

**A MATHEMATICAL MODEL DEMONSTRATING THE EFFECT
OF QUARANTINE TECHNIQUE ON BOVINE
TUBERCULOSIS IN CATTLE**

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Abstract: Bovine Tuberculosis(BTB) is an infectious disease caused by a bacteria called Mycobacterium Bovis. It affects the lungs and lymph nodes of mammals which results in death. A mathematical model describing the BTB in Cattle incorporating both within-herd transmission and external infection is proposed. The external infection is caused by infected cattle from other herds. The true reactors to BTB are quarantined and the effect of quarantine in the control of BTB is analysed. A system of differential equations is used to formulate the mathematical model. The spread of BTB in the cattle is represented by a bilinear transmission. Stability analysis of the disease free state and endemic state of the system is carried out locally and globally. Analysis of the proposed model shows that the disease free state of the system is globally asymptotically stable when the basic reproduction number of the system is less than 1 and the endemic state of the system is globally asymptotically stable when the basic reproduction number is greater than 1. Numerical simulations reveal that, by quarantining true reactor cattle, the infection in herd can be controlled upto 60% effectively.

Keywords and Phrases: Mathematical Modelling, Bovine Tuberculosis, Differential Equations.

2020 Mathematics Subject Classification: 00A71, 93A30.

1. Introduction

The dynamics of Bovine Tuberculosis transmission are generally poorly understood, and the conditions under which a tuberculous animal becomes an effective disseminator of infection are still unknown. Although field research shows a broad range of transmission rates, the spread of infection is still commonly thought to proceed slowly. It has been demonstrated that the slaughter of sick calves found by tuberculin testing and meat plant inspection is an effective strategy for eliminating tuberculosis, given that no further infection reservoirs exist and that all parties involved in the cattle business are dedicated to this strategy. Case-control studies and other epidemiological methods appear to be the most effective. This encourages us to carry out research and construct the Bovine tuberculosis models in order to analyse the disease spread.

Bovine Tuberculosis(BTB) occurs commonly in cattle. It is also found in wildlife species and hoofed mammals. Although BTB is controlled in many countries with several control strategies like culling, thorough eradication of BTB is very challenging since there is a risk of disease spread from wildlife. BTB remains endemic in India because of the absence of control strategies and socio-economic conditions. The occurrence of BTB is likely to increase in future due to increasing complexities in cattle farming [11]. India is one of the countries which have massive cattle population thereby producing the highest quantity of milk on the planet. Therefore sustaining the quality and health of livestock is of most importance. The prevalence of BTB is assessed using SIT(single intradermal tuberculin) and gamma interferon (IFN- γ) assay. About 18% of the milk production in India is produced by Uttar Pradesh. Thakur *et al.* (2016) conducted a study and observed that 16.1% of the tested cattle were found to be positive for BTB in Uttar Pradesh [13].

Livestock sector plays a pivotal role in creating sustainable, gainful employment opportunities and supplementing income to small farmers and landless labourers. Moreover, it provides much needed balanced nutritious food and improves the household's food security. BTB is a serious disease that hinders the health of the cattle thereby causing low productivity in milk and animal husbandry [14]. BTB is not only a livestock issue but affects public health, wildlife, international trade, tourism, and many other areas of public and private interest. The call for a re-thinking of control efforts and their economic consequences is crucial. An economic assessment attempting to consider the societal effect of BTB must somehow address all of these issues. Consideration must be made for the incidence and prevalence of BTB disease, and hence its economic effect changes in a nonlinear way during an intervention [17]. Focussing on this issue, a literature review of BTB was conducted.

BTB spreads between species including humans. It may spread from wildlife to cattle or from cattle to buffalo or from cattle to humans. Mathematical modelling of BTB spread from one species to another has been an interesting area of research. Transmission routes of BTB from cattle to Buffalo was studied by Phepa *et al.*(2016). The studies show that cattle populations are more infected by BTB than buffalo populations [6]. Cattle that are tested for BTB are either in the P_r class (that have tested positive to the CDF test and are infected with Mycobacterium Bovis) or in the F_p class of false-positive cattle that have tested positive to BTB but are infected with environmental Mycobacterium or Mycobacterium avium; they are detected following a comparative test that distinguishes reactions to Mycobacterium Bovis from reactions to environmental Mycobacterium avium [2]. In this paper, a mathematical model of BTB spread in cattle incorporating quarantine of infected cattle is constructed and the efficiency of the control strategy is analysed. The infected cattle are identified by carrying out the tests for the detection of BTB. In this testing, the cattle that are true reactors are identified and quarantined. The model incorporates external infection and within-herd transmission. A bilinear incidence rate is used to model the transmission process.

