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Mathematical Modelling of Bovine Tuberculosis Transmission Dynamics: Role of Combination of Control Measure

Sylas Oswald[1*], Theresia Crispin Marijani[1], Goodluck Mika Mlay[1], Winifrida Benedict Kidima[2]

[1]*Department of Mathematics, University of Dar-es-Salaam, P.O.Box 35062, Dar-es-salaam, Tanzania*

[2]*Department of Zoology and Wildlife Conservation, University of Dar-es-Salaam, P.O.Box 35064, Dar-es-salaam, Tanzania*

ABSTRACT

Bovine Tuberculosis (BTB) is a contagious and potentially life-threatening infectious disease of cattle caused by *Mycobacterium bovis* (*M. bovis*) that lives in various environment depending on prevailing weather conditions (Temperature and humidity). Globally, the BTB is one of the diseases with utmost public health challenges which places economic and financial burdens on the society. To curb the disease, a mathematical model for the spread of BTB among human and cattle populations including preventive measures was formulated. The model analysis focused on the existence of disease-free and endemic equilibria points and their stabilities. The study used normalized forward sensitivity index method to analyze the model, and results revealed that the most sensitive parameter is the contaminated environment or inter-cattle transmission. Moreover, the study determined the best way of curbing the spread of BTB disease in the human and cattle populations using three interventions: public health education campaign, treatment and vaccination. Subsequently, the study performed numerical simulations whose results affirm the positive effects of a combination of control measures on the magnitude of infections among human and cattle population.

Keywords: *Mycobacterium bovis*; Bovine Tuberculosis (BTB), Spectral radius; Most sensitive; Bifurcation coefficients; Stability analysis; Runge-Kutta fourth (RK4) order

INTRODUCTION

Bovine Tuberculosis (BTB) is a contagious and life-threatening infectious disease of cattle. The bacterium *Mycobacterium bovis* (*M. bovis*) that causes disease, can also afflict many other mammals such as humans, goats, pigs, cats and dogs. BTB remains a significant challenge in both human and animal health worldwide. It poses a substantial economic burden, particularly in regions heavily reliant on live-stock agriculture.

In 1993, the disease was declared a global state of emergency. Despite our knowledge of how to effectively prevent and cure BTB through half a century of development and progress, more than 1.6 million people have still died from it. In 2014, BTB claimed the lives of 1.5 million people, including 890,000 men, 480,000 women, and 140,000 children. India, Indonesia, and China had the largest number of cases, accounting for 23%, 10%, and 10% of the global total, respectively. Traditional control measures, such as culling infected animals

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Corresponding author: Sylas Oswald, Department of Mathematics, University of Dar-es-Salaam, P.O.Box 35062, Dar-es-salaam, Tanzania; E-mail: sylasossy@gmail.com

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and movement restrictions, have had limited success in curbing the transmission of BTB. To address this complex issue, researchers have increasingly turned to mathematical modelling to gain insights into the dynamics of BTB transmission and assess the potential impact of various control strategies. This study delves into the mathematical modelling of BTB transmission dynamics, with a novel focus on the synergistic role of a combination of vaccination, treatment, and education campaigns as control measures [1].

The transmission route entails direct, close contact through inhaling sputum droplets (thick mucus produced in the lungs) contaminated with *M. bovis* bacteria exhaled by infected animals. On the other hand, indirect transmission of the disease can occur when one comes into contact with an infected animal or ingests material heavily contaminated with *M. bovis*, such as sputum, pus, urine, feces, and other excrement of infected animals. There are inherent practical difficulties associated with detecting *M. bovis* in environmental samples. However, the bacteria have been traced in diverse sources, such as soil, excrement, hay, and pasture.

Bovine Tuberculosis: A Global Challenge

Bovine tuberculosis poses a substantial threat to both animal and human populations. In cattle, it results in reduced milk and meat production, increased slaughterhouse condemnation rates, and trade restrictions, contributing significantly to economic losses in affected regions. In humans, zoonotic transmission of BTB remains a concern, particularly in areas where close contact with infected cattle occurs. Given its impact on both public health and the livestock industry, BTB demands innovative and effective control measures [2-5].

Mathematical Modelling in Infectious Disease Control

Mathematical modelling has proven to be a powerful tool in understanding and predicting the dynamics of infectious diseases. In the context of BTB, previous studies have employed compartmental models, network models, and agent-based models to simulate disease spread and evaluate control strategies. These models have provided valuable insights into the role of factors such as cattle demographics, wildlife reservoirs, and testing protocols in BTB transmission dynamics.

Individual Control Measures

Many studies have assessed the efficacy of individual control measures, such as vaccination and antimi-crobial treatment, in reducing BTB prevalence. Vaccination with Bacillus Calmette-Gu´erin (BCG) has shown promise in reducing the severity of BTB in cattle. Antimicrobial treatment can help clear infections but is often logistically challenging due to the lengthy treatment duration and concerns about antibiotic resistance.

MATERIALS AND METHODS

Education Campaigns as a Control Measure

Education campaigns aimed at promoting best practices in cattle management, biosecurity measures, and early disease detection have been recognized as crucial components of BTB control. These campaigns empower farmers and stakeholders with knowledge to reduce the risk of BTB transmission within and between herds.

In Africa and Europe, bovine tuberculosis is a serious threat to the economy as well as human and animal health. For example, presented a deterministic model for examining the impact of separating two large human populations based on the probability of coming into contact with cattle for the prevention of bovine tuberculosis. The model encompasses three types of incidents: Those occurring between cattle, between humans, and between cattle and humans. The results of the study, based on the stability of the disease-free equilibrium and sensitivity analysis of the model's parameters, showed that quarantine measures for cattle and the parameter related to medical masks significantly contributed to reducing the basic reproduction number and, consequently, decreasing the disease transmission rate. The effects of vaccines on cattle, treatments, public health education campaigns for humans, and communal water source sharing, in relation to disease transmission, were not fully considered.

The study by also presented a dynamic mathematical model for the transmission of bovine BTB. The results from the model simulations supported vaccine administration as the best strategy for reducing BTB infections. Furthermore, they suggested that coupling a public health education campaign with treatment for humans could maintain R_0 below one, significantly reducing the spread of bovine tuberculosis. However, their study did not address the impact of treatment as a measure for controlling the transmission dynamics of bovine tuberculosis.

A study by revealed that the use of slaughter and quarantine methods to reduce the number of infectious cattle was found to be the most important control measures to minimize the prevalence of BTB disease.

The study done by revealed that a relatively high proportion of BTB infections in the Ngorongoro district are due to the husbandry practices of a semi-nomadic system where the Maasai search for water and pasture during the dry season. It is recommended that implementing an education campaign for cattle owners would be the best solution.

In developing countries such as Tanzania, Bovine Tuberculosis (BTB) remains a serious threat to human health because the disease is still largely hidden in society. Reported cases of BTB in Tanzania are concentrated in specific regions, including the Northern part of the country (Arusha, Kilimanjaro, and Manyara), dairy farms in Kibaha, and some areas in Morogoro districts. The prevalence of the disease varies from one region to another, depending on the concentration of cattle herds in a particular place, ranging from 0.2 percent to 13.3 percent.

Novelty of the Study

While previous research has explored individual control measures in isolation, this study stands out for its innovative approach in combining vaccination, treatment, and education campaigns within a mathematical modelling framework. Investigating the synergistic effects of these measures can provide a more comprehensive understanding of their potential impact on BTB transmission dynamics. Moreover, it addresses the practical question of how these measures can be integrated into a cohesive control strategy to reduce both the prevalence of BTB and its economic burden [6-10].

Model Formulation

The model under consideration here entails two populations: humans and cattle, which often come into contact. The management system for grazing is a ranching system in which only cattle are confined. The model operates on the assumption that at any time t, the human population, denoted by Nh(t), is divided into five classes: Susceptible non-educated ($S_{nh}(t)$), Susceptible educated ($S_{eh}(t)$), Exposed ($E_h(t)$), Infectious ($I_h(t)$), and Recovered ($R_h(t)$) individuals.

The total human population $N_h(t)$ is given by $N_h(t)=S_{nh}(t)+S_{eh}(t)+E_h(t)+I_h(t)+R_h(t)$.

The recruitment of humans into the susceptible non-educated class occurs at a constant rate π_h. The assumption is that the education strategy is executed at a rate of ψ only for susceptible, non-educated humans to reduce the disease's transmissibility. Moreover, the study assumes that the education given to a targeted group does not necessarily guarantee lifelong protection. Susceptible non-educated humans can acquire infection through the consumption of cattle products and aerosols from infected cattle, as well as through inter-human transmission at the rate ϕ_1, and then move to the exposed class. The variable ϕ_1 is the force of infection given by

$$\varphi_1 = (1-\theta)\left(a_1\frac{I_c}{N_h} + a_2\frac{I_h}{N_h}\right),$$

where a_1 and a_2 are probabilities that infectious cattle and human infects susceptible non educated humans, respectively and $\theta \in [0, 1]$ is the efficacy of the education campaign that is being implemented. Furthermore, an educated human may contract the disease by consuming contaminated cattle products and inhaling aerosols from infected cattle at the rate ϕ_2, leading to a transition to the exposed class. The variable ϕ_2 represents the force of infection for susceptible educated humans.

$$\varphi_2 = a\theta\frac{I_c}{N_h},$$

where a is the probability that infectious cattle infects susceptible educated human. If no education campaign is extended to susceptible non-educated human, then $\theta=0$. Both educated and non-educated susceptible humans may leave their respective classes following the occurrence of natural death at a rate of μ_h. Some of the exposed individuals leave the compartment upon gaining full recovery, denoted as ρ_h, due to treatment and a natural or disease-induced death rate of μ_h and α_h, respectively. Considering the transmission dynamics of BTB disease, the study assumes that the treatment does not guarantee permanent protection. As such, recovered humans may either progress to the exposed class at a rate of τ_h or leave the compartment following the occurrence of natural death at a rate of μ_h. This study presupposes that at any time t, the cattle population denoted by $N_c(t)$ is divided into four classes, of Susceptible non-vaccinated $S_{nv}(t)$, Susceptible vaccinated $S_v(t)$, Exposed $E_c(t)$ and Infectious $I_c(t)$ cattle. The total cattle population $N_c(t)$ is given by $N_c(t)=S_{nc}(t)+S_v(t)+E_c(t)+I_c(t)$. At any given time t, it is also presumed that cattle are recruited into the non-vaccinated cattle group at a constant rate π_c. Healthier cattle are vaccinated at a rate ω to reduce the transmission of *M. bovis* from the contaminated environment and inter-cattle transmission. Susceptible non-vaccinated cattle can contract infections from their respective environment, inter-cattle transmission, and infection from infected humans. The progression to the exposed class occurs at a rate σ_1. The variable σ_1 represents the force of infection for non-vaccinated cattle.

$$\sigma_1 = (1-\lambda)\left(\varepsilon_1\frac{I_h}{N_c} + \varepsilon_2\frac{I_c}{N_c}\right),$$

Where ε_1 is the probability of an infected human infecting susceptible, non-vaccinated cattle. Moreover, the presumption is that it is difficult to differentiate infected cattle from a contaminated environment or from inter-cattle transmission. ε_2 is the rate at which cattle get infected through nose-to-nose contact, aerosol inhalation, grazing areas, and all other environmental elements that can infect cattle. $\lambda \in [0, 1]$ represents the vaccine efficacy. If no susceptible cattle are vaccinated, then $\lambda=0$. Furthermore, vaccinated cattle may contract infection from both a contaminated environment and inter-cattle transmission at the rate σ_2, leading them to move to the exposed class. The variable σ_2 constitutes the force of infection for susceptible vaccinated cattle, as given by

$$\sigma_2 = \varepsilon\lambda\frac{I_h}{N_c},$$

Where ε represents the probability that an infected human will transmit the disease to susceptible vaccinated cattle. Both vaccinated and non-vaccinated susceptibles may leave their respective classes due to natural death at a rate of μ_c. Exposed cattle also exit this class, either because of natural death 5 at a rate of μ_c or due to progressive incubation into the infectious class at a rate of γ_c. Furthermore, infected

cattle exit the class through natural death at a rate of μ_c and disease-induced death at a rate of α_c (Figure 1).

Model Diagram

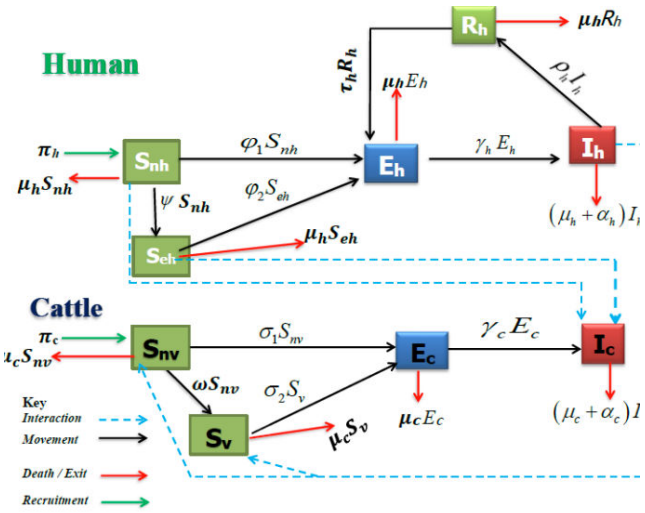

Figure 1: Schematic flow diagram for the dynamics of bovine tuberculosis disease in the presence of some intervention strategies for model.

