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A mathematical model for treatment of bovine brucellosis in cattle population

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Abstract. Brucellosis is an infectious bacterial zoonosis of public health and economic significance. In this paper, a mathematical model describing the propagation of bovine brucellosis within cattle population is formulated. Model analysis is carried out to obtain and establish the stability of the equilibrium points. A threshold parameter referred to as the basic reproduction number $\mathcal{R}_0$ is calculated and the conditions under which bovine brucellosis can be cleared in the cattle population are established. It is found out that when $\mathcal{R}_0 < 1$, the disease can be eliminated in the cattle population or persists when $\mathcal{R}_0 > 1$. Using Lyapunov function and Poincairé-Bendixson theory, we prove that the disease-free and endemic equilibrium, respectively are globally asymptotic stable. Numerical simulation reveals that control measures should aim at reducing the magnitude of the parameters for contact rate of infectious cattle with the susceptible and recovered cattle, and increasing treatment rate of infected cattle.

Keywords: Bovine brucellosis, endemic equilibrium, global stability, Lyapunov function, vertical transmission.
AMS Subject Classification: 34D23, 92D30, 92B05, 93A030.

1 Introduction

Brucellosis is an infectious and contagious zoonotic bacterial disease of animals and humans caused by a bacterium of genus *Brucella.* The four *Brucella* species responsible for the disease in decreasing order are: *B. meliten-*

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sis in small ruminants such as goats and sheep, *Brucella abortus* typically found in cattle, *Brucella suis* in swine, and *Brucella canis* in dogs [16]. It is endemic in low income countries of south sub-Saharan Africa and South Asia where weak control actions are still common. Most of the economic losses and high number of human infections are reported the regions of Africa, Asia, Syria, Iran, Iraq, and Saud Arabia. Bovine brucellosis is responsible for economic losses due to abortion, infertility and reproduction, weight loss, loss of calves, reduced meat and milk production [4], and also due to time lost by patients from normal daily activities.

Bovine brucellosis transmission to susceptible livestock occurs through direct contact with infected animal tissues, urine, and blood or with the environment that has been contaminated with discharges from infected cattle. It can also be vertically transmitted from infected mothers to their newborns.

Human brucellosis main sources include infected livestock and *Brucella* in the environment. Transmission of brucellosis to humans occurs through contact with infected animals or food of animal origin. Such contacts may be with secretions, placenta, calves and aborted fetuses. The disease can also be indirectly transmitted through consumption of contaminated milk or dairy products such as soft cheeses, yogurt and ice-creams prepared from unpasteurized milk that may be contaminated with the bacterial agent [7,8,14]. Other possible sources human brucellosis poor handling of manure from infected cattle and occupational exposure (for example veterinarians and abattoir workers) and inhalation of the causative agent. Human to human transmission of brucellosis is rare [22], though it may occur through contaminated blood transfusion.

The primary symptoms of bovine brucellosis in cattle are drop in the milk production of the cow and affected herd, infertility, abortion and weakened calves. The most signs and symptoms of human brucellosis are clinical manifestations that mimic other infectious diseases such as malaria, typhoid and rheumatic fever [5, 11]. They include fever, asthenia, mylgia, sweat, headache, chills, hepatomegaly, splenomelagy, fatigue and joint pain that can last for weeks to months.

Currently, no human vaccine for brucellosis exists and the occurrence of brucellosis in a region is directly linked to the status of animal brucellosis. It is therefore necessary to have interventions that may control zoonotic infection in animal reservoirs or prevent disease transmission from animals to humans that may offer more effective and economically viable approaches to disease management than those focusing on the human population alone [11, 12, 19].

Human brucellosis prevention and control strategies should aim reduction of animal-to-human transmission. They isolation of the infected animals, disinfection of contaminated areas, mass vaccination of livestock at risk, sustained removal of the infected animals, test and slaughter of infected animals/herds [12, 26] should be enforced. There is also need for public awareness campaigns for populations living in agro-pastrol communities to disseminate knowledge about brucellosis and associated risks such as consumption of unpasteurized dairy products, eating half-cooked meat, and use of protective measures in high-risk occupational groups such as livestock farmers, veterinarians, diary workers, slaughter house workers and laboratory personnel should be encouraged [18]. Treatment of infected cattle is an important method in controlling the spread of the brucellosis. The infected cattle successfully treated translate to recovered class.