2. Mathematical Model

The proposed system consists of six compartments, $S(t)$ - Susceptible population, $E(t)$ -Latent population, $I(t)$ - Infected population, $F_p(t)$ - False reactors to the test, $P_r(t)$ - True reactors to the test, $Q(t)$ - Quarantined population. The basic reproduction number (R_0) in the proposed system is the average number of cattle to which one infected cattle can spread the disease primarily during the course of infection. The total population is denoted by $N(t)$.

$$N(t) = S(t) + E(t) + I(t) + F_p(t) + P_r(t) + Q(t) \quad (2.1)$$

The following conditions are taken into consideration: (i) The population of cattle is generated by stocking or re-birth. (ii) $N(t)$ is a single herd. External infection rate is taken as Γ . (iii) There is no infection during stocking of herd or from infected wildlife.

The model description is as follows:

2.1. Susceptible Compartment: We assume that $0 < \tau < 1$. A is the number of cattle in the herd entering the Susceptible compartment. Γ is the rate of external infection that the herd acquires from neighboring cattle. βSI is the rate at which the susceptible cattle move to infected compartment due to infection within the herd. ϕ_1 is the rate at which the cattle in the susceptible compartment get tested falsely for BTB. μ is the natural death rate of the cattle. The equation can be

written as:

$$\frac{dS}{dt} = A\tau - (\beta I + \Gamma)S - (\phi_1 + \mu)S.$$

Table 1: List of parameters used in the system.

Parameter	Description
A	Number of cattle in a single herd
τ	Rate of rebirthing or stocking
β	Rate of transmission
Γ	External Infection
ϕ_1	Progression rate to F_p Class
ϕ_2	Progression rate to P_r Class from E
ϕ_3	Progression rate to P_r Class from I
η	Rate of quarantine
μ	Rate of natural death of cattle
γ	Progression rate from E to I
δ	Rate of death due to infection in cattle

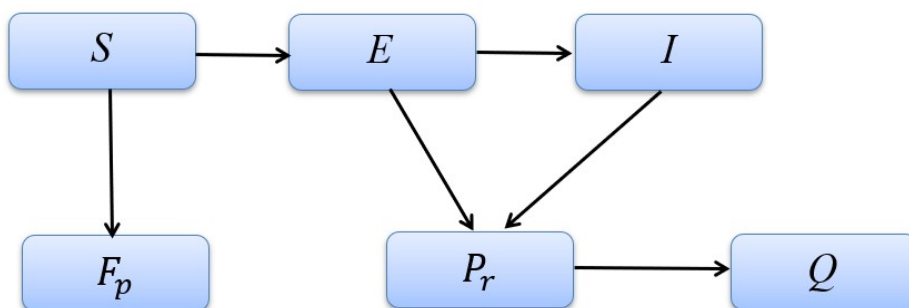


Figure 1: Mathematical model of Bovine Tuberculosis. Compartments are denoted by rectangles and parameters are denoted by circles.

2.2. Latent Compartment: The infected cattle enter the E compartment. It is denoted by the term $(\beta I + \Gamma)S$. The cattle remain in latent compartment for about 87–226 days to 7 years. ϕ_2 is the rate at which the cattle in the latent compartment get tested positive for BTB. The positively tested cattle in this latent compartment are true reactors to the test. γ is the progression rate from E to I . The equation

is given by

$$\frac{dE}{dt} = (\beta I + \Gamma) S - (\phi_2 + \gamma + \mu) E.$$

2.3. Infected Compartment: γ is the progression rate from E to I . ϕ_3 is the rate at which the cattle in the infected compartment get tested positive for BTB. The positively tested cattle in the infected compartment are true reactors to the test. The equation is given by

$$\frac{dI}{dt} = \gamma E - (\phi_3 + \mu) I.$$

2.4. False reactor Compartment: ϕ_1 is the rate at which the cattle in the susceptible compartment get tested false for bTB. The equation is given by

$$\frac{dF_p}{dt} = \phi_1 S - \mu F_p.$$

2.5. True reactor Compartment: ϕ_2 and ϕ_3 is the rate at which the cattle in the latent and infected compartment get tested positively for bTB. True reactors to the BTB are quarantined at a rate of η . It is denoted by

$$\frac{dP_r}{dt} = \phi_2 E + \phi_3 I - (\eta + \mu) P_r.$$

2.6. Quarantine Compartment: True reactors to the BTB are quarantined at a rate of η . δ is the death rate of cattle due to infection. It is denoted by

$$\frac{dQ}{dt} = \eta P_r - (\delta + \mu) Q$$

The proposed system is given by:

$$\begin{aligned} \frac{dS}{dt} &= A\tau - (\beta I + \Gamma) S - (\phi_1 + \mu) S \\ \frac{dE}{dt} &= (\beta I + \Gamma) S - (\phi_2 + \gamma + \mu) E \\ \frac{dI}{dt} &= \gamma E - (\phi_3 + \mu) I \\ \frac{dP_r}{dt} &= \phi_2 E + \phi_3 I - (\eta + \mu) P_r \\ \frac{dF_p}{dt} &= \phi_1 S - \mu F_p \\ \frac{dQ}{dt} &= \eta P_r - (\delta + \mu) Q \end{aligned} \tag{2.2}$$

Theorem 2.1. For the initial conditions, $S_n(t) > 0, E_n(t) > 0, I_n(t) > 0, (F_p)_n(t) > 0, (P_r)_n(t) > 0, Q_n(t) > 0$, the solutions of the system $(S(t), E(t), I(t), F_p(t), P_r(t), Q(t))$ are non negative for $t \geq 0$. Moreover, $\lim_{t \rightarrow \infty} N(t) \leq \frac{A\tau}{\mu}$.

Proof. Adding all the equations of system (2.2),

$$\frac{dN}{dt} \leq A\tau - \mu N \quad (2.3)$$

Applying limit on both sides, $\frac{dN}{dt} \rightarrow 0$ as $n \rightarrow \infty$

$$\lim_{\{t \rightarrow \infty\}} N(t) \leq \frac{A\tau}{\mu}$$

The region in which the solutions of the system (2.2) lie can be described as:

$$\Omega = \left\{ (S, E, I, F_p, P_r, Q) \in R_+^6 \mid N \leq \frac{A\tau}{\mu} \right\} \quad (2.4)$$

Theorem 2.2. The basic reproduction number of the system (2.2) is given by

$$R_0 = \frac{\beta\gamma\phi_1 A}{\mu} \quad (2.5)$$

Where $\alpha_1 = \Gamma + \phi_1 + \mu, \alpha_2 = \phi_2 + \gamma + \mu, \alpha_3 = \phi_3 + \mu$. Assume that $\alpha_1\alpha_2\alpha_3 < 1$.

3. Disease free equilibrium

The disease free equilibrium of system (2.2) is given by $\varpi^0 = (S^0, E^0, I^0, P_r^0, F_p^0, Q^0)$. The disease free equilibrium can be expressed as

$$\varpi^0 = \left(\frac{A\tau}{\Gamma + \phi_1 + \mu}, 0, 0, 0, \frac{A\phi_1\tau}{\Gamma + \phi_1 + \mu}, 0 \right)$$

Theorem 3.1. The Disease free equilibrium (DFE) of system (2.2) is locally asymptotically stable if $R_0 < 1$ and the following condition is satisfied

$$(\alpha_1 + \alpha_2 + \alpha_3) < 0 \quad (3.1)$$

Proof. Consider the Jacobian matrix of system (2.2) about ϖ^0 ,

$$J_1 = \begin{pmatrix} -(\Gamma + \phi_1 + \mu) & 0 & -\beta S^0 & 0 & 0 & 0 \\ \Gamma & -(\phi_2 + \gamma + \mu) & \beta S^0 & 0 & 0 & 0 \\ 0 & \gamma & -(\phi_3 + \mu) & 0 & 0 & 0 \\ 0 & \phi_2 & \phi_3 & -(\eta + \mu) & 0 & 0 \\ \phi_1 & 0 & 0 & 0 & -\mu & 0 \\ 0 & 0 & 0 & \eta & 0 & -(\delta + \mu) \end{pmatrix}$$

The three eigen values of the above matrix are $\lambda_1 = -(\eta + \mu)$, $\lambda_2 = -\mu$, $\lambda_3 = -(\delta + \mu)$. $|\lambda_i| \leq 1$ for $i = 1, 2, 3$. The remaining matrix can be written as,

$$J_1^* = \begin{pmatrix} -(\Gamma + \phi_1 + \mu) & 0 & -\beta S^0 \\ \Gamma & -(\phi_2 + \gamma + \mu) & \beta S^0 \\ 0 & \gamma & -(\phi_3 + \mu) \end{pmatrix}$$

