MODELING THE TRANSMISSION DYNAMICS OF BOVINE TUBERCULOSIS WITH CONTROL PARAMETERS

Theresia Shirima Sabini

A Dissertation Submitted in Partial Fulfillment of the Requirements for the Degree of Master's in Mathematical and Computer Sciences and Engineering of the Nelson Mandela African Institution of Science and Technology

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ABSTRACT

Bovine tuberculosis (bTB) is a bacterial and zoonotic disease which is transmitted through; consumption of unpasteurized milk, raw meat and inhalation of aerosols. This study used a deterministic mathematical model to assess the impact of each parameter in the transmission of bTB. The basic reproduction number R_0 computed to determine the behaviour of the disease. The disease-free equilibrium exists and is locally asymptotically stable when $R_0 < 1$, and it is unstable otherwise. However, there is a possibility for the diseases free equilibrium to coexist with endemic equilibrium when $R_0 = 1$. The parameters which drive the dynamics of bTB computed and sensitivity analysis performed. The analysis shows that the basic reproduction number R_0 increases proportionally as the most positive sensitive parameters are increases. However, the rate of animal deaths due to the disease mortality, the rate of natural animal deaths and the rate of leaking for unused dairy products are conversely proportional to the basic reproduction number R_0. Numerical analysis performed to analyse how sensitive each parameter is to the disease. Results show that bTB will increase when we increase rates of consuming dairy products and contacts with infected humans and animals, respectively. The basic model then extended by including control parameters to reduce bTB transmission. The effective reproduction number R_e decreases as we increase treatment of infected humans, quarantine of infected animals and inspection of the dairy product. However, the standard requirement of effective reproduction number Re to be less than a unit for the disease to clear is not enough because the model undergoes backward bifurcation when $R_e = 1$. Numerical analysis carried out to study the long term behaviour of bTB. Simulations show that when control parameters increase, the number of susceptible humans and animals increases, while the number of infected humans and animals decreases. To contained Bovine tuberculosis, there should be the treatment of infected humans, are quarantine of infected animals and dairy products should be inspected.

Key words: Bovine tuberculosis; Dairy products; Sensitivity analysis; Bifurcation analysis.

DECLARATION

I, **Theresia Shirima Sabini,** do hereby declare to the Senate of Nelson Mandela African Institution of Science and Technology that this dissertation is my own original work and that it has neither been submitted nor presented for similar award in any other institution.

Theresia Shirima Sabini (Candidate)	Date

The above declaration is confirmed

Dr. Jacob Ismail Irunde (Supervisor 1)	Date
Prof. Dmitry Kuznetsov (Supervisor 2)	Date

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CERTIFICATION

The undersigned certify that they have read and hereby recommend for acceptance by the Nelson Mandela African Institution of Science and Technology the dissertation entitled: Modeling Transmission Dynamics of Bovine Tuberculosis with Control Parameters, in fulfillment of the requirements for the degree of Master's in Mathematical and Computer Sciences and Engineering of the Nelson Mandela African Institution of Science and Technology.

Dr. Jacob Ismail Irunde
(Supervisor 1)

Date: .....

Prof. Dmitry Kuznetsov
(Supervisor 2)

Date: .....

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DEDICATION

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ABBREVIATIONS

bTB	Bovine tuberculosis
CoCSE	Communication and Computational Science and Engineering
DFE	Disease Free Equilibrium
EE	Endemic Equlibrium
FAO	Food and Agriculture Organization
NM-AIST	The Nelson Mandela African Institution of Science and Technology
ODE	Ordinary Differential Equation
OIE	World Organization for Animal Health
PCR	Polymerase chain reaction
R_0	Basic Reproduction number
R_e	Effective Reproduction number
SICCT	Single Intra-dermal Comparative Cervical
TB	Tuberculosis
WHO	World Health Organization

CHAPTER ONE

INTRODUCTION

1.1 Background of the Problem

Tuberculosis (TB) is a global health problem which is among the top 10 diseases which lead by causing deaths of many people (WHO, 2018). World Health Organization (WHO) have conducted several meeting on how to end TB, but still, the disease is leading by taking the lives of many people. According to the WHO report of 2018, TB kills 1.3 million people among HIV negative, and there were an additional of 300 000 people with HIV who died with TB. The estimation shows that annually 10 million people get ill with TB, of which 5.8 million are men, 3.2 million are women, and 1.0 million are children (WHO, 2018). Africa reported to have the highest number of cases, followed by India, China and Indonesia, with percentages: 72%, 27%, 9% and 8% respectively (WHO, 2018). Although TB is a worldwide health problem, other kinds of TB, including zoonotic tuberculosis, are still neglected especially in developing countries.

Bovine tuberculosis (bTB) is a bacterial and zoonotic disease which was initially transmitted to cattle from wild animals especially buffalo and badger, and then spread to other domestic animals like cows, goats, pigs, horses, and sheep (WHO, 2016). The disease has a tremendous negative economic impact due to the death of livestock when they acquire bTB Durnez *et al.* (2011); Ramos *et al.* (2015) and it causes human health problems which cost their lives. The disease can lead to loss of self-employment for some workers, especially those who depend on livestock keeping as their primary source of income (De Garine-Wichatitsky *et al.*, 2013). Bovine tuberculosis is transmitted from animal to animal through inhalation of aerosols when there is close contact (Menzies, 2000). The infections occur when the salivary, faeces and urine of infected animal drop on grasses, since bTB is a bacterial disease once animals eat grasses which contain bacteria they acquire bTB infection (Tschopp *et al.*, 2009). The disease is also transmitted from animal to human beings through inhalation of aerosols, drinking of unpasteurized milk, eating infectious dairy products or when the blood of infected animal gets into someone who is having scratches (Katale *et al.*, 2012; Kilale, 2016).

Bovine tuberculosis is a threatening disease to the economy, human and animal health in European countries. The study conducted in England from 2002-2014 shows that 357 human bTB new cases are reported annually (Davidson *et al.*, 2017). A large number of people, about 74% were at risk to be exposed to bTB due to the consumption of unpasteurized milk frequently (Davidson *et al.*, 2017; Menzies, 2000). In Britain, 50 000 new cases of people reported

whereby 2500 people die with bTB due to the consumption of unpasteurized milk (McCulloch, 2017). However, European countries succeeded to contain the spread of bTB by reducing the transmission from 4.8% to 2% collaborative efforts between veterinarians and public health workers (Pavlik, 2006). In Ethiopia, many people acquire bTB due to the consumption of infectious raw milk (Ameni *et al.*, 2000, 2007; Demelash *et al.*, 2009; Gumi *et al.*, 2011). The study conducted by Regassa *et al.* (2008) in Ethiopia revealed that about 16% of cattle owners reported having *M. bovis* which cause bTB due to the consumption of unpasteurized milk while 46% of cattle had *M. bovis* which cause bTB. The availability of bTB data in African countries is still a problem since there is no surveillance information. Most of the African countries have no bTB surveillance data, Phepa *et al.* (2016), although the disease is a threat to human and animal health. Disease control in developing countries is still a challenge due to limited data and expensive control options (Dejene *et al.*, 2016).

Symptoms of bovine tuberculosis for livestock include reduced productivity, weight loss and lack of appetite, while for some animals lymph nodes may expand gradually and sometimes may burst (Centers for Disease Control & Prevention, 2011; Phepa *et al.*, 2016). Sometimes it may take up to one month for livestock to develop symptoms of bTB, and most of the time they might be latent for few years or when they are under stress or at old age (Michel *et al.*, 2006; Hassan *et al.*, 2014; Phepa *et al.*, 2016). In human, bTB has the following symptoms: loss of weight, general body weakness, poor appetite, fever, a productive cough, and night sweats. It mostly affects extra-pulmonary sites such as lymph nodes, joints, backbone and neck (Bowong, 2010; Centers for Disease Control & Prevention, 2011).

Mycobacterium bovis (*M. bovis*), which cause bovine tuberculosis (bTB) to animals can survive in various places depending on weather condition (Ramos *et al.*, 2015). These bacteria can survive in cold and dark places where there is a moist condition (Jemal, 2016). From the fact that bTB is a neglected disease, it has received little attention, and this makes the control of disease to be weak despite its negative impact to the society and close interaction between wild animals, domestic animals and humans which makes its transmission easier (Katale *et al.*, 2012). Bovine tuberculosis is neglected despite being among the diseases which take the lives of many people and causing the economic problem. This study aims to formulate a mathematical model to study the transmission dynamics of the disease and suggest ways of controlling the transmissions.

Globally, the estimation shows that 147 000 new cases of bTB in humans reported whereby 12 000 people die annually due to bTB (WHO, 2016). In Uruguay bTB in livestock reported to increases in the average of 19% of animals in the herds annually. Besides, at least 70% of the world population, especially in sub-Sahara African countries such as Ethiopia, South

Africa, Tanzania and Kenya, are at risk of being infected with bovine tuberculosis due to closer interaction between human and livestock (WHO, 2016; De Garine-Wichatitsky *et al.*, 2013). In Tanzania, the disease prevalence varies from region to region depending on the number of livestock in a particular place, and it ranges from 0.2%-13.3% (Shirima *et al.*, 2003; Katale *et al.*, 2013). Places where bTB is likely to exist, include Northern Tanzania (Arusha, Kilimanjaro, and Manyara), dairy farms in Kibaha and some areas in Morogoro districts (Durnez *et al.*, 2011; Katale *et al.*, 2013).

The diagnosis of bTB helps to know the dynamics of the disease and identify ways of controlling the transmission factors before it becomes endemic. Several methods used to diagnose bTB, including Tuberculin skin test, interferon-gamma test Assembly (2009), polymerase chain reaction (PCR) and gene sequencing of culture isolate (Mathews *et al.*, 2006; Kilale, 2016), are widely and commonly used diagnostic methods in developed countries compared to developing countries. Post mortem examination, which focuses on lymph nodes, is also used since bTB affects lymph nodes parts (OIE, 2016). Single intradermal comparative cervical test (SICCT) is another diagnostic tool for the early stages of bTB in cattle (OHagan *et al.*, 2015). From the external examination, it shows that there is a possibility of carcasses from slaughtered cattle to contain bTB pathogens (Biet *et al.*, 2005).

Various organisations such as World Health Organization (WHO), World Organization for Animal Health (OIE), Food and Agriculture Organization (FAO) joined together to fight against transmission of bTB in order to eradicate the disease (WHO, 2016). Most of the European countries managed to eliminate the transmission of bTB while countries like Britain and Ireland still the disease is a problem (Allen *et al.*, 2018). Though different approaches such as "one health approach, together we can save lives and secure lively-hoods" are used to control the transmission, but bTB is a problem. Although bTB controlled by treating livestock using various medicines such as pyrazinamide, it develops resistance to pyrazinamide because it used to treat patients with pulmonary TB (WHO, 2016).

Mathews *et al.* (2006), Agusto *et al.* (2011), Phepa *et al.* (2016) and Liu *et al.* (2016) developed mathematical models to study the transmission dynamics of bTB and its control strategies. Most of these studies did not consider the impacts of dairy products in the transmission dynamics of bTB. According to WHO (2016), it is crucial to conduct more research on bTB to find good ways of controlling the transmission of the disease. This study uses a mathematical model to study the transmission dynamics of bTB in human beings and livestock by considering dairy products as a risk factor for the transmission of bTB.

1.2 Statement of the Problem

Various studies such as those by Wilkinson *et al.* (2004), Agusto *et al.* (2011), Phepa *et al.* (2016) Liu *et al.* (2016), Mathews *et al.* (2006) and Brooks-Pollock and Danon (2017), have been conducted to investigate the transmission dynamics of bTB and suggested ways of controlling the disease. Results from these studies show the existence of bTB and recommended slaughtering as the best way of controlling the disease. However, none of these studies considered dairy products as the risk factor for disease transmission. Dairy products is among the factor which drive the transmission of bTB since these products consumed with a large number of people worldwide (Bonsu *et al.*, 2000; Perez *et al.*, 2002). Dairy products in one among the factors which lead in the spread of bTB as some of the findings reported the products to contain *M. bovis* (Ramos *et al.*, 2015). This study investigated the transmission dynamics of bTB in humans and animals by taking into consideration dairy products as a risk factor for disease transmission and suggested control strategies for disease transmission.

1.3 Rationale of the Study

Bovine tuberculosis is a significant disease to humans and animals health and the economy of many countries. The disease has a tremendous negative impact on many dairy products industries worldwide since many cattle slaughtered due to bTB (Ramos *et al.*, 2015). Many people from Ethiopia get infected with bTB when they consume unpasteurized infectious dairy products (Jemal, 2016). Also, bTB reported to found in dairy farms in Uruguay (Perez *et al.*, 2002; Picasso *et al.*, 2017). Therefore there is a need to investigate the contribution of dairy products on the transmission of bovine tuberculosis.

1.4 Research Objectives

1.4.1 General Objective

The general objective of this study is to develop and analyze a mathematical model for the transmission dynamics of bovine tuberculosis in livestock and human with control parameters.