Model Equations

The biological description and schematic flow diagram in Figure 1 together result in the following system of nine nonlinear ordinary differential equations (Table 1).

$$\begin{cases} \frac{dS_{nh}}{dt} &= \pi_h - (\mu_h + \psi + \varphi_1)S_{nh}, \\ \frac{dS_{eh}}{dt} &= \psi S_{nh} - (\mu_h + \varphi_2)S_{eh}, \\ \frac{dE_h}{dt} &= \varphi_1 S_{nh} + \varphi_2 S_{eh} - (\mu_h + \gamma_h)E_h + \tau_h R_h, \\ \frac{dI_h}{dt} &= \gamma_h E_h - (\mu_h + \alpha_h + \rho_h)I_h, \\ \frac{dR_h}{dt} &= \rho_h I_h - (\mu_h + \tau_h)R_h, \\ \frac{dS_{nv}}{dt} &= \pi_c - (\mu_c + \omega + \sigma_1)S_{nv}, \\ \frac{dS_v}{dt} &= \omega S_{nv} - (\mu_c + \sigma_2)S_v, \\ \frac{dE_c}{dt} &= \sigma_1 S_{nv} + \sigma_2 S_v - (\mu_c + \gamma_c)E_c, \\ \frac{dI_c}{dt} &= \gamma_c E_c - (\mu_c + \alpha_c)I_c, \end{cases} \quad (1)$$

where $\varphi_1 = (1-\theta)\left(a_1\frac{I_c}{N_h} + a_2\frac{I_h}{N_h}\right)$, $\varphi_2 = a\theta\frac{I_c}{N_h}$, $\sigma_1 = (1-\lambda)\left(\varepsilon_1\frac{I_h}{N_c} + \varepsilon_2\frac{I_c}{N_c}\right)$, $\sigma_2 = \varepsilon\lambda\frac{I_h}{N_c}$.

Table 1: Parameters and their descriptions.

Parameter	Description	Values
π_h	Human recruitment rate	36
π_c	Cattle recruitment rate	200
a_1	Probability that infectious cattle infects susceptible non-educated human	0.55
a_2	Contaminated environment/inter-human transmission	0.35
a	Probability that an infectious cattle infect a susceptible educated human	0.000010995
ε_1	Probability that an infectious human infects a susceptible cattle	0.000057803
ε_2	Contaminated environment/inter-cattle transmission	0.908
ε	Probability that an infected human infects a susceptible vaccinated cattle	0.000016252
ρ_h	Human recovery rate with treatment	0.098
τ_h	Removal rate to latent state	0.01
γ_h	Progression rate to infectious state	0.18
γ_c	Progression rate to infectious state	0.18
μ_h	Human natural death rate	0.0023
μ_c	Cattle natural death rate	0.013
α_h	Human death rate due to disease	0.139
α_c	Cattle death rate due to disease	0.12

θ	Education efficacy	0.75
λ	Vaccine efficacy	0.25
ψ	Per capita education rate	0.85
ω	Per capita vaccination rate	0.67

Basic Properties of the Model

Positivity of the solutions: To ensure that the model (1) is epidemiologically meaningful and well-posed, it is necessary to demonstrate that all state variables are non-negative ∀t ≥ 0.

Lemma 1. Let {$S_{nh}(0) \geq 0$, $S_{eh}(0) \geq 0$, $E_h(0) \geq 0$, $I_h(0) \geq 0$, $R_h(0) \geq 0$, $S_{nv}(0) \geq 0$, $S_v(0) \geq 0$, $E_c(0) \geq 0$ and $I_c(0) \geq 0$ } of themodel system (1) are satisfied then the solutions {S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c } are non-negative for all t ≥ 0 in Ω.

Proof. From the first equation of system (1),

$$\frac{dS_{nh}}{dt} = \pi_h - (\mu_h + \psi + \varphi_1)S_{nh} \geq -(\mu_h + \psi + \varphi_1)\, S_{nh}.$$

In the absence of human recruitment π_h it follows that

$$\frac{dS_{nh}}{S_{nh}} \geq -(\mu_h + \psi + \varphi_1)\, dt.$$

Upon integration gives,

$$\frac{dS_{nh}}{S_{nh}} \geq -(\mu_h + \psi + \varphi_1)\, dt \implies \int_0^t \frac{dS_{nh}}{S_{nh}} ds \geq -\int_0^t (\mu_h + \psi + \varphi_1)\, ds.$$

$$\ln S_{nh}(s)|_0^t \geq -\int_0^t (\mu_h + \psi + \varphi_1) ds \implies \ln S_{nh}(t) - \ln S_{nh}(0) \geq -\int_0^t (\mu_h + \psi + \varphi_1) ds.$$

$$\log_e\left(\frac{S_{nh}(t)}{S_{nh}(0)}\right) \geq -\int_0^t (\mu_h + \psi + \varphi_1) ds \implies \left(\frac{S_{nh}(t)}{S_{nh}(0)}\right) \geq \exp\left(-\int_0^t (\mu_h + \psi + \varphi_1) ds\right),$$

$$S_{nh}(t) \geq S_{nh}(0) \exp\left(-\int_0^t (\mu_h + \psi + \varphi_1) ds\right) \geq 0.$$

In the same way, the remaining state variables give;

$$\begin{aligned}
S_{nh}(t) &\geq S_{nh}(0) \exp\left(-\int_0^t (\mu_h + \psi + \varphi_1) dt\right) \geq 0, \\
S_{eh}(t) &\geq S_{eh}(0) \exp\left(-\int_0^t (\mu_h + \varphi_2) dt\right) \geq 0, \\
E_h(t) &\geq E_h(0) \exp\left(-(\mu_h + \gamma_h)t\right) \geq 0, \\
I_h(t) &\geq I_h(0) \exp\left(-(\mu_h + \alpha_h + \rho_h)\, t\right) \geq 0, \\
R_h(t) &\geq R_h(0) \exp\left(-(\mu_h + \tau_h)t\right) \geq 0, \\
S_{nv}(t) &\geq S_{nv}(0) \exp\left(-\int_0^t (\mu_c + \omega + \sigma_1) dt\right) \geq 0, \\
S_v(t) &\geq S_v(0) \exp\left(-\int_0^t (\mu_c + \sigma_2) dt\right) \geq 0, \\
E_c(t) &\geq E_c(0) \exp\left(-(\mu_c + \gamma_c)t\right) \geq 0, \\
I_c(t) &\geq I_c(0) \exp\left(-(\mu_c + \alpha_c)t\right) \geq 0.
\end{aligned} \tag{2}$$

Therefore, the solution set {S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c} of the model system (1) is non negative for all t ≥ 0 in Ω.

Invariant Region

Lemma 2. The feasible region defined by $\Omega = \Omega_h \cup \Omega_c \in R^5_+ \times R^4_+$ where $\Omega_h = \{S_{nh}, S_{eh}, E_h, I_h, R_h \in R^5_+ : S_{nh}+S_{eh}+E_h+I_h+R_h = N_h \leq \pi_h/\mu_h\}$ and $\Omega_c = \{S_{nv}, S_v, E_c, I_c \in R^4_+ : S_{nv}+S_v+E_c+I_c = N_c \leq \pi_c/\mu_c\}$, is positively invariant and attracting with regard to the model system (1).

Proof. Given $S_{nh}(0) \geq 0$, $S_{eh}(0) \geq 0$, $E_h(0) \geq 0$, $I_h(0) \geq 0$, $R_h(0) \geq 0$, $S_{nv}(0) \geq 0$, *vs.* $(0) \geq 0$, $E_c(0) \geq 0$ and $I_c(0) \geq 0$, it is sufficient to prove that {$S_{nh}(t)$, Seh(t), $E_h(t)$, $I_h(t)$, $R_h(t)$, $S_{nv}(t)$, $S_v(t)$, $E_c(t)$, $I_c(t)$} $\in R^9_+$ is bounded.

Case 1: By summing equations for human population from model system gives;

$$\frac{dN_h}{dt} = \pi_h - \mu_h N_h - \alpha_h\, I_h \leq \pi_h - \mu_h N_h.$$

Separating variables and applying anti-derivative of an integrating factor it gives;

$$\int \frac{d}{dt}\left(N_h(t)e^{\mu_h t}\right) dt \leq \int \pi_h e^{\mu_h t} dt \implies \int_0^t \frac{d}{ds}\left(N_h(s)e^{\mu_h s}\right) ds \leq \int_0^t \pi_h e^{\mu_h s} ds.$$

$$N_h(s)e^{\mu_h s}|_0^t \leq \frac{\pi_h}{\mu_h} e^{\mu_h s}|_0^t \implies N_h(t)e^{\mu_h t} - N_h(0) \leq \frac{\pi_h}{\mu_h} e^{\mu_h t} - \frac{\pi_h}{\mu_h}.$$

$$N_h(t)e^{\mu_h t} \leq N_h(0) + \frac{\pi_h}{\mu_h} e^{\mu_h t} - \frac{\pi_h}{\mu_h} \implies N_h(t) \leq \frac{\pi_h}{\mu_h} - \left(\frac{\pi_h - \mu_h N_h(0)}{\mu_h}\right) e^{-\mu_h t}.$$

$$N_h(t) \leq \frac{\pi_h}{\mu_h} - \left(\frac{\pi_h - \mu_h N_h(0)}{\mu_h}\right) e^{-\mu_h t}. \tag{3}$$

The same procedure can be used to prove that;

$$N_c(t) \leq \frac{\pi_c}{\mu_c} - \left(\frac{\pi_c - \mu_c N_c(0)}{\mu_c}\right) e^{-\mu_c t}. \tag{4}$$

Since the total population $N_h(t)$ as well as $N_c(t)$ is positive for all t ≥ 0. It is well defined that,

$$\lim_{t\to\infty} sup N_h(t) \leq \frac{\pi_h}{\mu_h}.$$

$$\lim_{t\to\infty} sup N_c(t) \leq \frac{\pi_c}{\mu_c}.$$

$$0 \leq N_h(t) \leq \frac{\pi_h}{\mu_h} \text{ and } 0 \leq N_c(t) \leq \frac{\pi_c}{\mu_c}, \text{ for all } t > 0.$$

This proves that all solutions of the BTB model system (1) with initial conditions in Ω for all t>0.

Model Analysis

Disease free equilibrium: Disease-Free Equilibrium (DFE) of the mathematical model system (1) is the point where there is no disease. Disease-free equilibrium point is obtained by setting $E_h=I_h=R_h=E_c=I_c=0$ in all equations. Now, Let E_0 be disease-free equilibrium point. Therefore, the Disease Free Equilibrium Point (DFE) denoted by E_0 can be expressed as

$$E_0 = \left[\frac{\pi_h}{\mu_h+\psi}, \frac{\psi}{\mu_h}\left(\frac{\pi_h}{\psi+\mu_h}\right), 0, 0, 0, \frac{\pi_c}{\mu_c+\omega}, \frac{\omega}{\mu_c}\left(\frac{\pi_c}{\omega+\mu_c}\right), 0, 0\right].$$

The Effective Reproduction Number

The Effective Reproduction Number (R_e) is the expected number of secondary cases produced by a single (typical) infection in a completely susceptible population and in non-susceptible hosts [6,7,10]. Effective reproduction number (R_e) is the the spectral radius of the next-generation matrix, denoted 10 by $R_e=\rho(FV^{-1})$ at DFE with F and V, respectively given by

$$F = \left[\frac{\partial \mathcal{F}_i}{\partial(E_h, I_h, E_c, I_c)}\right]_{E_0} = \begin{bmatrix} 0 & \frac{a_2(1-\theta)\mu_h}{\psi+\mu_h} & 0 & \frac{a_1(1-\theta)\mu_h+a\psi\theta}{\psi+\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\varepsilon_1(1-\lambda)\mu_c+\varepsilon\omega\lambda}{\omega+\mu_c} & 0 & \frac{\varepsilon_2(1-\lambda)\mu_c}{\omega+\mu_c} \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad (5)$$

and

$$V = \left[\frac{\partial \mathcal{V}_i}{\partial(E_h, I_h, E_c, I_c)}\right]_{E_0} = \begin{bmatrix} (\mu_h+\gamma_h) & 0 & 0 & 0 \\ -\gamma_h & (\mu_h+\alpha_h+\rho_h) & 0 & 0 \\ 0 & 0 & (\mu_c+\gamma_c) & 0 \\ 0 & 0 & -\gamma_c & (\mu_c+\alpha_c) \end{bmatrix}. \quad (6)$$

From which,

$$FV^{-1} = \begin{bmatrix} b_{11} & b_{12} & b_{13} & b_{14} \\ 0 & 0 & 0 & 0 \\ b_{31} & b_{32} & b_{33} & b_{34} \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad (7)$$

where, $b_{11} = \frac{(1-\theta)\gamma_h\mu_h a_2}{(\psi+\mu_h)(\gamma_h+\mu_h)(\alpha_h+\mu_h+\rho_h)}$, $b_{12} = \frac{(1-\theta)\mu_h a_2}{(\psi+\mu_h)(\alpha_h+\mu_h+\rho_h)}$,
$b_{13} = \frac{\gamma_c(a\psi\theta+(1-\theta)\mu_h a_1)}{(\alpha_c+\mu_c)^2(\psi+\mu_h)}$, $b_{14} = \frac{a\psi\theta+(1-\theta)\mu_h a_1}{(\alpha_c+\mu_c)(\psi+\mu_h)}$,
$b_{31} = \frac{\gamma_h(\varepsilon\omega\lambda+(1-\lambda)\mu_c\varepsilon_1)}{(\omega+\mu_c)(\gamma_h+\mu_h)(\alpha_h+\mu_h+\rho_h)}$, $b_{32} = \frac{\varepsilon\omega\lambda+(1-\lambda)\mu_h\varepsilon_1}{(\omega+\mu_c)(\alpha_h+\mu_h+\rho_h)}$,
$b_{33} = \frac{\gamma_c\varepsilon_2\mu_c(1-\lambda)}{(\omega+\mu_c)(\alpha_c+\mu_c)^2}$ and $b_{34} = \frac{\mu_c\varepsilon_2(1-\lambda)}{(\omega+\mu_c)(\alpha_c+\mu_c)}$

It follows that the effective reproduction number of the model system (1), is given by

$$R_e = \frac{1}{2}\left(b_{11}+b_{33}+\sqrt{(b_{11}-b_{33})^2+4b_{13}b_{31}}\right), \quad (8)$$

such that,

$$b_{11} = \frac{(1-\theta)\gamma_h\mu_h a_2}{(\psi+\mu_h)(\gamma_h+\mu_h)(\alpha_h+\mu_h+\rho_h)}, b_{13} = \frac{\gamma_c(a\psi\theta+(1-\theta)\mu_h a_1)}{(\alpha_c+\mu_c)^2(\psi+\mu_h)},$$
$$b_{31} = \frac{\gamma_h(\varepsilon\omega\lambda+(1-\lambda)\mu_c\varepsilon_1)}{(\omega+\mu_c)(\gamma_h+\mu_h)(\alpha_h+\mu_h+\rho_h)} \text{ and } b_{33} = \frac{\gamma_c\varepsilon_2\mu_c(1-\lambda)}{(\omega+\mu_c)(\alpha_c+\mu_c)^2}.$$