Modeling is a valuable tool in planning and evaluating of intervention measures for disease control and prevention. Mathematical models based on transmission dynamics of animal diseases have long provided important insights to guide their prevention and control [24]. This is because they can help to figure out decisions that are of significant importance on the outcomes and provide comprehensive examinations that enter into decision making. They can be used to evaluate control strategies against the infection in order to determine the optimal control measures.

Mathematical models for the spread infectious diseases through direct contact have been reviewed in [13]. Mathematical models that incorporate both direct and indirect transmissions of brucellosis have been studied in [2, 15, 20, 23, 27]. Direct transmission dynamics of brucellosis among bison (*Bison bison*) herds and elk have been considered in Dobson and Meagher [6] using an SIR epidemic model and among bison alone in Abatih et al. [1].

Li et al. [15] proposed a model to evaluate control strategies for brucellosis and found out that a combination of animal vaccination, environment disinfection, and elimination of infected animals were necessary to ensure cost-effective control for brucellosis. In the model by Ainseba et al. [2] an SI epidemic model for ovine brucellosis incorporating direct and indirect transmissions is considered in which the direct transmission follows a bilinear incidence rate and assumes no disease-related mortality and control interventions.

In this paper, a SIR epidemic model for bovine brucellosis in cattle population is presented. The successfully treated infected cattle translate to the recovered class. When the treatment wanes off, the recovered cattle may become infected again through interaction with the infectious cattle. Both the susceptible and recovered cattle acquire infection through direct

contact with the infectious cattle. The infection may also be transmitted from infectious mothers to their newborns. The transmission of brucellosis via direct follows standard incidence law.

This paper is organized as follows: In Section 2, we formulate the model based on the assumptions, definitions of the variables and parameters. In Section 3, model analysis is carried out. The equilibrium points of the model and their stability are established and numerical simulation is done as well. The discussion of results and conclusion are done in Section 4.

2 Mathematical model

2.1 Formulation of the Model

A mathematical model to study transmission dynamics of bovine brucellosis in the cattle population is formulated. The cattle population is divided into three epidemiological classes $S(t)$, $I(t)$ and $R(t)$ that denote the number of the cattle population that susceptible, infectious, and recovered at time t, respectively. The total cattle population size at t is given by $N(t) = S(t) + I(t) + R(t)$. Infectious cattle transmit bacterium to susceptible and recovered cattle through either direct contact or vertically to their newborns. Direct transmission of bovine brucellosis to susceptible and recovered cattle are given by the standard incidence rates $(\beta SI)/N$ and $(\beta SR)/N$, respectively. Recruitment into the cattle population is only through birth at a constant rate λ. Vertical transmission of brucellosis is indicated by the inflow of new infective cattle proportional to the number of infective individuals already in the population at a rate $\epsilon\lambda I$. Recovered cattle give birth to susceptible calves. Infected cattle are treated at a rate τ and have a disease-induced death rate α. The model assumes the disease independent mortality that is a function of the population density and has the form $(\mu + (bN)/K)$ [17] and is shared proportionally by all subpopulations.

The following assumptions are made in the formulation of the model:

(i) Recruitment into the population is only via birth.

(ii) There is homogeneous mixing in the cattle population.

(iii) Population birth rate and natural mortality rate are constant.

(iv) Treated cattle give birth to susceptible calves.

(v) Population birth rate is greater than the natural mortality rate.

(vi) Cattle that show symptoms and test positive to *brucellosis* are treated.

(vii) Grazing space is not so large and as such the effect of congestion contributes on death rate of the cattle.

(viii) The infected animals can be treated to remain carriers but after a period, they are re-infected as treatment wanes off.

(ix) Treatment rate is proportional to the number of infective cattle.

(x) All the parameters are positive constants.

(xi) $\beta > \alpha$.

Parameters used in the model are defined as follows:

α disease-related death rate of the cattle population.

β average contact rate / transmission rate.

ϵ proportion of newborns (from infected mothers) that are infected.

μ natural death rate of the cattle population.

λ per natural birth rate of the cattle population.

b intrinsic growth rate of the cattle population.

K carrying capacity of the environment.

τ treatment rate of the disease.

σ rate at which treatment wanes off.