Consider $|J_1^* - \lambda I| = 0$. The characteristic polynomial can be written as

$$\rho(\lambda) = \lambda^3 + g_1\lambda^2 + g_2\lambda + g_3 = 0 \tag{3.2}$$

where

$$\begin{aligned} g_1 &= -(\alpha_1 + \alpha_2 + \alpha_3) \\ g_2 &= \frac{1}{\alpha_1\alpha_2\alpha_3} \left[\frac{R_0}{\phi_1(\Gamma + \phi_1 + \mu)} - (\alpha_1 + \alpha_2 + \alpha_3) \right] \\ g_3 &= -\alpha_1\alpha_2\alpha_3 \left(\frac{R_0}{\phi_1(\Gamma + \phi_1 + \mu)} + 1 \right) \end{aligned}$$

From Jury’s conditions, the eigen values of the above characteristic equation is less than 1 iff $\rho(1) > 0$, $\rho(-1) < 0$ and $|J_1^*| < 1$ [8]. Hence the proof. It is observed that, in addition to the condition $R_0 < 1$, condition (8) must also be satisfied. The local stability of the system fails if either of the condition fails.

Theorem 3.2. *The Disease free equilibrium (DFE) of system (2.2) is globally asymptotically stable(GAS) if $R_0 < 1$.*

Proof. Consider the Lyapunov function, $\chi = \gamma\phi_1S + \beta\gamma E + \beta\alpha_1I + P_r + \gamma\alpha_1F_p + Q$
The Lyapunov derivative with respect to t can be expressed as,

$$\begin{aligned} \dot{\chi} &= \gamma\phi_1 \{A\tau - (\beta I + \Gamma) S - (\phi_1 + \mu) S\} + \beta\gamma \{(\beta I + \Gamma) S - \alpha_2 E\} \\ &\quad + \beta\alpha_2 \{\gamma E - \alpha_3 I\} + \{\phi_2 E + \phi_3 I - (\eta + \mu) P_r\} + \gamma\alpha_1 \{\phi_1 S - \mu F_p\} \\ &\quad \eta P_r - (\delta + \mu) Q \\ &< \gamma\phi_1 A\tau - \gamma\phi_1\beta IS + \beta\gamma(\beta I + \Gamma) S - \alpha_2\alpha_3\beta I \\ &\quad \phi_2 E + \phi_3 I - (\eta + \mu) P_r - \gamma\alpha_1\mu F_p - (\delta + \mu) Q \\ &< \beta I (R_0 - 1) + \gamma\phi_1 A\tau \left[1 - \frac{\beta\mu\alpha_1 + I}{R_0} \right] - (\eta + \mu) P_r - (\delta + \mu) Q \\ &< 0 \end{aligned}$$

Since the parameters in the system are non negative, for $R_0 < 1$, $\dot{\chi} \leq 0$. By LaSalle’s invariance principle, the solutions of the system (2.2) approach a disease

free equilibrium when $R_0 < 1$ [8]. Therefore, the Disease free equilibrium of system (2.2) is globally asymptotically stable.

4. Endemic equilibrium

Let $\hat{\omega} = (\hat{S}, \hat{E}, \hat{I}, \hat{P}_r, \hat{F}_p, \hat{Q})$ be the endemic equilibrium of the system (2.2), where $\hat{S} = \frac{A\tau}{\beta\hat{I} + \Gamma + \phi_1 + \mu}$, $\hat{E} = \frac{(\beta\hat{I} + \Gamma)\hat{S}}{\alpha_1}$, $\hat{P}_r = \frac{\phi_2\hat{E} + \phi_3\hat{I}}{\eta}$, $\hat{F}_p = \frac{\phi_1\hat{S}}{\mu}$, $\hat{Q} = \frac{\eta\hat{P}_r}{\delta + \mu}$, where

$$\hat{I} = \frac{\gamma A}{\alpha_1 \alpha_2 \alpha_3 \mu} = \frac{R_0}{\beta \phi_1}$$

By Theorem 2.1, the equilibrium point \hat{I} exists only if $R_0 > 1$.

Theorem 4.1. *The endemic equilibrium of system (2.2) is locally asymptotically stable(LAS) if $R_0 > 1$ and $\alpha_1 > \Gamma$.*

Proof. Consider the Jacobian matrix of system (2.2) about $\hat{\omega}$,

$$\hat{J} = \begin{pmatrix} -(\beta\hat{I} + \Gamma + \phi_1 + \mu) & 0 & -\beta\hat{S} & 0 & 0 & 0 \\ \beta\hat{I} + \Gamma & -(\phi_2 + \gamma + \mu) & \beta\hat{S} & 0 & 0 & 0 \\ 0 & \gamma & -(\phi_3 + \mu) & 0 & 0 & 0 \\ 0 & \phi_2 & \phi_3 & -(\eta + \mu) & 0 & 0 \\ \phi_1 & 0 & 0 & 0 & -\mu & 0 \\ 0 & 0 & 0 & \eta & 0 & -(\delta + \mu) \end{pmatrix}$$