So, when there is no intervention strategy such that ψ=0 and ω=0," which refers to the education campaign and vaccination rate," and without any treatment for individual humans being implemented (ρ_h=0), then the basic reproduction number R_0 will be

$$R_0 = \frac{1}{2}\left(z_{11}+z_{33}+\sqrt{(z_{11}-z_{33})^2+4z_{13}z_{31}}\right),$$

where,

$$z_{11} = \frac{\gamma_h a_2}{(\gamma_h+\mu_h)(\alpha_h+\mu_h)}, z_{13} = \frac{\gamma_c a_1}{(\alpha_c+\mu_c)^2}, z_{31} = \frac{\gamma_h\varepsilon_1}{(\gamma_h+\mu_h)(\alpha_h+\mu_h)}$$
$$\text{and } z_{33} = \frac{\gamma_c\varepsilon_2}{(\alpha_c+\mu_c)^2}.$$

Biologically, it is meaningful to compare the threshold values: the basic reproduction number without a vaccine, denoted as R_0, and the effective reproduction numbers with and without a vaccine, denoted as R_v and R_e respectively, such that $R_e<R_v<R_0$. The basic reproduction number, often denoted as R_0, plays a crucial role in epidemiological studies by facilitating predictions of future infections of interest. When the basic reproduction number is less than one ($R_0<1$), it implies that, on average, an infectious individual leads to the infection of fewer than one other individual. Consequently, over time, the bTB disease could naturally die out, leading to a population free from BTB. In other words, both the human and cattle populations would remain free from the disease invasion if $R_0<1$ [11-15].

$$\frac{1}{2}\left(z_{11}+z_{33}+\sqrt{(z_{11}-z_{33})^2+4z_{13}z_{31}}\right) < 1.$$

Conversely, if the basic reproduction number (R_0) is greater than one, it implies that each infected individual, on average, will lead to more than one newly infected individual. In this scenario, the infection persists, causing the disease to continue spreading in the population. Therefore, if $R_0>1$, BTB will continue to spread

$$\frac{1}{2}\left(z_{11}+z_{33}+\sqrt{(z_{11}-z_{33})^2+4z_{13}z_{31}}\right) > 1.$$

Local Stability of Disease-Free Equilibrium (DFE)

The local stability analysis of DFE enables us to understand how a system behaves near an equilibrium point, but not necessarily at a specific equilibrium point. In other words, local stability investigates the nature of the system in the vicinity of the equilibrium point.

Theorem 3.1. The BTB model system at DFE (E_0) is locally asymptotically stable if $R_e<1$ and unstable if $R_e>1$.

Proof. The Jacobian matrix of the model system at disease free equilibrium point (E_0) is given by

$$J_{E_0} = \begin{pmatrix} -y_{11} & 0 & 0 & y_{14} & 0 & 0 & 0 & 0 & y_{19} \\ \psi & -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & y_{29} \\ 0 & 0 & -y_{33} & y_{34} & 0 & 0 & 0 & 0 & y_{39} \\ 0 & 0 & \gamma_h & -y_{44} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \rho_h & -y_{55} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & y_{64} & 0 & -y_{66} & 0 & y_{68} & 0 \\ 0 & 0 & 0 & y_{74} & 0 & \omega & -\mu_c & 0 & 0 \\ 0 & 0 & 0 & y_{84} & 0 & 0 & 0 & -y_{88} & y_{89} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_c & -y_{99} \end{pmatrix},$$

where,

$$y_{11} = \mu_h+\psi, y_{14} = \frac{-a_2(1-\theta)\mu_h}{\mu_h+\psi}, y_{19} = \frac{-a_1(1-\theta)\mu_h}{\mu_h+\psi}, y_{29} = \frac{-a\theta\psi}{\mu_h+\psi},$$

$$y_{33} = \mu_h+\gamma_h, y_{34} = \frac{a_2(1-\theta)\mu_h}{\mu_h+\psi}, y_{39} = \frac{a_1(1-\theta)\mu_h}{\mu_h+\psi}, y_{44} = \mu_h+\alpha_h+\rho_h,$$

$$y_{55} = \mu_h + \tau_h,\ y_{64} = \frac{-(1-\lambda)\varepsilon_1\mu_c}{\mu_c+\omega},\ y_{66} = \mu_c + \omega,\ y_{68} = \frac{-(1-\lambda)\varepsilon_2\mu_c}{\mu_c+\omega},$$
$$y_{74} = \frac{-\lambda\varepsilon\omega}{\mu_c+\omega},\ y_{84} = \frac{(1-\lambda)\varepsilon_1\mu_c}{\mu_c+\omega},\ y_{88} = (\mu_c+\gamma_c),\ y_{89} = \frac{(1-\lambda)\varepsilon_2\mu_c}{\mu_c+\omega}$$
and $y_{99} = \mu_c + \alpha_c$.

From the Jacobian matrix J_{E0}, the first, fifth and seventh columns contains diagonal entries. Therefore, the diagonals $-\mu_h$, $-(\mu_h + \tau_h)$, and $-\mu_c$ are the three eigenvalues of the Jacobian J_{E0}. Thus, excluding these columns and the corresponding rows, the remaining eigenvalues are computed. Then the reduced 6 × 6 matrix from J_{E0} becomes.

$$\xi = \begin{pmatrix} -y_{11} & 0 & y_{14} & 0 & 0 & y_{19} \\ 0 & -y_{33} & y_{34} & 0 & 0 & y_{39} \\ 0 & \gamma_h & -y_{44} & 0 & 0 & 0 \\ 0 & 0 & y_{64} & -y_{66} & y_{68} & 0 \\ 0 & 0 & y_{84} & 0 & -y_{88} & y_{89} \\ 0 & 0 & 0 & 0 & \gamma_c & -y_{99} \end{pmatrix}.$$

From the Jacobian matrix ξ, the first and fourth columns contains diagonal entries. Therefore, the diagonals $-(\mu_h + \psi)$ and $-(\mu_c + \omega)$ are the two eigenvalues of the Jacobian ξ. Thus, excluding these columns and the corresponding rows, the remaining eigenvalues are computed. Then the reduced 4 × 4 matrix from ξ bec becomes.

$$\Upsilon = \begin{pmatrix} -y_{33} & y_{34} & 0 & y_{39} \\ \gamma_h & -y_{44} & 0 & 0 \\ 0 & y_{84} & -y_{88} & y_{89} \\ 0 & 0 & \gamma_c & -y_{99} \end{pmatrix} = \begin{pmatrix} G_1 & G_3 \\ G_4 & G_2 \end{pmatrix}.$$

Matrices G_1, G_2, G_3 and G_4 are defined as follows

$$G_1 = \begin{pmatrix} -y_{33} & y_{34} \\ \gamma_h & -y_{44} \end{pmatrix},\ G_2 = \begin{pmatrix} -y_{88} & y_{89} \\ \gamma_c & -y_{99} \end{pmatrix},\ G_3 = \begin{pmatrix} 0 & y_{39} \\ 0 & 0 \end{pmatrix},\ G_4 = \begin{pmatrix} 0 & y_{84} \\ 0 & 0 \end{pmatrix}.$$

Apparently, matrices G_3 and G_4 are singular matrices with $|G_3| = 0$ and $|G_4| = 0$. Now, it is sufficient to demonstrate that the disease-free equilibrium point of the model system (1) is stable only when the trace and determinant of matrices G_1 and G_2 are negative and positive, respectively. Trace and determinant of matrix G_1 denoted by $Tr(G_1)$ and $det(G_1)$ are respectively given by.

$$\mathrm{Tr}(G_1) = -(y_{33} + y_{44}) = -(2\mu_h + \gamma_h + \alpha_h + \rho_h) < 0.$$
$$\det(G_1) = y_{33}y_{44} - \gamma_h y_{34} = (\mu_h + \gamma_h)(\mu_h + \alpha_h + \rho_h) - \frac{a_2(1-\theta)\mu_h\gamma_h}{\mu_h + \psi},$$
such that $\frac{a_2(1-\theta)\mu_h\gamma_h}{\mu_h+\psi} = A(\gamma_h + \mu_h)(\alpha_h + \mu_h + \rho_h)$,
where,
$$A = R_e - \frac{\gamma_c\gamma_h\mu_c\mu_h\varepsilon_1 a_1(a\psi\theta + 1 - \theta)(\varepsilon\omega\lambda + 1 - \lambda)}{(\gamma_h + \mu_h)(\alpha_h + \mu_h + \rho_h)(\psi + \mu_h)\left[(\omega + \mu_c)(\alpha_c + \mu_c)^2 R_e - \gamma_c\mu_c\varepsilon_2(1-\lambda)\right]}.$$

Trace and determinant of matrix G_2 denoted by $Tr(G_2)$ and $det(G_2)$ are respectively given by

$$\mathrm{Tr}(G_2) = -(y_{88} + y_{99}) = -(2\mu_c + \gamma_c + \alpha_c) < 0.$$
$$\det(G_2) = y_{88}y_{99} - \gamma_c y_{89} = (\mu_c + \gamma_c)(\mu_c + \alpha_c) - \frac{\varepsilon_2(1-\lambda)\mu_c\gamma_c}{\mu_c + \omega},$$
such that $\frac{\varepsilon_2(1-\lambda)\mu_c\gamma_c}{\mu_c+\omega} = B(\alpha_c + \mu_c)^2$,
where,
$$B = R_e - \frac{\gamma_c\gamma_h\mu_c\mu_h\varepsilon_1 a_1(a\psi\theta + 1 - \theta)(\varepsilon\omega\lambda + 1 - \lambda)}{(\omega + \mu_c)(\alpha_c + \mu_c)^2\left[(\gamma_h + \mu_h)(\alpha_h + \mu_h + \rho_h)(\psi + \mu_h)R_e - \gamma_h\mu_h a_2(1-\theta)\right]}.$$

Thus $det(G_1) > 0$ and $det(G_2) > 0$ if and only if $R_e < 1$. Since the traces of matrix G_1 and G_2 are negative and the determinants are strictly greater than zero when $R_e < 1$, then the disease-free equilibrium point E_0 is locally asymptotically stable when $R_e < 1$ and unstable otherwise. Epidemiologically, it implies that the bTB disease can be eliminated in the endemic area when $R_e < 1$, particularly when the vaccination rate (ω) is kept constant without limitation, and the education campaign rate (ψ) is also executed for every individual. Conversely, if $R_e > 1$, then each infectious individual produces more than one new infected individual, implying that the disease can spread rapidly in the entire population [16-20].

Global Stability of Disease-Free Equilibrium (DFE)

This subsection analyses the global stability pertaining to DFE aimed to establish the asymptotic behaviour of the model system (1) beyond just the neighbourhood points of the model disease-free steady state.

Lemma 3. The disease-free equilibrium point is globally asymptotically stable if $R_e < 1$ and unstable if $R_e > 1$.

Proof. The approach of [6] is used to analyse the global stability of DFE of the model system (1). Using this approach, the model system (1) is written as follows.

$$\begin{cases} \dfrac{d\zeta_n}{dt} = M_1(\zeta_n - \zeta_{E_0}) + M_2\zeta_i, \\ \dfrac{d\zeta_i}{dt} = M_3\zeta_i. \end{cases} \quad (9)$$

where ζ_n stands for classes that do not transmit bTB disease, that is $\zeta_n = (S_{nh}, S_{eh}, R_h, S_{nv}, S_v)$ T and ζ_i stands for classes that can transmit bTB disease, that is $\zeta_i = (I_h, E_h, E_c, I_c)$ T. Here, T stands for the transposition of ζ_n and ζ_i, and also ζ_{E0} is ζ_n at DFE (E_0). The matrix M_1 is obtained by differentiating the non-transmitting equations of the model system (1) with respect to non-transmitting variables at E_0, whereas the matrix M_2 is obtained by differentiating the non-transmitting equations of the model system (1) with respect to the transmitting variables. The disease-free equilibrium point (E_0) is globally asymptotically stable if the eigenvalues of M_1 are real and negative, and if M_3 is a Metzler stable matrix with non-negative off-diagonal elements. Therefore, the model system (1) yields the following.

$$\zeta_n - \zeta_{E_0} = \begin{pmatrix} S_{nh} \\ S_{eh} \\ R_h \\ S_{nv} \\ S_v \end{pmatrix} - \begin{pmatrix} \frac{\pi_h}{\mu_h+\psi} \\ \frac{\psi}{\mu_h}\left(\frac{\pi_h}{\psi+\mu_h}\right) \\ 0 \\ \frac{\pi_c}{\mu_c+\omega} \\ \frac{\omega}{\mu_c}\left(\frac{\pi_c}{\omega+\mu_c}\right) \end{pmatrix} = \begin{pmatrix} S_{nh} - \frac{\pi_h}{\mu_h+\psi} \\ S_{eh} - \frac{\psi}{\mu_h}\left(\frac{\pi_h}{\psi+\mu_h}\right) \\ R_h \\ S_{nv} - \frac{\pi_c}{\mu_c+\omega} \\ S_v - \frac{\omega}{\mu_c}\left(\frac{\pi_c}{\omega+\mu_c}\right) \end{pmatrix}. \quad (10)$$

Also,

$$M_1 = \frac{\partial \zeta_n(E_0)}{\partial(S_{nh}, S_{eh}, R_h, S_{nv}, S_v)} = \begin{pmatrix} -(\mu_h+\psi) & 0 & 0 & 0 & 0 \\ \psi & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & -(\mu_h+\tau_h) & 0 & 0 \\ 0 & 0 & 0 & -(\mu_c+\omega) & 0 \\ 0 & 0 & 0 & \omega & -\mu_c \end{pmatrix},$$

$$M_2 = \frac{\partial \zeta_n}{\partial(E_h, I_h, E_c, I_c)} = \begin{pmatrix} 0 & -(1-\theta)a_2\frac{S_{nh}}{N_h} & 0 & -(1-\theta)a_1\frac{S_{nh}}{N_h} \\ 0 & 0 & 0 & \theta a\frac{S_{nh}}{N_h} \\ 0 & -(1-\lambda)\varepsilon_2\frac{S_{nv}}{N_c} & 0 & -(1-\lambda)\varepsilon_1\frac{S_{nv}}{N_c} \\ 0 & \lambda\varepsilon\frac{S_v}{N_c} & 0 & 0 \end{pmatrix},$$

Since the matrix M_1 is a lower triangular matrix, then the eigenvalues will be the diagonal entries presented as follows: $\xi_1=-(\mu_h+\psi)$, $\xi_2=-\mu_h$, $\xi_3=-(\mu_c+\omega)$ and $\xi_4=-\mu_c$ which are all negative and real. This shows that at the DFE the system $d\zeta_n/dt = M_1(\zeta_n-\zeta_{E0})+M_2\zeta_i$ is globally asymptotically stable. Furthermore, the matrix M_3 is obtained by differentiating the transmitting equations of the model system (1) with respect to the transmitting variables which gives

$$M_3 = \begin{pmatrix} -(\mu_h+\gamma_h) & 0 & 0 & a\theta\frac{S_{nh}}{N_h} + a_1(1-\theta)\frac{S_{nh}}{N_h} \\ \gamma_h & -(\mu_h+\alpha_h+\rho_h) & 0 & 0 \\ 0 & \varepsilon\lambda\frac{S_v}{N_c} + \varepsilon_1(1-\lambda)\frac{S_{nv}}{N_c} & -(\mu_c+\gamma_c) & \varepsilon_2(1-\lambda)\frac{S_{nv}}{N_c} \\ 0 & 0 & \gamma_c & -(\mu_c+\alpha_c) \end{pmatrix}.$$

Testing whether the matrix M_3 is a Metzler stable matrix requires applying the approach as propounded by [16,11]. The following Lemma is used.