Based on the assumptions and definitions of variables and parameters, our SIR model can be written as a set of three coupled system of differential equations as follows:

$$\begin{aligned} \frac{dS}{dt} &= \lambda S + \lambda R + (1-\epsilon)\lambda I - \beta S\frac{I}{N} - \left(\mu + \frac{bN}{K}\right) S, \\ \frac{dI}{dt} &= \beta S\frac{I}{N} + \epsilon\lambda I - \left(\mu + \frac{bN}{K}\right) I - (\tau+\alpha)I + \sigma\beta\frac{RI}{N}, \\ \frac{dR}{dt} &= \tau I - \left(\mu + \frac{bN}{K}\right) R - \sigma\beta\frac{RI}{N}, \end{aligned} \tag{1}$$

together with $S + I + R = N$ and

$$\frac{dN}{dt} = bN\left(1 - \frac{N}{K}\right) - \alpha I.$$

This is a logistic growth for the cattle population in absence of brucellosis. N is the total number of animals, K is the carrying capacity of the environment at equilibrium and b is the intrinsic growth rate of the cattle population. Therefore, the feasible region solution set of system (1) enters the region $\Omega = \{(S, I, R) \in \mathbb{R}^3_+ : N \leq K\}$. In this case, whenever $N > K$, $\frac{dN}{dt} < 0$. This means that the cattle population reduces asymptotically to the carrying capacity, K. On the other hand, whenever $N < K$, every solution with the initial condition in $\mathbb{R}^3_+$ remains in that region for all $t > 0$.

Thus, the region Ω is positively invariant and the model is well posed and biologically meaningful.

Defining $s(t) = S(t)/N(t), i(t) = I(t)/N(t)$ and $r(t) = R(t)/N(t)$ as the proportions for the classes $S(t), I(t)$ and $R(t)$, respectively, where $s(t) + i(t) + r(t) = 1$. Then differentiating with respect to t, system (1) gives in terms of proportions the following system equations.

$$\begin{aligned}
\frac{ds}{dt} &= \lambda r(t) + (1-\epsilon)\lambda i(t) + (\alpha - \beta) i(t) s(t), \\
\frac{di}{dt} &= \beta s(t) i(t) - [(1-\epsilon)\lambda + \tau + \alpha] i(t) + \sigma\beta r(t) i(t) + \alpha i^2(t), \qquad (2) \\
\frac{dr}{dt} &= \tau i(t) - \lambda r(t) + (\alpha - \sigma\beta) r(t) i(t),
\end{aligned}$$

together with $s(t) + i(t) + r(t) = 1$. Using the simplification $r(t) = 1 - s(t) - i(t)$, the following system of equations is obtained.

$$\begin{aligned}
\frac{ds}{dt} &= \lambda(1 - s(t) - i(t)) + (1-\epsilon)\lambda i(t) + (\alpha - \beta) i(t) s(t), \qquad (3) \\
\frac{di}{dt} &= \beta s(t) i(t) - [(1-\epsilon)\lambda + \tau + \alpha] i(t) + \sigma\beta(1 - s(t) - i(t)) i(t) + \alpha i^2(t).
\end{aligned}$$

It be can verified that the region

$$T = \{(s(t), i(t)) \in \mathbb{R}_+ : 0 \le s(t), 0 \le i, s(t) + i(t) \le 1\},$$

is positively invariant with respect to system (3), where $\mathbb{R}^2_+$ denotes the non-negative cone of $\mathbb{R}^2$ including its lower dimensional faces. We denote the boundary and the interior of T by ∂T and $\overset{\circ}{T}$ respectively.

3 Analysis of the model

The equilibrium points are obtained by setting the right hand side of system (3) to equal zero as follows:

$$\lambda(1 - s(t) - i(t)) + (1-\epsilon)\lambda i(t) + (\alpha - \beta) i(t) s(t) = 0, \quad (4)$$

$$\beta s(t) i(t) - [(1-\epsilon)\lambda + \tau + \alpha] i(t) + \sigma\beta(1 - s(t) - i(t)) i(t) + \alpha i^2(t) = 0. \quad (5)$$

From Eq. (5), we get $i(t) = 0$, or

$$\begin{aligned}
\beta s(t) - [(1-\epsilon)\lambda + \tau + \alpha] + \sigma\beta(1 - s(t) - i(t)) + \alpha i(t) &= 0. \\
\beta s(t) - [(1-\epsilon)\lambda + \tau + \alpha] + \sigma\beta(1 - s(t) - i(t)) + \alpha i(t) &= 0, \\
\beta s(t) - [(1-\epsilon)\lambda + \tau + \alpha] + \sigma\beta - \sigma\beta s(t) - \sigma\beta i(t) + \alpha i(t) &= 0, \\
(\beta - \sigma\beta) s(t) + (\alpha - \sigma\beta) i(t) + \sigma\beta - [(1-\epsilon)\lambda + \tau + \alpha] &= 0. \qquad (6)
\end{aligned}$$