The three eigen values of the above matrix are $\lambda_1 = -(\eta + \mu)$, $\lambda_2 = -\mu$, $\lambda_3 = -(\delta + \mu)$. $|\lambda_i| \leq 1$ for $i = 1, 2, 3$. The remaining matrix can be written as,

$$\hat{J}_1 = \begin{pmatrix} -(\beta\hat{I} + \Gamma + \phi_1 + \mu) & 0 & -\beta\hat{S} \\ \beta\hat{I} + \Gamma & -(\phi_2 + \gamma + \mu) & \beta\hat{S} \\ 0 & \gamma & -(\phi_3 + \mu) \end{pmatrix}$$

Consider $|\hat{J}_1 - \lambda I| = 0$. The characteristic polynomial can be written as

$$\sigma(\lambda) = \lambda^3 + f_1\lambda^2 + f_2\lambda + f_3 = 0 \tag{4.1}$$

where

$$\begin{aligned} f_1 &= -\left(\frac{R_0}{\phi_1} + \alpha_1 + \alpha_2 + \alpha_3\right) \\ f_2 &= \frac{\gamma\beta A\tau\phi_1}{R_0 + \phi_1\alpha_1} - (\alpha_1\alpha_2 + \alpha_2\alpha_3 + \alpha_1\alpha_3) \\ f_3 &= \frac{\gamma\beta A\tau\phi_1}{R_0 + \phi_1\alpha_1} \left[\frac{R_0}{\phi_1} + \alpha_1 - \Gamma\right] \end{aligned}$$

From Jury's conditions, the eigen values of the above characteristic equation is less than 1 iff $\sigma(1) > 0$, $\sigma(-1) < 0$ and $|\hat{J}_1| < 1$ [8]. The conditions $\sigma(1) > 0$, $\sigma(-1) < 0$ and $|\hat{J}_1| < 1$ iff $R_0 > 1$ and $\alpha_1 > \Gamma$. Hence the proof.

Theorem 4.2. *The endemic equilibrium of system (2.2) is globally asymptotically stable(GAS) if $R_0 > 1$.*

Proof. Let us consider a lyapunov function

$$\begin{aligned} \chi_1 = & \frac{(S - \hat{S})^2}{2} + \frac{(E - \hat{E})^2}{2} + \frac{(I - \hat{I})^2}{2} \\ & \frac{(F_p - \hat{F}_p)^2}{2} + \frac{(P_r - \hat{P}_r)^2}{2} + \frac{(Q - \hat{Q})^2}{2} \end{aligned} \tag{4.2}$$

Taking the derivative on both sides,

$$\begin{aligned} \frac{d\chi_1}{dt} &= (S - \hat{S}) \frac{dS}{dt} + (E - \hat{E}) \frac{dE}{dt} + (I - \hat{I}) \frac{dI}{dt} \\ &= (S - \hat{S}) \left\{ -(\beta\hat{I} + \Gamma) - (\phi_1 + \mu) \right\} (S - \hat{S}) + \\ & \quad (E - \hat{E}) \left\{ (\beta\hat{I} + \Gamma)\hat{S} - (\phi_2 + \gamma + \mu)(E - \hat{E}) \right\} \\ & \quad + (I - \hat{I}) \left\{ \gamma(E - \hat{E}) - (\phi_3 + \mu)(I - \hat{I}) \right\} \end{aligned}$$

Where $q_{11}\beta\hat{I} + \Gamma + \phi_1 + \mu$, $q_{22} = \phi_2 + \gamma + \mu$, $q_{33} = \phi_3 + \mu$

$$\begin{aligned} \frac{d\chi_1}{dt} &< -\frac{1}{2}q_{11} (S - \hat{S})^2 - \frac{1}{2}q_{22} (E - \hat{E})^2 - \frac{1}{2}q_{33} (I - \hat{I})^2 \\ & \quad -\frac{1}{2}q_{11} (S - \hat{S})^2 - \frac{1}{2}q_{22} (E - \hat{E})^2 - \frac{1}{2}q_{33} (I - \hat{I})^2 \\ & \quad + (\beta\hat{I} + \Gamma) (S - \hat{S}) (E - \hat{E}) + \gamma (I - \hat{I}) (E - \hat{E}) \\ & \quad - \beta\hat{S} (I - \hat{I}) (S - \hat{S}) \\ & \quad - \eta (P_r - \hat{P}_r) (F_p - \hat{F}_p) - (\delta + \mu) (Q - \hat{Q})^2 \end{aligned}$$