Lemma 4. Let G be a square Metzler matrix stable which can be written in block form

$$G = \begin{pmatrix} A & B \\ C & D \end{pmatrix}$$

where A and D are square matrices. Then, G is a Metzler stable matrix if and only if matrix A and $D-CA^{-1}B$ or matrix D and $A-BD^{-1}C$ are Metzler stable.

By comparing the matrix M_3 and a square Metzler matrix G, then the matrices A, B, C and D are expressed as follows:

$$A = \begin{pmatrix} -(\mu_h+\gamma_h) & 0 \\ \gamma_h & -(\mu_h+\alpha_h+\rho_h) \end{pmatrix}, D = \begin{pmatrix} -(\mu_c+\gamma_h) & \varepsilon_2(1-\lambda)\frac{S_{nv}}{N_c} \\ \gamma_c & -(\mu_c+\alpha_c) \end{pmatrix},$$

$$B = \begin{pmatrix} 0 & a\theta\frac{S_{eh}}{N_h} + a_1(1-\theta)\frac{S_{nh}}{N_h} \\ 0 & 0 \end{pmatrix}, C = \begin{pmatrix} 0 & \varepsilon\lambda\frac{S_v}{N_c} + \varepsilon_1(1-\lambda)\frac{S_{nv}}{N_c} \\ 0 & 0 \end{pmatrix}.$$

After some computations and simplifications,

$$BD^{-1}C = \begin{pmatrix} 0 & 0 \\ 0 & 0 \end{pmatrix},$$

$$A - BD^{-1}C = \begin{pmatrix} -(\mu_h+\gamma_h) & 0 \\ \gamma_h & -(\mu_h+\alpha_h+\rho_h) \end{pmatrix}$$

$A - BD^{-1}C$ is said to be Metzler stable matrix if and only if $(\mu_h+\gamma_h)(\mu_h+\alpha_h+\rho_h) \geq 0$. As already noted, it can be seen that matrix M_1 has all the eigenvalues that are real and negative and matrix M_3 is the Metzler matrix as its off-diagonal elements are so non-negative, that $|A-BD^{-1}C| \geq 0 \Rightarrow M_3(i,j) \geq 0$ for all indices $i \neq j$. Implicitly, that the disease-free equilibrium point is globally asymptotically stable when $R_e<1$, otherwise it is unstable. In biological terms, the bTB disease will eventually die out in the population given $R_e<1$ no matter how huge the disease invaded. Otherwise, if $R_e>1$ the disease will spread even more as there will be many new infections in the population.

Endemic Equilibrium Point (EEP)

Existence of the equilibrium solutions: The Endemic Equilibrium (EE) denoted by E_* such that $E_*=(S^*_{nh}, S^*_{eh}, E^*_h, I^*_h, R^*_h, S^*_{nv}, S^*_v, E^*_c, I^*_c)$ is the state where the population is not free from the infection. At this point the disease persists in the population. In other words, the disease cannot be eradicated from the population. In the case of bTB, the number of infectious cases is not equal to zero such that, $E_h \neq 0$, $I_h \neq 0$, $R_h \neq 0$, $E_c \neq 0$, $I_c \neq 0$. Setting each equation in the model system (1) equal to zero, then the steady state of the system becomes.

$$\begin{cases} \pi_h - (\mu_h+\psi+\varphi_1^*)S^*_{nh} = 0, \\ \psi S^*_{nh} - (\mu_h+\varphi_2^*)S^*_{eh} = 0, \\ \varphi_2^* S^*_{eh} + \varphi_1^* S^*_{nh} - (\mu_h+\gamma_h)E^*_h + \tau_h R^*_h = 0, \\ \gamma_h E^*_h - (\mu_h+\alpha_h+\tau_h)I^*_h = 0, \\ \tau_h I^*_h - (\mu_h+\rho_h)R^*_h = 0, \\ \pi_c - (\mu_c+\omega+\sigma_1^*)S^*_{nv} = 0, \\ \omega S^*_{nv} - (\mu_c+\sigma_2^*)S^*_v = 0, \\ \sigma_1^* S^*_{nv} + \sigma_2^* S^*_v - (\mu_c+\gamma_c)E^*_c = 0, \\ \gamma_c E^*_c - (\mu_c+\alpha_c)I^*_c = 0. \end{cases} \quad (11)$$

where, $\varphi_1^* = (1-\theta)\left(a_1\frac{I^*_c}{N^*_h} + a_2\frac{I^*_h}{N^*_h}\right)$, $\sigma_1^* = (1-\lambda)\left(\varepsilon_1\frac{I^*_h}{N^*_c} + \varepsilon_2\frac{I^*_c}{N^*_c}\right)$,

$\varphi_2^* = a\theta\frac{I^*_c}{N^*_h}$ and $\sigma_2^* = \varepsilon\lambda\frac{I^*_h}{N^*_c}$.

Finally, when solving for the corresponding variable from equation (11) the Endemic Equilibrium denoted by $E_*=(S^*_{nh}, S^*_{eh}, E^*_h, I^*_h, R^*_h, S^*_{nv}, S^*_v, E^*_c, I^*_c)$ is solved and given by

$$
\begin{aligned}
S_{nh}^* &= \frac{\pi_h}{\mu_h+\psi+\varphi_1^*},\\
S_{eh}^* &= \frac{\psi\pi_h}{(\mu_h+\psi+\varphi_1^*)(\mu_h+\varphi_2^*)},\\
E_h^* &= \frac{(\psi\pi_h\varphi_1^*\varphi_2^*)(\mu_h+\alpha_h+\rho_h)(\mu_h+\tau_h)+\psi\varphi_2^*)}{(\psi+\mu_h+\varphi_1^*)(\mu_h+\alpha_h+\rho_h)(\mu_h+\tau_h)(\mu_h+\varphi_2^*)+\gamma_h\rho_h\tau_h(\mu_h+\varphi_2^*)},\\
I_h^* &= \frac{(\pi_h\gamma_h(\mu_h+\tau_h)(\mu_h\varphi_1^*+\varphi_1^*\varphi_2^*+\psi\varphi_2^*)}{(\psi+\mu_h+\varphi_1^*)(\mu_h+\alpha_h+\rho_h)(\mu_h+\tau_h)(\mu_h+\varphi_2^*)+\gamma_h\rho_h\tau_h(\mu_h+\varphi_2^*)},\\
R_h^* &= \frac{\pi_h\gamma_h\rho_h(\mu_h\varphi_1^*+\varphi_2^*\varphi_1^*+\psi\varphi_2^*)}{(\psi+\mu_h+\varphi_1^*)(\mu_h+\alpha_h+\rho_h)(\mu_h+\tau_h)(\mu_h+\varphi_2^*)+\gamma_h\rho_h\tau_h(\mu_h+\varphi_2^*)},\\
S_{nv}^* &= \frac{\pi_c\omega}{\mu_c+\omega+\sigma_1^*},\\
S_v^* &= \frac{\omega\pi_c}{(\mu_c+\omega+\sigma_1^*)(\mu_c+\sigma_2^*)},\\
E_c^* &= \frac{\pi_c(\mu_c\sigma_1^*+\omega\sigma_2^*+\sigma_1^*\sigma_2^*)}{(\mu_c+\omega+\sigma_1^*)(\mu_c+\gamma_c)(\mu_c+\sigma_2^*)},\\
I_c^* &= \frac{\gamma_h\pi_c(\mu_c\sigma_1^*+\omega\sigma_2^*+\sigma_1^*\sigma_2^*)}{(\mu_c+\alpha_c)(\gamma_c+\mu_c)(\mu_c+\omega+\sigma_1^*)(\mu_c+\sigma_2^*)}.
\end{aligned}
\tag{12}
$$

where,

$$\varphi_1^* = (1-\theta)\left(a_1\frac{I_c^*}{N_h^*}+a_2\frac{I_h^*}{N_h^*}\right),\ \varphi_2^* = a\theta\frac{I_c^*}{N_h^*},\ \sigma_1^* = (1-\lambda)\left(\varepsilon_1\frac{I_h^*}{N_c^*}+\varepsilon_2\frac{I_c^*}{N_c^*}\right)\text{ and }\sigma_2^* = \varepsilon\lambda\frac{I_h^*}{N_c^*}.$$

From equation (12), the value of I^*_h and I^*_c is substituted into

$$\sigma_1^* - (1-\lambda)\left(\varepsilon_1\frac{I_h^*}{N_c^*}+\varepsilon_2\frac{I_c^*}{N_c^*}\right) = 0,$$

and after some algebraic computations and simplifications, a polynomial of degree three results $A\sigma_1^{*3}+B\sigma_1^{*2}+C\sigma_1^{*}=0$. Upon algebraic simplification, $A\sigma_1^{*3}+B\sigma_1^{*2}+C\sigma_1^{*}=0$ results to $\sigma_1^{*}*A\sigma_1^{*2}+B\sigma^{*}_1+C=0$, where $\sigma_1^{*}=0$ or $A\sigma_1^{*2}+B\sigma^{*}_1+C=0$

$$
\begin{aligned}
A &= \mu_c+\frac{a\theta\gamma_h\pi_c}{N_h(\mu_c+\alpha_c)(\gamma_c+\mu_c)(\mu_c+\sigma_2^*)}(\mu_c+\sigma_2^*),\\
B &= -\frac{a\theta\gamma_h^2\pi_c^2(\mu_c^2+2\mu_c\sigma_2^*+\sigma_2^{*^2})}{N_h[(\mu_c+\alpha_c)(\gamma_c+\mu_c)(\mu_c+\sigma_2^*)]^2}+\omega\sigma_2^*+(\mu_c+\omega)(\mu_c+\mu_h)\\
&\quad+\frac{\gamma_h\pi_c(-N_h\mu_c^2-\mu_c\sigma_2^*+2a\theta\mu_c(\omega+\mu_c)+a\theta\omega\sigma_2^*)}{N_h(\mu_c+\alpha_c)(\gamma_c+\mu_c)(\mu_c+\sigma_2^*)}\\
&\quad-\frac{2a\theta(1-\lambda)\gamma_h^2\pi_h\pi_c\varepsilon_1\mu_c(\psi+\varphi_1^*)(\mu_h+\tau_h)}{N_hN_c(\mu_c+\alpha_c)(\gamma_c+\mu_c)(\mu_c+\sigma_2^*)(\rho_h\tau_h\gamma_h+(\rho_h+\alpha_h+\mu_h)(\psi+\mu_h+\varphi_1^*)(\mu_h+\tau_h))}\\
&\quad-\frac{(1-\lambda)\gamma_h\pi_h\varepsilon_1\mu_h(\mu_h+\tau_h)\varphi_1^*}{N_c(\rho_h\tau_h\gamma_h+(\rho_h+\alpha_h+\mu_h)(\psi+\mu_h+\varphi_1^*)(\mu_h+\tau_h))},
\end{aligned}
$$

and

$$
\begin{aligned}
C &= -\frac{\gamma_h\pi_c^2(\mu_c\mu_h(\omega+\mu_c)+\mu_h(\omega+\mu_c)\sigma_2^*+\omega\sigma_2^{*^2}+\omega\sigma_2^*\mu_c)}{(\mu_c+\alpha_c)(\gamma_c+\mu_c)(\mu_c+\sigma_2^*)}\\
&\quad-\frac{\gamma_h\pi_c(N_h\mu_c^2+\mu_c\sigma_2^*+2a\theta\mu_c(\omega+\mu_c)+a\theta\omega\sigma_2^*)}{N_h(\mu_c+\alpha_c)(\gamma_c+\mu_c)(\mu_c+\sigma_2^*)}\\
&\quad-\frac{a\theta(1-\lambda)\gamma_h^2\pi_h\pi_c\varepsilon_1\mu_c(\psi+\varphi_1^*)(\mu_h+\tau_h)}{N_hN_c(\mu_c+\alpha_c)(\gamma_c+\mu_c)(\mu_c+\sigma_2^*)(\rho_h\tau_h\gamma_h+(\rho_h+\alpha_h+\mu_h)(\psi+\mu_h+\varphi_1^*)(\mu_h+\tau_h))}\\
&\quad-\frac{2(1-\lambda)\gamma_h\pi_h\varepsilon_1\mu_h(\mu_h+\tau_h)(\omega+\mu_c)\varphi_1^*(1+2\omega-2\mu_c)}{N_c(\rho_h\tau_h\gamma_h+(\rho_h+\alpha_h+\mu_h)(\psi+\mu_h+\varphi_1^*)(\mu_h+\tau_h))}\\
&\quad-\frac{2a\theta\gamma_h^2\pi_c^2\omega\sigma_2^*(\mu_c^2+\mu_c\sigma_2^*+\sigma_2^{*^2})}{N_h[(\mu_c+\alpha_c)(\gamma_c+\mu_c)(\mu_c+\sigma_2^*)]^2}\\
&\quad+\left(\frac{\omega\sigma_2^*}{\mu_c+\mu_h}+\mu_h\right)\frac{(\mu_c+\omega)^2\gamma_h\pi_c a\theta\omega\sigma_2^*(\rho_h+\alpha_h+\mu_h)(\psi+\mu_h)(\mu_h+\tau_h)}{(\mu_c^2+2\mu_c\sigma_2^*+\sigma_2^{*^2})N_h(\mu_c+\alpha_c)(\gamma_c+\mu_c)(\mu_c+\sigma_2^*)}(1-R_e)R_e
\end{aligned}
\tag{13}
$$

$$\text{For } \sigma_1^* = (1-\lambda)\left(\varepsilon_1\frac{I_h^*}{N_c^*}+\varepsilon_2\frac{I_c^*}{N_c^*}\right) = 0,$$

corresponds to Disease Free Equilibrium (DFE), whereas $A\sigma_1^{*2}+B\sigma^{*1}+C=0$, can also be written in the form:

$$\sigma_1^* = \frac{-B\pm\sqrt{B^2-4AC}}{2},$$

which satisfies Endemic Equilibrium. The value of A is strictly positive. Depending on the signs of B and C, there are three cases to consider as having positive real root of the force of infection (σ^*_1) as follows:

Case 1: If B<0 then model system (1) has a stable endemic equilibrium point when C<0. This equilibrium happens when R_e>1 as interpreted from (13). In this case backward bifurcation is not possible due to the absence of multiple equilibria.