Dividing $(\beta - \sigma\beta)$ through Eq. (6) gives

$$s(t) + \frac{(\alpha - \sigma\beta)i(t)}{\beta - \sigma\beta} - \left[\frac{(1-\epsilon)\lambda + \tau + \alpha - \sigma\beta}{\beta - \sigma\beta}\right] = 0. \tag{7}$$

This can be written as $s(t) + \rho i(t) - \phi = 0$, where

$$\rho = \frac{\alpha - \sigma\beta}{\beta - \sigma\beta} \text{ and } \phi = \frac{(1-\epsilon)\lambda + \tau + \alpha - \sigma\beta}{\beta - \sigma\beta}.$$

From $s(t) + \rho i(t) - \phi = 0$, we obtain, $s(t) = \phi - \rho i(t)$.

In the absence of the disease, $i(t) = 0$, and substituting $i(t) = 0$ into Eq. (4) gives $E_0(1, 0)$. This represents the state in which there is no infection in the population and is known as disease-free equilibrium.

The endemic equilibrium is obtained by substituting the expression for $s(t)$ into Eq. (4) to give

$$\begin{aligned}
\lambda(1 - i(t)) - \lambda[\phi - \rho i(t)] + (1-\epsilon)\lambda i(t) + (\alpha - \beta)i(t)[\phi - \rho i(t)] = 0,\\
\lambda - \lambda i(t) - \lambda\phi + \lambda\rho i(t) + (1-\epsilon)\lambda i(t) + \phi(\alpha - \beta)i(t) - \rho(\alpha - \beta)i^2(t) = 0,\\
(\lambda - \lambda\phi) + [\lambda\rho - \lambda + (1-\epsilon)\lambda + \phi(\alpha - \beta)]i(t) - \rho(\alpha - \beta)i^2(t) = 0,\\
\lambda(1 - \phi) + [\lambda(\rho - \epsilon) + \phi(\alpha - \beta)]i(t) - \rho(\alpha - \beta)i^2(t) = 0.
\end{aligned}$$

Thus,

$$\rho(\beta - \alpha)i^2(t) - [\lambda(\epsilon - \rho) + \phi(\beta - \alpha)]i(t) + \lambda(1 - \phi) = 0.$$

When the constant term in the characteristic equation is negative, then $\beta < (1-\epsilon)\lambda + \tau + \alpha$, and we get one negative root and one positive root. The positive root is given by

$$i^*(t) = \frac{\lambda(\epsilon - \rho) + (\beta - \alpha)\phi + \sqrt{[(\lambda(\epsilon - \rho) + \phi(\beta - \alpha))^2 - 4\rho\lambda(\beta - \alpha)(1 - \phi)]}}{2\rho(\beta - \alpha)}.$$

The negative root is considered to be biologically meaningless. Hence, $i^*(t) = \frac{\psi}{2\rho(\beta-\alpha)}$, where

$$\psi = \lambda(\epsilon - \rho) + (\beta - \alpha)\phi + \sqrt{[(\lambda(\epsilon - \rho) + \phi(\beta - \alpha))^2 - 4\rho\lambda(\beta - \alpha)(1 - \phi)]}.$$

From $s^*(t) = \phi - \rho i^*(t)$, we obtain

$$s^*(t) = \phi - \rho i^*(t) = \phi - \frac{\rho\psi}{2\rho(\beta - \alpha)} = \frac{2\phi(\beta - \alpha) - \psi}{2(\beta - \alpha)}. \tag{8}$$

Thus, we obtain an endemic equilibrium $E_1(s^*(t), i^*(t))$ given by

$$E_1\left(\frac{2\phi(\beta - \alpha) - \psi}{2(\beta - \alpha)}, \frac{\psi}{2\rho(\beta - \alpha)}\right).$$

