)
b_B	$0.0002999\ \left(\frac{1}{\text{day}}\right)$
b_T	$0.001609\ \left(\frac{1}{\text{day}}\right)$
τ_B	$0.000265\ \left(\frac{1}{\text{day}}\right)$
α_B	$0.001000\ \left(\frac{1}{\text{day}}\right)$
β_{BT}	$0.000610\ \left(\frac{1}{\text{day}}\right)$
β_{TB}	$0.000480\ \left(\frac{1}{\text{day}}\right)$
p	0.100000

If $\widetilde{\mathcal{R}}_0 > 1$, then $C > 0$ and by Descartes' Rule of Signs, there exists one positive real root to this quadratic; thus, $F(I_T)$ has one positive real zero. At $I_T = N_T$, it is obvious that $F(N_T) < 0$, and at $I_T = 0$, it follows that $F(0) = C$, and hence, if $\widetilde{\mathcal{R}}_0 > 1$, then $F(0) > 0$. By the intermediate value property, $0 < I_T^* < N_T$. Since $I_T^* > 0$, from (13) $A_{BJ}^* > 0$. Using (13), elementary arguments show that $I_{BA}^* > 0$ and $S_{BA}^* > 0$. Therefore, there exists a unique endemic equilibrium P^* when $\mathcal{R}_0 > 1$. From (1) at equilibrium, it then follows that S_T^*, S_{BJ}^* and R_{BA}^* are all positive. □

The following numerical simulations were done using the Columbia data presented in Aranda et al. (2012), summarized in Table 2, and taking $\epsilon = 0.5$. Since we consider cattle to be juveniles up until 9 months of age, $m_{BJ} = \frac{1}{270}\frac{1}{\text{day}}$. The same initial conditions for I_{BA} and I_T (and assuming that $A_{BJ}(0) = 0$) were also used in accordance with Aranda et al. (2012), namely, $I_{BA}(0)/N_{BA} = 0.52$ and $I_T(0)/N_T = 0.60$. Taking $R_{BA}(0) = 0$ gives $I_{BA}(0) = N_{BA} - S_{BA}(0)$. To simplify our calculations, we also assume $N_{BA} + N_{BJ} = N_T$ (i.e., the cattle population is equal to the tick population, as in Aranda et al. (2012)). Note that Figs. 2 and 3 are based on a β_{BT} value of 0.006, a high value estimate (Aranda et al. 2012), while Fig. 4 has $\beta_{BT} = 0.000610$ as in Table 2.

First, the values of Aranda et al. (2012) as in Table 2 but with $\beta_{BT} = 0.006$ are used to obtain Fig. 2. The endemic equilibrium is calculated to be

$$P^* = (0.068N_{BA},\ 0.943N_{BJ},\ 0.594N_{BA},\ 0.761N_T)$$

Numerically, it is possible to investigate the local stability of the endemic equilibrium with different parameter values. Simple calculations show that the eigenvalues of the system's Jacobian matrix have negative real parts, and it follows that P^* is locally asymptotically stable.

Now, if the birth rate of the cattle is doubled (this could possibly be done by artificial insemination), i.e., $b_B = 2 \times 0.0002999$, the percentage of infectious cattle is less, as can be seen in Fig. 3. The endemic equilibrium is calculated to be

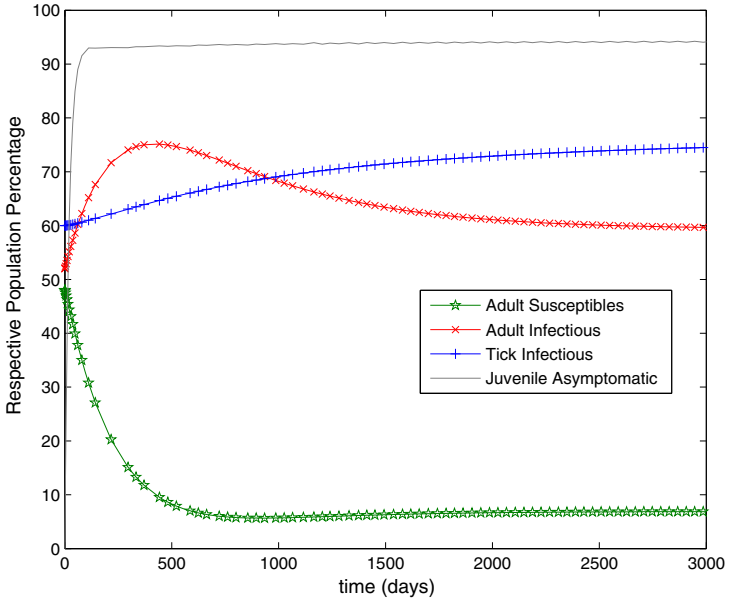

Fig. 2 S_{BA}/N_{BA}, A_{BJ}/N_{BJ}, I_{BA}/N_{BA}, and I_T/N_T versus time, $\mathcal{R}_0 = 3.07$, $\widetilde{\mathcal{R}}_0 = 8.16$. Parameter values as in Table 2 except $\beta_{BT} = 0.006$ (Color Figure Online)

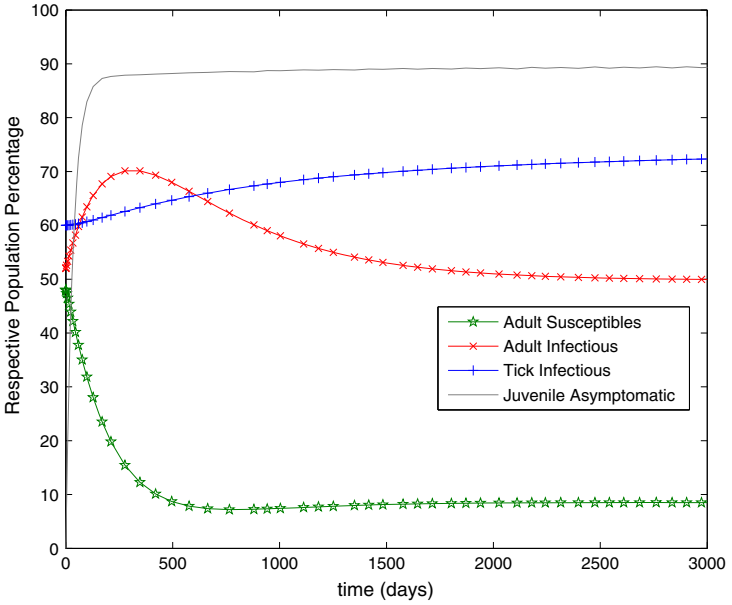

Fig. 3 S_{BA}/N_{BA}, A_{BJ}/N_{BJ}, I_{BA}/N_{BA}, and I_T/N_T versus time, $\mathcal{R}_0 = 2.53$, $\widetilde{\mathcal{R}}_0 = 6.43$. Parameter values as in Table 2 except $b_B = 2 \times 0.0002999$ and $\beta_{BT} = 0.006$ (Color Figure Online)

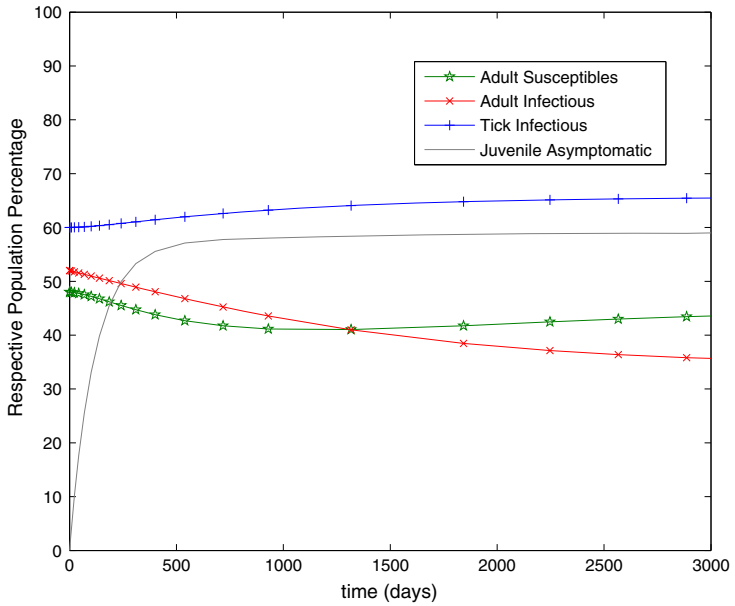

Fig. 4 S_{BA}/N_{BA}, A_{BJ}/N_{BJ}, I_{BA}/N_{BA}, and I_T/N_T versus time, $\mathcal{R}_0 = 1.39$, $\widetilde{\mathcal{R}}_0 = 2.60$. Parameter values as in Table 2 (Color Figure Online)

$$P^* = (0.084N_{BA},\ 0.895N_{BJ}\ , 0.498N_{BA},\ 0.738N_T)$$

Eigenvalues of its Jacobian matrix at P^* have negative real parts; thus, P^* is locally asymptotically stable. It is interesting to note that with b_B doubled, the infectious adult cattle decrease by almost $0.1N_{BA}$, while the infectious ticks remain relatively the same. Note also that the endemic equilibria achieved with juvenile cattle included have a lower percentage of the adult bovine population that is infectious than in the model presented by Aranda et al. (2012). The $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$ also decrease with the increased cattle birth rate. These qualitative features become even more apparent in numerical simulations with a larger birth rate.