1.4.2 Specific Objectives

This study has the following specific objectives:

(i) To formulate a mathematical model for transmission dynamics of bTB in human and livestock that include dairy product.

(ii) To compute the basic reproduction number and determine the relative impact of each parameter in the basic reproduction number.

(iii) To determine the conditions for existence and stability of equilibrium points.

(iv) To determine how control parameters can help to contain the disease.

1.5 Research Questions

The research objectives achieved by analyzing and answering the following questions:

(i) How can a mathematical model for the transmission dynamics of bovine tuberculosis in human and livestock be formulated?

(ii) How can basic reproduction number be computed and which parameters are sensitive to the disease?

(iii) What are the conditions for existence and stability of equilibrium points?

(iv) Which are the effective control parameters for bTB?

1.6 Significance of the Research

(i) The outcome of the study will help public health workers and veterinarian to determine if the inspection of dairy products can help to reduce the transmission of bTB.

(ii) This study will help to know how the quarantine of infected animals can help to reduce the transmission of the disease.

1.7 Delineation of the Study

This study conducted to investigate the transmission dynamics of bTB in humans and animals before controls and after controls. The chapters organized as follows; chapter one which consists of a general introduction and background of the problem, statement of the problem, rationale of the study, research objectives, significance of the study and delineation of the study. Chapter two contains a literature review whereby the author relates other studied to this work and address what they did not do in their work. Chapter three consists of material and methods. This chapter discusses the methodology used, model analysis, numerical simulation for the basic model and conclusion. Chapter four discussed the extended model from the basic model. The chapter contains a brief introduction, extension of the basic model, analysis of the model, numerical simulation and conclusion. Chapter five includes a summary of the study, conclusion, and future works. Also, reference and appendices which contain codes and output of this work.

CHAPTER TWO

LITERATURE REVIEW

Many studies have been conducted to investigate the transmission dynamics of bovine tuberculosis. To provide a base for this study, few studies are reviewed in this section to point out what has been done and what has been left out. This will help to delineate the research gap that this study is addressing.

Liu *et al.* (2016) formulated a mathematical model for transmission dynamics of bTB in humans and cows in Urumqi, Xingjian China. In their study, the results show that the existence of bTB is not only a critical world health problem but also hinders the development of dairy products industries. They recommended test and slaughter to be the effective control strategies for the transmission of the disease. However, in their formulated model, they did not include dairy products as a factor for disease transmission.

Palmer *et al.* (2012) investigated the transmission dynamics of bTB when the interaction between livestock-wildlife and humans is considered. The study recommends that for bTB to be eradicated the interaction between wildlife, livestock and human beings should be controlled. On the contrary, controlling the interaction between animal and human populations is difficult, especially for pastoralists who tend to move from one place to another, searching for pastures.

Phepa *et al.* (2016) formulated a mathematical model to assess the transmission dynamics of bTB in buffalo and cattle populations in South Africa. Model analysis shows that buffaloes are the carrier of *M. bovis* and can spread the infection to animal species, which is a threat to animals and human beings as well. The study did not consider dairy products as an essential factor for the transmission of the disease, although many people consume these products. This study includes dairy products as a risk factor for disease transmission.

Durnez *et al.* (2011) investigated the possibility of small mammals like rodents and insects in carrying *M. bovis* to cattle. Data analysis shows that bTB can be transmitted to cattle from other species easily compared to small mammals. However, high preference of *M. bovis* to these small mammals impose a high risk to human health, especially those with HIV positive (Durnez et al., 2011). Though their interest was to study whether small mammals and insects can be the carriers of *M. bovis*, they did not think whether dairy products can be the carrier of *M. bovis*. Also, they did not give contributions to how the transmission of the disease can be controlled. The fascinating thing from their study is the confirmation of the presence of bTB in cattle.

Wilkinson *et al.* (2004) developed a spatial stochastic model for controlling the transmission of

bTB in badger and cattle using different vaccination strategies. Their model was effective for about 75% on the group of badgers while in cattle, it did not work. The study calls for more mathematical models that will give out the cost-effective way of controlling the disease in cattle. Their study, however, was based on cattle and badger only and did not include humans. This study intended to study the dynamics of bTB in the presence of human beings.

Ssematimba *et al.* (2015) conducted a study on the transmission Dynamics of Contagious Bovine Pleuropneumonia. A mathematical modelling approach was employed to assess the effects of the vaccine on cattle during their early stage of development. The model simulation shows that vaccination is the most effective way of controlling bovine for at least 18 months. Additionally, the study suggested that regular checkups will play a big role in controlling bovine tuberculosis. However, the model failed to give out the contribution of dairy products as a risk factor for the transmission of the disease. They also did not include humans in the transmission dynamics of bTB.

Agusto *et al.* (2011) developed a deterministic model that incorporates the imported infected cattle to investigate the transmission dynamics of bTB in a single cattle herd. In their study, they found out that the importation of infected cattle may lead to the endemic condition of the disease. However, dairy products are neglected, although they are factors for bTB transmission. Hence this study developed a mathematical model that incorporated dairy products as a risk factor.

Leo and Natalini (2015) investigated bTB transmission dynamics using a stochastic model to assess the presence of *M. bovis* in dairy cattle. The study based on three ways which are; routine test on each farm carried out after every three years, tuberculin skin test, test and slaughter method. Among all three methods, slaughtering suggested as the best way of controlling disease transmissions. However, they did not consider the economic impact of slaughtering since it is practised mostly in developed countries where there is a good economy. Also, they did not take into consideration whether the method is affordable for those who depend on livestock activities.

Griffin *et al.* (2000) investigated the presence of *M. bovis* within a herd. Their results showed strong evidence that transmission of bTB may occur within a herd and then spread easily to other species, including human beings. However, the study did not pay attention on dairy products as the factors for disease transmission. Also, they did not suggest the way of controlling the disease as they were just making a numerical estimation of the importance of within-herd transmission. So this study concentrated on dairy products as one among the important factor for disease transmission and suggesting an effective way of controlling bTB.

Though researches have conducted to investigate the dynamics of bTB and controls, most of the studies did not include dairy products as a factor for disease transmission. Most of these studies recommended the slaughtering of animals as a way of controlling the spread of the disease. However, this method is commonly practised in developed countries than in developing countries. This study has investigated the transmission dynamics of bTB by including dairy products as a significant factor for disease transmission. The study also has proposed ways of controlling the spread of the disease.

CHAPTER THREE

MATERIALS AND METHODS

This section describes the methods that are used to achieve the stated objectives and the area where data are collected. Justification for selecting the area for data collection is also provided in this section.

3.1 Methodology

To achieve objective one, a mathematical model which includes humans and animals populations formulated with the aid of ordinary differential equations.

The basic reproduction number R_0 computed by the next-generation matrix method, and forward normalized sensitivity index is applied to determine the sensitivity index of each parameter with respect to basic reproduction number R_0.

Linearization method, which also involves trace and determinant, is used to deduce local stability of the disease free-equilibrium. The model is analyzed to determine whether it undergoes backward bifurcation when $R_0 = 1$. To determine whether the model undergoes backward bifurcation when $R_0 = 1$, we used centre manifold theory.

To determine the dynamics of bTB, the model is solved numerically by using the Runge-Kutta method. Simulation of the model results is carried out using MATLAB or Mapple software.

3.1.1 Model Formulation

The model formulated by modifying the tuberculosis model for human and cows in Urumqi, Xinjiang China which was developed by Liu *et al.* (2016).

The current model includes animal and human populations. Human population is divided into: Susceptible S_h, Exposed E_h and Infected I_h, (SEI) and animal population is divided into Susceptible S_a, Exposed E_a and Infected I_a compartments. The variable D represents Dairy products which are produced by infected animals. The proposed model does not include recovery class because it assumed that, there is no natural recovery (Assembly, 2009).

Susceptible humans recruited through birth and migration at a rate Λ_h, and they acquire bovine tuberculosis latent infection following contacts with infected human, animals and after consuming dairy products from infected animals at a rate: $\lambda_h = \dfrac{(\beta_1 I_h + \beta_2 I_a + \beta_3 D)}{N_h}$.

Exposed compartment E_h increases following latent infection of susceptible humans S_h at a rate of λ_h and it decreases due to progression to the infectious stage at a rate of γ_h. Infectious

humans I_h increase at a rate γ_h and diminish due to disease-induced mortality at a rate α_h. All individual compartments suffer natural mortality at a rate of μ_h.

Susceptible animals S_a are recruited through birth and migration at a rate Λ_a and acquire bovine tuberculosis latent infection following contacts with infectious humans and animals, and after consuming dairy products at rates: $\lambda_a = \dfrac{\beta_4 I_h + \beta_5 I_a + \beta_6 D}{N_a}$

Exposed animals E_a increase following latent infection of susceptible animals S_a at a rate λ_a. However, they decrease due to progression to the infectious stage at a rate of γ_a.

Infectious animals I_a increase at a rate γ_a and diminish due to disease-induced mortality at a rate of α_a. All animal compartments suffer natural mortality at a rate of μ_a. Dairy products are produced by infectious animals at a rate of ρ and the remaining products leak at rate ω.

In the model, we assume all humans and animals are susceptible to the disease. Susceptible human S_h contact bTB when they consume dairy products D such milk and meat from infected animals; when they inhale aerosols from infected animals and human, and direct contact with the dairy product from infected animals through scratches (Dejene *et al.*, 2016). Susceptible animal acquires infection when they interact with infected animals and humans, through breast-feeding from infectious animals and inhalation of aerosols. There is constant natural death to both animals and human beings. There is no natural recovery for infected individuals.

Figure 1 demonstrates the interaction of state variables, Tables 1 and 2 describe state variables and parameters, respectively.

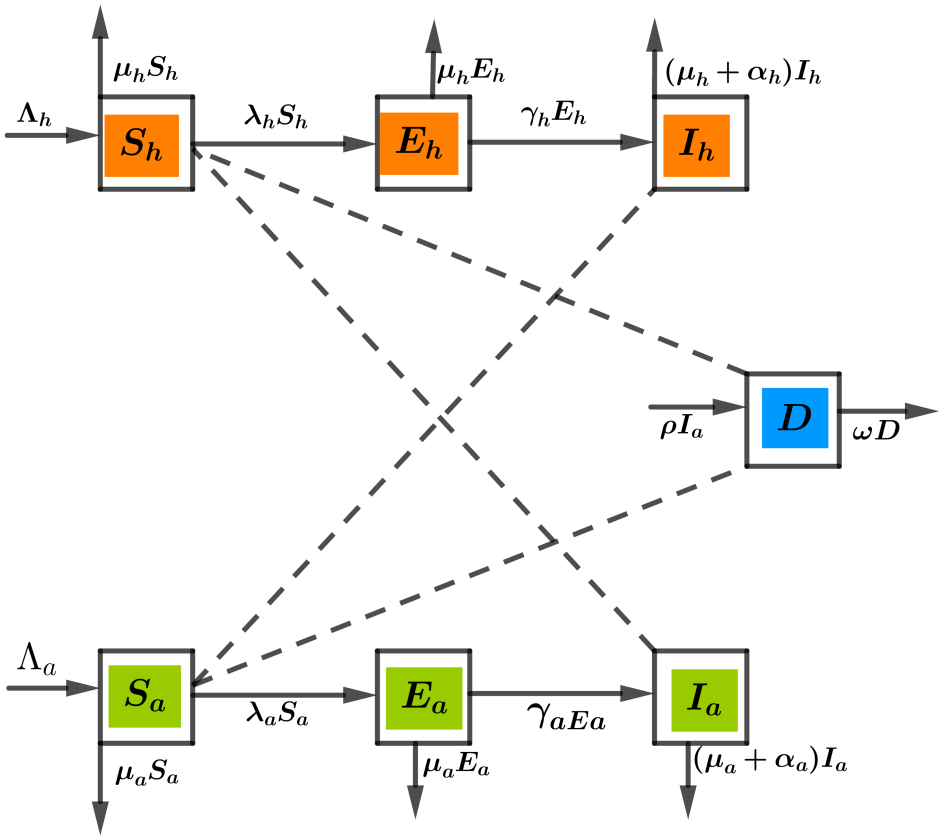


Figure 1: Model flow diagram

Table 1: Basic Model Variables Description

Symbol	Description
$S_h(t)$	Number of susceptible human at time t.
$S_a(t)$	Number of susceptible animal at time t.
$E_h(t)$	Number of Exposed human beings at time t.
$E_a(t)$	Number of Exposed animals at time t.
$I_h(t)$	Number of infected human at time t.
$I_a(t)$	Number of infected animals at time t
$D(t)$	Amount of dairy products produced at time t

3.1.2 Model Equations

Basing on the assumption during model formulation and compartmental diagram we have the following system of differential equations:

$$\frac{dS_h}{dt} = \Lambda_h - \left(\frac{\beta_1 I_h + \beta_2 I_a + \beta_3 D}{N_h}\right) S_h - \mu_h S_h. \tag{3.1a}$$

$$\frac{dE_h}{dt} = \left(\frac{\beta_1 I_h + \beta_2 I_a + \beta_3 D}{N_h}\right) S_h - (\gamma_h + \mu_h) E_h. \tag{3.1b}$$

$$\frac{dI_h}{dt} = \gamma_h E_h - (\mu_h + \alpha_h) I_h. \tag{3.1c}$$

$$\frac{dS_a}{dt} = \Lambda_a - \left(\frac{\beta_4 I_h + \beta_5 I_a + \beta_6 D}{N_a}\right) S_a - \mu_a S_a. \tag{3.1d}$$

$$\frac{dE_a}{dt} = \left(\frac{\beta_4 I_h + \beta_5 I_a + \beta_6 D}{N_a}\right) S_a - (\gamma_a + \mu_a) E_a. \tag{3.1e}$$

$$\frac{dI_a}{dt} = \gamma_a E_a - (\mu_a + \alpha_a) I_a. \tag{3.1f}$$

$$\frac{dD}{dt} = \rho I_a - \omega D. \tag{3.1g}$$

Subject to their initial conditions:

$$S_h(0) > 0; E_h(0) \geq 0; I_h(0) \geq 0; S_a(0) > 0; E_a(0) \geq 0; I_a(0) \geq 0; D(0) \geq 0.$$

Table 2: Basic Model Parameters' Descriptions

Parameter	Descriptions
Λ_h	Human recruitment rate.
Λ_a	Animals recruitment rate.
μ_h	Human natural death rate.
γ_h	Progression rate from E_h to I_h.
α_h	Human disease induced death rate.
$\beta_1, \beta_2, \beta_3$	Humans infection rate from I_h, I_a, and D respectively.
μ_a	Animal natural death rate.
γ_a	Progression rate from E_a to I_a.
α_a	Animals disease induced death rate.
ρ	Rate of producing dairy products from infected animals.
ω	Amount of decaying dairy products.
$\beta_4, \beta_5, \beta_6$	Animals infection rate from I_h, I_a, and D respectively.