3.1 Local and global stability of the disease-free equilibrium E_0

We discuss the local stability of the disease-free equilibrium by examining the linearized form of system (3) at the equilibrium $E_0(1,0)$. The Jacobian matrix of system (3) is given by

$$\mathbf{J} = \begin{bmatrix} -\lambda + (\alpha-\beta)i^*(t) & -\epsilon\lambda + (\alpha-\beta)s^*(t) \\ (\beta-\sigma\beta)i^*(t) & (\beta-\sigma\beta)s^*(t) + 2(\alpha-\sigma\beta)i^*(t) - \eta \end{bmatrix}, \tag{9}$$

where, $\eta = [(1-\epsilon)\lambda + \tau + \alpha - \sigma\beta]$. The Jacobian matrix (9) evaluated at the disease-free equilibrium $E_0(1,0)$ gives

$$J_{E_0} = \begin{bmatrix} -\lambda & (\alpha-\beta) - \epsilon\lambda \\ 0 & \beta(1-\sigma) - [(1-\epsilon)\lambda + \tau + \alpha - \sigma\beta] \end{bmatrix}. \tag{10}$$

The trace and determinant of the Jacobian matrix (10) at the disease free equilibrium are given by

$$\begin{aligned} tr(J_{E_0}) &= -\lambda + \beta(1-\sigma) - [(1-\epsilon)\lambda + \tau + \alpha - \sigma\beta] \\ &= -[\lambda + (1-\epsilon)\lambda + \tau + \alpha] + \beta = -\lambda + \beta - [(1-\epsilon)\lambda + \tau + \alpha], \end{aligned}$$

and

$$\begin{aligned} \det(J_{E_0}) &= -\lambda[\beta - \sigma\beta - (1-\epsilon)\lambda - (\tau+\alpha) + \sigma\beta] \\ &= -\lambda[\beta - [(1-\epsilon)\lambda + \tau + \alpha]]. \end{aligned}$$

It can be noted that $tr(J_{E_0}) < 0$ and $\det(J_{E_0}) > 0$ if $\beta < (1-\epsilon)\lambda + \tau + \alpha$, that is, $\frac{\beta}{(1-\epsilon)\lambda+\tau+\alpha} < 1$. Defining $\mathcal{R}_0 = \frac{\beta}{(1-\epsilon)\lambda+\tau+\alpha}$, it is easy to see that $tr(J_{E_0}) < 0$ and $\det(J_{E_0}) > 0$ if $\mathcal{R}_0 < 1$. Therefore, we have established Lemma 1 below.

Lemma 1. *The disease free equilibrium $E_0(1,0)$ is locally stable if $R_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

$\mathcal{R}_0$ is called the basic reproduction number [3], defined as the number of secondary infectious cases produced by one primary case introduced into an entirely susceptible population at the disease-free equilibrium.

Theorem 1. *The disease-free equilibrium $E_0 = (1,0)$ is globally asymptotically stable if $\mathcal{R}_0 \leq 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. Consider the Lyapunov function defined by $L(z)$ such that: $L(t) = i(t)$. Its derivative along solutions to the system (3) is

$$\begin{aligned} L' &= i'(t) = \beta s(t)i(t) - ((1-\epsilon)\lambda + \tau + \alpha)i(t) + \sigma\beta r(t)i(t) + \alpha i^2(t) \\ &= i(t)[\beta s(t) - ((1-\epsilon)\lambda + \tau + \alpha) + \sigma\beta(1 - i(t) - s(t)) + \alpha i(t)] \\ &= i(t)[(1-\epsilon)\lambda + \tau + \alpha)\left(\frac{\beta}{(1-\epsilon)\lambda + \tau + \alpha}s(t) - 1\right) \\ &\quad +\sigma\beta(1 - i(t) - s(t)) + \alpha i(t)] \\ &= i(t)[(1-\epsilon)\lambda + \tau + \alpha)\,(\mathcal{R}_0 s(t) - 1) + \sigma\beta(1 - i(t) - s(t)) + \alpha i(t)] \\ &\leq i(t)[(1-\epsilon)\lambda + \tau + \alpha)\,(\mathcal{R}_0 - 1)] \leq 0, \end{aligned}$$

if $\mathcal{R}_0 \leq 1$. We have established that $L' \leq 0$, if $\mathcal{R}_0 \leq 1$ and the equality, $L' = 0$ holds if $\mathcal{R}_0 = 1$ and $i(t) = 0$. If $\mathcal{R}_0 > 1$, then $L' > 0$ when $s(t)$ is sufficiently close to 1 except when $i(t) = 0$. From the Lyapunov-LaSalle Theorem [9], it follows that all the paths in T approach the largest positive invariant subset of the set where $L' = 0$ is $\{(s(t), i(t)) \in T \mid L' = 0\}$. On the boundary of T where $i(t) = 0$ (s(t)-axis), $s'(t) = \lambda(1 - s(t)$ so that $s(t) = (1 + e^{-\lambda t}) \to 1$ as $t \to +\infty$. Thus all solution paths in T will approach the disease-free equilibrium E_0. Therefore, the disease-free equilibrium E_0 is globally asymptotically and this completes the proof of Theorem 1. □