Another interesting comparison that can be done as in Aranda et al. (2012) where a different value for β_{BT} is taken. Instead of the high value of 0.006, we take $\beta_{BT} = 0.000610$ in Fig. 4 and see how this affects P^*, $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$. The endemic equilibrium is

$$P^* = (0.449N_{BA},\ 0.590N_{BJ},\ 0.334N_{BA},\ 0.656N_T)$$

The eigenvalues of its Jacobian matrix at P^* have negative real parts; thus, P^* is locally asymptotically stable. As expected, I^*_{BA}, I^*_T, $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$ decrease with a decrease in β_{BT}.

2.4 Control Measures for Model J

2.4.1 Sensitivity Analysis

In order to determine the effect that the different parameters have on both $\mathcal{R}_0$ and on $\widetilde{\mathcal{R}}_0$, the normalized forward sensitivity index is introduced as in Chitnis et al. (2008). It is defined as follows, where u is the variable and ω is the parameter:

$$\Upsilon_{\omega}^{u} = \frac{\partial u}{\partial \omega} \times \frac{\omega}{u} \tag{14}$$

In other works, such as in Caswell (2001), this is referred to as the elasticity of a parameter, since it measures the proportional perturbation that changes in parameters have on the variable. This allows us to determine the importance of an increase or decrease of a parameter on both $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$. These indices are also useful because they provide insight into the relative accuracy of parameter values needed in order for this model to be as realistic as possible. All of the normalized forward sensitivity indices are calculated for both $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$ using the data in Table 2. For $\widetilde{\mathcal{R}}_0$

$$\Upsilon_{\beta_{BT}}^{\widetilde{\mathcal{R}}_0} = \Upsilon_{\beta_{TB}}^{\widetilde{\mathcal{R}}_0} = 0.5 \text{ and } \Upsilon_{p}^{\widetilde{\mathcal{R}}_0} = \Upsilon_{b_T}^{\widetilde{\mathcal{R}}_0} = -0.5$$

The other indices with respect to $\widetilde{\mathcal{R}}_0$ are:

$$\Upsilon_{\epsilon}^{\widetilde{\mathcal{R}}_0} = \frac{\epsilon(\tau_B + b_B)}{2[\epsilon(\tau_B + b_B) + b_B]}, \Upsilon_{\tau_B}^{\widetilde{\mathcal{R}}_0} = -\frac{b_B}{2[\epsilon(\tau_B + b_B) + b_B]} \frac{\tau_B}{\tau_B + b_B}$$
$$\Upsilon_{b_B}^{\widetilde{\mathcal{R}}_0} = -\frac{(\tau_B + b_B)b_B}{2[\epsilon(\tau_B + b_B) + b_B]} \left(\frac{\epsilon}{b_B} + \frac{b_B}{(\tau_B + b_B)^2} \right)$$

For $\mathcal{R}_0$, the forward sensitivity index for each parameter is calculated to be

$$\Upsilon_{\beta_{TB}}^{\mathcal{R}_0} = \Upsilon_{\beta_{BT}}^{\mathcal{R}_0} = \frac{1}{2\mathcal{R}_0[\mathcal{R}_0 - \frac{1}{2}(1-p)]} \frac{\beta_{BT}\beta_{TB}N_T}{b_T N_{BA}} \left(\frac{\epsilon}{b_B} + \frac{1}{\tau_B + b_B} \right) = -\Upsilon_{b_T}^{\mathcal{R}_0}$$
$$\Upsilon_{p}^{\mathcal{R}_0} = -\frac{p}{\mathcal{R}_0} \left[\frac{1}{2} + \frac{(1-p)}{\mathcal{R}_0 - \frac{1}{2}(1-p)} \right], \Upsilon_{\epsilon}^{\mathcal{R}_0} = \frac{1}{2\mathcal{R}_0[\mathcal{R}_0 - \frac{1}{2}(1-p)]} \frac{\epsilon\beta_{BT}\beta_{TB}N_T}{b_B b_T N_{BA}}$$
$$\Upsilon_{\tau_B}^{\mathcal{R}_0} = -\frac{1}{2\mathcal{R}_0[\mathcal{R}_0 - \frac{1}{2}(1-p)]} \frac{\beta_{BT}\beta_{TB}N_T}{b_T N_{BA}} \frac{\tau_B}{(\tau_B + b_B)^2}$$
$$\Upsilon_{b_B}^{\mathcal{R}_0} = -\frac{1}{2\mathcal{R}_0[\mathcal{R}_0 - \frac{1}{2}(1-p)]} \frac{\beta_{BT}\beta_{TB}N_T}{b_T N_{BA}} \left(\frac{\epsilon}{b_B} + \frac{b_B}{(\tau_B + b_B)^2} \right)$$

Note that all indices are sign determined since $\mathcal{R}_0 > \frac{1}{2}(1-p)$. Using the values reported in Aranda et al. (2012) and $\epsilon = 0.5$, these indices were calculated and are shown in Table 3. It is interesting to note that the biggest disparity between normalized forward sensitivity indices for each respective parameter for $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$ occurs for p. This is most likely due to the fact that the sole difference between $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$ is that

Table 3 Normalized forward sensitivity indices for Model J

Parameter	Value of parameter	N. F. S. index of $\mathcal{R}_0$	N. F. S. index of $\widetilde{\mathcal{R}}_0$
β_{BT}	0.0006100	0.2600	0.5000
β_{TB}	0.0004800	0.2600	0.5000
b_T	0.0016090	−0.2600	−0.5000
b_B	0.0002999	−0.1972	−0.3792
ϵ	0.5000000	0.1261	0.2425
p	0.1000000	−0.1052	−0.5000
τ_B	0.0002650	−0.0628	−0.1208

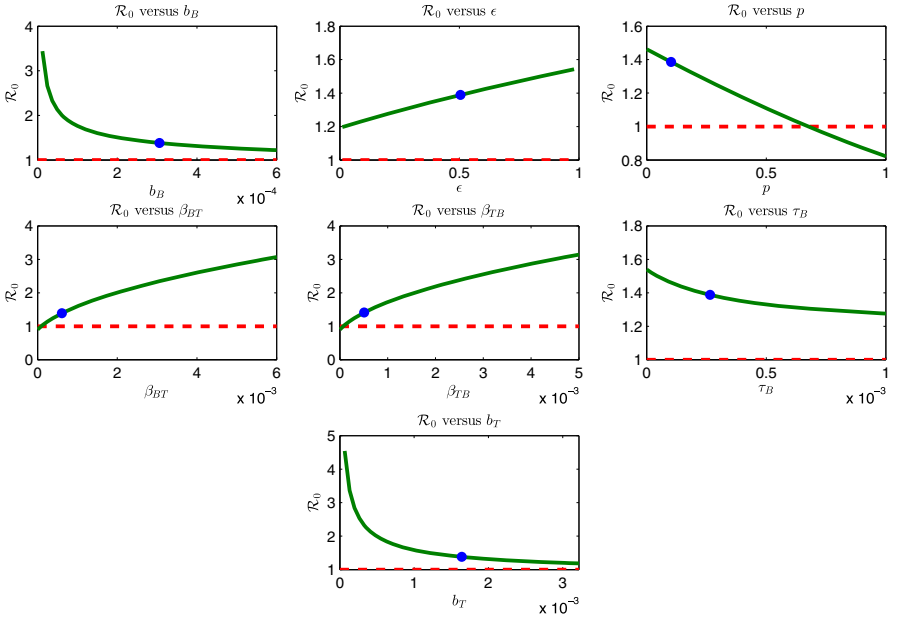

Fig. 5 Variations of parameters and their effect on $\mathcal{R}_0$. All other parameter values are as given in Table 2. Note that we assume that $N_T = N_{BA} + N_{BJ}$. The *circular marker* represents the value of the parameter assigned by Aranda et al. (2012) (Color figure online)

vertical transmission is taken to be a secondary infection for $\mathcal{R}_0$, and as transfer for $\widetilde{\mathcal{R}}_0$. Also, all the magnitudes of the normalized forward sensitivity indices, except for b_B, are larger for $\widetilde{\mathcal{R}}_0$ than for $\mathcal{R}_0$.

While the local sensitivity analysis offers insight into the relative change in $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$ caused by small variations of the parameters, a more global perspective across a wider range of parameter values can be achieved as in Manore et al. (2014) by graphing each specific parameter against $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$ as that parameter varies through a feasible region. For p, it follows that this feasible region is $0 < p \leq 1$, and for ϵ, $0 < \epsilon < 1$. It is more complicated to obtain a specific feasible region for b_B, b_T, β_{TB}, and β_{BT}. In Fig. 5, b_B ranges from 0 to 2×0.0002999, the same upper limit value used in the

numerical simulations of Sect. 2.3, and similarly for b_T. Aranda et al. (2012) suggest that $\beta_{BT} = 0.006$ is a high value estimate for this parameter, and for our purposes, we consider this to be the parameter's upper limit. Since the upper limit of β_{BT} is $10\times$ the value reported in Aranda et al. (2012), we set the upper limit of β_{TB} to also be $10\times$ its value as reported in Aranda et al. (2012).