Case 2: Exactly one endemic equilibrium point. Suppose B<0 and C=0 or $B^2-4AC=0$. In other words, the polynomial $A\sigma_1^{*2}+B\sigma^{*}_1+C=0$ has just one positive root and hence the model system (1) has unique endemic equilibrium point.

Case 3: Two endemic equilibria. If B<0, C>0 and $B^2-4AC>0$, then the polynomial $A\sigma_1^{*2}+B\sigma^{*}_1+C=0$ has two positive real roots. In other words, the model system (1) has two endemic equilibria and hence there is a possibility of backward bifurcation. These three cases are summarized under theorem 3.2.

Theorem 3.2. The number of positive endemic equilibria of bovine tuberculosis model (1) is hereunder summarised thus:

If C<0, R_e>1, then the system has a unique endemic equilibrium.

If B<0 and C=0 or $B^2-4AC=0$, then the system has exactly one endemic equilibrium.

If B<0, C>0 and $B^2-4AC>0$, then the system has exactly two endemic equilibria.

Otherwise there are no endemic equilibria, *i.e.*, when AC>0 and B>0.

Stability of Endemic Equilibrium Point (EEP) of Model with Interventions

In epidemiological models, forward or backward bifurcation has vital implications for the biological control measures of infectious diseases. Bifurcation analysis of the equilibrium points reveals whether the disease is completely reducible or persistent in the afflicted population. This analysis occurs at the disease-free equilibrium using Center Manifold theory, as presented in [6]. It is done by renaming the state variables of the model system as follows.

$$\text{Let } x_1 = S_{nh}, x_2 = S_{eh}, x_3 = E_h, x_4 = I_h, x_5 = R_h, x_6 = S_{nv}, x_7 = S_v, x_8 = E_c \text{ and } x_9 = I_c$$

Resulting system can be written in the form of

$$\frac{dX_i}{dt} = F(X_i),$$

where, $X_i = (x_1, x_2, ..., x_9)^T$, $F = (f_1, f_2, ..., f_9)^T$ and $(.)^T$ denotes a matrix transpose. system (1) takes the form:

$$
\begin{cases}
\frac{dx_1}{dt} &= f_1 = \pi_h-(\mu_h+\psi)x_1-(1-\theta)\left(a_1\frac{x_9x_1}{N_h}+a_2\frac{x_4x_1}{N_h}\right),\\
\frac{dx_2}{dt} &= f_2 = \psi x_1-\mu_h x_2-a\theta\frac{x_9x_2}{N_h},\\
\frac{dx_3}{dt} &= f_3 = (1-\theta)\left(a_1\frac{x_9x_1}{N_h}+a_2\frac{x_4x_1}{N_h}\right)+a\theta\frac{x_9x_2}{N_h}-(\mu_h+\gamma_h)x_3+\tau_h x_5,\\
\frac{dx_4}{dt} &= f_4 = \gamma_h x_3-(\mu_h+\alpha_h+\rho_h)x_4,\\
\frac{dx_5}{dt} &= f_5 = \rho_h x_4-(\mu_h+\tau_h)x_5,\\
\frac{dx_6}{dt} &= f_6 = \pi_c-(\mu_c+\omega)x_6-(1-\lambda)\left(\varepsilon_1\frac{x_4x_6}{N_c}+\varepsilon_2\frac{x_9x_6}{N_c}\right),\\
\frac{dx_7}{dt} &= f_7 = \omega x_6-\mu_c x_7-\varepsilon\lambda\frac{x_4x_7}{N_c},\\
\frac{dx_8}{dt} &= f_8 = (1-\lambda)\left(\varepsilon_1\frac{x_4x_6}{N_c}+\varepsilon_2\frac{x_9x_6}{N_c}\right)+\varepsilon\lambda\frac{x_4x_7}{N_c}-(\mu_c+\gamma_c)x_8,\\
\frac{dx_9}{dt} &= f_9 = \gamma_c x_8-(\mu_c+\alpha_c)x_9,
\end{cases}
\tag{14}
$$

where $N_h = x_1+x_2+x_3+x_4+x_5$ and $N_c = x_6+x_7+x_8+x_9$.

Let ε^*_2 be the bifurcation parameter, then, the system is a system of model equations at disease free equilibrium point when $\varepsilon_2=\varepsilon^*_2$ with $R_0=1$. Hence, solving for ε^*_2 from $R_0=1$ in

$$R_0 = \frac{z_{11} + z_{33} + \sqrt{(z_{11} - z_{33})^2 + 4z_{13}z_{31}}}{2},$$

such that, $z_{11} = \frac{\gamma_h a_2}{(\gamma_h + \mu_h)(\alpha_h + \mu_h)}$, $z_{13} = \frac{\gamma_c a_1}{(\alpha_c + \mu_c)^2}$, $z_{31} = \frac{\gamma_h \varepsilon_1}{(\gamma_h + \mu_h)(\alpha_h + \mu_h)}$ and $z_{33} = \frac{\gamma_c \varepsilon_2}{(\alpha_c + \mu_c)^2}$.
which gives

$$\varepsilon_2^* = \frac{(\alpha_c + \mu_c)^2}{\gamma_c} + \frac{\gamma_h a_1 \varepsilon_1}{\gamma_h a_2 - (\gamma_h + \mu_h)(\alpha_h + \mu_h)}.$$

Next, the system is transformed with $\varepsilon_2=\varepsilon^*_2$, which possesses a simple zero eigenvalue. Center Manifold Theory is employed to analyze the dynamics of in the vicinity of $\varepsilon_2=\varepsilon^*_2$. Consequently, the Jacobian matrix of the system at the disease-free equilibrium, denoted as $J(\varepsilon^*_2)$, is given by

$$J(\varepsilon_2^*) = \begin{pmatrix} -R_{11} & 0 & 0 & -R_{14} & 0 & 0 & 0 & 0 & -R_{19} \\ \psi & -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & -R_{29} \\ 0 & 0 & -R_{33} & R_{34} & 0 & 0 & 0 & 0 & R_{39} \\ 0 & 0 & \gamma_h & -R_{44} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \rho_h & -R_{55} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -R_{64} & 0 & -R_{66} & 0 & -R_{68} & 0 \\ 0 & 0 & 0 & -R_{74} & 0 & \omega & -\mu_c & 0 & 0 \\ 0 & 0 & 0 & R_{84} & 0 & 0 & 0 & -R_{88} & R_{89} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_c & -R_{99} \end{pmatrix},$$

$$R_{11} = \mu_h + \psi,\ R_{14} = R_{34} = \frac{a_2(1-\theta)\mu_h}{\mu_h + \psi},\ R_{19} = R_{39} = \frac{a_1(1-\theta)\mu_h}{\mu_h + \psi},$$
$$R_{29} = \frac{a\theta\psi}{\mu_h + \psi},\ R_{33} = \mu_h + \gamma_h,\ R_{64} = R_{84} = \frac{(1-\lambda)\varepsilon_1\mu_c}{\mu_c + \omega},\ R_{66} = \mu_c + \omega,$$
$$R_{74} = \frac{\lambda\varepsilon\omega}{\mu_c + \omega},\ R_{88} = (\mu_c + \gamma_c),\ R_{68} = R_{89} = \frac{(1-\lambda)\varepsilon_2\mu_c}{\mu_c + \omega} \text{ and } R_{99} = \mu_c + \alpha_c.$$

Now, the right and left eigenvectors associated with the zero eigenvalues are calculated. The right eigenvector associated with the zero eigenvalue is given by $w=(w_1, w_2, ..., w_9)^T$, which results in the following equations:

$$\begin{cases} -R_{11}w_1 - R_{14}w_4 - R_{19}w_9 = 0, \\ \psi w_1 - \mu_h w_2 - R_{29}w_9 = 0, \\ -R_{33}w_3 + R_{34}w_4 + R_{39}w_9 = 0, \\ \gamma_h w_3 - R_{44}w_4 = 0, \\ \rho_h w_4 - R_{55}w_5 = 0, \\ -R_{64}w_4 - R_{66}w_6 - R_{68}w_8 = 0, \\ -R_{74}w_4 + \omega w_6 - \mu_c w_7 = 0, \\ R_{84}w_4 - R_{88}w_8 + R_{89}w_9 = 0, \\ \gamma_c w_8 - R_{99}w_9 = 0. \end{cases} \quad (15)$$

Solving the system (15) for w_i's, i=1, 2, ..., 9, gives the following right eigenvectors

$$\begin{aligned} w_1 &= \frac{-1}{R_{11}}(R_{14}w_4 + R_{19}w_9) \implies w_1 < 0, \\ w_2 &= \frac{-1}{\mu_h R_{11}}\left[(R_{11}R_{29} + \psi R_{19})w_9 + \psi R_{14}w_4\right] \implies w_2 < 0, \\ w_3 &= \frac{1}{R_{33}}(R_{34}w_4 + R_{39}w_9) \implies w_3 > 0, \\ w_4 &= \frac{\gamma_h w_3}{R_{44}} \implies w_4 > 0, \\ w_5 &= \frac{\rho_h\gamma_h}{R_{33}R_{44}R_{55}}(R_{34}w_4 + R_{39}w_9) \implies w_5 > 0, \\ w_6 &= \frac{-1}{R_{66}}\left[\frac{R_{64}\gamma_h}{R_{33}R_{44}}(R_{34}w_4 + R_{39}w_9) + \frac{R_{68}}{R_{88}}(R_{84}w_4 + R_{89}w_9)\right] \implies w_6 < 0, \\ w_7 &= \frac{-R_{74}w_4}{\mu_c} - \omega\left[\frac{R_{64}\gamma_h(R_{34}w_4 + R_{39}w_9)}{\mu_c R_{33}R_{44}R_{66}} + \frac{R_{68}(R_{84}\omega_4 + R_{89}w_9)}{\mu_c R_{66}R_{88}}\right] \implies w_7 < 0, \\ w_8 &= \frac{1}{R_{88}}(R_{84}w_4 + R_{89}w_9) \implies w_8 > 0, \\ w_9 &= \frac{\gamma_c}{R_{88}R_{99}}(R_{84}w_4 + R_{89}w_9) \implies w_9 > 0. \end{aligned}$$

furthermore, to calculate left eigenvector given by $v=(v_1, v_2, ..., v_9)^T$, which satisfy v. w=1, the matrix $J(\varepsilon^*_2)$ is transposed and becomes.

$$J(\varepsilon_2^*)^T = \begin{pmatrix} -R_{11} & \psi & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\mu_h & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -R_{33} & \gamma_h & 0 & 0 & 0 & 0 & 0 \\ R_{14} & 0 & R_{34} & -R_{44} & \rho_h & R_{64} & R_{74} & R_{84} & 0 \\ 0 & 0 & 0 & 0 & -R_{55} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -R_{66} & \omega & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\mu_c & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & R_{68} & 0 & -R_{88} & \gamma_c \\ -R_{19} & -R_{29} & R_{39} & 0 & 0 & 0 & 0 & R_{89} & -R_{99} \end{pmatrix},$$

Then solving $J(\varepsilon^*_2)^T$, the following are obtained

$$\begin{aligned}
-R_{11}v_1 + \psi v_2 &= 0,\\
-\mu_h v_2 &= 0,\\
-R_{33}v_3 + \gamma_h v_4 &= 0,\\
R_{14}v_1 + R_{34}v_3 - R_{44}v_4 + \rho_h v_5 + R_{64}v_6 + R_{74}v_7 + R_{84}v_8 &= 0,\\
-R_{55}v_5 &= 0,\\
-R_{66}v_6 + \omega v_7 &= 0,\\
-\mu_c v_7 &= 0,\\
R_{68}v_6 - R_{88}v_8 + \gamma_c v_9 &= 0,\\
R_{19}v_1 + R_{29}v_2 + R_{39}v_3 + R_{89}v_8 + R_{99}v_9 &= 0.
\end{aligned} \tag{16}$$

Solving the system (16) for v_i's, i = 1, 2, ..., 9, yields the following left eigenvectors

$$\begin{aligned}
v_1 &= 0,\\
v_2 &= 0,\\
v_3 &= \frac{\gamma_h}{R_{33}}v_4\\
v_4, &= \frac{R_{34}}{R_{44}}v_3 + \frac{R_{84}\gamma_c}{R_{44}R_{88}}v_9,\\
v_5 &= 0,\\
v_6 &= 0,\\
v_7 &= 0,\\
v_8 &= \frac{\gamma_c}{R_{88}}v_9,\\
v_9 = v_9 &> 0.
\end{aligned}$$

From [6], Theorem 4:1 is used to establish the conditions for the existence of forward or backward bifurcations of the endemic equilibrium point near R_0=1.