3.2 Model Analysis

To determine whether the model is mathematically meaningful, we find the invariant region and test positivity of the solution. The model is biologically and mathematically meaningful when its solutions are positive and bounded.

3.2.1 Invariant Region

Invariant region shows the feasibility of the model solutions. To find the invariant region, we denote humans and livestock populations by N_h and N_a respectively. Beginning with human population we have:

$$\begin{aligned} N_h &= S_h + E_h + I_h, \\ \frac{dN_h}{dt} &\leq \Lambda_h - \mu_h N_h, \end{aligned} \tag{3.2}$$

From (3.2), we have:

$$\begin{aligned} \frac{dN_h}{dt} &\leq \Lambda_h - \mu_h N_h, \\ \text{which gives} & \\ N_h(t) &\leq \frac{\Lambda_h}{\mu_h} + \left(N_h(0) - \frac{\Lambda_h}{\mu_h} \right) e^{-\mu_h t}, \end{aligned} \tag{3.3}$$

Analysis of N_h consider two cases:

$$\text{when } \; N_h(0) > \frac{\Lambda_h}{\mu_h} \; \text{ and when } \; N_h(0) < \frac{\Lambda_h}{\mu_h},$$

When $N_h(0) > 0$:

$$N_h(t) \leq \frac{\Lambda_h}{\mu_h} \leq \frac{\Lambda_h}{\mu_h} + \left(N_h(0) - \frac{\Lambda_h}{\mu_h} \right) e^{-\mu_h t}, \tag{3.4}$$

and when $N_h(0) < 0$:

$$\begin{aligned} N_h(t) &\leq \frac{\Lambda_h}{\mu_h} + \left(N_h(0) - \frac{\Lambda_h}{\mu_h} \right) e^{-\mu_h t} \leq \frac{\Lambda_h}{\mu_h}, \\ \text{since } & \lim_{t \to \infty} \left(N_h(0) - \frac{\Lambda_h}{\mu_h} \right) e^{-\mu_h t} \to 0, \end{aligned} \tag{3.5}$$

For all two cases, we have:

$$0 \leq N_h \leq \frac{\Lambda_h}{\mu_h}. \tag{3.6}$$

Animals population is given by:

$$N_a = S_a + E_a + I_a,$$

where:

$$\frac{dN_a}{dt} \leq \Lambda_a - \mu_a N_a,$$

$$\frac{dN_a}{dt} \leq \Lambda_a - \mu_a N_a,$$

whose solution is

$$N_a(t) \leq \frac{\Lambda_a}{\mu_a} + \left(N_a(0) - \frac{\Lambda_a}{\mu_a}\right) e^{-\mu_a t}, \tag{3.7}$$

The analysis of N_a consider two cases:

$$\text{When } N_a(0) > \frac{\Lambda_a}{\mu_a} \text{ and when } N_a(0) < \frac{\Lambda_a}{\mu_a},$$

For

$$N_a(0) > \frac{\Lambda_a}{\mu_a}: \ N_a(t) \leq \frac{\Lambda_a}{\mu_a} + \left(N_a(0) - \frac{\Lambda_a}{\mu_a}\right) e^{-\mu_a t}, \tag{3.8}$$

and for

$$\begin{aligned} N_a(0) \leq \frac{\Lambda_a}{\mu_a}: \ N_a(t) \leq \frac{\Lambda_a}{\mu_a} + \left(N_a(0) - \frac{\Lambda_a}{\mu_a}\right) e^{-\mu_a t} \leq \frac{\Lambda_a}{\mu_a}, \\ \text{As } \lim_{t\to\infty} \left(N_a(0) - \frac{\Lambda_a}{\mu_a}\right) e^{-\mu_a t} \to 0, \end{aligned} \tag{3.9}$$

all two cases gives:

$$0 \leq N_a \leq \frac{\Lambda_a}{\mu_a}. \tag{3.10}$$

For the case of dairy products when we have:

$$\frac{dD}{dt} \leq \rho \frac{\Lambda_a}{\mu_a} - \omega D. \tag{3.11}$$

From (3.11) we have:

$$\frac{dD}{dt} + \omega D \leq \rho \frac{\Lambda_a}{\mu_a},$$

whose solution is given by:

$$D \leq \frac{\Lambda_a}{\mu_a}\left(\frac{\rho}{\omega}\right) + \left(D(0) - \frac{\Lambda_a}{\mu_a}\left(\frac{\rho}{\omega}\right)\right) e^{-\omega t}. \tag{3.12}$$

But as $t \to \infty$, we obtain:

$$D(t) \leq \frac{\Lambda_a}{\mu_a}\left(\frac{\rho}{\omega}\right). \tag{3.13}$$

Therefore the model (3.1) is positive invariant at the region:

$$Z = \left\{ (S_h, E_h, I_h, S_a, E_a, Ia, D) \in R_+^7 : 0 \leq N_h \leq \frac{\Lambda_h}{\mu_h}; 0 \leq N_a \leq \frac{\Lambda_a}{\mu_a}; 0 \leq D \leq \frac{\Lambda a}{\mu_a}\left(\frac{\rho}{\omega}\right) \right\}. \tag{3.14}$$

The model (3.1) is mathematically and epidemiologically meaningful, therefore we can consider the flow generated by the model for analysis.

3.2.2 Positivity of Solutions

Theorem 1: *Let the initial values for the state variables for the model* (3.1) *be* $S_h(0) > 0, E_h(0) \geq 0, I_h(0) \geq 0, S_a(0) > 0, E_a(0) \geq 0, I_a(0) \geq 0$ *and* $D \geq 0$ *then the solutions of the model* (3.1) *are positive* $\forall t > 0$.

Proof: Let's consider the equations (3.1a) of the model system (3.1) which is:

$$\begin{aligned} \frac{dS_h}{dt} &= \Lambda_h - \left(\frac{\beta_1 I_h + \beta_2 I_a + \beta_3 D}{N_h}\right) S_h - \mu_h S_h, \\ \frac{dS_h}{dt} &\geq -\left(\frac{\beta_1 I_h + \beta_2 I_a + \beta_3 D}{N_h}\right) S_h - \mu_H S_h. \end{aligned} \tag{3.15}$$

By separating variables (3.15) and integrating we get:

$$\begin{aligned} \frac{dS_h}{S_h} &\geq -\left(\frac{\beta_1 I_h + \beta_2 I_a + \beta_3 D}{N_h} + \mu_h\right) dt, \\ \int \frac{dS_h}{S_h} &\geq -\int_0^t \left(\frac{\beta_1 I_h(s) + \beta_2 I_a(s) + \beta_3 D(s)}{N_h(s)} + \mu_h\right) ds, \\ \ln S_h &\geq -\int_0^t \left(\frac{\beta_1 I_h(s) + \beta_2 I_a(s) + \beta_3 D(s)}{N_h(s)} + \mu_h\right) ds + C, \\ S_h &\geq Ce^{\int_0^t -\left(\frac{\beta_1 I_h(s) + \beta_2 I_a(s) + \beta_3 D(s)}{N_h(s)} + \mu_h\right) ds}. \end{aligned} \tag{3.16}$$

At initial condition we get:

$$S_h(t) \geq S_h(0) e^{\int_0^t -\left(\frac{\beta_1 I_h(s) + \beta_2 I_a(s) + \beta_3 D(s)}{N_h(s)} + \mu_h\right) ds}. \tag{3.17}$$

Then $S_h(t) \geq 0, \forall\, t \geq 0$.

From equation (3.1b) of the model (3.1) we have:

$$\begin{aligned} \frac{dE_h}{dt} &= \left(\frac{\beta_1 I_h + \beta_2 I_a + \beta_3 D}{N_h}\right) S_h - (\gamma_h + \mu_h) E_h, \\ \frac{dE_h}{dt} &\geq -(\gamma_h + \mu_h) E_h. \end{aligned} \tag{3.18}$$

By separating variables and integrating equation (3.18) we get:

$$\begin{aligned} \frac{dE_h}{E_h} &\geq -(\gamma_h + \mu_h) dt, \\ \int \frac{dE_h}{E_h} &\geq \int_0^t -(\gamma_h + \mu_h) ds + C, \\ \ln E_h &\geq \int_0^t -(\gamma_h + \mu_h) ds + C, \\ E_h(t) &\geq Ce^{-(\gamma_h + \mu_h)t}. \end{aligned} \tag{3.19}$$

At initial condition we get:

$$E_h(t) \geq E_h(0)e^{-(\gamma_h+\mu_h)t}. \tag{3.20}$$

Then $E_h \geq 0 \; \forall \; t \geq 0$.

From equation (3.1c) of the model (3.1) we have:

$$\begin{aligned} \frac{dI_h}{dt} &= \gamma_h E_h - (\mu_h+\alpha_h)I_h, \\ \frac{dI_h}{dt} &\geq -(\mu_h+\alpha_h)I_h. \end{aligned} \tag{3.21}$$

By separating variables and solving equation (3.21) we get:

$$\begin{aligned} \frac{dI_h}{I_h} &\geq -(\mu_h+\alpha_h)dt. \\ \int \frac{dI_h}{I_h} &\geq \int_0^t -(\mu_h+\alpha_h)ds + C, \\ \ln I_h &\geq \int_0^t -(\mu_h+\alpha_h)ds + C, \\ I_h(t) &\geq I_h(0)e^{-(\mu_h+\alpha_h)t}. \end{aligned} \tag{3.22}$$

Then, $I_h \geq 0 \; \forall \; t \geq 0$.

Consider model equation (3.1d) from the model system (3.1):

$$\begin{aligned} \frac{dS_a}{dt} &= \Lambda_a - \left(\frac{\beta_4 I_h + \beta_5 Ia + \beta_6 I_D}{N_a} + \mu_a\right) S_a, \\ \frac{dS_a}{dt} &\geq -\left(\frac{\beta_4 I_h + \beta_5 I_a + \beta_6 D}{N_a} + \mu_a\right) S_a. \end{aligned} \tag{3.23}$$

By separating variables and integrating equation (3.23) we get:

$$\begin{aligned} \frac{dS_a}{S_a} &\geq -\left(\frac{\beta_4 I_h + \beta_5 I_a + \beta_6 D}{N_a} + \mu_a\right) ds, \\ \int \frac{dS_a}{S_a} &\geq \int_0^t -\left(\frac{\beta_4 I_h(s) + \beta_5 I_a(s) + \beta_6 D(s)}{N_a(s)} + \mu_a\right) ds + C, \\ \ln S_a &\geq \int_0^t -\left(\frac{\beta_4 I_h(s) + \beta_5 I_a(s) + \beta_6 D(s)}{N_a(s)} + \mu_a\right) ds + C, \\ S_a &\geq Ce^{\int_0^t -\left(\frac{\beta_4 I_h(s) + \beta_5 I_a(s) + \beta_6 D(s)}{N_a(s)} + \mu_a\right) ds}. \end{aligned} \tag{3.24}$$

At initial condition we get:

$$S_a(t) \geq S_a(0)e^{\int_0^t -\left(\frac{\beta_4 I_h(s) + \beta_5 I_a(s) + \beta_6 D(s)}{N_a(s)} + \mu_a\right) ds}. \tag{3.25}$$

So, $S_a \geq 0 \; \forall \; t \geq 0$.