Figure 5 shows the variation of $\mathcal{R}_0$ as each parameter is varied and all other values are kept at their respective values reported by Aranda et al. (2012), given in Table 1. The curves that are close to linear illustrate that the normalized forward sensitivity index is a good approximation for relative change in $\mathcal{R}_0$ for even large perturbations of the parameter. First, we remark that variation of parameters results in variations of $\widetilde{\mathcal{R}}_0$ similar to those reported for $\mathcal{R}_0$. Second, note that for $\mathcal{R}_0$ it is obvious that τ_B has little effect. In fact, using the values presented in Aranda et al. (2012), $\lim_{\tau_B \to 0^+} \mathcal{R}_0 = 1.54$, and $\lim_{\tau_B \to \infty} \mathcal{R}_0 = 1.18$. Clearly, variations in τ_B, no matter their magnitudes, do not cause large changes in $\mathcal{R}_0$. Biologically, this means that the natural recovery rate does not affect the number of secondary infections caused by one infection. From Fig. 5, it is evident that slight changes in only one parameter value cannot insure $\mathcal{R}_0 < 1$; moreover, in order that $\mathcal{R}_0 < 1$ by controlling only one parameter, either β_{TB} or β_{BT} needs to be decreased drastically, or p needs to be increased beyond 0.6, a sixfold increase from $p = 0.1$. This analysis illustrates that targeting only one parameter is not a feasible method to eradicate Bovine Babesiosis. However, using the method developed in Shuai et al. (2013), it is possible to calculate target reproduction numbers that can be used to more rigorously evaluate the effort needed to eradicate Bovine Babesiosis by various other control measures.

2.4.2 Target Reproduction Numbers

Target reproduction numbers allow us to quantify the percentage of a certain population that needs to be removed from the susceptibles in order to eradicate the disease (i.e., make $\mathcal{R}_0 \leq 1$). All of the following target reproduction numbers are calculated using the method described in Shuai et al. (2013). First, we calculate the target reproduction number associated with vaccination of both the adult and juvenile cattle. Using the notation in Shuai et al. (2013) and the next generation matrix $F_1 V_1^{-1}$ (5), the target set $S = \{(1, 3), (2, 3)\}$. The target reproduction number is then

$$\mathcal{T}_S = \frac{\beta_{BT}\beta_{TB}N_T}{pb_T}\left(\frac{\epsilon}{m_{BJ}N_{BJ}} + \frac{1}{N_{BA}(\tau_B + b_B)}\right) = \widetilde{\mathcal{R}}_0^{\,2} \tag{15}$$

Using the next generation matrix $F_2 V_2^{-1}$ (9) gives this same target reproduction number. Biologically, this means that if a proportion of $1 - \frac{1}{\mathcal{T}_S}$ of the juvenile and the adult bovines could be effectively vaccinated, then Bovine Babesiosis would be eradicated. Another possible method of control of cattle would be to douse them in acaricide, although this method is known to be less permanent and more intensive than vaccination, as ticks become immune to various chemicals present in acaricides (Abbas et al. 2014).

Next, we calculate the target reproduction number associated with controlling solely the adult bovine population perhaps by means of vaccination; thus, $S = \{(2, 3)\}$. Using $F_1 V_1^{-1}$, this target reproduction number is:

$$\mathcal{T}_{23} = \frac{\beta_{BT}\beta_{TB}N_T m_{BJ}N_{BJ}}{N_{BA}(\tau_B + b_B)(m_{BJ}N_{BJ}pb_T - \epsilon\beta_{BT}\beta_{TB}N_T)} \text{ if } m_{BJ}N_{BJ}pb_T - \epsilon\beta_{BT}\beta_{TB}N_T > 0 \tag{16}$$

The same calculations for this target reproduction number can be done using $\widetilde{\mathcal{R}}_0$, and $\widetilde{\mathcal{T}}_{23} = \mathcal{T}_{23}$. This means that a proportion $1 - \frac{1}{\mathcal{T}_{23}}$ of the adult cattle needs to be controlled (perhaps by vaccination) in order for Bovine Babesiosis to be eradicated.

The target reproduction number associated with control of the tick population is also calculated, where $S = \{(3, 1), (3, 2), (3, 3)\}$. This is actually a type reproduction number as introduced by Roberts and Heesterbeek (2003) and Heesterbeek and Roberts (2007) and is denoted by $\mathcal{T}_{3*}$, where

$$\mathcal{T}_{3*} = \frac{\beta_{BT}\beta_{TB}N_T}{b_T}\left(\frac{\epsilon}{m_{BJ}N_{BJ}} + \frac{1}{N_{BA}(\tau_B + b_B)}\right) + (1 - p) \tag{17}$$

As done above, we also calculate the type reproduction number based on the matrix $F_2 V_2^{-1}$, used to calculate $\widetilde{\mathcal{R}}_0$ instead, and this leads to

$$\widetilde{\mathcal{T}}_{3*} = \frac{\beta_{BT}\beta_{TB}N_T}{pb_T}\left(\frac{\epsilon}{m_{BJ}N_{BJ}} + \frac{1}{N_{BA}(\tau_B + b_B)}\right) = \widetilde{\mathcal{R}}_0^{\,2} = \mathcal{T}_S \tag{18}$$

This means that in order to eradicate Bovine Babesiosis, there needs to be an eradication of a proportion of $1 - \frac{1}{\widetilde{\mathcal{T}}_{3*}} = 1 - \frac{1}{\mathcal{T}_S}$ of the total tick population N_T (this will reduce the control reproduction number below one which will result in the populations going to the DFE). It is important to note that de Waal and Combrink (2006) state that control of the tick population in order to eradicate Bovine Babesiosis is perhaps not feasible (if tick control is other then total eradication) as it suffices to have only a few infected ticks in a region in order to cause an outbreak of Bovine Babesiosis in cattle. Therefore, it is probably more relevant to target bovine populations for control of Bovine Babesiosis, or to eradicate the tick population, as in the US (Schnittger et al. 2012).

Values for the various target/type reproduction numbers discussed above can be computed using the same data for the relevant parameters as Aranda et al. (2012). Note that ϵ is varied to investigate its effect, as well as β_{BT}. Just as in the numerical simulations of Model J in Sect. 2.3, it is assumed that $N_T = N_{BJ} + N_{BA}$ (so the total tick population equals the total bovine population). Aranda et al. (2012) reported that $\beta_{BT} = 0.00061$ in North Colombia, and only used $\beta_{BT} = 0.006$ as a high value for β_{BT}. The same was done here. In Table 4, the target reproduction numbers are as defined above. If $\mathcal{T}_{23}$ is omitted in Table 4 for specific ϵ and β_{BT} values, it is because $m_{BJ}N_{BJ}pb_T - \epsilon\beta_{BT}\beta_{TB}N_T \leq 0$, and $\mathcal{T}_{23}$ is therefore irrelevant, as biologically this control measure alone cannot lead to eradication of Bovine Babesiosis.

Table 4 Target reproduction numbers for numerical simulations of Model J

ϵ value	β_{BT} value	Target rep. number	Value	% of pop. needed to control
0.1	0.00061	$\mathcal{T}_{23}$	10.12	90
0.1	0.00061	$\mathcal{T}_{3*}$	1.31	24
0.1	0.00061	$\mathcal{T}_S$	4.14	76
0.5	0.00061	$\mathcal{T}_{3*}$	1.58	37
0.5	0.00061	$\mathcal{T}_S$	6.76	85
0.9	0.00061	$\mathcal{T}_{3*}$	1.84	46
0.9	0.00061	$\mathcal{T}_S$	9.39	89
0.1	0.006	$\mathcal{T}_{3*}$	4.97	80
0.1	0.006	$\mathcal{T}_S$	40.70	98
0.5	0.006	$\mathcal{T}_{3*}$	7.55	87
0.5	0.006	$\mathcal{T}_S$	66.51	99
0.9	0.006	$\mathcal{T}_{3*}$	10.13	90
0.9	0.006	$\mathcal{T}_S$	92.32	99

3 Model with Groups (Model G)

3.1 Formulation of Model G

This model divides the cattle into two groups: the *B. indicus* and the *B. taurus* group but does not distinguish juvenile cattle. According to Jonsson et al. (2008), the *B. indicus* breed of cattle have a higher resistance to ticks, as well as lower parasitaemia in their blood stream following a successful infection by either *B. bigemina* or by *B. bovis*. Furthermore, Jonsson et al. (2008) stipulated that vaccination of *B. indicus* cattle is perhaps not necessary to eradicate Bovine Babesiosis from the overall cattle population. Model G investigates this. Here S_{BU}, I_{BU}, R_{BU} and N_{BU} represent the susceptible, infectious, recovered and total population of *B. taurus* cattle; S_{BD}, I_{BD}, R_{BD} and N_{BD} represent the susceptible, infectious, recovered and total population of *B. indicus* cattle. Also, since the *B. indicus* cattle are more tick resistant and have lower parasitaemia in their blood following an infection, we introduce new parameters ζ_{BT}, $0 < \zeta_{BT} < 1$ to lower the transmission rate of Bovine Babesiosis from ticks to *B. indicus* cattle, and ζ_{TB}, $0 < \zeta_{TB} < 1$ to lower the transmission rate of Bovine Babesiosis from *B. indicus* cattle to ticks. This means that $\zeta_{TB}\beta_{TB}$ is the transmission rate of Bovine Babesiosis from *B. indicus* cattle to ticks, and that $\zeta_{BT}\beta_{BT}$ is the transmission rate of Bovine Babesiosis from ticks to *B. indicus* cattle. Piper et al. (2008) reported that on average, *B. indicus* cattle had 10 times as many ticks attached as *B. taurus* cattle (to show this, Brahman and Holstein–Friesian cattle, types of *B. indicus* and *B. taurus*, respectively, were used). This implies that $\zeta_{BT} = 0.1$, if we assume that the *B. indicus* cattle resistance to Bovine Babesiosis is solely brought upon by less tick attachment. Bock et al. (1999b) reported that the mean total parasitaemia score of *B. bigemina* present in *B. taurus* cattle is about five times larger than that present in *B. indicus*, and Bock et al. (1997) concluded that the mean total parasitaemia score