Lemma 5. Consider the following general system of ordinary differential equations with a parameter β such that

$$\frac{dx}{dt} = f(x,\beta),\ \ f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R} \ and\ f \in \mathbb{C}^2(\mathbb{R}^n \times \mathbb{R}), \tag{17}$$

such that f (0, β) ≡ 0, where 0 is an equilibrium point of the system with the following conditions:

$$A = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_i}(0,0)\right)$$

is the linearization matrix of the model system (1) around the equilibrium 0 with β evaluated at 0.

Zero is a simple eigenvalue of A, and all other eigenvalues of A have negative real parts.

Matrix A has a non-negative right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue.

Let f_k be the k^{th} components of f such that

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0) \ \text{and}\ b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}(0,0).$$

The sign of a and b always determines the local dynamics of the system around the equilibrium point.

a>0, b>0. When β <0 with |β| ≪ 1; 0 is locally asymptotically stable and there exists a positive unstable equilibrium; when 0< β ≪ 1; 0 is unstable and there exists a negative, locally asymptotically stable equilibrium.

a<0, b<0. When β <0 with |β| ≪ 1; 0 is locally asymptotically stable and there exists a positive unstable equilibrium; when 0< β ≪ 1; 0 is asymptotically stable equilibrium, and there exists a positive unstable equilibrium.

a<0, b<0. When β <0 with |β| ≪ 1; 0 is unstable, and a positive unstable equilibrium appears.

a<0, b>0. When β changes from positive to negative, 0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium becomes positive and locally asymptotically stable. Particularly, if a>0 and b>0, then, a subcritical (or backward) bifurcation occurs at β =0.

Computation of bifurcation coefficients to determine the local dynamics of the transformed system (14), the value of an and b are computed to probe whether the model system (14) shows forward or backward bifurcation. Since v_1=v_2=v_5=v_6=v_7=0 for k=1; 2; 5; 6; 7 then k=3; 4; 8; 9 are considered. For the system (14), the associated non-zero second order partial derivatives at disease-free equilibrium and at ε_2= ε^*_2 are given by

$$\frac{\partial^2 f_3}{\partial x_1 \partial x_9} = \frac{(1-\theta)a_1\psi\mu_h}{\pi_h(\psi+\mu_h)},\ \frac{\partial^2 f_3}{\partial x_1 \partial x_4} = \frac{(1-\theta)a_2\psi\mu_h}{\pi_h(\psi+\mu_h)},\ \frac{\partial^2 f_3}{\partial x_2 \partial x_9} = \frac{\theta a\mu_h}{\pi_h},$$

$$\frac{\partial^2 f_8}{\partial x_4 \partial x_6} = \frac{(1-\lambda)\varepsilon_1\omega\mu_c}{\pi_c(\omega+\mu_c)},\ \frac{\partial^2 f_8}{\partial x_6 \partial x_9} = \frac{(1-\lambda)\varepsilon_2^*\psi\mu_c}{\pi_c(\omega+\mu_c)},\ \frac{\partial^2 f_8}{\partial x_4 \partial x_7} = \frac{\lambda\varepsilon\mu_c}{\pi_c}.$$

Therefore,

$$\begin{aligned}
a =& v_3 w_1 w_9 \frac{(1-\theta)a_1\psi\mu_h}{\pi_h(\psi+\mu_h)} + v_3 w_1 w_4 \frac{(1-\theta)a_2\psi\mu_h}{\pi_h(\psi+\mu_h)} + v_3 w_2 w_9 \frac{\theta a\mu_h}{\pi_h} + v_8 w_4 w_6 \frac{(1-\lambda)\varepsilon_1\omega\mu_c}{\pi_c(\omega+\mu_c)}\\
&+ v_8 w_6 w_9 \frac{(1-\lambda)\varepsilon_2^*\psi\mu_c}{\pi_c(\omega+\mu_c)} + v_8 w_4 \omega_7 \frac{\lambda\varepsilon\mu_c}{\pi_c},\\
a =& -\left[w_9\left(A_1\frac{(1-\theta)a_1\psi\mu_h}{\pi_h(\psi+\mu_h)} + A_2\frac{\theta a\mu_h}{\pi_h} + A_3\frac{(1-\lambda)\varepsilon_2^*\psi\mu_c}{\pi_c(\omega+\mu_c)}\right) + w_4\left(B_1\frac{(1-\theta)a_2\psi\mu_h}{\pi_h(\psi+\mu_h)} + \right.\right.\\
&\left.\left. B_2\frac{(1-\lambda)\varepsilon_1\omega\mu_c}{\pi_c(\omega+\mu_c)} + B_3\frac{\lambda\varepsilon\mu_c}{\pi_c}\right)\right] < 0,
\end{aligned}$$

where;

$$A_1 = B_1 = -\frac{\gamma_h v_4}{R_{11}R_{33}}(R_{14}w_4 + R_{19}w_9),$$

$$A_2 = -\frac{\gamma_h v_4}{\mu_h R_{11}R_{33}}\left[(R_{11}R_{29} + \psi R_{19})w_9 + \psi R_{14}w_4\right],$$

$$A_3 = B_2 = -\frac{\gamma_c v_9}{R_{66}R_{88}}\left[\frac{R_{64}\gamma_h}{R_{33}R_{44}}(R_{34}w_4 + R_{39}w_9) + \frac{R_{68}}{R_{88}}(R_{84}w_4 + R_{89}w_9)\right],$$

$$A_4 = -\frac{\gamma_h v_4}{R_{33}R_{66}}\left[\frac{R_{64}\gamma_h}{R_{33}R_{44}}(R_{34}w_4 + R_{39}w_9) + \frac{R_{68}}{R_{88}}(R_{84}w_4 + R_{89}w_9)\right],$$

$$B_3 = -\frac{R_{74}\omega_4\gamma_c v_9}{\mu_c R_{88}} - \omega\left[\frac{R_{64}\gamma_h\gamma_c v_9(R_{34}wa_4 + R_{39}w_9)}{\mu_c R_{33}R_{44}R_{66}R_{88}} + \frac{R_{68}\gamma_c v_9(R_{84}w_4 + R_{89}w_9)}{\mu_c R_{66}R_{88}^2}\right]$$

For the value of b, it can be shown that there exists a non-vanishing partial derivative.

$$\frac{\partial^2 f_8}{\partial x_9 \partial \varepsilon_2^*} = \frac{(1-\lambda)\varepsilon_2^*\psi\mu_c}{\omega+\mu_c}, \text{ which follows that } b = v_8 w_9 \frac{(1-\lambda)\varepsilon_2^*\psi\mu_c}{\omega+\mu_c} > 0.$$

Therefore, a<0 and b>0, hence the model system (1) exhibit a forward bifurcation.

Lemma 6. The unique endemic equilibrium is guaranteed and by 3.2, the model is locally asymptotically stable for R0>1. In addition, by 3.2 item (i), the model undergoes backward bifurcation when a>0. This holds only if v3<0 and v8<0, otherwise it undergoes forward bifurcation.

RESULTS AND DISCUSSION

Bifurcation Diagram

Figure 2 illustrates the forward bifurcation (transcritical bifurcation) at R_0=1 when a<0 and b>0 for a model system described by equation (1). Thus, the bifurcation diagram for the model system shows that the disease-free equilibrium and endemic equilibria exchange stability when R_0=1. This scenario indicates biologically that the model system (1) is globally asymptotically stable at the disease-free level. The equilibrium point emerges when R_0<1, and a unique endemic equilibrium point results whenever R_0>1. The unique endemic equilibrium point is locally asymptotically stable when R_0 is near one. Observably, as R_0 decreases below one (R_0<1), no endemicity exists, and the disease wanes. As R_0 rises above one (R_0>1), the disease spreads through the population.

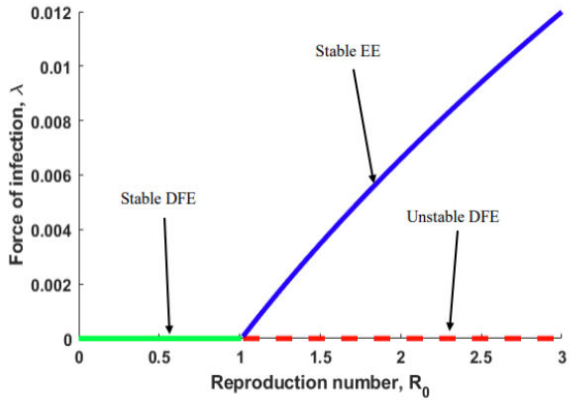

Figure 2: Forward bifurcation diagram for the model system (1) illustrating how that the disease-free equilibrium and endemic equilibria exchange stability when R_0=1. The cyan and blue curve indicate stable equilibria whereas the dashed red curve signifies unstable equilibrium.

Global Stability of Endemic Equilibrium Point

This subsection deploys the Lyapunov function method to study the global stability of the Endemic Equilibrium Point (EEP) beyond just the issue of neighborhood equilibrium points. The Lyapunov 25 function enables the extension of the analysis to capture an enormous region, as opposed to relying only on a strip region.

Theorem 3.3. If R_e>1, then the bovine tuberculosis disease model system (1) has a unique equilibrium point E_* which is globally asymptotically stable.

Proof. Since a suitable Lyapunov function is developed using approach, the Lyapunov function will then be applied to analyze the global stability of the endemic equilibrium point of the model system (1). Therefore, the Lyapunov function is developed using the general form as illustrated below:

$$L=\sum_{i=1}^{9} c_i(x_i-x_i^*\ln x_i)=c_1(x_1-x_1^*\ln x_1)+c_2(x_2-x_2^*\ln x_2)+c_3(x_3-x_3^*\ln x_3)$$
$$+c_4(x_4-x_4^*\ln x_4)+c_5(x_5-x_5^*\ln x_5)+c_6(x_6-x_6^*\ln x_6)+c_7(x_7-x_7^*\ln x_7)$$
$$+c_8(x_8-x_8^*\ln x_8)+c_9(x_9-x_9^*\ln x_9).$$

where c_i is a positive constant, x_i is the population of the i^{th} compartment and x^*_i is the endemic equilibrium point. Thus, the following Lyapunov function is constructed:

$$L=c_1(S_{nh}-S_{nh}^*\ln S_{nh})+c_2(S_{eh}-S_{eh}^*\ln S_{eh})+c_3(E_h-E_h^*\ln E_h)$$
$$+c_4(I_h-I_h^*\ln I_h)+c_5(R_h-R_h^*\ln R_h)+c_6(S_{nv}-S_{nv}^*\ln S_{nv})$$
$$+c_7(S_v-S_v^*\ln S_v)+c_8(E_c-E_c^*\ln E_c)+c_9(I_c-I_c^*\ln I_c).$$

Since the chosen function L and its constant are always differentiable and continuous, the differentiating L regarding time yields the following results:

$$\begin{aligned}\frac{dL}{dt}=&c_1\left(1-\frac{S_{nh}^*}{S_{nh}}\right)\frac{dS_{nh}}{dt}+c_2\left(1-\frac{S_{eh}^*}{S_{eh}}\right)\frac{dS_{eh}}{dt}+c_3\left(1-\frac{E_h^*}{E_h}\right)\frac{dE_h}{dt}\\&+c_4\left(1-\frac{I_h^*}{I_h}\right)\frac{dI_h}{dt}+c_5\left(1-\frac{R_h^*}{R_h}\right)\frac{dR_h}{dt}+c_6\left(1-\frac{S_{nv}^*}{S_{nv}}\right)\frac{dS_{nv}}{dt}\\&+c_7\left(1-\frac{S_v^*}{S_v}\right)\frac{dS_v}{dt}+c_8\left(1-\frac{E_c^*}{E_c}\right)\frac{dE_c}{dt}+c_9\left(1-\frac{I_c^*}{I_c}\right)\frac{dI_c}{dt}.\end{aligned}\tag{18}$$

Substituting the model equations of the model system (1) into the equation (18) to obtain

$$\begin{aligned}\frac{dL}{dt}=&c_1\left(1-\frac{S_{nh}^*}{S_{nh}}\right)(\pi_h-(\mu_h+\psi+\varphi_1)S_{nh})\\&+c_2\left(1-\frac{S_{eh}^*}{S_{eh}}\right)(\psi S_{nh}-(\mu_h+\varphi_2)S_{eh})\\&+c_3\left(1-\frac{E_h^*}{E_h}\right)(\varphi_2 S_{eh}+\varphi_1 S_{nh}-(\mu_h+\gamma_h)E_h+\tau_h R_h)\\&+c_4\left(1-\frac{I_h^*}{I_h}\right)(\gamma_h E_h-(\mu_h+\alpha_h+\rho_h)I_h)\\&+c_5\left(1-\frac{R_h^*}{R_h}\right)(\rho_h I_h-(\mu_h+\tau_h)R_h)\\&+c_6\left(1-\frac{S_{nv}^*}{S_{nv}}\right)(\pi_c-(\mu_c+\omega+\sigma_1)S_{nv})\\&+c_7\left(1-\frac{S_v^*}{S_v}\right)(\omega S_{nv}-(\mu_c+\sigma_2)S_v)\\&+c_8\left(1-\frac{E_c^*}{E_c}\right)(\sigma_1 S_{nv}+\sigma_2 S_v-(\mu_c+\gamma_c)E_c)\\&+c_9\left(1-\frac{I_c^*}{I_c}\right)(\gamma_c E_c-(\mu_c+\alpha_c)I_c).\end{aligned}\tag{19}$$

Now, at the endemic equilibrium point of the model system (1) the following are results.