Consider equation (3.1e) of the model system (3.1) which is:

$$\begin{aligned} \frac{dE_a}{dt} &= \left(\frac{\beta_4 I_h + \beta_5 I_a + \beta_6 D}{N_a}\right) S_a - (\gamma_a+\mu_a)E_a, \\ \frac{dE_a}{dt} &\geq -(\gamma_a+\mu_a)E_a. \end{aligned} \tag{3.26}$$

Separate variables and integrate equation (3.26) to get:

$$\begin{aligned} \frac{dE_a}{E_a} &\geq -(\gamma_a+\mu_a)dt, \\ \int \frac{dE_a}{E_a} &\geq \int_0^t -(\gamma_a+\mu_a)ds+C, \\ \ln E_a &\geq \int_0^t -(\gamma_a+\mu_a)ds+C, \\ E_a(t) &\geq Ce^{-(\gamma_a+\mu_a)t}. \end{aligned} \tag{3.27}$$

At initial we get:

$$E_a(t) \geq E_a(0)e^{-(\gamma_a+\mu_a)t}. \tag{3.28}$$

Hence $E_a \geq 0 \; \forall \; t \geq 0$.

Again from the model equation (3.1f) of the model (3.1) we have:

$$\begin{aligned} \frac{dI_a}{dt} &= \gamma_a E_a - (\mu_a+\alpha_a)I_a, \\ \frac{dI_a}{dt} &\geq -(\mu_a+\alpha_a)I_a. \end{aligned} \tag{3.29}$$

By solving the differential equation (3.29) we get:

$$\begin{aligned} \frac{dI_a}{I_a} &\geq -(\mu_a+\alpha_a)dt, \\ \int \frac{dI_a}{I_a} &\geq \int_0^t -(\mu_a+\alpha_a)ds+C, \\ \ln I_a &\geq \int_0^t -(\mu_a+\alpha_a)ds+C, \\ I_a(t) &\geq Ce^{-(\mu_a+\alpha_a)t}. \end{aligned} \tag{3.30}$$

Initially we get:

$$I_a(t) \geq I_a(0)e^{-(\mu_a+\alpha_a)t}. \tag{3.31}$$

Then, $I_a \geq 0 \; \forall \; t \geq 0$.

Lastly from the equation (3.1g) of the model (3.1) we have:

$$\begin{aligned} \frac{dD}{dt} &= \rho I_a - \omega D, \\ \frac{dD}{dt} &\geq -\omega D. \end{aligned} \tag{3.32}$$

By solving the equation (3.32) we get:

$$\begin{aligned} \frac{dD}{D} &\geq -\omega dt, \\ \int \frac{dD}{D} &\geq \int_0^t -\omega dt+C, \\ \ln D &\geq \int_0^t -\omega ds+C, \\ D(t) &\geq Ce^{-\omega t}. \end{aligned} \tag{3.33}$$

At time zero we get:

$$D(t) \geq D(0)e^{-\omega t}. \tag{3.34}$$

Then, $D \geq 0 \; \forall \; t \geq 0$.
Therefore all solutions are positive $\forall t > 0$.

3.3 Disease free equilibrium

The disease-free equilibrium point is the state when there is no disease in the population. When there is no bTB in human and animal populations, the disease-free equilibrium is given by:

$$DF^0 = (S_h, E_h, I_h, S_a, E_a, I_a, D) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_a}{\mu_a}, 0, 0, 0\right). \tag{3.35}$$

3.3.1 The Basic Reproduction Number R_0

The basic reproduction number refers to the average number of new cases that single infectious individual causes when introduced into an entirely susceptible population (Diekmann *et al.*, 1990). It determines whether the disease persists or clears out in the population. When the basic reproduction number $R_0 < 1$, the disease clears out in the population. It persists when the basic reproduction number $R_0 > 1$. When an infectious individual introduced into an entirely susceptible population, he/she infects more than one individuals hence the disease persists (Diekmann *et al.*, 1990; Van, 2002). To compute the basic reproduction number R_0, we use the next generation matrix method where we consider new infections and transfer terms as used by Diekmann *et al.* (1990) and Van (2002). If bTB new infectious and transfer terms are denoted by F_i and V_i respectively, then the basic reproduction number R_0 is given as the maximum eigenvalue. That is:

$$R_0 = \rho(FV^{-1}), \tag{3.36}$$

where:

$$F = \frac{\partial F_i}{\partial X_j}\left(DF^0\right) \;\; and \;\; V = \frac{\partial V_i}{\partial X_j}(DF^0).$$

From the model system (3.1), F_i and V_i are defined as:

$$F_i = \begin{bmatrix} \left(\frac{\beta_1 I_h + \beta_2 I_a + \beta_3 D}{N_h}\right) S_h \\ 0 \\ \left(\frac{\beta_4 I_h + \beta_5 I_a + \beta_6 D}{N_a}\right) S_a \\ 0 \\ 0 \end{bmatrix}, \tag{3.37}$$

(3.38)

and

$$V_i = \begin{bmatrix} (\mu_h + \gamma_h) E_h \\ \gamma_h E_h - (\mu_h + \alpha_h) I_h \\ (\gamma_a + \mu_a) I_a \\ \gamma_a E_a - (\mu_a + \alpha_a) I_a \\ \rho I_a - \omega \end{bmatrix}. \tag{3.39}$$

Jacobian of F_i and V_i at disease free equilibrium is given by:

$$F = \begin{bmatrix} 0 & \beta_1 & 0 & \beta_2 & \beta_3 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_4 & 0 & \beta_5 & \beta_6 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \tag{3.40}$$

and

$$V = \begin{bmatrix} \mu_h + \gamma_h & 0 & 0 & 0 & 0 \\ -\gamma_h & \mu_h + \alpha_h & 0 & 0 & 0 \\ 0 & 0 & \gamma_a + \mu_a & 0 & 0 \\ 0 & 0 & -\gamma_a & \mu_a + \alpha_a & 0 \\ 0 & 0 & 0 & -\rho & \omega \end{bmatrix}. \tag{3.41}$$

Inverse of the matrix (3.41) works out to be:

$$V^{-1} = \begin{bmatrix} \frac{1}{(\gamma_h+\mu_h)} & 0 & 0 & 0 & 0 \\ \frac{\gamma_h}{(\gamma_h+\mu_h)(\alpha_h+\mu_h)} & \frac{1}{(\alpha_h+\mu_h)} & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{(\gamma_a+\mu_a)} & 0 & 0 \\ 0 & 0 & \frac{\gamma_a}{(\gamma_a+\mu_a)(\alpha_a+\mu_a)} & \frac{1}{(\alpha_a+\mu_a)} & 0 \\ 0 & 0 & \frac{\rho\,\gamma_a}{(\gamma_a+\mu_a)(\alpha_a+\mu_a)\,\omega} & \frac{\rho}{(\alpha_a+\mu_a)\,\omega} & \frac{1}{\omega} \end{bmatrix}, \tag{3.42}$$

and the product of matrices (3.40) and (3.42) is:

$$FV^{-1} = \begin{bmatrix} \frac{\beta_1\gamma_h}{(\gamma_h+\mu_h)(\alpha_h+\mu_h)} & q & \frac{\beta_2\gamma_a}{(\alpha_a+\mu_a)(\gamma_a+\mu_a)} + \frac{\beta_3\rho\,\gamma_a}{(\alpha_a+\mu_a)(\gamma_a+\mu_a)\,\omega} & d & \frac{\beta_3}{\omega} \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_4\gamma_h}{(\gamma_h+\mu_h)(\mu_h+\alpha_h)} & f & \frac{\beta_5\gamma_a}{(\gamma_a+\mu_a)(\alpha_a+\mu_a)} + \frac{\beta_6\rho\,\gamma_a}{(\alpha_a+\mu_a)(\gamma_a+\mu_a)\,\omega} & h & \frac{\beta_6}{\omega} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}. \tag{3.43}$$

where

$$\begin{aligned} q &= \frac{\beta_1}{\alpha_h+\mu_h}, \\ d &= \frac{\beta_2}{\alpha_a+\mu_a} + \frac{\beta_3\rho}{(\alpha_a+\mu_a)\,\omega}, \\ f &= \frac{\beta_4}{\alpha_h+\mu_h}, \\ h &= \frac{\beta_5}{\alpha_a+\mu_a} + \frac{\beta_6\rho}{(\alpha_a+\mu_a)\,\omega}. \end{aligned} \tag{3.44}$$

Therefore the basic reproduction number R_0 is given by:

$$\begin{aligned} R_0 =& \frac{1}{2}\left(\frac{\gamma_a\,(\omega\,\beta_5+\rho\,\beta_6)}{(\gamma_a+\mu_a)\,(\mu_a+\alpha_a)\,\omega} + \frac{\beta_1\gamma_h}{(\mu_h+\gamma_h)\,(\mu_h+\alpha_h)}\right) \\ &+\frac{1}{2}\left(\sqrt{\left(\frac{\gamma_a\,(\omega\,\beta_5+\rho\,\beta_6)}{(\gamma_a+\mu_a)\,(\mu_a+\alpha_a)\,\omega} - \frac{\beta_1\gamma_h}{(\mu_h+\gamma_h)\,(\mu_h+\alpha_h)}\right)^2 + 4ce}\right). \end{aligned} \tag{3.45}$$

where, $ce = \dfrac{\gamma_h\gamma_a(\omega\beta_2+\rho\beta_3)}{\omega(\gamma_h+\mu_h)(\alpha_h+\mu_h)(\gamma_a+\mu_a)(\alpha_a+\mu_a)}$.

The terms:

(i) $\frac{1}{\gamma_h + \mu_h}$ is the average period an individual human spent in exposed class.

(ii) $\frac{1}{\gamma_a + \mu_a}$ the average period an individual animal spends in exposed class.

(iii) $\frac{1}{\alpha_h + \mu_h}$ is the average period an infectious human spends in their infectious class.

(iv) $\frac{1}{\alpha_a + \mu_a}$ is the average period an infectious animal spends in their infectious class.

(v) $\frac{\beta_1 \gamma_h}{(\gamma_h + \mu_h)(\alpha_h + \mu_h)}$ and $\frac{\beta_4 \gamma_h}{(\gamma_h + \mu_h)(\alpha_h + \mu_h)}$ are the proportions of E_h that move into I_h.

(vi) $\frac{(\omega\beta_5 + \rho\beta_6)\gamma_a}{\omega(\gamma_a + \mu_a)(\alpha_a + \mu_a)}$ is the sum of proportions of infected animals that progress from E_a to I_a after coming into contact with infectious animals and after consuming infectious dairy products.

(vii) $\frac{\gamma_h \gamma_a (\omega\beta_2 + \rho\beta_3)}{\omega(\gamma_h + \mu_h)(\alpha_h + \mu_h)(\gamma_a + \mu_a)(\alpha_a + \mu_a)}$ is the sum of proportions of infected humans who develop bTB by contacting infectious animals and after consuming infectious dairy products.

3.3.2 Sensitivity Analysis of basic Reproduction number R_0

According to Fellin *et al.* (2005) and Silva and Torres (2013) sensitivity analysis of R_0 helps to understand effects of each parameter on the model output and their influence in the spread of disease in the population. We adopted normalized forward sensitivity index as used by Chitnis *et al.* (2008) and Silva and Torres (2013) to perform sensitivity analysis of R_0. A normalized forward index of variable β with respect to basic reproduction number R_0 is defined as:

$$\Upsilon_{\beta}^{R_0} = \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0}. \tag{3.46}$$

Using estimated parameters and from related literature, sensitivity index of each parameter with respect to basic reproduction number R_0 is computed and summarized in Table 3.

Table 3: Sensitivity Indices for R_0

Parameter	Index value
β_1	0.0271.
β_2	0.0530.
β_3	0.1177.
β_4	0.1708.
β_5	0.3601.
β_6	0.2713.
γ_h	0.0892.
μ_h	-0.2728.
α_h	-0.0144.
γ_a	0.1671.
α_a	-0.5793.
μ_a	-0.3898.
ρ	0.3890.
ω	-0.3890.

Sensitivity analysis shows that humans infection rate due to the consumption of dairy products β_3 and contact rate with infected animals β_2, animal infection rates due to contact with infectious animals β_5, and the rate at which animal consume dairy product β_6 drive the dynamics of bTB. Generally, the most sensitive parameter is the rate of producing dairy products, ρ. The sensitivity indices of R_0 with respect to ρ, β_5 and β_6 are 0.3898, 0.3601 and 0.2713, respectively. The increases of these parameters by 10%, lead to an increase in basic reproduction number R_0 by 38.9%, 36% and 27.1%, respectively. However, when animals mortality rate due to disease α_a, the natural death rate for animals μ_a, humans disease-induced death rate α_h, the natural death rate for humans μ_h and dairy products decaying rate ω increase, the basic reproduction number R_0 decreases consequently.

3.3.3 Stability Analysis for Disease Free Equilibrium (DFE)

In this section, we use the linearization method to establish local stability of disease-free equilibrium since the model has the possibility of undergoing backward bifurcation when $R_0 = 1$, therefore global stability of DFE is not considered.