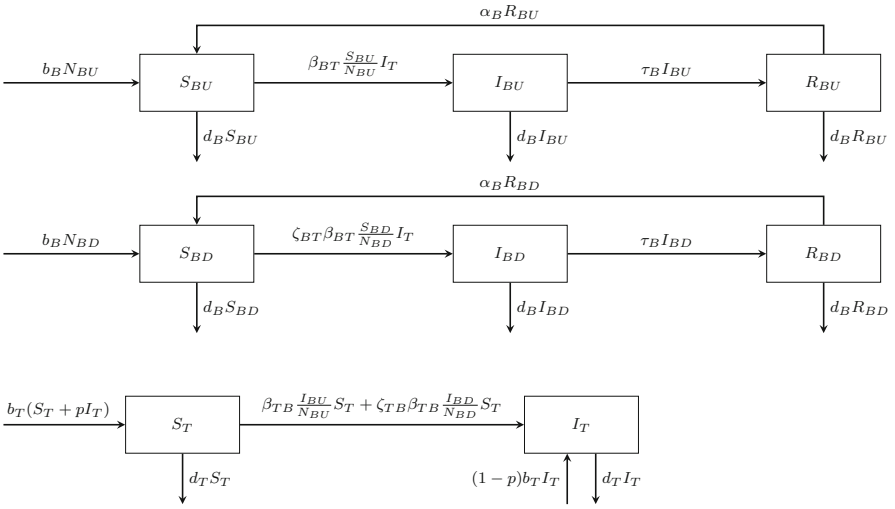

Fig. 6 Bovine and tick populations flowchart for Model G

of *B. bovis* is also about 5 times larger in *B. taurus* cattle than in *B. indicus* cattle. Since there are about 10 times fewer ticks on the *B. indicus* cattle, and the mean total parasitaemia score of both *B. bovis* and *B. bigemina* for *B. indicus* cattle is about five times less than that of *B. taurus* cattle, we assume that $\zeta_{TB} = 0.1(0.2) = 0.02$. In order to visualize Model G, Fig. 6 is useful. We formulate the dynamics of Model G as follows:

$$\begin{aligned}
\frac{\mathrm{d}S_{BD}}{\mathrm{d}t} &= b_B N_{BD} + \alpha_B R_{BD} - \zeta_{BT}\beta_{BT}\frac{S_{BD}}{N_{BD}} I_T - d_B S_{BD} \\
\frac{\mathrm{d}I_{BD}}{\mathrm{d}t} &= \zeta_{BT}\beta_{BT}\frac{S_{BD}}{N_{BD}} I_T - (\tau_B + d_B) I_{BD} \\
\frac{\mathrm{d}R_{BD}}{\mathrm{d}t} &= \tau_B I_{BD} - (\alpha_B + d_B) R_{BD} \\
\frac{\mathrm{d}S_{BU}}{\mathrm{d}t} &= b_B N_{BU} + \alpha_B R_{BU} - \beta_{BT}\frac{S_{BU}}{N_{BU}} I_T - d_B S_{BU} \\
\frac{\mathrm{d}I_{BU}}{\mathrm{d}t} &= \beta_{BT}\frac{S_{BU}}{N_{BU}} I_T - (\tau_B + d_B) I_{BU} \\
\frac{\mathrm{d}R_{BU}}{\mathrm{d}t} &= \tau_B I_{BU} - (\alpha_B + d_B) R_{BU} \\
\frac{\mathrm{d}S_T}{\mathrm{d}t} &= p b_T I_T + b_T S_T - \beta_{TB} S_T \frac{I_{BU}}{N_{BU}} - \zeta_{TB}\beta_{TB} S_T \frac{I_{BD}}{N_{BD}} - d_T S_T \\
\frac{\mathrm{d}I_T}{\mathrm{d}t} &= (1-p) b_T I_T + \beta_{TB} S_T \frac{I_{BU}}{N_{BU}} + \zeta_{TB}\beta_{TB} S_T \frac{I_{BD}}{N_{BD}} - d_T I_T
\end{aligned} \tag{19}$$

The above equations directly imply the following:

$$\frac{\mathrm{d}N_{BD}}{\mathrm{d}t} = b_B N_{BD} - d_B N_{BD}$$

$$\frac{\mathrm{d}N_{BU}}{\mathrm{d}t} = b_B N_{BU} - d_B N_{BU}$$
$$\frac{\mathrm{d}N_T}{\mathrm{d}t} = (b_T - d_T) N_T$$

Using the constraints that $S_{BD} + I_{BD} + R_{BD} = N_{BD}$, $S_T + I_T = N_T$, and $S_{BU} + I_{BU} + R_{BU} = N_{BU}$, along with assumptions that $b_B = d_B$ and that $b_T = d_T$, this model can be reduced to the following 5-dimensional one:

$$\begin{aligned}
\frac{\mathrm{d}S_{BD}}{\mathrm{d}t} &= b_B N_{BD} + \alpha_B (N_{BD} - S_{BD} - I_{BD}) - \zeta_{BT}\beta_{BT}\frac{S_{BD}}{N_{BD}} I_T - b_B S_{BD} \\
\frac{\mathrm{d}I_{BD}}{\mathrm{d}t} &= \zeta_{BT}\beta_{BT}\frac{S_{BD}}{N_{BD}} I_T - (\tau_B + b_B) I_{BD} \\
\frac{\mathrm{d}S_{BU}}{\mathrm{d}t} &= b_B N_{BU} + \alpha_B (N_{BU} - S_{BU} - I_{BU}) - \beta_{BT}\frac{S_{BU}}{N_{BU}} I_T - b_B S_{BU} \\
\frac{\mathrm{d}I_{BU}}{\mathrm{d}t} &= \beta_{BT}\frac{S_{BU}}{N_{BU}} I_T - (\tau_B + b_B) I_{BU} \\
\frac{\mathrm{d}I_T}{\mathrm{d}t} &= (1-p) b_T I_T + \beta_{TB}(N_T - I_T)\frac{I_{BU}}{N_{BU}} + \zeta_{TB}\beta_{TB}(N_T - I_T)\frac{I_{BD}}{N_{BD}} - b_T I_T
\end{aligned} \tag{20}$$

It is easy to show that this 5-dimensional model is well posed within the feasible region

$$\begin{aligned}
\Gamma = \{(S_{BD}, I_{BD}, S_{BU}, I_{BU}, I_T) \in \mathbb{R}^5_+ \mid 0 \le S_{BD} + I_{BD} \le N_{BD}, 0 \le S_{BU} \\
+ I_{BU} \le N_{BU}, 0 \le I_T \le N_T\}.
\end{aligned}$$

3.2 Basic Reproduction Numbers for Model G

It is obvious that when $S_{BU} = N_{BU}$, $S_{BD} = N_{BD}$, $S_T = N_T$ and $I_{BD} = I_{BU} = I_T = 0$ the system is at equilibrium, and this point P_0 is the DFE. Taking the infected classes, the Jacobian matrix about P_0 is

$$J(P_0) = \begin{bmatrix} -(\tau_B + b_B) & 0 & \zeta_{BT}\beta_{BT} \\ 0 & -(\tau_B + d_B) & \beta_{BT} \\ \zeta_{TB}\beta_{TB}\frac{N_T}{N_{BD}} & \beta_{TB}\frac{N_T}{N_{BU}} & (1-p)b_T - b_T \end{bmatrix}$$

It follows that

$$F_1 = \begin{bmatrix} 0 & 0 & \zeta_{BT}\beta_{BT} \\ 0 & 0 & \beta_{BT} \\ \zeta_{TB}\beta_{TB}\frac{N_T}{N_{BD}} & \beta_{TB}\frac{N_T}{N_{BU}} & (1-p)b_T \end{bmatrix} \text{ and } V_1 = \begin{bmatrix} (\tau_B + b_B) & 0 & 0 \\ 0 & (\tau_B + b_B) & 0 \\ 0 & 0 & b_T \end{bmatrix}$$

Thus,

$$\mathcal{R}_0 = \rho\left(\begin{bmatrix} 0 & 0 & \frac{\zeta_{BT}\beta_{BT}}{b_T} \\ 0 & 0 & \frac{\beta_{BT}}{b_T} \\ \frac{\zeta_{TB}\beta_{TB}}{\tau_B+b_B}\frac{N_T}{N_{BD}} & \frac{\beta_{TB}}{\tau_B+b_B}\frac{N_T}{N_{BU}} & (1-p) \end{bmatrix}\right) \tag{21}$$

Alternatively, as in Model J, if vertical transmission is taken as transfer, then

$$\widetilde{\mathcal{R}}_0 = \sqrt{\frac{\beta_{TB}\beta_{BT}N_T}{pb_T(\tau_B+b_B)}\left(\frac{\zeta_{TB}\zeta_{BT}}{N_{BD}}+\frac{1}{N_{BU}}\right)} \tag{22}$$

3.3 Equilibria and Stability of Model G

The following theorem about the local stability of P_0 immediately follows from Theorem 2 in van den Driessche and Watmough (2002), since Model G satisfies the required assumptions.