Case 1: Considering the differential equations of human population, then making π_h, ψ, τ_h, γ_h and ρ_h the subject yields the following:

$$\begin{cases} \pi_h = (\mu_h + \psi + \varphi_1^*)S_{nh}^*, \\ \psi = (\mu_h + \varphi_2^*)\dfrac{S_{eh}^*}{S_{nh}^*}, \\ \tau_h = -\dfrac{1}{R_h^*}\left(\varphi_2^* S_{eh}^* + \varphi_1^* S_{nh}^* + (\mu_h + \gamma_h)E_h^*\right), \\ \gamma_h = (\mu_h + \alpha_h + \rho_h)\dfrac{I_h^*}{E_h^*}, \\ \rho_h = (\mu_h + \tau_h)\dfrac{R_h^*}{I_h^*}. \end{cases} \tag{20}$$

Case 2: Considering the differential equations of cattle population and making π_c, ω, $(\mu_c+\gamma_c)$ and γ_c the subject yields the following:

$$\begin{cases} \pi_c = (\mu_c + \omega + \sigma_1^*)S_{nv}^*, \\ \omega = (\mu_c + \sigma_2^*)\dfrac{S_v^*}{S_{nv}^*}, \\ (\mu_c + \gamma_c) = \dfrac{1}{E_c^*}(\sigma_2^* S_v^* + \sigma_1^* S_{nv}^*), \\ \gamma_c = (\mu_h + \alpha_c)\dfrac{I_c^*}{E_c^*}. \end{cases} \tag{21}$$

Now, substituting equations (20) and (21) into the equation, then dL/dt becomes.

$$\begin{aligned} \frac{dL}{dt} =& c_1\left(1 - \frac{S_{nh}^*}{S_{nh}}\right)\left[(\mu_h + \psi + \varphi_1^*)S_{nh}^* - (\mu_h + \psi + \varphi_1^*)S_{nh})\right] \\ &+ c_2\left(1 - \frac{S_{eh}^*}{S_{eh}}\right)\left[(\mu_h + \varphi_2^*)\frac{S_{eh}^*}{S_{nh}^*}S_{nh} - (\mu_h + \varphi_2^*)S_{eh}\right] \\ &+ c_3\left(1 - \frac{E_h^*}{E_h}\right)(\varphi_2 S_{eh} + \varphi_1 S_{nh} - (\mu_h + \gamma_h)E_h \\ &- c_3\left(\frac{1}{R_h^*}\left(\varphi_2^* S_{eh}^* + \varphi_1^* S_{nh}^* + (\mu_h + \gamma_h)E_h^*\right)R_h\right) \\ &+ c_4\left(1 - \frac{I_h^*}{I_h}\right)\left[(\mu_h + \alpha_h + \rho_h)\frac{I_h^*}{E_h^*}E_h - (\mu_h + \alpha_h + \rho_h)I_h\right] \\ &+ c_5\left(1 - \frac{R_h^*}{R_h}\right)\left[(\mu_h + \tau_h)\frac{R_h^*}{I_h^*}I_h - (\mu_h + \tau_h)R_h\right] \\ &+ c_6\left(1 - \frac{S_{nv}^*}{S_{nv}}\right)\left[(\mu_c + \omega + \sigma_1^*)S_{nv}^* - (\mu_c + \omega + \sigma_1^*)S_{nv}\right] \\ &+ c_7\left(1 - \frac{S_v^*}{S_v}\right)\left[(\mu_c + \sigma_2^*)\frac{S_v^*}{S_{nv}^*}S_{nv} - (\mu_c + \sigma_2)S_v\right] \\ &+ c_8\left(1 - \frac{E_c^*}{E_c}\right)\left[\sigma_1 S_{nv} + \sigma_2 S_v - \frac{1}{E_c^*}(\sigma_2^* S_v^* + \sigma_1^* S_{nv}^*)E_c\right] \\ &+ c_9\left(1 - \frac{I_c^*}{I_c}\right)\left[(\mu_h + \alpha_c)\frac{I_c^*}{E_c^*}E_c - (\mu_c + \alpha_c)I_c\right]. \end{aligned} \tag{22}$$

To investigate the global stability of the endemic equilibrium using the Lyapunov function approach at the steady state, set $dN^*_h/dt=0$ and $dN^*_c/dt=0$, such that:

$$\frac{dN_h^*}{dt} = \pi_h - \mu_h N_h^* - \alpha_h I_h^* = 0 \implies N_h^* = \frac{\pi_h - \alpha_h I_h^*}{\mu_h}, \tag{23}$$

$$\frac{dN_c^*}{dt} = \pi_c - \mu_c N_c^* - \alpha_c I_c^* = 0 \implies N_c^* = \frac{\pi_c - \alpha_c I_c^*}{\mu_c}. \tag{24}$$

Substituting equation (23) and (24) into forces of infections to obtain:

$$\begin{aligned} \varphi_1^* &= \frac{\mu_h(1-\theta)}{\pi_h - \alpha_h I_h^*}(a_1 I_c^* + a_2 I_h^*),\ \varphi_2^* = a\theta\frac{\mu_h I_c^*}{\pi_h - \alpha_h I_h^*}. \\ \sigma_1^* &= \frac{\mu_c(1-\lambda)}{\pi_c - \alpha_c I_c^*}(\varepsilon_1 I_h^* + \varepsilon_2 I_c^*),\ \sigma_2^* = \varepsilon\lambda\frac{\mu_c I_h^*}{\pi_c - \alpha_c I_c^*}. \end{aligned} \tag{25}$$

After substituting equation (25) into (26), the equation becomes

$$\begin{aligned} \frac{dL}{dt} =& c_1\left(1 - \frac{S_{nh}^*}{S_{nh}}\right)\left[\left(\mu_h + \psi + \frac{\mu_h(1-\theta)(a_1 I_c^* + a_2 I_h^*)}{\pi_h - \alpha_h I_h^*}\right)S_{nh}^*\right. \\ &\left. - \left(\mu_h + \psi + \frac{\mu_h(1-\theta)(a_1 I_c^* + a_2 I_h^*)}{\pi_h - \alpha_h I_h^*}\right)S_{nh}\right] \\ &+ c_2\left(1 - \frac{S_{eh}^*}{S_{eh}}\right)\left[\left(\mu_h + \frac{a\theta\mu_h I_c^*}{\pi_h - \alpha_h I_h^*}\right)\frac{S_{eh}^*}{S_{nh}^*}S_{nh} - \left(\mu_h + \frac{a\theta\mu_h I_c^*}{\pi_h - \alpha_h I_h^*}\right)S_{eh}\right] \\ &+ c_3\left(1 - \frac{E_h^*}{E_h}\right)\left(\frac{a\theta\mu_h I_c^*}{\pi_h - \alpha_h I_h^*}\right)S_{eh} + \frac{\mu_h(1-\theta)(a_1 I_c^* + a_2 I_h^*)}{\pi_h - \alpha_h I_h^*}S_{nh} - (\mu_h + \gamma_h)E_h \\ &- \left(\frac{1}{R_h^*}\left(\frac{a\theta\mu_h I_c^*}{\pi_h - \alpha_h I_h^*}S_{eh}^* + \frac{\mu_h(1-\theta)(a_1 I_c^* + a_2 I_h^*)}{\pi_h - \alpha_h I_h^*}S_{nh}^* + (\mu_h + \gamma_h)E_h^*\right)R_h\right) \\ &+ c_4\left(1 - \frac{I_h^*}{I_h}\right)\left[(\mu_h + \alpha_h + \rho_h)\frac{I_h^*}{E_h^*}E_h - (\mu_h + \alpha_h + \rho_h)I_h\right] \\ &+ c_5\left(1 - \frac{R_h^*}{R_h}\right)\left[(\mu_h + \tau_h)\frac{R_h^*}{I_h^*}I_h - (\mu_h + \tau_h)R_h\right] \\ &+ c_6\left(1 - \frac{S_{nv}^*}{S_{nv}}\right)\left[\left(\mu_c + \omega + \frac{\mu_c(1-\lambda)(\varepsilon_1 I_h^* + \varepsilon_2 I_c^*)}{\pi_c - \alpha_c I_c^*}\right)S_{nv}^*\right. \\ &\left. - \left(\mu_c + \omega + \frac{\mu_c(1-\lambda)(\varepsilon_1 I_h^* + \varepsilon_2 I_c^*)}{\pi_c - \alpha_c I_c^*}\right)S_{nv}\right] \\ &+ c_7\left(1 - \frac{S_v^*}{S_v}\right)\left[\left(\mu_c + \frac{\varepsilon\lambda\mu_c I_h^*}{\pi_c - \alpha_c I_c^*}\right)\frac{S_v^*}{S_{nv}^*}S_{nv} - \left(\mu_c + \frac{\varepsilon\lambda\mu_c I_h^*}{\pi_c - \alpha_c I_c^*}\right)S_v\right] \\ &+ c_8\left(1 - \frac{E_c^*}{E_c}\right)\left[\frac{\mu_c(1-\lambda)(\varepsilon_1 I_h^* + \varepsilon_2 I_c^*)}{\pi_c - \alpha_c I_c^*}S_{nv} + \frac{\varepsilon\lambda\mu_c I_h^*}{\pi_c - \alpha_c I_c^*}S_v\right. \\ &\left. - \frac{1}{E_c^*}\left(\frac{\varepsilon\lambda\mu_c I_h^*}{\pi_c - \alpha_c I_h^*}S_v^* + \frac{\mu_c(1-\lambda)(\varepsilon_1 I_h^* + \varepsilon_2 I_c^*)}{\pi_c - \alpha_c I_c^*}S_{nv}^*\right)E_c\right] \\ &+ c_9\left(1 - \frac{I_c^*}{I_c}\right)\left[(\mu_h + \alpha_c)\frac{I_c^*}{E_c^*}E_c - (\mu_c + \alpha_c)I_c\right]. \end{aligned} \tag{26}$$

Simplifying equation (26), it gives

$$\begin{aligned} \frac{dL}{dt} =& c_1\left(1 - \frac{S_{nh}^*}{S_{nh}}\right)\left[(\mu_h + \psi)S_{nh}^* - (\mu_h + \psi)S_{nh})\right] \\ &+ c_6\left(1 - \frac{S_{nv}^*}{S_{nv}}\right)\left[(\mu_c + \omega +)S_{nv}^* - (\mu_c + \omega)S_{nv}\right] \\ &+ \Phi(P) \end{aligned} \tag{27}$$

$$\frac{dL}{dt} = -c_1(\mu_h + \psi)\left(1 - \frac{S_{nh}^*}{S_{nh}}\right)^2 S_{nh} - c_6(\mu_c + \omega)\left(1 - \frac{S_{nv}^*}{S_{nv}}\right)^2 S_{nv} + \Phi(P) \tag{28}$$

where $P = (S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c)$ such that P is non negative. Therefore

$$\begin{aligned}\Phi(P) = &- c_1\left(\frac{\mu_h(1-\theta)(a_1 I_c^* + a_2 I_h^*)}{\pi_h - \alpha_h I_h^*}\right)\left(1 - \frac{S_{nh}^*}{S_{nh}}\right)^2 S_{nh} \\ &- c_2\left(\mu_h + \frac{a\theta\mu_h I_c^*}{\pi_h - \alpha_h I_h^*}\right)\left(1 - \frac{S_{eh}^*}{S_{eh}}\right)\left(1 - \frac{S_{eh}^* S_{nh}}{S_{eh} S_{nh}^*}\right) S_{eh} \\ &- c_3\left(\frac{\mu_h(1-\theta)(a_1 I_c^* + a_2 I_h^*)}{\pi_h - \alpha_h I_h^*}\right)\left(1 - \frac{E_h^*}{E_h}\right)\left(1 - \frac{S_{nh}}{S_{nh}^*}\right)\frac{R_h}{R_h^*} S_{nh}^* \\ &- c_3\left(\frac{a\theta\mu_h I_c^*}{\pi_h - \alpha_h I_h^*}\right)\left(1 - \frac{E_h^*}{E_h}\right)\left(1 - \frac{S_{nh}}{S_{nh}^*}\right)\frac{R_h}{R_h^*} S_{nh}^* \\ &- c_3(\mu_h + \gamma_h)\left(1 - \frac{E_h^*}{E_h}\right)^2 \frac{R_h}{R_h^*} E_h \\ &- c_4(\mu_h + \alpha_h + \rho_h)\left(1 - \frac{I_h^*}{I_h}\right)\left(1 - \frac{I_h^* E_h}{I_h E_h^*}\right) I_h \\ &- c_5(\mu_h + \tau_h)\left(1 - \frac{R_h^*}{R_h}\right)\left(1 - \frac{R_h^* I_h}{R_h I_h^*}\right) R_h \\ &- c_6\left(\frac{\mu_c(1-\lambda)(\varepsilon_1 I_h^* + \varepsilon_2 I_c^*)}{\pi_c - \alpha_c I_c^*}\right)\left(1 - \frac{S_{nv}^*}{S_{nv}}\right)^2 S_{nv} \\ &- c_7\left(\mu_c + \frac{\varepsilon\lambda\mu_c I_h^*}{\pi_c - \alpha_c I_c^*}\right)\left(1 - \frac{S_v^*}{S_v}\right)\left(1 - \frac{S_v^* S_{nv}}{S_v S_{nv}^*}\right) S_v \\ &- c_8\left(\frac{\mu_c(1-\lambda)(\varepsilon_1 I_h^* + \varepsilon_2 I_c^*)}{\pi_c - \alpha_c I_c^*}\right)\left(1 - \frac{E_c^*}{E_c}\right)\left(1 - \frac{E_c S_{nv}^*}{E_c^* S_{nv}}\right) E_c S_{nv} \\ &- c_8\left(\frac{\varepsilon\lambda\mu_c I_h^*}{\pi_c - \alpha_c I_c^*}\right)\left(1 - \frac{E_c^*}{E_c}\right)\left(1 - \frac{S_v^*}{E_c^* S_v}\right) S_v E_c \\ &- c_9(\mu_h + \alpha_c)\left(1 - \frac{I_c^*}{I_c}\right)\left(1 - \frac{I_c^* E_c}{I_c E_c^*}\right) I_c.\end{aligned}$$

Therefore, following the idea of the function Φ (S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c) is non positive, that is Φ (S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c) ≤ 0 for all (S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c). Then, this implies that dL/dt ≤ 0 for all (S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c) and it is zero only when $S_{nh}=S^*_{nh}$, $S_{eh}=S^*_{eh}$, $E_h=E*_h$, $I_h=I^*_h$, $R_h=R^*_h$, $S_{nv}=S^*_{nv}$, $S_v=S^*_v$, $E_c=E^*_c$, $I_c=I^*_c$. Therefore, the largest compact invariant set in S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c such that dL/dt=0 is the singleton (E_*) which is the endemic equilibrium point of the model system (1). [20] principle, indicates that (E_*) is globally asymptotically in the interior region of S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c. Now recalling the definition of Lyapunov stability, if L(S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c)>0 ∀(S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c) ∈ Ω \{E∗}, L(E∗)=0 and L′(S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c) ≤ 0 ∀(S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c) ∈ Ω \{E_*} then, $E_*=(S^*_{nh}, S^*_{eh}, E^*_h, I^*_h, R^*_h, S^*_{nv}, S^*_v, E^*_c, I^*_c)$ is stable. Furthermore, if L(S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c)>0 ∀(S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c) ∈ Ω\ {E_*}, L(E_*)=0 and L′ (S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, I_c)< 0 ∀(S_{nh}, S_{eh}, E_h, I_h, R_h, S_{nv}, S_v, E_c, Ic) ∈ Ω\{E_*} then, $E_*=(S^*_{nh}, S^*_{eh}, E^*_h, I^*_h, R^*_h, S^*_{nv}, S^*_v, E^*_c, I^*_c)$ is asymptotically stable.