Disease-free equilibrium is locally asymptotically stable when $R_0 < 1$. Negative eigenvalues from the linearized system at disease-free equilibrium show that the disease-free equilibrium is locally stable. By using the linearization method, the Jacobian of the system (3.1) at DFE is given by;

$$J=\begin{bmatrix} -\mu_h & 0 & -\beta_1 & 0 & 0 & -\beta_2 & -\beta_3 \\ 0 & -\mu_h-\gamma_h & \beta_1 & 0 & 0 & \beta_2 & \beta_3 \\ 0 & \gamma_h & -\mu_h-\alpha_h & 0 & 0 & 0 & 0 \\ 0 & 0 & -\beta_4 & -\mu_a & 0 & -\beta_5 & -\beta_6 \\ 0 & 0 & \beta_4 & 0 & -\mu_a-\gamma_a & \beta_5 & \beta_6 \\ 0 & 0 & 0 & 0 & \gamma_a & -\mu_a-\alpha_a & 0 \\ 0 & 0 & 0 & 0 & 0 & \rho & -\omega \end{bmatrix}. \tag{3.47}$$

From first and fourth columns, the eigenvalues are $-\mu_h$ and $-\mu_a$. Matrix (3.47) now reduces to

$$K=\begin{bmatrix} -\mu_h-\gamma_h & \beta_1 & 0 & \beta_2 & \beta_3 \\ \gamma_h & -\mu_h-\alpha_h & 0 & 0 & 0 \\ 0 & \beta_4 & -\mu_a-\gamma_a & \beta_5 & \beta_6 \\ 0 & 0 & \gamma_a & -\mu_a-\alpha_a & 0 \\ 0 & 0 & 0 & \rho & -\omega \end{bmatrix}. \tag{3.48}$$

We analyze matrix K by using trace tr and determinant det. Disease free equilibrium is locally stable if trace is negative $tr(K)<0$ and $det(K)>0$. From (3.48) trace of the matrix K is given by

$$tr(K)=-((\mu_h+\gamma_h)+(\mu_h+\gamma_h)+(\mu_a+\gamma_a)+(\mu_a+\alpha_a)+\omega)<0. \tag{3.49}$$

Determinant $det(K)$ is given by

$$\begin{aligned} det(K)=&(\gamma_h+\mu_h)(\alpha_h+\mu_h)\omega\beta_5\gamma_a+(\gamma_h+\mu_h)(\mu_h+\alpha_h)\rho\beta_6\gamma_a \\ &+(\gamma_a+\mu_a)(\alpha_a+\mu_a)\omega\beta_1\gamma_h+\omega\beta_2\beta_4\gamma_h\gamma_a+\rho\beta_3\beta_4\gamma_h\gamma_a \\ &-\rho\beta_1\beta_6\gamma_h\gamma_a(\gamma_h+\mu_h)(\alpha_h+\mu_h)(\gamma_a+\mu_a)(\alpha_a+\mu_a)\omega+\omega\beta_1\beta_5\gamma_h\gamma_a. \end{aligned} \tag{3.50}$$

$\det(K)>0$ if

$$\begin{aligned} \frac{\beta_1\gamma_h}{(\mu_h+\gamma_h)(\mu_h+\alpha_h)}+\frac{\gamma_a(\omega\beta_5+\rho\beta_6)}{\omega(\gamma_a+\mu_a)(\mu_a+\alpha_a)}+\frac{\beta_4\gamma_H(\omega\beta_2\gamma_a+\rho\beta_3\gamma_a)}{\omega(\gamma_h+\mu_h)(\alpha_h+\mu_h)(\gamma_a+\mu_a)(\alpha_a+\mu_a)} \\ -\frac{\gamma_h\gamma_a\beta_1(\omega\beta_5+\rho\beta_6)}{\omega(\gamma_h+\mu_h)(\alpha_h+\mu_h)(\gamma_a+\mu_a)(\alpha_a+\mu_a)}>1. \end{aligned} \tag{3.51}$$

Theorem 2: *The disease free equilibrium is locally asymptotically stable if* $R_0<1$ *and condition* (3.51) *holds.*

However, the disease-free equilibrium may not be globally asymptotically stable due to the possibility of the model (3.1) to undergo backward bifurcation when $R_0 = 1$.

Since the trace of matrix K is negative, and its determinant is positive provided condition (3.51) holds, then DFE is locally asymptotically stable.

3.4 Endemic Equilibrium

Endemic equilibrium is a state when the disease prevails in the population. To compute endemic equilibrium, right side of each equation in model system (3.1) is set to zero. That is:

$$\Lambda_h - \left(\frac{\beta_1 I_h + \beta_2 I_a + \beta_3 D}{N_h}\right) S_h - \mu_h S_h = 0. \tag{3.52a}$$

$$\left(\frac{\beta_1 I_h + \beta_2 I_a + \beta_3 D}{N_h}\right) S_h - (\gamma_h + \mu_h) E_h = 0. \tag{3.52b}$$

$$\gamma_h E_h - (\mu_h + \alpha_h) I_h = 0. \tag{3.52c}$$

$$\Lambda_a - \left(\frac{\beta_4 I_h + \beta_5 I_a + \beta_6 D}{N_a}\right) S_a - \mu_a S_a = 0. \tag{3.52d}$$

$$\left(\frac{\beta_4 I_h + \beta_5 I_a + \beta_6 D}{N_a}\right) S_a - (\gamma_a + \mu_a) E_a = 0. \tag{3.52e}$$

$$\gamma_a E_a - (\mu_a + \alpha_a) I_a = 0. \tag{3.52f}$$

$$\rho I_a - \omega D = 0. \tag{3.52g}$$

Solving system (3.52) in terms of force of infection, we obtain;

$$\begin{aligned}
S_h^* &= \frac{\Lambda_h}{\lambda_h^* + \mu_h} \\
E_h^* &= \frac{\lambda_h \Lambda_h}{(\mu_h + \gamma_h)\left(\lambda_h^* + \mu_h\right)} \\
I_h^* &= \frac{\gamma_h \lambda_h^* \Lambda_h}{(\mu_h + \alpha_h)(\mu_h + \gamma_h)\left(\lambda_h^* + \mu_h\right)} \\
S_a^* &= \frac{\Lambda_a}{\lambda_a^* + \mu_a} \\
E_a^* &= \frac{\lambda_a^* \Lambda_a}{(\mu_a + \gamma_a)(\lambda_a^* + \mu_a)} \\
I_a^* &= \frac{\gamma_a \lambda_a^* \Lambda_a}{(\mu_a + \alpha_a)(\mu_a + \gamma_a)(\lambda_a^* + \mu_a)} \\
D^* &= \frac{\rho\, \gamma_a \lambda_a^* \Lambda_a}{\omega\,(\mu_a + \alpha_a)(\mu_a + \gamma_a)(\lambda_a^* + \mu_a)}.
\end{aligned} \tag{3.53}$$

3.4.1 Bifurcation Analysis

To determine the possibility of model (3.1) to undergo backward bifurcation when $R_0 = 1$, we rename the state variables $S_h, E_h, I_h, S_a, E_a, I_a, D$ to be $x_1, x_2, x_3, x_4, x_5, x_6, x_7$ respectively, where $N_h = x_1 + x_2 + x_3$ and $N_a = x_4 + x_5 + x_7$. By introducing the vector notations $X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7)^T$, the model system (3.1) is now written as $\frac{dX}{dt} = F(X)$, where $F(X) = (f_1, f_2, f_3, f_4, f_5, f_6, f_7)^T$. The model system (3.1) is then re-written as:

$$\begin{aligned}
\frac{dx_1}{dt} &= f_1 = \Lambda_h - \left(\frac{\beta_1 x_3 + \beta_2 x_6 + \beta_3 x_7}{x_1 + x_2 + x_3}\right) x_1 - \mu_h x_1, \\
\frac{dx_2}{dt} &= f_2 = \left(\frac{\beta_1 x_3 + \beta_2 x_6 + \beta_3 x_7}{x_1 + x_2 + x_3}\right) x_1 - (\gamma_h + \mu_h) x_2, \\
\frac{dx_3}{dt} &= f_3 = \gamma_h x_2 - (\mu_h + \alpha_h) x_3, \\
\frac{dx_4}{dt} &= f_4 = \Lambda_a - \left(\frac{\beta_4 x_3 + \beta_5 x_6 + \beta_6 x_7}{x_4 + x_5 + x_6}\right) x_4 - \mu_a x_4, \\
\frac{dx_5}{dt} &= f_5 = \left(\frac{\beta_4 x_3 + \beta_5 x_6 + \beta_6 x_7}{x_4 + x_5 + x_6}\right) x_4 - (\gamma_a + \mu_a) x_5, \\
\frac{dx_6}{dt} &= f_6 = \gamma_a x_5 - (\mu_a + \alpha_a) x_6, \\
\frac{dx_7}{dt} &= f_7 = \rho x_6 - \mu_D x_7.
\end{aligned} \tag{3.54}$$

The Jacobian of the system (3.1) at disease free equilibrium is given by:

$$J = \begin{bmatrix}
-\mu_h & 0 & -\beta_1 & 0 & 0 & -\beta_2 & -\beta_3 \\
0 & -\mu_h - \gamma_h & \beta_1 & 0 & 0 & \beta_2 & \beta_3 \\
0 & \gamma_h & -\mu_h - \alpha_h & 0 & 0 & 0 & 0 \\
0 & 0 & -\beta_4 & -\mu_a & 0 & -\beta_5 & -\beta_6 \\
0 & 0 & \beta_4 & 0 & -\mu_a - \gamma_a & \beta_5 & \beta_6 \\
0 & 0 & 0 & 0 & \gamma_a & -\mu_a - \alpha_a & 0 \\
0 & 0 & 0 & 0 & 0 & \rho & -\omega
\end{bmatrix}. \tag{3.55}$$

To determine whether the system (3.1) undergoes backward bifurcation at $R_0 = 1$, we adopt the theorem in Gumel and Song (2008) which is restated as follows;

Theorem 3: *Consider the following general system of ordinary differential equations with a parameter β^*.*
$\frac{dx}{dt} = f(x, \beta^*), f : \Re \times \Re^n \mapsto \Re^n$ *and* $f \in C^2(\Re^n \times \Re)$ *where 0 is an equilibrium point of the system (that is $f(0, \beta^*) \equiv 0 \ \forall \beta^*$ and*

1. $A = D_x f(0,0) = \frac{\partial f_i}{\partial x_j}(0,0)$ *is a linearization matrix of the system around the equilibrium 0 with β^* at 0.*

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

3. *Matrix A has a right eigenvectors w and left eigenvectors v corresponding to the zero eigenvalues.*

Let f_k be the k^{th} component of f and

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \tag{3.56}$$

$$b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(0,0). \tag{3.57}$$

Then, the local dynamics of the system around the equilibrium point is totally determined by the signs of a and b. Particularly if $a > 0$ and $b > 0$ then a backward bifurcation occurs at $\beta^* = 0$.
The local dynamics at (3.54) *around 0 are totally determined by signs of a and b.*

i. $a > 0$, $b > 0$. When $\beta^* < 0$ with $|\beta^*| << 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0 < \beta^* << 1$, 0 is unstable and there exists a negative and locally asymptotically stable equilibrium;

ii. $a < 0$, $b < 0$. When $\beta^* < 0$ with $|\beta^*| << 1$, 0 is unstable; when $0 < \beta^* << 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium;

iii. $a > 0$, $b < 0$. When $\beta^* < 0$ with $|\beta^*| << 1$, 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0 < \beta^* << 1$, 0 is stable, and a positive unstable equilibrium appears;

iv. $a < 0$, $b > 0$. When β^* changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.

Suppose we choose the bifurcation parameter to be $\beta_3 = \beta^*$ when $R_0 = 1$. Now, solving for $\beta_3 = \beta^*$ when $R_0 = 1$ we get:

$$\begin{aligned} \beta_3 = \beta^* = M &\left(2 - \frac{\beta_4 \gamma_h}{(\gamma_h + \mu_h)(\alpha_h + \mu_h)} - \frac{\gamma_a(\rho\beta_6 + \omega\beta_2)}{(\gamma_a + \mu_a)(\alpha_a + \mu_a)}\right)^2 \\ + M &\left(\frac{\beta_1 \gamma_h}{(\gamma_h + \mu_h)(\alpha_h + \mu_h)} - \frac{\gamma_a(\rho\beta_6 + \omega\beta_2)}{(\gamma_a + \mu_a)(\alpha_a + \mu_a)}\right)^2. \end{aligned} \tag{3.58}$$

where;

$$M = \frac{(\gamma_a+\mu_a)(\alpha_a+\mu_a)(\gamma_h+\mu_h)(\alpha_h+\mu_h)\omega}{\gamma_h\gamma_a\rho\beta_4}.$$

From (3.55), right eigenvectors $w = (w_i)^T$ where $i = 1,2...7$ are:

$$\begin{aligned}
w_5 &= \frac{\omega(\alpha_a+\mu_a)w_7}{\gamma_a\rho}, \quad w_6 = \frac{\omega w_7}{\rho},\\
w_3 &= \left(\frac{\omega(\gamma_a+\mu_a)(\alpha_a+\mu_a)-\gamma_a(\omega\beta_5+\rho\beta_6)}{\gamma_a\rho\beta_4}\right)w_7,\\
w_4 &= \left(\frac{\omega(\gamma_a+\mu_a)(\alpha_a+\mu_a)-2(\omega\beta_5+\rho\beta_6)\gamma_a}{\gamma_a\mu_a\rho}\right)w_7,\\
w_1 &= -\left(\frac{\omega M_1+(\omega\beta_5+\rho\beta_6)+\gamma_a\beta_4(\omega\beta_2+\rho\beta_3)}{\beta_4\gamma_a\mu_h\rho}\right)w_7,\\
w_2 &= \left(\frac{\omega(\gamma_a+\mu_a)(\alpha_a+\mu_a)-\gamma_a(\omega\beta_5+\rho\beta_6)(\alpha_h+\mu_h)}{\gamma_h\gamma_a\beta_4\rho}\right)w_7.
\end{aligned} \tag{3.59}$$

where $w_7 > 0$ is free right eigenvector and $M_1 = (\gamma_a+\mu_a)(\alpha_a+\mu_a)$.