Theorem 6 *For Model G, if $\mathcal{R}_0 < 1$, then the DFE P_0 is locally asymptotically stable, and if $\mathcal{R}_0 > 1$, then it is unstable.*

Theorem 7 *For Model G, if $\mathcal{R}_0 \leq 1$, then the DFE P_0 is globally asymptotically stable; if $\mathcal{R}_0 > 1$, then Model G is uniformly persistent and there exists at least one endemic equilibrium.*

Proof Let $x = (I_{BD}, I_{BU}, I_T)^T$ denote the infected classes. From (19), it follows that $x' \leq (F_1 - V_1)x$ and $V_1^{-1}F_1$ is irreducible. As in the proof of Theorem 3, following Theorem 2.1 in Shuai and van den Driessche (2013), consider a Lyapunov function $L = w^T V_1^{-1}x$, where w^T is the left Perron eigenvector of $V_1^{-1}F_1$. It follows that $L' \leq w^T V_1^{-1}(F_1 - V_1)x = (\mathcal{R}_0 - 1)w^T x$ as $\mathcal{R}_0 = \rho(F_1V_1^{-1}) = \rho(V_1^{-1}F_1)$. Thus, $L' \leq 0$ provided $\mathcal{R}_0 \leq 1$. An application of LaSalle's invariance principle (LaSalle 1976) establishes that if $\mathcal{R}_0 \leq 1$, then P_0 is globally asymptotically stable. The Lyapunov function constructed can be used as in Theorem 4 to prove the stated results for $\mathcal{R}_0 > 1$. Specifically, $L' > 0$ in a neighborhood of P_0 provided $\mathcal{R}_0 > 1$. Thus, instability of P_0 implies the uniform persistence of Model G and the existence of at lease one endemic equilibrium, following similar arguments as those in Sect. 2.3. □

Theorem 8 *For Model G, if $\alpha_B = 0$ (i.e., recovered cattle have permanent immunity) and $\mathcal{R}_0 > 1$, then the endemic equilibrium is unique and globally asymptotically stable in the interior of Γ.*

Proof The S_T and I_T equations of (19) can be written as

$$\begin{aligned} \frac{\mathrm{d}S_T}{\mathrm{d}t} &= pb_T N_T - (d_T - b_T(1-p))S_T - \beta_{TB}S_T\frac{I_{BU}}{N_{BU}} - \zeta_{TB}\beta_{TB}S_T\frac{I_{BD}}{N_{BD}} \\ \frac{\mathrm{d}I_T}{\mathrm{d}t} &= \beta_{TB}S_T\frac{I_{BU}}{N_{BU}} + \zeta_{TB}\beta_{TB}S_T\frac{I_{BD}}{N_{BD}} - (d_T - b_T(1-p))I_T \end{aligned}$$

Since $d_T = b_T$, it follows that $d_T - b_T(1-p) > 0$. Thus, system (19) can be regarded as a 3-group SIR model in the form

$$\begin{aligned} \frac{\mathrm{d}S_i}{\mathrm{d}t} &= \Lambda_i - d_i S_i - \sum_{j=1}^{3} \beta_{ij} S_i I_j \\ \frac{\mathrm{d}I_i}{\mathrm{d}t} &= \sum_{j=1}^{3} \beta_{ij} S_i I_j - (d_i + \gamma_i) I_i \end{aligned} \tag{23}$$

with $\Lambda_i > 0$, $d_i > 0$, $\gamma_i \geq 0$, and the contact matrix $[\beta_{ij}]$ irreducible. Using Theorem 3.3 of Guo et al. (2006), the result holds. □

3.4 Target Reproduction Numbers for Model G

Just as was done for Model J, target reproduction numbers are calculated to evaluate the effort needed to eradicate Bovine Babesiosis by controlling various populations in Model G. This allows us to determine which control strategy (for Model G) would be most suitable and feasible. These reproduction numbers are calculated as in Shuai et al. (2013). The target reproduction numbers are calculated from both next generation matrices (used to calculate $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$), but are found to be equal.

The first target reproduction number calculated targets only the *B. taurus* cattle, as they are more highly susceptible to infection by Bovine Babesiosis:

$$\begin{aligned} \mathcal{T}_{23} = \widetilde{\mathcal{T}}_{23} = \frac{\beta_{BT}\beta_{TB}N_T N_{BD}}{N_{BU}[(\tau_B + b_B)N_{BD}pb_T - \zeta_{BT}\zeta_{TB}\beta_{BT}\beta_{TB}N_T]} \\ \text{if } (\tau_B + b_B)N_{BD}pb_T - \zeta_{BT}\zeta_{TB}\beta_{BT}\beta_{TB}N_T > 0 \end{aligned} \tag{24}$$

From this, it follows that in order to eradicate Bovine Babesiosis by only controlling the *B. taurus* cattle, there would need to be vaccination of a proportion of $1 - \frac{1}{\mathcal{T}_{23}}$ of the *B. taurus* cattle.

The next target reproduction number targets both cattle breeds; thus, $S = \{(1,3),(2,3)\}$, and

$$\mathcal{T}_S = \widetilde{\mathcal{T}}_S = \frac{\beta_{BT}\beta_{TB}N_T(\zeta_{BT}\zeta_{TB}N_{BU} + N_{BD})}{pb_T(\tau_B + b_B)N_{BU}N_{BD}} \tag{25}$$

It follows that in order to eradicate Bovine Babesiosis, there would need to be vaccination of a proportion of $1 - \frac{1}{\mathcal{T}_S}$ of both breeds of cattle.

The above target reproduction numbers are calculated using the parameter values presented in Aranda et al. (2012) as in Table 2, and the results are shown in Table 5. Note that $\mathcal{T}_{23}$ and $\mathcal{T}_S$ are quite close: this is due to the low values of ζ_{TB} and ζ_{BT} that were used. If ζ_{TB} and ζ_{BT} are increased, then $\mathcal{T}_{23}$ increases faster than $\mathcal{T}_S$.

Table 5 Target reproduction numbers for numerical simulations of Model J

Target rep. number	Value	% of pop. needed to control
$\mathcal{T}_{23}$	6.53	85
$\mathcal{T}_S$	6.46	85

3.5 Model by Aranda et al. (2012), a Special Case

Model G can be simplified to the model presented in Aranda et al. (2012) for one cattle breed, e.g., *B. taurus*. The simplification of Model G to the model in Aranda et al. (2012) is done by taking $\zeta_{BT} = \zeta_{TB} = 0$, $N_B = N_{BU}$, $S_B = S_{BU}$, $I_B = I_{BU}$, $R_B = R_{BU}$. Note that $\mathcal{R}_0$ for this model after simplifications is the same (with no vertical transmission in cattle) as in Friedman and Yakubu (2014), whereas Aranda et al. (2012) use $\widetilde{\mathcal{R}}_0^2$ as the threshold parameter. After the above simplifications, Model G becomes

$$\begin{aligned}
\frac{\mathrm{d}S_B}{\mathrm{d}t} &= b_B N_B + \alpha_B R_B - \beta_{BT}\frac{S_B}{N_B} I_T - d_B S_B \\
\frac{\mathrm{d}I_B}{\mathrm{d}t} &= \beta_{BT}\frac{S_B}{N_B} I_T - (\tau_B + d_B) I_B \\
\frac{\mathrm{d}R_B}{\mathrm{d}t} &= \tau_B I_B - (\alpha_B + d_B) R_B \\
\frac{\mathrm{d}S_T}{\mathrm{d}t} &= b_T (S_T + p I_T) - \beta_{TB} S_T \frac{I_B}{N_B} - d_T S_T \\
\frac{\mathrm{d}I_T}{\mathrm{d}t} &= (1-p) b_T I_T + \beta_{TB} S_T \frac{I_B}{N_B} - d_T I_T
\end{aligned} \tag{26}$$

These imply the next set of differential equations for N_B and N_T:

$$\begin{aligned}
\frac{\mathrm{d}N_B}{\mathrm{d}t} &= (b_B - d_B) N_B \\
\frac{\mathrm{d}N_T}{\mathrm{d}t} &= (b_T - d_T) N_T
\end{aligned}$$

As in Aranda et al. (2012), this model can be reduced to a 3-dimensional one by substituting $S_T = N_T - I_T$ and $R_B = N_B - S_B - I_B$. Also, we assume that $b_B = d_B$ and $b_T = d_T$ so both the bovine and the tick populations are constant. The model becomes:

$$\begin{aligned}
\frac{\mathrm{d}S_B}{\mathrm{d}t} &= b_B (N_B - I_B) + \alpha_B (N_B - S_B - I_B) - \beta_{BT}\frac{S_B}{N_B} I_T + b_B I_B - b_B S_B \\
\frac{\mathrm{d}I_B}{\mathrm{d}t} &= \beta_{BT}\frac{S_B}{N_B} I_T - (\tau_B + b_B) I_B \\
\frac{\mathrm{d}I_T}{\mathrm{d}t} &= (1-p) b_T I_T + \beta_{TB} (N_T - I_T)\frac{I_B}{N_B} - b_T I_T
\end{aligned} \tag{27}$$

The region $\Gamma = \{(S_B, I_B, I_T) \in \mathbb{R}^3_+ \mid 0 \leq S_B + I_B \leq N_B, 0 \leq I_T \leq N_T\}$ is the feasible region for the above system, and it is easy to show that the model is well posed within this feasible region, as stated in Aranda et al. (2012).

Local and global asymptotic stability of the DFE of this model were proved in Aranda et al. (2012). A unique endemic equilibrium P^* if $\mathcal{R}_0 > 1$ was also determined; see Eqs. (5)–(7) in Aranda et al. (2012). The following new result addresses the local stability of P^* for $\alpha_B \geq 0$ (i.e., recovered cattle may have temporary immunity).