3.2 Local and global stability of the endemic equilibrium E_1

The Jacobian matrix evaluated at the endemic equilibrium $E_1\,(s^*(t), i^*(t))$ is given by

$$J_{E_1} = \begin{bmatrix} -\lambda + (\alpha - \beta)i^*(t) & -\epsilon\lambda + (\alpha - \beta)s^*(t) \\ \\ (\beta - \sigma\beta)i^*(t) & (\beta - \sigma\beta)s^*(t) + 2(\alpha - \sigma\beta)i^*(t) - \eta \end{bmatrix},$$

where $\eta = [(1-\epsilon)\lambda + \tau + \alpha - \sigma\beta]$. From Eq. (6) and $(\beta - \sigma\beta)s^*(t) + (\alpha - \sigma\beta)i^*(t) + \sigma\beta - [(1-\epsilon)\lambda + \tau + \alpha] = 0$, we obtain the Jacobian matrix given by

$$J_{E_1} = \begin{bmatrix} -\lambda + (\alpha - \beta)i^*(t) & -\epsilon\lambda + (\alpha - \beta)s^*(t) \\ \beta(1 - \sigma)i^*(t) & (\alpha - \sigma\beta)i^*(t) \end{bmatrix}, \tag{11}$$

Let

$$J_{11} = -\lambda + (\alpha-\beta)i^*(t) = -\lambda + (\alpha-\beta)\frac{-\psi}{2\rho(\alpha-\beta)} = \frac{-(\psi+2\rho\lambda)}{2\rho}.$$

$$J_{12} = -\epsilon\lambda + (\alpha-\beta)s^*(t) = -\epsilon\lambda + (\alpha-\beta)\frac{[-2\phi(\alpha-\beta)-\psi]}{-2(\alpha-\beta)}$$

$$= \frac{1}{2}[2\phi(\alpha-\beta)+\psi-2\epsilon\lambda] = \frac{1}{2}\psi - \epsilon\lambda - \phi(\beta-\alpha).$$

$$J_{21} = \beta(1-\sigma)i^*(t) = (1-\sigma)\beta\frac{-\psi}{2\rho(\alpha-\beta)} = \frac{\beta\psi(1-\sigma)}{2\rho(\beta-\alpha)}.$$

$$J_{22} = (\alpha-\sigma\beta)i^*(t) = (\alpha-\sigma\beta)\frac{\psi}{2\rho(\beta-\alpha)}.$$

The Jacobian matrix (11) can thus be written as

$$J_{E_1} = \begin{bmatrix} J_{11} & J_{12} \\ \\ J_{21} & J_{22} \end{bmatrix},$$

with

$$\begin{aligned} tr(J_{E_1}) &= J_{11} + J_{22} = \frac{-\psi-2\rho\lambda}{2\rho} + (\alpha-\sigma\beta)\frac{\psi}{2\rho(\beta-\alpha)} \\ &= -\lambda - \frac{\psi}{2\rho}\left[(\beta-\alpha)+(\sigma\beta-\alpha)\right]. \\ \det(J_{E_1}) &= J_{11}J_{22} - J_{12}J_{21}, \\ &= \frac{-(\psi+2\rho\lambda)}{2\rho}(\alpha-\sigma\beta)\frac{\psi}{2\rho(\beta-\alpha)} - \frac{\beta\psi(1-\sigma)}{2\rho(\beta-\alpha)}\left[\frac{1}{2}\psi - \epsilon\lambda - \phi(\beta-\alpha)\right] \\ &= \frac{(\psi+2\rho\lambda)}{2\rho}(\sigma\beta-\alpha)\frac{\psi}{2\rho(\beta-\alpha)} + \frac{\beta\psi(1-\sigma)}{4\rho(\beta-\alpha)}\left[2(\epsilon\lambda+\phi(\beta-\alpha))-\psi\right]. \end{aligned}$$

Since $\beta > \alpha$, $\mathrm{tr}(J_{E_1}) < 0$ and $\det(J_{E_1}) > 0$ and the endemic equilibrium E_1 is a stable node.