The left eigenvectors $v = (v_i)^T$ where $i = 1,2...7$ are:

$$\begin{aligned}
v_1 &= v_4 = 0. \quad v_2 = \frac{\gamma_h v_3}{\gamma_h+\mu_h},\\
v_5 &= \left(\frac{(\gamma_h+\mu_h)(\alpha_h+\mu_h)-\gamma_h\beta_2}{\beta_4(\gamma_h+\mu_h)}\right)v_3,\\
v_6 &= \left(\frac{(\gamma_a+\mu_a)((\gamma_h+\mu_h)(\alpha_h+\mu_h)-\gamma_h\beta_2)}{\gamma_a\beta_4(\gamma_h+\mu_h)}\right)v_3,\\
v_7 &= \left(\frac{(\gamma_h\beta_3\beta_4+\beta_6((\gamma_h+\mu_h)(\alpha_h+\mu_h)-\gamma_h\beta_2)}{\omega(\gamma_h+\mu_h)\beta_4}\right)v_3.
\end{aligned} \tag{3.60}$$

where $v_3 > 0$ is free left eigenvector.

Computation of a

From the model system (3.1) the associated non-zero partial derivatives of F at disease free equilibrium are given by:

$$\begin{aligned}
&\frac{\partial^2 f_2}{\partial x_3^2} = -\frac{2\beta_1\mu_h}{\Lambda_h}, \frac{\partial^2 f_2}{\partial x_2\partial x_3} = -\frac{\beta_1\mu_h}{\Lambda_h}, \frac{\partial^2 f_2}{\partial x_2\partial x_6} = -\frac{\beta_2\mu_h}{\Lambda_h}, \frac{\partial^2 f_2}{\partial x_2\partial x_7} = \frac{-\beta^*\mu_h}{\Lambda_h},\\
&\frac{\partial^2 f_2}{\partial x_3\partial x_6} = -\frac{\beta_2\mu_h}{\Lambda_h}, \frac{\partial^2 f_2}{\partial x_3\partial x_7} = \frac{-\beta^*\mu_h}{\Lambda_h}, \frac{\partial^2 f_5}{\partial x_6^2} = -\frac{2\beta_5\mu_a}{\Lambda_a} \frac{\partial^2 f_5}{\partial x_3\partial x_5} = -\frac{2\beta_4\mu_a}{\Lambda_a},\\
&\frac{\partial^2 f_5}{\partial x_3\partial x_6} = -\frac{\beta_4\mu_a}{\Lambda_a}, \frac{\partial^2 f_5}{\partial x_5\partial x_6} = \frac{-\beta_5\mu_a}{\Lambda_a}, \frac{\partial^2 f_5}{\partial x_5\partial x_7} = \frac{-\beta_6\mu_a}{\Lambda_a}, \frac{\partial^2 f_5}{\partial x_6\partial x_7} = \frac{-\beta_6\mu_a}{\Lambda_a}.
\end{aligned} \tag{3.61}$$

Since $v_1 = v_4 = 0$ it follows that,

$$a = v_2\sum_{i,j=1}^{n} w_i w_j \frac{\partial^2 f_2}{\partial x_i\partial x_j} + v_5\sum_{i,j=1}^{n} w_i w_j \frac{\partial^2 f_5}{\partial x_i\partial x_j}. \tag{3.62}$$

To compute the values of a we substitute the partial derivatives from (3.61) into (3.62) to get:

$$\begin{aligned} a = &\left(\frac{2\mu_h\Lambda_h w_3 M_2\beta_1\gamma_a(\omega\beta_5+\rho\beta_6)}{\Lambda_h\gamma_h\Lambda_a\gamma_a\rho\beta_4}\right)w_7v_2 \\ &-\left(\frac{2\mu_h\Lambda_a w_3 M_1(\beta_1\omega M_1+\gamma_a\beta_4(\omega\beta_2+\rho\beta_3))}{\Lambda_h\gamma_h\Lambda_a\gamma_a\rho\beta_4}\right)w_7v_2 \\ &-\left(\frac{2\Lambda_h\gamma_h\omega\beta_4\mu_a(w_5+w_6)M_1}{\Lambda_h\gamma_h\Lambda_a\gamma_a\rho\beta_4}\right)w_7v_5. \end{aligned}$$

where $M_2 = \alpha_h+\mu_h+\gamma_h$.

To analyze the sign of a we consider two cases.

Case I:

$a<0$ if

$$\frac{\gamma_a(\omega\beta_5+\rho\beta_6)}{\omega(\gamma_a+\mu_a)(\alpha_a+\mu_a)}<1$$

$$\text{and } \frac{\Lambda_h\gamma_h\mu_a\omega\beta_4(w_5+w_6)M_1v_5+\Lambda_a\mu_h w_3(\omega M_1\beta_1+(\omega\beta_2+\rho\beta_3)\gamma_a\beta_4)M_1v_2}{\Lambda_h\gamma_a\mu_h M_2\beta_1(\omega\beta_5+\rho\beta_6)w_3v_2}>1 \quad (3.63)$$

Case II:

$a>0$ if

$$\frac{\gamma_a(\omega\beta_5+\rho\beta_6)}{\omega(\gamma_a+\mu_a)(\alpha_a+\mu_a)}>1$$

$$\text{and } \frac{\Lambda_h\gamma_h\mu_a\omega\beta_4(w_5+w_6)M_1v_5+\Lambda_a\mu_h w_3(\omega M_1\beta_1+(\omega\beta_2+\rho\beta_3)\gamma_a\beta_4)M_1v_2}{\Lambda_h\gamma_a\mu_h M_2\beta_1(\omega\beta_5+\rho\beta_6)w_3v_2}<1 \quad (3.64)$$

Computation of b

Recall from (3.60) since $v_1=v_4=0$, b becomes:

$$\begin{aligned} b &= v_2\sum_{i=1}^{n} w_i\frac{\partial^2 f_k}{\partial x_i\partial\beta^*}(0,0), \\ b &= v_2w_7\frac{\partial^2 f_2}{\partial x_7\partial\beta^*}, \\ b &= \frac{\gamma_h w_7v_3}{\gamma_H+\mu_h}>0. \end{aligned} \quad (3.65)$$

From the computation of a and b we can establish the following results.

Theorem 4: *If*

$$\frac{\gamma_a(\omega\beta_5+\rho\beta_6)}{\omega(\gamma_a+\mu_a)(\alpha_a+\mu_a)}>1$$

and

$$\frac{\Lambda_h\gamma_h\mu_a\omega\beta_4(w_5+w_6)M_1v_5+\Lambda_a\mu_h w_3(\omega M_1\beta_1+(\omega\beta_2+\rho\beta_3)\gamma_a\beta_4)M_1v_2}{\Lambda_h\gamma_a\mu_h M_2\beta_1(\omega\beta_5+\rho\beta_6)w_3v_2}<1$$

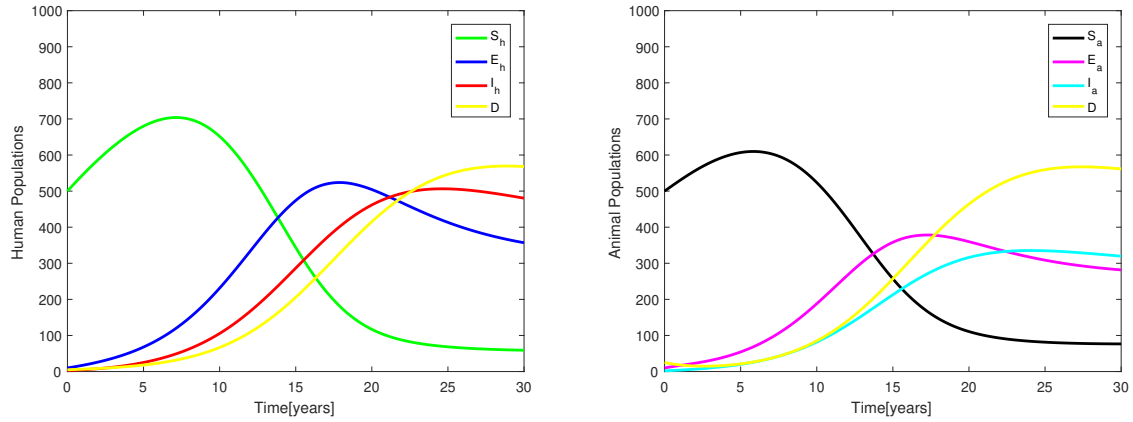
then the model system (3.1) *undergoes backward bifurcation when* $R_0=1$.

3.5 Numerical Simulation

In this section, we discuss the dynamics of bTB in humans and animal population by considering parameters which drive the transmission dynamics of bTB. The initial condition we assumed to be $S_h=530, E_h=15, I_h=4, S_a=500, E_a=25, I_a=10$ and $D=13$. We use estimated parameters and some from related literature as summarized in Table 4.

Table 4: Parameter Values of the Model system (3.1)

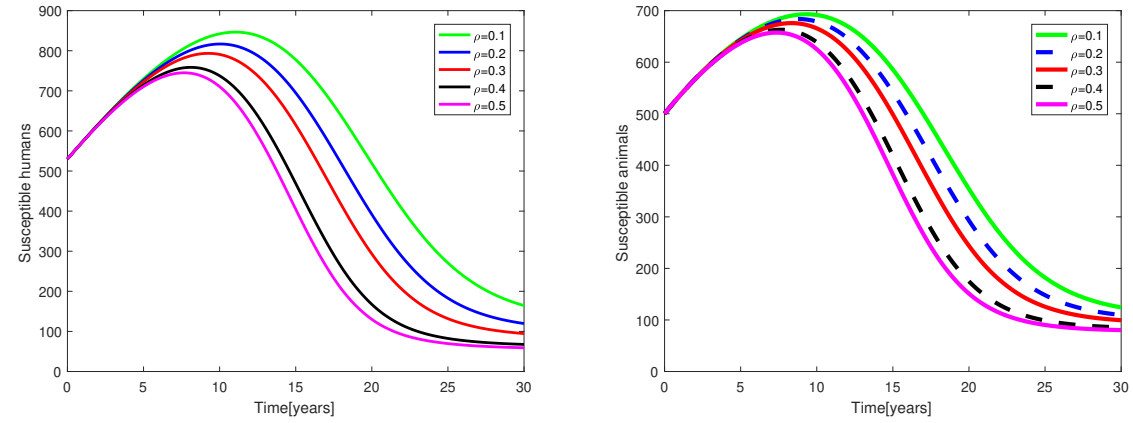
Parameter	Interpretation	Value yr^{-1}	Source.
β_2	human infection rate from infected animals	0.55	Hassan *et al.* (2014).
β_5	rate of cow infected via animal	0.6	Agusto *et al.* (2011).
β_6	rate of animals infected via dairy products	0.34	Estimated.
γ_h	progression rate from E_h to I_h	0.15	Dye and Williams (2008).
μ_h	human natural death rate	0.01	Liu *et al.* (2016).
α_h	human death rate due to disease induced	0.139	Liu *et al.* (2016).
γ_a	progression rate from E_a to I_a	0.18	Ssematimba *et al.* (2015).
α_a	animal death due to disease induced	0.0304	Agusto *et al.* (2011).
μ_a	animal natural death rate	0.05	Mariner *et al.* (2006).
ρ	dairy production rate	0.69	Estimated.
ω	rate of decaying dairy products	0.4	Estimated.
β_3	human infection rate from infected dairy products	0.999	Estimated.
β_4	rate of cow infected via human	0.25	Estimated.
β_1	human infection rate from infected human	0.35	Estimated.