Theorem 9 *If $\mathcal{R}_0 > 1$, then the endemic equilibrium P^* of (27) is locally asymptotically stable.*

Proof Let $\gamma_B = \alpha_B + b_B$ and consider

$$J(P^*) = \begin{bmatrix} -\left(\gamma_B + \beta_{BT} I_T^* \frac{1}{N_B}\right) & -\alpha_B & -\beta_{BT} \frac{S_B^*}{N_B} \\ \beta_{BT} \frac{I_T^*}{N_B} & -(b_B + \tau_B) & \beta_{BT} \frac{S_B^*}{N_B} \\ 0 & \beta_{TB}(N_T - I_T^*) \frac{1}{N_B} & -\left(pb_T + \beta_{TB} \frac{I_B^*}{N_B}\right) \end{bmatrix} \tag{28}$$

From (28) $tr(J(P^*)) = -(\gamma_B + \beta_{BT} I_B^* \frac{1}{N_B} + b_B + \tau_B + pb_T + \beta_{TB} \frac{I_B^*}{N_B}) < 0$. By using $\frac{I_B^*}{I_T^*} = \beta_{BT} \frac{S_B^*}{N_B} \frac{1}{\tau_B + b_B}$ from (27), calculations show

$$\begin{aligned} det(J(P^*)) = -\gamma_B \left(\beta_{TB}\beta_{BT} I_T^* S_B^* \frac{1}{N_B}\frac{1}{N_B} + \beta_{BT}\beta_{TB} I_B^* \frac{N_T}{N_B}\frac{1}{N_B}\right) \\ -\beta_{BT}\beta_{TB}\tau_B I_B^* \frac{N_T}{N_B}\frac{1}{N_B} < 0 \end{aligned} \tag{29}$$

After simplification the sum of the 2×2 principal minors of $J(P^*)$, denoted by a_2 is

$$\begin{aligned} a_2 = \gamma_B \beta_{TB} \frac{I_B^*}{I_T^*} \frac{N_T}{N_B} + \beta_{BT}\beta_{TB} I_B^* \frac{I_B^*}{I_T^*} \frac{N_T}{N_B}\frac{1}{N_B} + \beta_{BT}\beta_{TB} I_T^* S_B^* \frac{1}{N_B}\frac{1}{N_B} \\ + \tau_B \gamma_B + \gamma_B b_B + \beta_{BT}\tau_B I_T^* \frac{1}{N_B} + \beta_{BT}\gamma_B I_T^* \frac{1}{N_B} \end{aligned}$$

Forming $tr(J(P^*))a_2 - det(J(P^*))$ and simplifying shows this expression is negative. Thus, by the Routh–Hurwitz conditions, the eigenvalues of $J(P^*)$ all have negative real parts, and so P^* is locally asymptotically stable when $\mathcal{R}_0 > 1$. □

4 Model with Juveniles and Groups (Model J+G)

4.1 Formulation of Model J+G

Jonsson et al. (2008) state that while *B. indicus* cattle are more highly resistant to Bovine Babesiosis than *B. taurus* cattle, outbreaks still occur among the *B. indicus* cattle if there is not a high exposure rate among juvenile *B. indicus* cattle (which

effectively lowers the amount of subsequent susceptible adult *B. indicus* cattle). Model J+G was formulated in order to investigate more accurately possible control measures for Bovine Babesiosis when both *B. indicus* and *B. taurus* cattle are present, and the juvenile resistance is taken into account. In the following model, S_{BDJ} and A_{BDJ} refer, respectively, to the susceptible and infectious juvenile *B. indicus* cattle, S_{BDA}, I_{BDA}, and R_{BDA} refer, respectively, to the susceptible, infectious, and recovered *B. indicus* adult cattle, N_{BDJ} refers to the total juvenile *B. indicus* cattle population, and N_{BDA} refers to the total adult *B. indicus* population. Also S_{BUJ} and A_{BUJ} refer, respectively, to the to the susceptible and infectious juvenile *B. taurus* cattle, and S_{BUA}, I_{BUA}, and R_{BDA} refer, respectively, to the susceptible, infectious, and recovered *B. taurus* adult cattle, N_{BUJ} refers to the total juvenile *B. taurus* population, and N_{BUA} refers to the total adult *B. taurus* population. The other parameters are as defined in Table 1, and ζ_{BT} and ζ_{TB} are as defined in Model G. Model J+G is formulated as follows:

$$
\begin{aligned}
\frac{\mathrm{d}S_{BDJ}}{\mathrm{d}t} &= b_B N_{BA} - m_{BJ} S_{BDJ} - \zeta_{BT}\beta_{BT} I_T \frac{S_{BDJ}}{N_{BDJ}} \\
\frac{\mathrm{d}A_{BDJ}}{\mathrm{d}t} &= \zeta_{BT}\beta_{BT} I_T \frac{S_{BDJ}}{N_{BDJ}} - m_{BJ} A_{BDJ} \\
\frac{\mathrm{d}S_{BDA}}{\mathrm{d}t} &= m_{BJ} S_{BDJ} + \alpha_B R_{BDA} - \zeta_{BT}\beta_{BT} \frac{S_{BDA}}{N_{BDA}} I_T - d_B S_{BDA} \\
\frac{\mathrm{d}I_{BDA}}{\mathrm{d}t} &= \zeta_{BT}\beta_{BT} \frac{S_{BDA}}{N_{BDA}} I_T - (\tau_B + d_B) I_{BDA} \\
\frac{\mathrm{d}R_{BDA}}{\mathrm{d}t} &= \tau_B I_{BDA} - (\alpha_B + d_B) R_{BDA} \\
\frac{\mathrm{d}S_{BUJ}}{\mathrm{d}t} &= b_B N_{BUA} - m_{BJ} S_{BUJ} - \beta_{BT} I_T \frac{S_{BUJ}}{N_{BUJ}} \\
\frac{\mathrm{d}A_{BUJ}}{\mathrm{d}t} &= \beta_{BT} I_T \frac{S_{BUJ}}{N_{BUJ}} - m_{BJ} A_{BUJ} \\
\frac{\mathrm{d}S_{BUA}}{\mathrm{d}t} &= m_{BJ} S_{BUJ} + \alpha_B R_{BUA} - \beta_{BT} \frac{S_{BUA}}{N_{BUA}} I_T - d_B S_{BU} \\
\frac{\mathrm{d}I_{BUA}}{\mathrm{d}t} &= \beta_{BT} \frac{S_{BUA}}{N_{BUA}} I_T - (\tau_B + d_B) I_{BUA} \\
\frac{\mathrm{d}R_{BUA}}{\mathrm{d}t} &= \tau_B I_{BUA} - (\alpha_B + d_B) R_{BUA} \\
\frac{\mathrm{d}S_T}{\mathrm{d}t} &= p b_T I_T + b_T S_T - \beta_{TB} S_T \frac{I_{BUA}}{N_{BUA}} - \zeta_{TB}\beta_{TB} S_T \frac{I_{BDA}}{N_{BDA}} \\
&\quad - \epsilon\beta_{TB} S_T \frac{A_{BUJ}}{N_{BUJ}} - \epsilon\zeta_{TB}\beta_{TB} S_T \frac{A_{BDJ}}{N_{BDJ}} - d_T S_T \\
\frac{\mathrm{d}I_T}{\mathrm{d}t} &= (1-p) b_T I_T + \beta_{TB} S_T \frac{I_{BUA}}{N_{BUA}} + \zeta_{TB}\beta_{TB} S_T \frac{I_{BDA}}{N_{BDA}} \\
&\quad + \epsilon\beta_{TB} S_T \frac{A_{BUJ}}{N_{BUJ}} + \epsilon\zeta_{TB}\beta_{TB} S_T \frac{A_{BDJ}}{N_{BDJ}} - d_T I_T
\end{aligned}
\tag{30}
$$

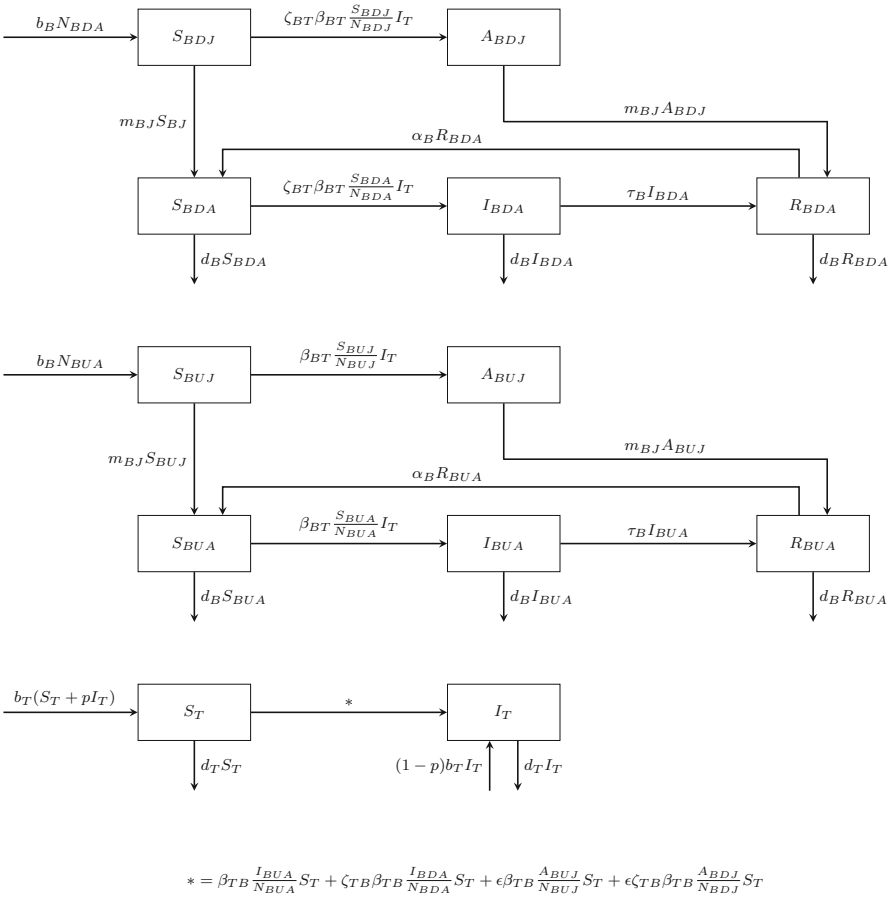

Fig. 7 Bovine and tick populations flowchart for Model J+G

In order to visualize Model J+G, Fig. 7 is useful. Equations (30) directly imply the next set of equations:

$$\begin{aligned}
\frac{\mathrm{d}N_{BDJ}}{\mathrm{d}t} &= b_B N_{BDA} - m_{BJ} N_{BDJ} \\
\frac{\mathrm{d}N_{BDA}}{\mathrm{d}t} &= m_{BJ} N_{BDJ} - d_B N_{BDA} \\
\frac{\mathrm{d}N_{BUJ}}{\mathrm{d}t} &= b_B N_{BUA} - m_{BJ} N_{BUJ} \\
\frac{\mathrm{d}N_{BUA}}{\mathrm{d}t} &= m_{BJ} N_{BUJ} - d_B N_{BUA} \\
\frac{\mathrm{d}N_T}{\mathrm{d}t} &= (b_T - d_T) N_T
\end{aligned}$$

As in Sect. 2, we assume that all of the total populations are constant. This means that not only $b_B = d_B$ and $b_T = d_T$, but also $b_B N_{BDA} = m_{BJ} N_{BDJ}$ and $b_B N_{BUA} = m_{BJ} N_{BUJ}$. Since $R_{BUA} = N_{BUA} - S_{BUA} - I_{BUA}$, $S_{BUJ} = N_{BUJ} - A_{BUJ}$, $R_{BDA} = N_{BDA} - S_{BDA} - I_{BDA}$, $S_{BDJ} = N_{BDJ} - A_{BDJ}$, and $S_T = N_T - I_T$, the above model can be reduced to the following 7-dimensional model:

$$\begin{aligned}
\frac{\mathrm{d}A_{BDJ}}{\mathrm{d}t} &= \zeta_{BT}\beta_{BT} I_T \frac{N_{BDJ} - A_{BDJ}}{N_{BDJ}} - m_{BJ} A_{BDJ} \\
\frac{\mathrm{d}S_{BDA}}{\mathrm{d}t} &= m_{BJ} S_{BDJ} + \alpha_B (N_{BDA} - S_{BDA} - I_{BDA}) - \zeta_{BT}\beta_{BT} \frac{S_{BDA}}{N_{BDA}} I_T - b_B S_{BDA} \\
\frac{\mathrm{d}I_{BDA}}{\mathrm{d}t} &= \zeta_{BT}\beta_{BT} \frac{S_{BDA}}{N_{BDA}} I_T - (\tau_B + b_B) I_{BDA} \\
\frac{\mathrm{d}A_{BUJ}}{\mathrm{d}t} &= \beta_{BT} I_T \frac{N_{BUJ} - A_{BUJ}}{N_{BUJ}} - m_{BJ} A_{BUJ} \\
\frac{\mathrm{d}S_{BUA}}{\mathrm{d}t} &= m_{BJ} S_{BUJ} + \alpha_B (N_{BDA} - S_{BDA} - I_{BDA}) - \beta_{BT} \frac{S_{BUA}}{N_{BUA}} I_T - b_B S_{BU} \\
\frac{\mathrm{d}I_{BUA}}{\mathrm{d}t} &= \beta_{BT} \frac{S_{BUA}}{N_{BUA}} I_T - (\tau_B + b_B) I_{BUA} \\
\frac{\mathrm{d}I_T}{\mathrm{d}t} &= (1-p) b_T I_T + \beta_{TB} (N_T - I_T) \frac{I_{BUA}}{N_{BUA}} + \zeta_{TB}\beta_{TB} (N_T - I_T) \frac{I_{BDA}}{N_{BDA}} \\
&\quad + \epsilon\beta_{TB} (N_T - I_T) \frac{A_{BDJ}}{N_{BDJ}} + \epsilon\zeta_{TB}\beta_{TB} (N_T - I_T) \frac{A_{BDJ}}{N_{BDJ}} - b_T I_T
\end{aligned} \tag{31}$$

This model is well posed within the feasible region

$$\begin{aligned}
\Gamma = \{ &(A_{BDJ}, S_{BDA}, I_{BDA}, A_{BUJ}, S_{BUA}, I_{BUA}, I_T) \in \mathbb{R}_+^7 \mid 0 \le A_{BDJ} \le N_{BDJ}, \\
&0 \le I_{BDA} + S_{BDA} \le N_{BDA},\ 0 \le I_{BUA} + S_{BUA} \le N_{BUA},\ 0 \le I_T \le N_T \}.
\end{aligned}$$

4.2 Calculation of Basic Reproduction Numbers

If $A_{BUJ} = A_{BDJ} = I_{BUA} = I_{BDA} = I_T = 0$, $S_{BUA} = N_{BUA} > 0$ and $S_{BDA} = N_{BDA} > 0$, the system is at equilibrium. This is the DFE, denoted by P_0. Taking the infected compartments $(A_{BDJ}, I_{BDA}, A_{BUJ}, I_{BUA}, I_T)$ and using the next generation matrix with

$$F_1 = \begin{bmatrix}
0 & 0 & 0 & 0 & \zeta_{BT}\beta_{BT} \\
0 & 0 & 0 & 0 & \zeta_{BT}\beta_{BT} \\
0 & 0 & 0 & 0 & \beta_{BT} \\
0 & 0 & 0 & 0 & \beta_{BT} \\
\epsilon\zeta_{TB}\beta_{TB}\frac{N_T}{N_{BDJ}} & \zeta_{TB}\beta_{TB}\frac{N_T}{N_{BDA}} & \epsilon\beta_{TB}\frac{N_T}{N_{BUJ}} & \beta_{TB}\frac{N_T}{N_{BUA}} & (1-p)b_T
\end{bmatrix}$$

and

$$V_1 = \begin{bmatrix} m_{BJ} & 0 & 0 & 0 & 0 \\ 0 & \tau_B + b_B & 0 & 0 & 0 \\ 0 & 0 & m_{BJ} & 0 & 0 \\ 0 & 0 & 0 & \tau_B + b_B & 0 \\ 0 & 0 & 0 & 0 & b_T \end{bmatrix}$$

give

$$\mathcal{R}_0 = \rho \left(\begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\zeta_{BT}\beta_{BT}}{b_T} \\ 0 & 0 & 0 & 0 & \frac{\zeta_{BT}\beta_{BT}}{b_T} \\ 0 & 0 & 0 & 0 & \frac{\beta_{BT}}{b_T} \\ 0 & 0 & 0 & 0 & \frac{\beta_{BT}}{b_T} \\ \epsilon\frac{\zeta_{TB}\beta_{TB}N_T}{m_{BJ}N_{BDJ}} & \frac{\zeta_{TB}\beta_{TB}N_T}{(\tau_B+b_B)N_{BDA}} & \epsilon\frac{\beta_{TB}N_T}{m_{BJ}N_{BUJ}} & \frac{\beta_{TB}N_T}{(\tau_B+b_B)N_{BUA}} & (1-p) \end{bmatrix} \right) \tag{32}$$

As for previous models, a different decomposition leads

$$\widetilde{\mathcal{R}}_0 = \sqrt{\frac{\beta_{TB}\beta_{BT}N_T}{pb_T}\left(\frac{\epsilon\zeta_{TB}\zeta_{BT}}{m_{BJ}N_{BDJ}} + \frac{\epsilon}{m_{BJ}N_{BUJ}} + \frac{\zeta_{TB}\zeta_{BT}}{N_{BDA}(\tau_B+b_B)} + \frac{1}{N_{BUA}(\tau_B+b_B)}\right)} \tag{33}$$

4.3 Equilibria and Stability of Model J+G

The following two theorems address the local and global stability of the DFE, with the first following immediately from van den Driessche and Watmough (2002).

Theorem 10 *For Model J+G, if $\mathcal{R}_0 < 1$, then the DFE P_0 is locally asymptotically stable, and if $\mathcal{R}_0 > 1$, then the DFE P_0 is unstable.*

Using a proof as for Theorem 7, but with $x = (A_{BDJ}, I_{BDA}, A_{BUJ}, I_{BUA}, I_T)^T$ denoting the infected classes and w^T being the left Perron eigenvector of $V_1^{-1}F_1$ with F_1, V_1 given in Sect. 4.2, the following result can be established.

Theorem 11 *For Model J+G, if $\mathcal{R}_0 \leq 1$, then the DFE P_0 is globally asymptotically stable; if $\mathcal{R}_0 > 1$, then Model J+G is uniformly persistent and there is at least one endemic equilibrium.*

4.4 Target Reproduction Numbers for Model J+G

This model was formulated mainly in order to see whether Bovine Babesiosis could be eradicated by solely vaccinating *B. taurus* cattle when both *B. taurus* and *B. indicus* adult and juvenile cattle are present. As stated before, according to Bock et al. (2004),

Hall et al. (1968) and Zintl et al. (2005), infected calves are asymptomatic and once recovered (we assume that this is the maturation rate) have natural immunity against reinfection. Since *B. taurus* cattle are more susceptible to infection by both *B. bovis* and *B. bigemina* than *B. indicus* cattle (Bock et al. 1999a; Jonsson et al. 2008), perhaps when calves are present it becomes feasible to only vaccinate *B. taurus* cattle in order to eradicate Bovine Babesiosis. Reproduction numbers are calculated to first target both the juvenile and adult *B. taurus*, and finally to target both the juvenile and adult *B. taurus*, along with the juvenile *B. indicus*. Reproduction numbers targeting adult cattle of both breeds and targeting both juvenile and adult cattle of both breeds are not calculated as the first would be equivalent to $\mathcal{T}_{23}$ for Model J, and the second would be equivalent to $\mathcal{T}_S$ for Model J. The type reproduction number associated with controlling the tick population is not calculated, as de Waal and Combrink (2006) state that tick control measures are much more intensive, extensive and temporary. However, note that the type reproduction number associated with controlling the tick population was determined for Model J in Sect. 2.4.2.