Theorem 2. *If $\mathcal{R}_0 > 1$, then the endemic equilibrium $E_1(s^*(t), i^*)(t)$ is globally asymptotically stable in the set $T = \{s(t), i(t) : s(t) > 0, i(t) > 0, s(t) + i(t) \leq 1\}$.*

Proof. Define the function $\phi(s,i) = i^{-1}$ for $(s(t), i(t)) \in T$, it follows that there can not be limit cycles. It is noted that,

$$\frac{\partial}{\partial s}\left(\frac{s'}{i}\right) + \frac{\partial}{\partial i}\left(\frac{i'}{i}\right) = \frac{-\lambda}{i} + \alpha - \beta + \alpha - \sigma\beta < 0.$$

Table 1: Parameter estimates for the model of brucellosis.

Symbol	Biological meaning	Value/day	Ref.
λ	Per-capita birth rate	0.00075	[27]
μ	Per-capita death rate	0.00062	[27]
α	Disease-related death rate	0.00001	[27]
ϵ	Proportion of infected newborns	0.00165	[2]
b	Intrinsic growth rate	0.00013	[27]
σ	Rate at which treatment wanes off	0.00274	[27]
K	Carrying capacity	100/sq mi	Assumed
β	Contact rate	0.00003-0.00009	Assumed
τ	Treatment rate	0.001-0.005	Assumed

The conditions of the Bendixson-Dulac criterion are satisfied and system (3) has no limit cycles in T. Thus by the Poincaré-Bendixson criterion, the endemic equilibrium $E_1(s^*(t), i^*(t))$ is globally asymptotically stable in T. □

The global stability of the endemic equilibrium E_1 is established by using Bendixson-Dulac criterion [10] to show that system (3) has no nontrivial orbit in T.

3.3 Numerical simulation

Numerical simulations are done using MATLAB computer software program. Parameter values given in Table 1 are obtained from epidemiological data in the literature while other parameters are allowed to vary within the possible intervals. Some parameters vary from country to country, and some are influenced by demographics, for instance, natural death rate, transmission/contact parameters, carrying capacity and treatment rate.

3.4 Variation of cattle population against time at various values of contact rate β

The effect of contact rate on the dynamics of the bovine brucellosis is studied for the following values of contact rates $\beta = 0.00003, 0.00006$ and 0.00009. It is observed that there is a drastic decrease in the number of susceptible cattle when the contact rate is higher, and for a lower contact rate, the susceptible cattle population reduces at slowly as shown in Figure 1a. For the infected cattle population as shown in Figure 1b, higher contact

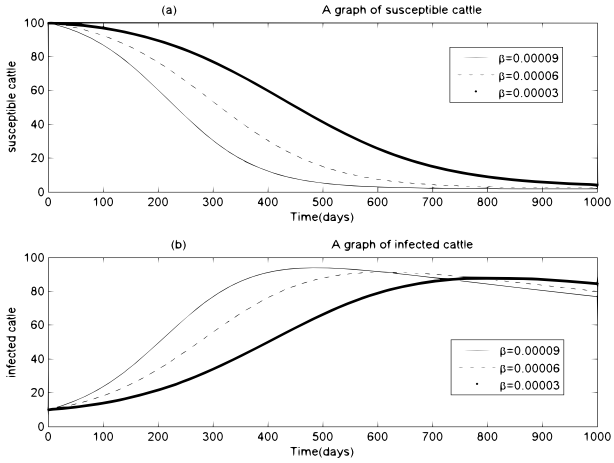

Figure 1: Changes in cattle population with time at various values of contact rate β.

rate results in a higher number of infected cattle than at a lower contact rate. The decline in the infected cattle population after some time is due to treatment of the infected cattle. This indicates that contact rate has a great effect on the transmission of the bovine brucellosis showing that for lower contact rate, the prevalence of bovine brucellosis decreases. This confirms the analytical results previously obtained on the basic reproduction number $\mathcal{R}_0$, that the contact rate should be as low as possible if $\mathcal{R}_0$ is to be less than one for the disease to die out.

3.5 Effect of varying treatment rate τ on different epidemiological classes

The role of treatment on the dynamics bovine brucellosis is carried out at different rates to show its effect on the different epidemiological classes. It is observed in Figure 2 that when treatment rate is low, the susceptible cattle population drops over time as the infected cattle population increases. It later decreases because of continued treatment of infected cattle population that recover increases. The decrease in the susceptible cattle population is because the recovered cattle population due to treatment never become susceptible again. The treatment temporally stops the disease symptoms as the treated cattle remain in the recovered class. The treated cattle become infected when they come into infectious cattle.