Simplifying,

$$\frac{d\chi_1}{dt} = - \left[\sqrt{\frac{q_{11}}{2}} (S - \hat{S}) - \sqrt{\frac{q_{22}}{2}} (E - \hat{E}) \right]^2$$

$$\begin{aligned}
 & - \left[\sqrt{\frac{q_{22}}{2}} (E - \hat{E}) - \sqrt{\frac{q_{33}}{2}} (I - \hat{I}) \right]^2 \\
 & - \left[\sqrt{\frac{q_{11}}{2}} (S - \hat{S}) - \sqrt{\frac{q_{33}}{2}} (I - \hat{I}) \right]^2 \\
 & - \beta \hat{S} (I - \hat{I}) (S - \hat{S}) \\
 & - \eta (P_r - \hat{P}_r) (F_p - \hat{F}_p) - (\delta + \mu) (Q - \hat{Q})^2 \\
 & \leq 0.
 \end{aligned} \tag{4.3}$$

It is observed that $S \rightarrow \hat{S}, E \rightarrow \hat{E}, I \rightarrow \hat{I}, F_p \rightarrow \hat{F}_p, P_r \rightarrow \hat{P}_r, Q \rightarrow \hat{Q}$ as $t \rightarrow \infty$. Since the parameters in the system are non negative, for $R_0 > 1, \frac{dx_1}{dt} \leq 0$. By LaSalle’s invariance principle, the solutions of the system (2.2) approach endemic equilibrium when $R_0 > 1$. Therefore, the endemic equilibrium of system (2.2) is globally asymptotically stable.

5. Numerical Simulation

The following values are considered for numerical simulation through MATLAB software. ODE45 solver is utilized since it implements a Runge-Kutta method with variable time step in order to achieve efficient results. The behaviour of the proposed system with various levels of quarantine technique and infection rate is observed. For the above values, the $R_0 = 2.9$. The endemic equilibrium \hat{E} has the

Table 2: Parametric values for numerical simulation

Parameter	Baseline Value	Reference
A	200	[8]
τ	0.0082 yr ⁻¹	[2]
β	0.3-0.6 yr ⁻¹	[2]
Γ	0 – 10 ⁻⁴	[2]
ϕ_1	1.3333e-004×365	[4]
ϕ_2	0.0025×365	[2]
ϕ_3	0.0037×365	[2]
η	0.12yr ⁻¹	[2]
μ	0.1yr ⁻¹	[8]
γ	0.32yr ⁻¹	Estimation
δ	0.0304yr ⁻¹	[2]

following solutions $\hat{S} = 0.00273, \hat{E} = 6.59, \hat{I} = 1000, \hat{P}_r = 11304.27, \hat{F}_p = 0.0249,$

$\hat{Q} = 10402.27$. The conditions $R_0 > 1$ and $\alpha_1 > \Gamma$ are satisfied. The dynamics of the system for different transmission rates β are observed.

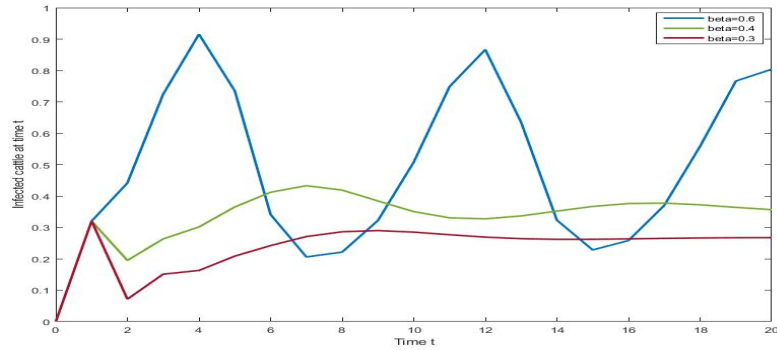


Figure 2: Infected cattle at time t for $\beta = 0.3$, $\beta = 0.4$ and $\beta = 0.6$

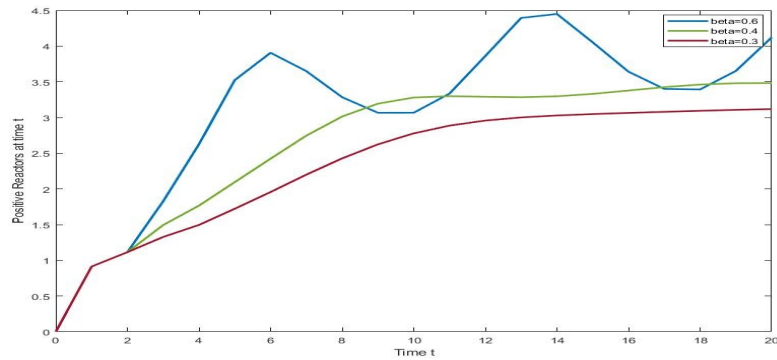


Figure 3: True reactor cattle at time t for $\beta = 0.3$, $\beta = 0.4$ and $\beta = 0.6$.