The first target reproduction number assesses the effort needed to eradicate Bovine Babesiosis by vaccinating both juvenile and adult *B. taurus* cattle. Denoting this type reproduction number as $\mathcal{T}_{S_1}$, where $S_1 = \{(3, 5), (4, 5)\}$, it is possible to calculate this explicitly, as in Shuai et al. (2013):

$$\mathcal{T}_{S_1} = \frac{N_{BDA}N_{BDJ}N_T\beta_{TB}\beta_{BT}[(\tau_B + b_B)N_{BUA}\epsilon + m_{BJ}N_{BUJ}]}{N_{BUJ}N_{BUA}[E - F]} \text{ if } E - F > 0 \quad (34)$$

where $E = (\tau_B + b_B)N_{BDA}N_{BDJ}pb_T m_{BJ}$ and $F = \zeta_{TB}\zeta_{BT}\beta_{TB}\beta_{BT}N_T[\epsilon N_{BDA}(\tau_B + b_B) + m_{BJ}N_{BDJ}]$. This means that in order to eradicate Bovine Babesiosis, there needs to be a vaccination of a proportion of $1 - \frac{1}{\mathcal{T}_{S_1}}$ of the total *B. taurus* population. If $E - F \leq 0$, then it is impossible to eradicate Bovine Babesiosis only through control of the total *B. taurus* population, and other methods of control need to be examined.

de Vos and Bock (2000) concluded that the benefit to cost ratio of vaccination against Bovine Babesiosis in Australia is positive for *B. taurus* cattle, and positive for *B. indicus* cattle only if just the juvenile *B. indicus* cattle are vaccinated, and the seroprevalence is <50 %. The next target reproduction number that is calculated has $S_2 = \{(1, 5), (3, 5), (4, 5)\}$: namely, targeting both juvenile and adult *B. taurus*, along with juvenile *B. indicus* cattle, to mirror the findings by de Vos and Bock (2000) and to determine the proportion of cattle to vaccinate to eradicate Bovine Babesiosis by this control. This target reproduction number, denoted $\mathcal{T}_{S_2}$ is

$$\mathcal{T}_{S_2} = \frac{\beta_{BT}\beta_{TB}N_T N_{BDA}[\epsilon(\tau_B + b_B)(\zeta_{TB}\zeta_{BT}N_{BUA}N_{BUJ} + N_{BDJ}N_{BUA}) + N_{BDJ}N_{BUJ}m_{BJ}]}{m_{BJ}N_{BUJ}N_{BUA}N_{BDJ}[(\tau_B + b_B)N_{BDA}pb_T - \zeta_{TB}\zeta_{BT}\beta_{TB}\beta_{BT}N_T]}$$
$$\text{if } (\tau_B + b_B)N_{BDA}pb_T - \zeta_{TB}\zeta_{BT}\beta_{TB}\beta_{BT}N_T > 0 \quad (35)$$

Therefore, in order to eradicate Bovine Babesiosis, there needs to be a vaccination of a proportion of $1 - \frac{1}{\mathcal{T}_{S_2}}$ of the total populations of juvenile and adult *B. taurus* cattle, and also of juvenile *B. indicus* cattle.

The above target reproduction numbers for Model J+G can be evaluated numerically and are shown in Table 6. We assume that the total bovine population equals the total

Table 6 Target reproduction numbers for numerical simulations of Model J+G

ϵ value	Target rep. number	Value	% of pop. needed to control
0.1	$\mathcal{T}_{S_1}$	8.42	88
0.1	$\mathcal{T}_{S_2}$	8.40	88
0.5	$\mathcal{T}_{S_1}$	13.90	93
0.5	$\mathcal{T}_{S_2}$	13.73	93
0.9	$\mathcal{T}_{S_1}$	19.50	95
0.9	$\mathcal{T}_{S_2}$	19.06	95

tick population, i.e., $N_T = N_{BUJ} + N_{BUA} + N_{BDJ} + N_{BDA}$, also $b_B N_{BUA} = m_{BJ} N_{BUJ}$ and $b_B N_{BDA} = m_{BJ} N_{BDJ}$. Moreover, we also assume that the respective juvenile and adult bovine populations are equal, i.e., $N_{BUJ} = N_{BDJ}$ and $N_{BUA} = N_{BDA}$. We set $\zeta_{TB} = 0.02$ and $\zeta_{BT} = 0.1$, with other parameters as in Table 2 and vary ϵ.

It is interesting to note from Table 6 that both $\mathcal{T}_{S_1}$ and $\mathcal{T}_{S_2}$ are very close for all three values of ϵ used. This could be attributed to the fact that we assumed that all populations were constant, and it follows that $N_{BDJ} = \frac{b_B}{m_{BJ}} N_{BDA}$. But since b_B is small, this means that N_{BDJ} is only a very small fraction of the total bovine population, and perhaps this explains why vaccination of the *B. indicus* juvenile does not greatly affect the target reproduction numbers. It is also important to remember that these target reproduction numbers were calculated assuming that there were equal numbers of *B. taurus* and *B. indicus* cattle, which may not be the case in every population.

5 Concluding Remarks

We first formulated an ODE model for Bovine Babesiosis caused by *B. bigemina* and *B. bovis* where the juvenile susceptible and asymptomatic cattle are separated from the adult susceptible, infectious, and recovered cattle. This generalizes the model of Aranda et al. (2012) in which there is no age separation for the cattle. We introduced the threshold value $\mathcal{R}_0$ and also proceeded to introduce another threshold value $\widetilde{\mathcal{R}}_0$, the only difference being that $\widetilde{\mathcal{R}}_0$ treats vertical transmission as transfer and not as a secondary infection. We showed that the disease-free equilibrium is globally asymptotically stable if $\mathcal{R}_0 \leq 1$ (i.e., Bovine babesiosis dies out), and if $\mathcal{R}_0 > 1$, then the disease-free equilibrium is unstable and there exists a unique endemic equilibrium. Using the Columbia data presented in Aranda et al. (2012), the endemic equilibrium for each numerical simulation was locally asymptotically stable. These numerical simulations illustrate a decline in $\mathcal{R}_0$ and in the number of infectious cattle for higher bovine birth rate, providing insight into possible means of lowering $\mathcal{R}_0$. Local sensitivity analysis was performed to determine the effect that small changes in the various parameters would have on $\mathcal{R}_0$. To further analyze this, $\mathcal{R}_0$ was graphed against each individual parameter as the parameter in question was varied within a predetermined feasible region, while all other parameters retained their value as assigned in Aranda et al. (2012). This illustrates that controlling just one parameter in order to drive $\mathcal{R}_0$ below one is most likely not feasible, and more suitable control methods would

probably try and control various parameters simultaneously. Then, target reproduction numbers were also calculated in order to determine the various effort required to eradicate Bovine Babesiosis by controlling different populations and their transmission of the disease. Numerics of these illustrate the possibility of eradicating Bovine Babesiosis by vaccinating 90 % of the adult cattle for some parameter values. We note that our results may not apply to Bovine Babesiosis caused by *B. divergens* since its vector is not a single host tick.

We next introduced an ODE model for Bovine Babesiosis where the cattle were not separated based on age, but rather based on their breed (i.e., *B. taurus* and *B. indicus*) with different susceptibility. We calculated the threshold values $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$ for this model and then proceeded to prove global asymptotic stability of the disease-free equilibrium for this model if $\mathcal{R}_0 \leq 1$, and of the endemic equilibrium if $\mathcal{R}_0 > 1$ and immunity is permanent in cattle. We concluded this section by calculating two target reproduction numbers, in order to evaluate various possible control methods for Bovine Babesiosis. This model also easily reduced to the model by Aranda et al. (2012), and we proved local asymptotic stability of the endemic equilibrium P^* (Aranda et al. 2012) showed P^* exists if $\mathcal{R}_0 > 1$).

The final section of the paper introduced an ODE model for Bovine Babesiosis that combined both previous models together. Both $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$ were determined for this model, with the ordering of $\mathcal{R}_0$ and $\widetilde{\mathcal{R}}_0$ as in Corollary 1. Asymptotic global stability of the disease-free equilibrium of this model is proved if $\mathcal{R}_0 \leq 1$, and disease persistence is established if $\mathcal{R}_0 > 1$. This combined model was formulated mainly to more accurately investigate possible control measures. Various target reproduction numbers were determined, and the values provided in Aranda et al. (2012) were used to numerically calculate these target reproduction numbers and the associated population percentage that would need to be controlled in order to eradicate Bovine Babesiosis. We expect, but have not proved, that for $\mathcal{R}_0 > 1$, this model has a unique endemic equilibrium that is globally asymptotically stable. Given accurate regional data, our target reproduction number formulas could be used to give realistic information for the control of Bovine Babesiosis in a particular region.

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