In Figure 3, it is observed that when the treatment rate is increase to a higher value, the infected cattle population drops rapidly. Thus, high

treatment rate reduces the infected cattle population whereas low treatment rate leads to an increase in the infected cattle population.

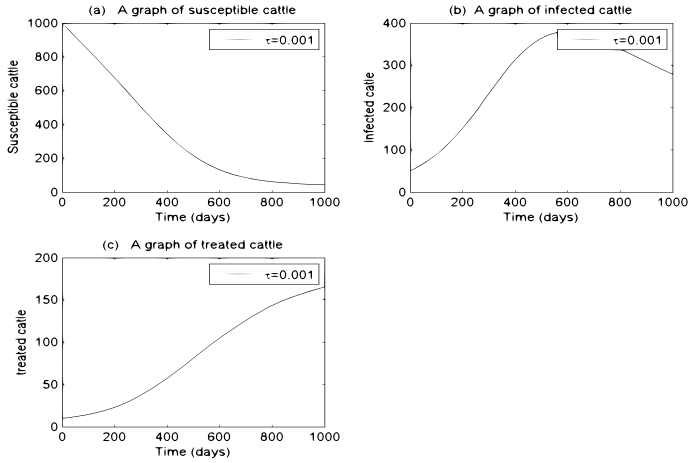

Figure 2: Change in cattle population with time at treatment rate $\tau = 0.001$.

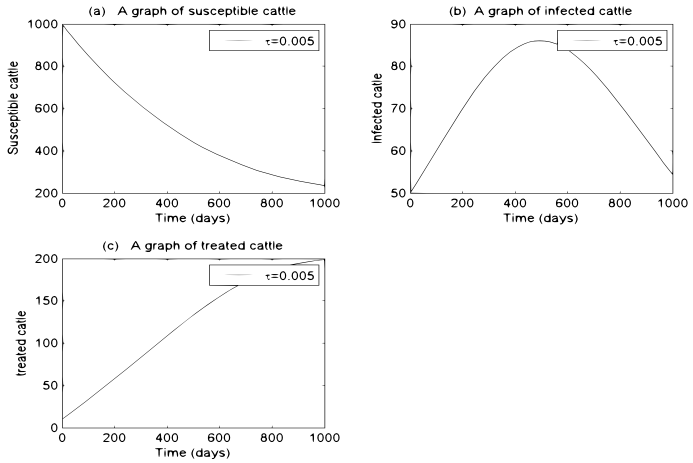

Figure 3: Change in cattle population with time at treatment rate $\tau = 0.005$.

4 Discussion and conclusion

Brucellosis is a contagious zoonotic disease that is transmitted to humans through direct or indirect contact with infectious animals and consumption

of contaminated animal products. Brucellosis in the cattle is transmitted either horizontally and vertically.

In this paper, an SIR epidemic model is proposed to study the effect of treatment of infected cattle on the transmission dynamics of brucellosis. The model was analyzed for equilibrium points and their stability. The basic reproduction number, $\mathcal{R}_0$ that describes the dynamics of the disease was obtained. It was established that for $\mathcal{R}_0 < 1$, the disease free equilibrium E_0 is locally asymptotically stable and the disease dies out. But when $\mathcal{R}_0 > 1$, the disease free equilibrium becomes unstable and the disease persists. The theory of Lyapunov function and Poincare were used to establish the global stability of the disease-free and endemic equilibrium, respectively. It was revealed that if $\mathcal{R}_0 \leq 1$, then the disease-free equilibrium E_0 is globally asymptotically stable. If $\mathcal{R}_0 > 1$, a unique endemic equilibrium E_1 is globally asymptotically stable in the interior of the feasible region and the disease will persist at the endemic equilibrium if it is initially present. The global stability of the endemic equilibrium E_1 was proved using the Poincaré-Bendixson theorem for 2-dimensional monotone systems.

The basic reproduction number $\mathcal{R}_0$ is directly proportional to the parameter β and inversely proportional to the parameters α, τ and ϵ. Thus, in order to reduce the basic reproduction number $\mathcal{R}_0$ below one, there is need to focus on reduction of the contact rate β, proportion of newborns (from infected mothers) that are infected ϵ and increase on the removal rate τ of the infectious cattle. This can be achieved by isolating any cow that aborts and then treating it and also ensuring that delivering animals are attended to by veterinary workers. Thus, there is need to conduct massive awareness campaigns in order to sensitize farmers on the significance of testing any animal that aborts and getting treatment from veterinary workers.

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