Sensitivity and Numerical Analysis

Sensitivity Analysis: The sensitivity analysis helps identify the influence of model parameters on the effective reproduction number (R_e) and disease transmission. It determines which parameters and initial conditions affect the model output. Additionally, it informs researchers about which parameters require more numerical attention. The normalized forward sensitivity index of the variable Re depends on the differentiability of a parameter p and is defined as follows:

$$\Upsilon_{\psi}^{R_e} = \frac{\partial R_e}{\partial \psi} \times \frac{\psi}{R_e}.$$

where ψ is a parameter present in effective reproduction number R_e. For example, the sensitivity index of R_e corresponding to the parameter α is given as

$$\Upsilon_{\gamma_h}^{R_e} = \frac{\partial R_e}{\partial \gamma_h} \times \frac{\gamma_h}{R_e} = +1.32496 \times 10^{-6}.$$

Other indices are calculated using a similar approach and the results are displayed in **Table 2 and Figure 3**.

Table 2: Sensitivity indices of Re using parameter values in Table 1.

Parameter	Sensitivity index	Parameter	Sensitivity index
a_1	+3.00524 ×10^{-6}	a_2	+3.4392 × 10^{-8}
a	+1.10214 × 10^{-6}	ε_1	+1.33268 × 10^{-6}
ε	+1.67366 × 10^{-6}	ε_2	0.999994
ρ_h	−2.10205 × 10^{-6}	γ_h	+1.32496 × 10^{-6}
γ_c	0.999997	μ_h	+7.8354 × 10^{-7}
μ_c	−0.0694459	α_h	−4.34908 × 10^{-7}
α_c	−0.999997	ψ	−2.61227 × 10^{-6}
λ	−0.33333	ω	−0.930551
θ	−9.11778 × 10^{-6}	-	-

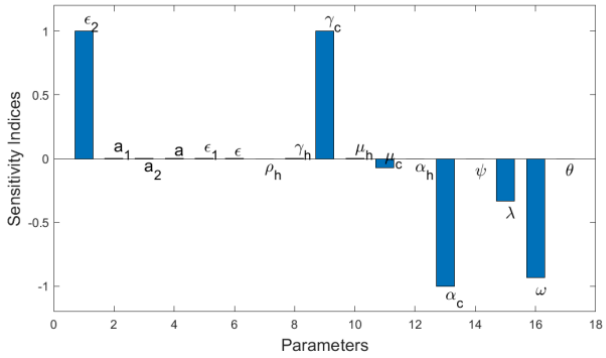

Figure 3: Graph of sensitivity indices of Re with respect to the model parameters.

Interpretation of the Sensitivity

Figure 3 displays the sensitivity profile of R_e concerning the model parameters found within R_e. Further analysis reveals that the parameters a_2, a_1, a, ε_1, ε_2, ε, γ_h, γ_c, and μ_h have positive indices, while μ_c, ρ_h, α_h, alpha$_c$, ψ, λ, ω, and θ have negative indices. Notably, parameters γ_c and ε_2 exhibit index values of +0.999997 and +0.999994, respectively, indicating that an increase in these parameters, while keeping other variables constant, elevates the effective reproduction number R_e.

In other words, increasing these parameters heightens the risk of a bTB outbreak in the wider population. To minimize infections in the cattle population, the rates γ_c and ε_2 must be kept sufficiently low to ensure that the removal rate from the exposed to infectious class remains minimal, while also maintaining a low contact rate in contaminated environments and inter-cattle transmission.

On the contrary, the vaccine parameter ω and the induced death rate α_c emerge as the most negatively sensitive parameters, with index values of α_c=−0.999997 and ω= −0.930551, respectively. This implies that increasing the value of ω by vaccinating healthy cattle and the value of α_c by culling infected animals, while keeping other parameters constant, reduces the effective reproduction number R_e, thus alleviating the disease burden among the cattle population and promoting disease free conditions in the human population.

Numerical Simulation

The most effective approach for accurately solving an Ordinary Differential Equation (ODE) is to carefully develop an exact solution, as discussed in. The chosen method must always uphold the standard properties of the approximated solution, including consistency and convergence, and it must also preserve the qualitative properties of the solution, such as boundedness and positivity, as highlighted. In this section, we employed the Runge-Kutta fourth (RK4) order method to obtain mathematical results for the model system due to its heuristic properties within the ODE framework. This method is known for its stability and practicality, even with large time steps, as demonstrated in previous studies. We utilized the ODE45 version in MATLAB software to execute the computations using the parameter values presented in **Table 1**. The initial conditions for the state variables were set as follows: S_{nh}=15000, S_{eh}=1000, E_h=1000, I_h=500, R_h=300, S_{nv}=15000, S_v=10, E_c=6000, and I_c=10. These initial conditions were arbitrarily chosen to illustrate specific behaviors of the model system (**Figure 4**).

Evolution of Population against Time

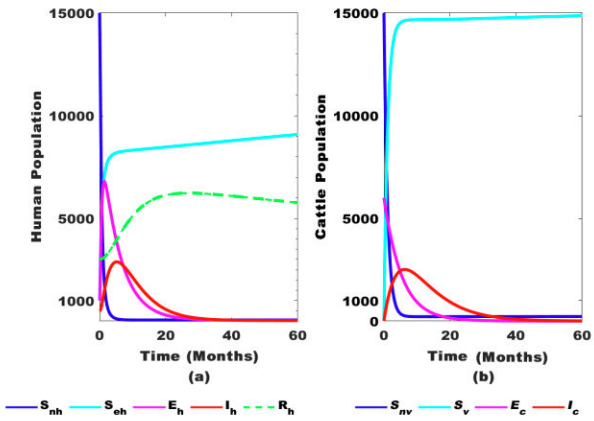

Figure 4: Dynamics of human and cattle populations over time with interventions.

Figure 4 depicts the behavior of the infected human and cattle populations as time progresses. **Figures 4(a) and 4(b)** indicate that interventions for both the human and cattle populations reduce the number of infected individuals significantly. This result attests to the existence of the DFE point in the model system (1). When ψ=0, ρ_h=0, and ω=0, it implies that interventions are not in place, meaning the populations of humans and cattle can never reach zero, no matter how long the EE point of the model system persists.

Simulation on the effects of varying some parameter values on the model this section presents data derived from the performance of the simulation of model system to explore the behavior of certain state variables over time. This simulation involved varying selected parameter values, as presented in **Table 1**. The simulation results are graphically displayed in **Figure 5**.

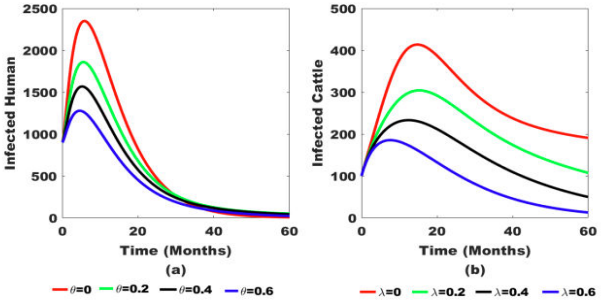

Figure 5: (a) Effect of education efficacy (θ) on infected human population and (b) Effect of vaccination efficacy (λ) on infected cattle population.

Figure 5 shows that education efficacy (θ) and vaccine efficacy (λ) play pivotal roles in reducing infectious hosts in humans and cattle. Thus, as education and vaccine efficacy approach 1, the number of infectious humans and cattle decreases.

From **Figure 6**, it can be surmised that the application of rifampicin, isoniazid, ethambutol, and pyrazinamide as medication for infected humans at the rate ρ_h and Bacille Calmette Gu´erin (BCG) vaccination for infected cattle at the rate ω leads to a reduction in the number of infected humans and cattle when employed on a large scale and applied effectively.

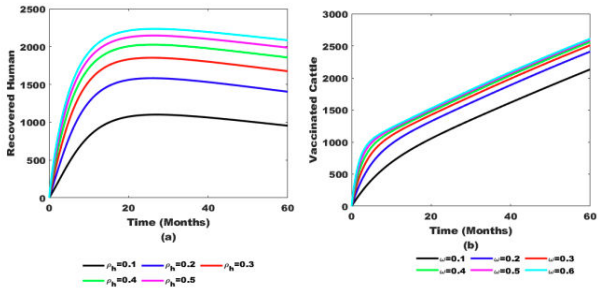

Figure 6: (a) Effect of treatment (ρh) on recovered human population and (b) Effect vaccine (ω) on cattle population.

Figure 6(b) reveals that when cattle are vaccinated to at least $\omega \geq 0.4$ with a minimum dose of 20 mg, the number of healthy cattle increases rapidly. Additionally, the number of recovered humans also rises, as shown in **Figure 6(a)**. Similarly, when treatment implementation reaches at least $\rho_h \geq 0.4$, it triggers a significant increase in the number of recovered humans.

Simulation on Environmental Contamination

This section presents results from the simulation of the infected human and cattle populations when pastures, soil, slurry, and hay are presumably heavily contaminated with *M. bovis*, with potential infections stemming from sputum, pus, urine, feces, and other excretions of infectious animals (**Figure 7**).

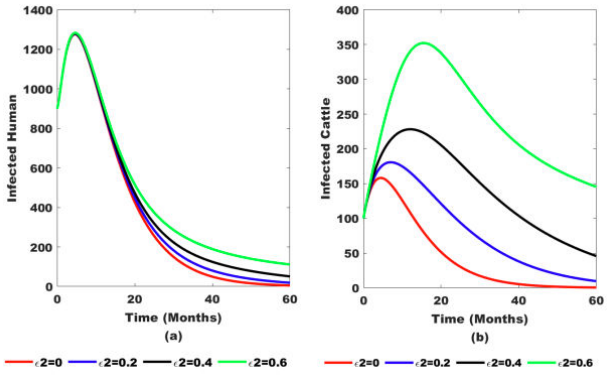

Figure 7: Impacts of contaminated environment (ϵ_2) on (a) Infected human population and (b) Infected cattle population.

Figure 7(b) illustrates that the number of infected cattle rises as the grazing rate (ϵ_2) on the heavily contaminated environment increases. However, after several months, the number of infected cattle decreases due to the disease.

Simulation on Multiple Intervention Strategies

This section presents data derived from simulating the infected human and cattle populations. It explores the outcomes resulting from the execution of various intervention strategies simultaneous (**Figure 8**).

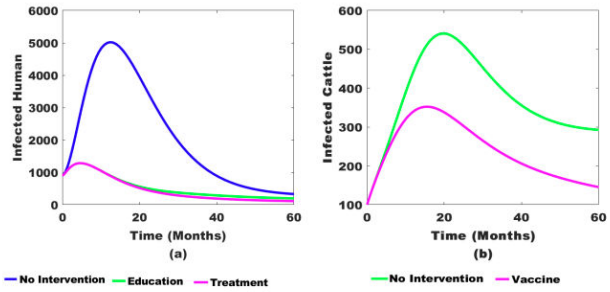

Figure 8: (a) Impacts of multiple interventions on infected human population and (b) Single intervention on infected cattle population.

Figure 8 shows the effects of a different combination of interventions (public health education campaigns, treatment, and vaccination) on the bTB transmission dynamics. Figure 8 shows that combining all three intervention strategies decreases disease transmission in the endemic area faster than using only two interventions.

CONCLUSION

This study has presented the formulation of the model for bTB disease transmission in the presence of control strategies, which include public health education campaigns, treatment, and vaccination. Specifically, it has clearly demonstrated the positivity and boundedness of the model system (1) for the model's domain to be biologically and mathematically meaningful. The results stemming from the sensitivity analysis on all model parameters, aimed at determining their relationship with the effective reproduction number (R_e), show that the probability of cattle contracting *M. bovis* from the contaminated environment, inter-cattle transmission (ε_2), and the removal rate from the exposed class to the infectious class (γ_c) are the most positively sensitive parameters for the effective reproduction number. In contrast, the most negatively sensitive parameters are ω (vaccination) and α_c (induced disease death rate), indicating that all infectious cattle are subject to culling in the absence of curative medication administration. Moreover, the numerical simulation of the model reveals that the combination of all interventions has the most significant impact on the control of bTB disease. Therefore, these control measures should be

implemented concurrently, especially in endemic areas, to effectively control bTB disease transmission.

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DECLARATION OF COMPETING INTEREST

Authors declare that they have no conflict of interest.

Credit authorship contribution statement

Sylas Oswald: Conceptualization, methodology, software, formal analysis, visualization, investigation and writing original draft

Theresia Crispin Marijan and Goodluck Mika Mlay: Conceptualization, methodology, formal analysis, investigation, writing, review, editing, resources and supervision.

Winifrida Benedict Kidima: Biology aspect visualization, investigation, writing, review, editing and supervision.

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