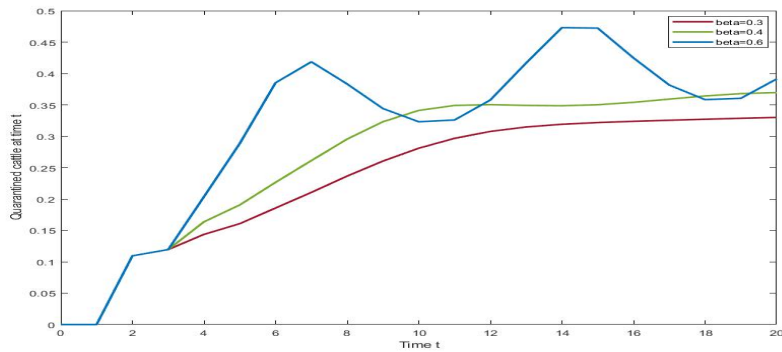


Figure 4: Quarantined cattle at time t for $\beta = 0.3$, $\beta = 0.4$ and $\beta = 0.6$.

The results show that as the transmission rate β increases, the infected cattle increases which leads to an increase in true reactors to the BTB. Figure 2,3 and 4 shows the infected, true reactor and quarantined cattle for transmission rate $\beta = 0.3$, $\beta = 0.4$ and $\beta = 0.6$ respectively. It is observed that number of quarantined cattle is directly proportional to the number of true reactors. The higher the true reactors to the BTB, the higher the cattle needs to be quarantined.

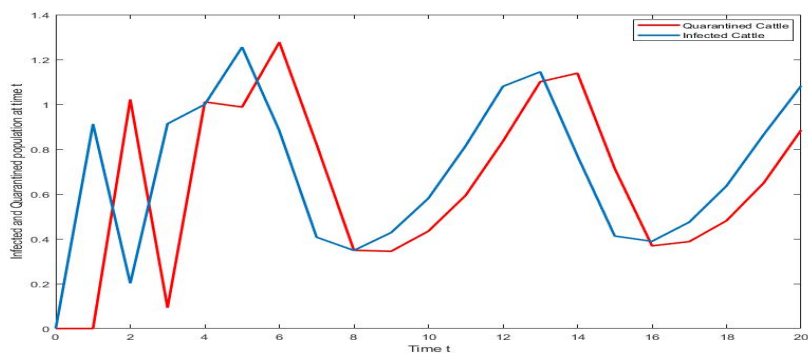


Figure 5: Infected and Quarantined cattle at time t for $\eta = 0.12$.

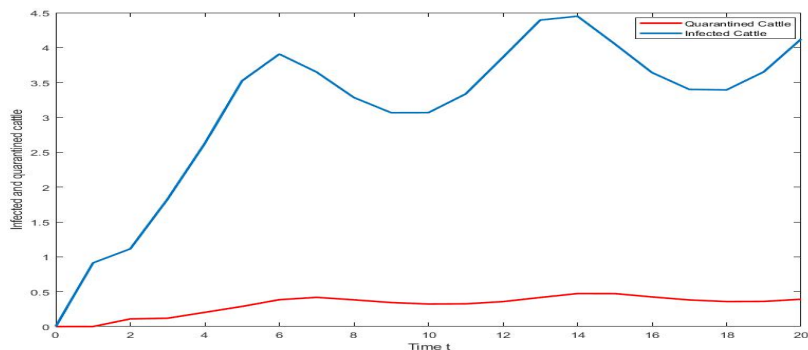


Figure 6: Infected and Quarantined cattle at time t for $\eta = 1.12$

For the above set of parametric values with $\beta = 0.6$, we compare the infected and quarantined cattle in Figures 5 and 6. In figure 5, the quarantine rate $\eta = 0.12$ is low and hence the infected cattle is higher. Whereas in figure 6, the rate of quarantine is increased to $\eta = 1.12$ and it is observed that when the quarantine rate increases, the infected cattle in the herd decreases. It is found that the infection decreases by 60% due to effective control strategy.