(a) Humans Population (b) Animals Population

Figure 2: Dynamics of humans and animals populations

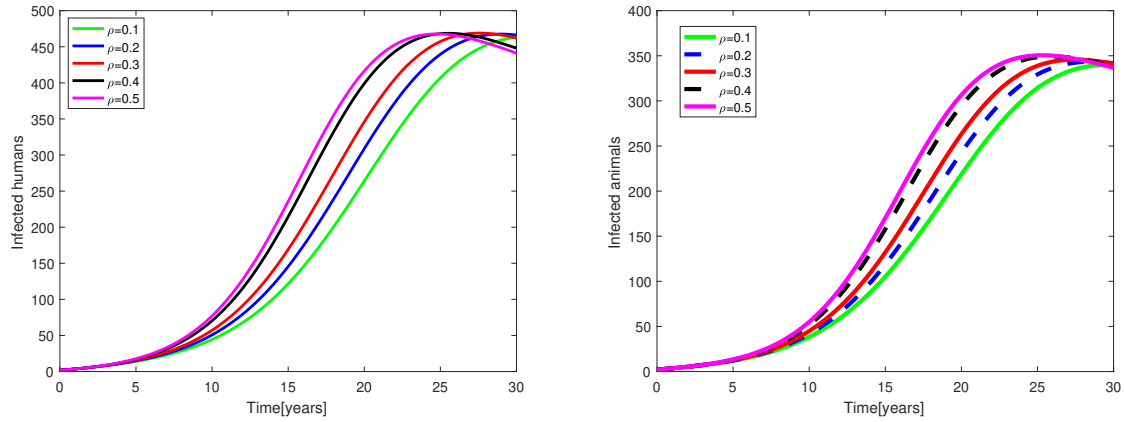
Susceptible humans and animals decrease after acquiring bTB when they come into contact with infectious humans and animals, and after consuming infectious dairy products, as shown in Fig. 2. However, infectious classes increase as individuals from susceptible class acquire bTB and move to the exposed class and then to infectious class.



(a) Susceptible humans (b) Susceptible animals

Figure 3: Variation of S_h and S_a due to consumption of dairy products

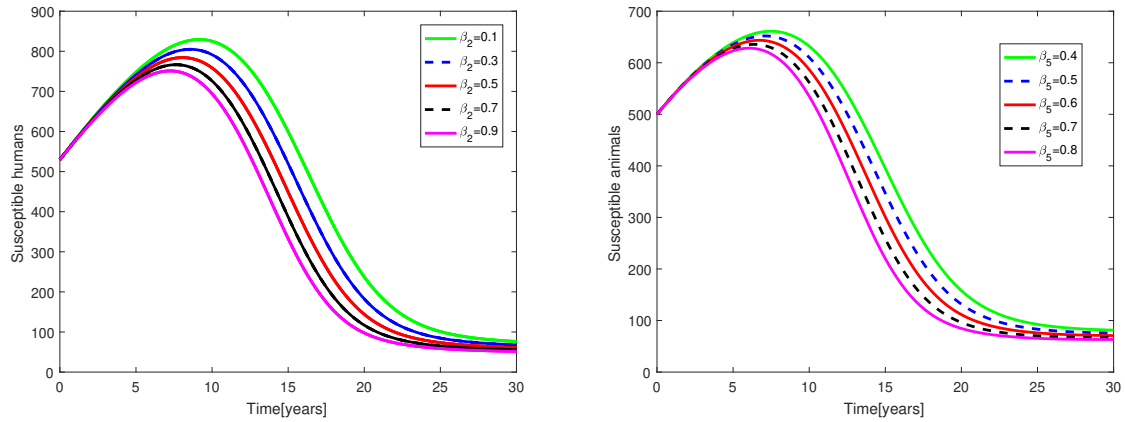
Figures 3 (a) and (b) show the variations in the rate of producing infectious dairy products. The increase in the rate of consumption of contaminated products leads to a decrease in the number of susceptible humans and susceptible animals. For example at $t = 10$ years an increase in the contaminated dairy products from 10% to 50% leads to a decrease in the number of susceptible humans from 800 to 700.



(a) Infected Humans (b) Infected animals

Figure 4: Variation of I_h and I_a due to consumption of dairy products

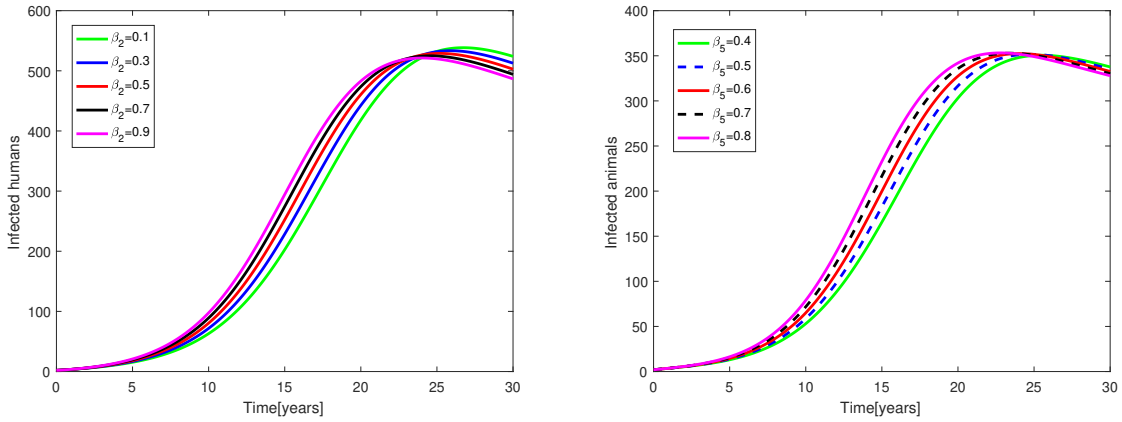
As the rate of producing contaminated dairy products increased, infected classes were increasing proportionally. For instance at $t = 15$ years, when the rate at which contaminated dairy products increased from 10% to 50%, the infected human class increased from 50 to 150 individuals while infected animals class increase from 100 to 150 as displayed in Fig. 4. This means that one infected animal double the number of infected humans.



(a) Susceptible humans (b) Susceptible animals

Figure 5: Variations of S_h and S_a when they interact with infected animals

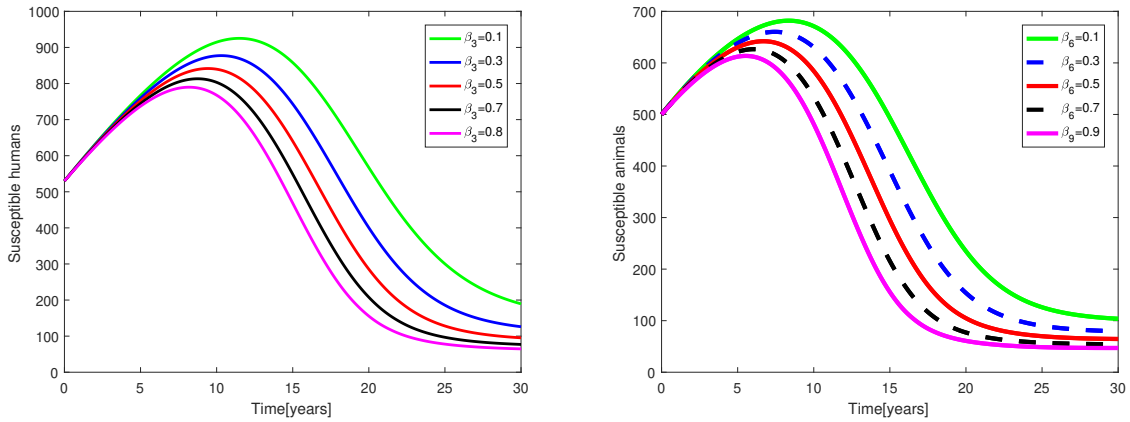
Figure 5 shows the effects of varying human and animal transmission rates from infected animals. The increase in the interaction between infected animals with susceptible humans and animals leads to decreases in susceptible classes, as shown in Fig. 5. For example, Fig. 5(a) shows that susceptible human class decreases as the interaction rate increases form 10%-90%. Also, Fig. 5(b) shows that susceptible animal class decreases as the interaction rates increase from 10%-80%.



(a) Infected Humans (b) Infected animals

Figure 6: Variation of I_h and I_a classes when they interact with infected animals

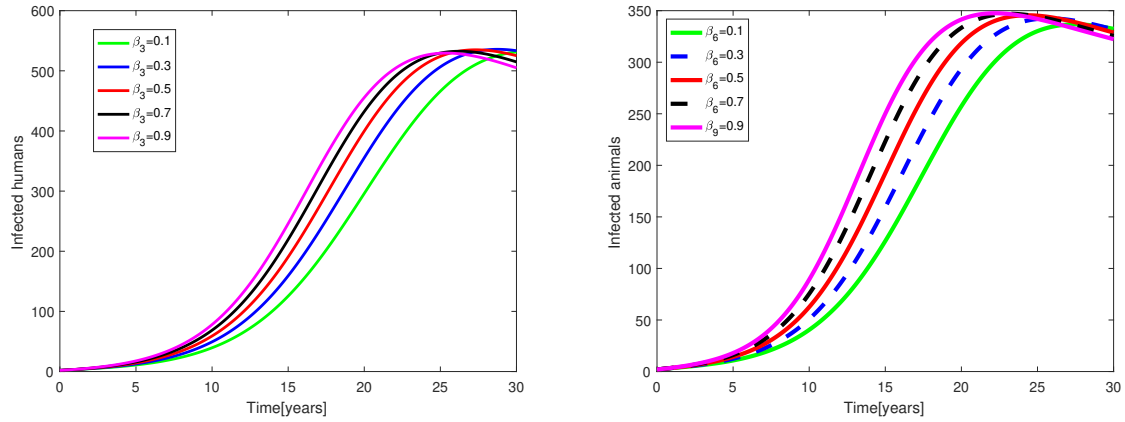
Infected human and animal classes increase over time as we vary infection rates. When the infection rate increased from 10% to 90% infected human class decreases, as shown in Fig. 6(a). Also, the infected animal class decreases as the rate of infection increased from 10% to 80%, as shown in Fig. 6(b).



(a) Susceptible humans (b) Susceptible animals

Figure 7: The impact of rates of transmission from dairy products to S_h and S_a.

Figure 7 shows the impacts of transmission rates due to the consumption of infectious dairy products β_3 and β_6. Susceptible human and animal classes decrease as the consumption rate increases from 10% to 80%, as shown in Fig. 7.



(a) Infected human (b) Infected animal

Figure 8: The impact of rates of transmission from dairy products to I_h and I_a.

Figure 8(a) and (b) show the impacts of increasing the consumption of infectious dairy products on infected human and animals. Infected human and animal classes increases as consumption of infectious dairy products increase from 10% to 90%, as shown in Fig. 8.

3.6 Conclusion

A deterministic model for transmission dynamics of bTB was developed and analyzed to determine parameters that drive the disease. We computed basic reproduction number R_0 and determined the sensitivity index for each parameter with respect to R_0. The sensitivity analysis shows that the animal infection rate from infectious animals β_5, production of infectious dairy products ρ, the human infection rate from dairy products β_3, and humans infection rate from infectious animals β_2, drive the dynamics of bTB. The stability of equilibrium states investigated, whereby disease-free equilibrium DFE is locally asymptotically stable when the basic reproduction number $R_0 < 1$. However, both disease-free and endemic equilibria are not globally stable due to the possibility of the model to undergo backward bifurcation when the basic reproduction number $R_0 = 1$. The disease can be contained if control strategies would target to reduce the most sensitive parameter values to the spread of the disease.

CHAPTER FOUR
RESULTS AND DISCUSSION

4.1 Bovine Tuberculosis Model with Control Strategies

Bovine tuberculosis is a form of zoonotic tuberculosis that has received little attention despite its impacts on human health and the economy. Pastoral communities, especially in Africa, do not know how bovine tuberculosis is transmitted and controlled. In such communities, the interaction between human beings and animals like cows, goats, sheep, and pigs is common. On top of that, the consumption of raw dairy products like unpasteurized milk and raw meat is a common practice. As if that is not enough, there are frequent contacts between domestic and wild animals like buffalo and badger, which occurs in communities that live around the national parks like Masai, Datooga, and Hadzabe to mention the few. Under these circumstances, when a domestic animal like cow, goat or sheep infected with bTB, it is easy to spread the disease to human beings, and other domestic and wild animals and vice versa. Through contacts between domestic and wild animals, bTB has spread from buffalo and badger to cattle, goats, pigs, horses and sheep (WHO, 2016). Domestic animals as well have spread bTB to human beings.

In order to contain the spread of bTB as recommended in chapter three, early diagnosis of the disease is essential. When an individual diagnosed with bTB, he/she advised undergoing treatment. The treatment which cures pulmonary tuberculosis (TB) also used to treat bTB. Liu *et al.* (2016), Cousins and Roberts (2001) in their study they suggested that quarantine and slaughtering of infected animals that is to remove infected animals from the herd reducing contacts with a human being and other animals and pasteurization of milk are the ways of controlling the transmission. In this study, we propose an inspection of dairy products, quarantine of infected animals and the treatment of infected humans to contain the transmission of bTB.

In this chapter, we extend the basic model by introducing control parameters and discuss how they can control the transmission of bTB. Treatment of infected humans and quarantine of infected animals helps to prevent the transmission of bTB from infected to susceptible individuals; hence the rates of transmission will be reduced. Also, an inspection of dairy products helps to know whether meat or milk is infected to take precautions.