6. Conclusion

The proposed mathematical model is used to analyze the effect of quarantine and isolation in control of BTB. The numerical results reveal that quarantine tech-

niques increased by 89.3% reduced the infection by 60%. It was also observed the local stability of the system (2.2) around the disease free and endemic equilibrium depended on the basic reproduction number (R_0) and other conditions. Whereas, the global stability of the system (2.2) around the disease free and endemic equilibrium depended on the basic reproduction number (R_0) alone. Finally, the numerical simulations revealed that there is a need for intensive quarantine and isolation methods when the incidence increases. The infection can be effectively controlled in a herd health practice when true reactors to the BTB is removed at the earliest. The limitation of the proposed model is that it utilises a bilinear incidence rate. However, a saturated or non-linear incidence rate will be more efficient in numerical computations.

References

- [1] Agustoa F. B., Suzanne Lenhartb, Abba B. Gumelc and Agricola Odoid, Mathematical analysis of a model for the transmission dynamics of bovine tuberculosis, *Mathematical Methods in Applied Sciences*, 34(2011), 1873-1887.
- [2] Barua, A. G. and Chandrani Goswami, H. R., Slaughter house surveillance for tuberculosis among cattle in Ri-Bhoi district of Meghalaya, *Culture*, 18(2016), 15-0.
- [3] Kao R. R., Roberts M. G. and Ryan T. J., A model of bovine tuberculosis control in domesticated cattle herds, *Proceedings of the Royal Society of London*, 264(1997), 1069-1076.
- [4] Liu S., Li A., Feng X., Zhang X., and Wang K., A Dynamic Model of Human and Livestock Tuberculosis Spread and Control in Urumqi, Xinjiang, China, *Computational and Mathematical Methods in Medicine*, (2016).
- [5] Neill, S. D., Hanna, J., Mackie, D. P., & Bryson, T. G., Isolation of *Mycobacterium bovis* from the respiratory tracts of skin test-negative cattle, *The Veterinary Record*, 131(3) (1992), 45-47.
- [6] Phepa P. B., Chirove F. and Govinder K. S., Modelling the role of multi-transmission routes in the epidemiology of bovine tuberculosis in cattle and buffalo populations, *Mathematical Biosciences*, 277(2016), 47-58.
- [7] Pollock E. B., Roberts G. O. and Keeling M. J., A dynamic model of bovine tuberculosis spread and control in Great Britain, *Nature*, 511(2014), 228-230.

- [8] Sebastian E., Victor Priyanka and Victor Preethi, Discrete-Time Eco-epidemiological Model with Disease in Prey and Holling Type III Functional Response, Proceedings of Fifth International Conference on Soft Computing for Problem Solving, Springer, Singapore, (2016)
- [9] Smith G. C., Models of *Mycobacterium bovis* in wildlife and cattle, *Tuberculosis*, 81(2001), 51-64.
- [10] Smith G. C. and Cheeseman C. L., A mathematical model for the control of diseases in wildlife populations: culling, vaccination and fertility control, *Ecological Modelling*, 150(2002), 45-53.
- [11] Srinivasan S., Easterling L., Bipin Rimal, Xiaoyue Maggie Niu, Andrew J. K. Conlan, Patrick Dudas, Kapur Vivek, Prevalence of Bovine Tuberculosis in India: A systematic review and meta-analysis, *Transboundary and Emerging Diseases*, 65(2018), 1627-1640.
- [12] Thakur A., Sharma M., Katoch V. C., Dhar P., Katoch R. C., A study on the prevalence of Bovine Tuberculosis in farmed dairy cattle in Himachal Pradesh, *Veterinary World*, 3(2010), 409-414.
- [13] Thakur, M. K., Sinha, D. K., & Singh, B. R., Evaluation of complementary diagnostic tools for bovine tuberculosis detection in dairy herds from India, *Veterinary world*, 9(8) (2016), 862.
- [14] Thornton, P. K., Livestock production: recent trends, future prospects, *Philosophical Transactions of the Royal Society B: Biological Sciences*, 365(1554) (2010), 2853-2867.
- [15] Wilkinson D., Smith G. C., Delahay R. J. and Cheesman C. L., A model of bovine tuberculosis in the badger *Meles* : an evaluation of different vaccination strategies, *Journal of Applied Ecology*, 41(2004), 492-501.
- [16] Zanella G., Bar-Hen A., Boschioli M.-L., Hars J., Moutou F., Garin-Bastuji B. and Durand B., Modelling Transmission of Bovine Tuberculosis in Red Deer and Wild Boar in Normandy, France, *Zoonoses Public Health*, 59(2012), 170-178.
- [17] Zinsstag, J., Schelling, E., Roth, F., Kazwala, R., Thoen, C. O., & Steele, J. H., Economics of bovine tuberculosis. *Mycobacterium bovis* infection in animals and humans, 2(2006), 68-83.