4.2 Model Formulation

The model of bTB in human and livestock is extended by including controls. Dynamics of bTB is grouped into human population and animals population. Human population is divided into susceptible class S_h exposed class E_h and infected class I_h. Animals population is divided into susceptible class S_a, exposed/latent class E_a and infectious class I_a.

Susceptible class S_h increases through birth and recovery at rates Λ_h and Π_h respectively. However, they acquire disease and become latent after coming into contact with infectious humans, infectious animals and by consuming infectious dairy products at a rate $X_h = \frac{(1-\tau_h)\beta_1 I_h + (1-\tau_a)\beta_2 I_a + \beta_3(1-\varepsilon)D}{N_h}$. Parameters τ_h, τ_a and ε represent the rates at which infected humans treated, infected animals quarantined, and dairy products inspected, respectively.

Exposed human class E_h increases when susceptible class acquire bTB and moves into class at the rate of X_h. However, individuals in the class decrease by dying naturally at the rate of μ_h, and when they develop symptoms and progress into infectious class at the rate of γ_h.

Infectious class increases when an individual from exposed class progress into infectious class at a rate of γ_h. However, they decrease due to disease-induced and by dying naturally at rates α_h and μ_h respectively. They also decrease following the quarantine of infected animals at the rate of τ_a.

Susceptible animals S_a increase through birth and migration at a rate Λ_a. They acquire bovine tuberculosis latent infection following contacts with infectious humans and animals, and after consuming infectious dairy products at a rate $X_a = \frac{\beta_4(1-\tau_h)I_h + \beta_5(1-\tau_a)I_a + \beta_6(1-\varepsilon)D}{N_a}$.

Exposed animals E_a increase following latent infection of susceptible animals S_a at a rate X_a. However, they decrease as they develop symptoms and progress into an infectious state at a rate of γ_a and by dying naturally at a rate of μ_a.

Infectious animals I_a increase at a rate γ_a and diminish due to disease-induced mortality at a rate α_a and by quarantine of infected animals at a rate τ_a. In this class animals also suffer natural mortality at a rate of μ_a.

Infectious animals produce dairy products at a rate of ρ the remaining products leak at rate ω.

In the control model, we assumed that there is no interaction between susceptible animals and quarantined animals. Treatment of infected human and permanent quarantine of infected animals helps to reduce the transmission rate of the disease. Inspection of dairy products helps to reduce the production of infectious dairy products; hence the rate of transmission from the dairy product can be reduced since the consumption of infectious dairy products decreases. On recovery, humans become susceptible to the disease again.

Figure 9 demonstrates the interaction of state variables, Tables 5 and 6 describe state variables and parameters, respectively.

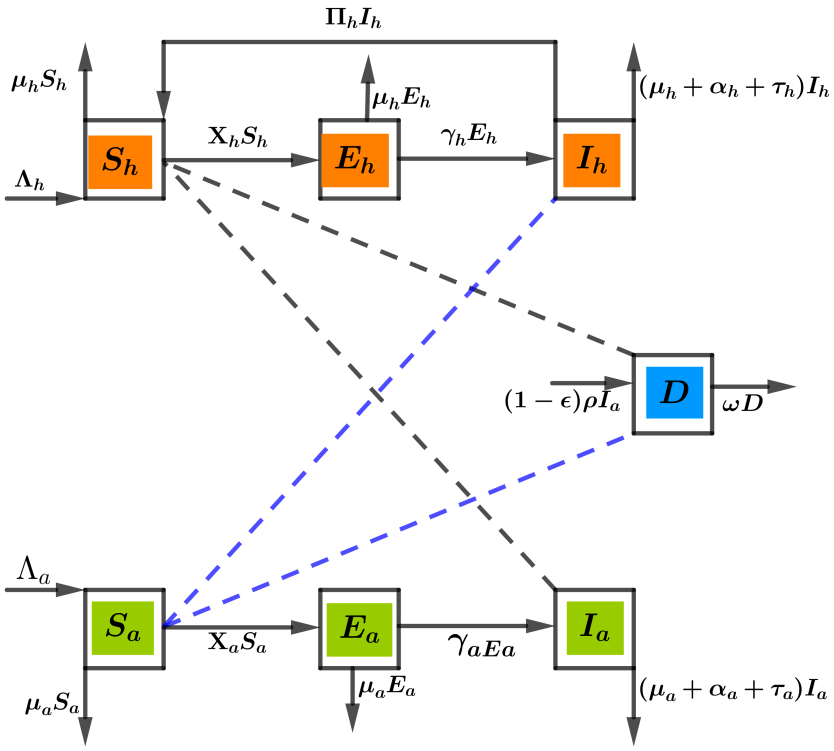


Figure 9: Model Flow Diagram Extended

Table 5: Model Variables Description

Symbol	Description
$S_h(t)$	Number of susceptible human at time t.
$S_a(t)$	Number of susceptible animal at time t.
$E_h(t)$	Number of Exposed human beings at time t.
$E_a(t)$	Number of Exposed animals at time t.
$I_h(t)$	Number of infected human at time t.
$I_a(t)$	Number of infected animals at time t
$D(t)$	Amount of producing dairy products at time t

4.2.1 Model Equations

The bTB model with controls is governed by the following system of differential equations:

$$\frac{dS_h}{dt} = \Lambda_h + \Pi_h I_h - \left(\frac{\beta_1(1-\tau_h)I_h + \beta_2(1-\tau_a)I_a + \beta_3(1-\varepsilon)D}{N_h}\right) S_h - \mu_h S_h. \tag{4.1a}$$

$$\frac{dE_h}{dt} = \left(\frac{\beta_1(1-\tau_h)I_h + \beta_2(1-\tau_a)I_a + \beta_3(1-\varepsilon)D}{N_h}\right) S_h - (\gamma_h + \mu_h)E_h. \tag{4.1b}$$

$$\frac{dI_h}{dt} = \gamma_h E_h - (\mu_h + \alpha_h + \tau_h)I_h. \tag{4.1c}$$

$$\frac{dS_a}{dt} = \Lambda_a - \left(\frac{\beta_4(1-\tau_h)I_h + \beta_5(1-\tau_a)I_a + \beta_6(1-\varepsilon)D}{N_a}\right) S_a - \mu_a S_a. \tag{4.1d}$$

$$\frac{dE_a}{dt} = \left(\frac{\beta_4(1-\tau_h)I_h + \beta_5(1-\tau_a)I_a + \beta_6(1-\varepsilon)D}{N_a}\right) S_a - (\gamma_a + \mu_a)E_a. \tag{4.1e}$$

$$\frac{dI_a}{dt} = \gamma_a E_a - (\mu_a + \alpha_a + \tau_a)I_a. \tag{4.1f}$$

$$\frac{dD}{dt} = \rho(1-\varepsilon)I_a - (\omega + \theta)D. \tag{4.1g}$$

Subject to their initial conditions:
$S_h(0) > 0; E_h(0) \geq 0; I_h(0) \geq 0; S_a(0) > 0; E_a(0) \geq 0; I_a(0) \geq 0; D(0) \geq 0.$

Table 6: Parameters' Descriptions

Parameter	Descriptions
Λ_h	Human recruitment rate.
μ_h	Human natural death.
γ_h	Progression rate from E_h to I_h.
α_h	Human death rate due to disease induced.
ε	Rate of inspecting dairy products.
$\beta_1, \beta_2, \beta_3$	Humans infection rate from I_h, I_a, and D respectively.
τ_a	Rate of quarantine infected animals.
μ_a	Animal natural death rate.
γ_a	Progression rate from E_a to I_a.
α_a	Mortality of animals due to disease
ρ	Rate of dairy products produced from I_a.
ω	Rate of decaying unconsumed dairy products.
$\beta_4, \beta_5, \beta_6$	Animals infection rate from I_h, I_a, and D respectively.
τ_h	Rate of treating infected humans.
Π_h	Human recovery rate.

4.3 Model Analysis

To show that the model is mathematically meaningful, we find the invariant region and test positivity of the solution. The model is biologically and mathematically meaningful if its solutions are positive and bounded.

4.3.1 Invariant Region

The invariant region shows the feasibility of the model solutions. To find the invariant region, we denote humans and livestock populations by N_h and N_a respectively. Beginning with the human population, we have:

$$N_h = S_h + E_h + I_h, \tag{4.2}$$

From (4.2), we have:

$$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h, \tag{4.3}$$

whose solution when $t = 0$ is:

$$N_h(0) \leq \frac{\Lambda_h}{\mu_h} + \left(N_h(0) - \frac{\Lambda_h}{\mu_h}\right) e^{-\mu_h t}. \tag{4.4}$$

Analysis of N_h consider two cases:

$$\text{when } N_h(0) > \frac{\Lambda_h}{\mu_h} \text{ and when } N_h(0) < \frac{\Lambda_h}{\mu_h}.$$

For

$$N_h(0) > 0: \quad N_h(t) \leq \frac{\Lambda_h}{\mu_h} \leq \frac{\Lambda_h}{\mu_h} + \left(N_h(0) - \frac{\Lambda_h}{\mu_h}\right) e^{-\mu_h t}, \tag{4.5}$$

and for

$$\begin{aligned} N_h(0) < 0: \quad N_h(t) &\leq \frac{\Lambda_h}{\mu_h} + \left(N_h(0) - \frac{\Lambda_h}{\mu_h}\right) e^{-\mu_h t} \leq \frac{\Lambda_h}{\mu_h}, \\ \text{Since } \lim_{t\to\infty} &\left(N_h(0) - \frac{\Lambda_h}{\mu_h}\right) e^{-\mu_h t} \to 0, \end{aligned} \tag{4.6}$$

then,

$$0 \leq N_h \leq \frac{\Lambda_h}{\mu_h}. \tag{4.7}$$

Animals population is given by:

$$N_a = S_a + E_a + I_a,$$

Thus

$$\frac{dN_a}{dt} \leq \Lambda_a - \mu_a N_a. \tag{4.8}$$

Using initial condition, the solution is:

$$N_a(t) \leq \frac{\Lambda_a}{\mu_a} + \left(N_a(0) - \frac{\Lambda_a}{\mu_a}\right) e^{-\mu_a t}. \tag{4.9}$$

The analysis of N_a consider two cases:

$$\text{When } \; N_a(0) > \frac{\Lambda_a}{\mu_a} \; \text{ and when } \; N_a(0) < \frac{\Lambda_a}{\mu_a},$$

For,

$$N_a(0) > \frac{\Lambda_a}{\mu_a} : \;\; N_a(t) \leq \frac{\Lambda_a}{\mu_a} + \left(N_a(0) - \frac{\Lambda_a}{\mu_a}\right) e^{-\mu_a t}. \tag{4.10}$$

and for

$$\begin{aligned} N_a(0) \leq \frac{\Lambda_a}{\mu_a} : \;\; N_a(t) \leq \frac{\Lambda_a}{\mu_a} + \left(N_a(0) - \frac{\Lambda_a}{\mu_a}\right) e^{-\mu_a t} \leq \frac{\Lambda_a}{\mu_a}, \\ \text{As } \lim_{t\to\infty} \left(N_a(0) - \frac{\Lambda_a}{\mu_a}\right) e^{-\mu_a t} \to 0. \end{aligned} \tag{4.11}$$

All the two cases give:

$$0 \leq N_a \leq \frac{\Lambda_a}{\mu_a}. \tag{4.12}$$

For dairy products we have:

$$\frac{dD}{dt} \leq \rho(1-\varepsilon) I_a - (\omega + \theta) D,$$

Since

$$I_a \leq \frac{\Lambda_a}{\mu_a} \; \text{ then,}$$

$$\frac{dD}{dt} \leq \rho(1-\varepsilon)\frac{\Lambda_a}{\mu_a} - (\omega + \theta) D, \tag{4.13}$$

From (4.13) we have:

$$\frac{dD}{dt} + (\omega + \theta) D \leq \rho(1-\varepsilon)\frac{\Lambda_a}{\mu_a}, \tag{4.14}$$

By using initial conditions we get:

$$D(t) \leq \frac{\Lambda_a}{\mu_a}\left(\frac{1-\varepsilon}{\omega+\theta}\right)\rho + \left(D(0) - \frac{\Lambda_a}{\mu_a}\left(\frac{1-\varepsilon}{\omega+\theta}\right)\rho\right) e^{-(\omega+\theta)t},$$

But as $t \to \infty$, we obtain:

$$D(t) \leq \frac{\Lambda_a}{\mu_a}\left(\frac{1-\varepsilon}{\omega+\theta}\right)\rho. \tag{4.15}$$

Therefore the model (4.1) is positive invariant in the region:

$$Z = \left\{ (S_h, E_h, I_h, S_a, E_a, Ia, D) \in R_+^7 : 0 \leq N_h \leq \frac{\Lambda_h}{\mu_h}; 0 \leq N_a \leq \frac{\Lambda_a}{\mu_a}; 0 \leq D \leq \frac{\Lambda_a}{\mu_a}\left(\frac{1-\varepsilon}{\omega+\theta}\right)\rho \right\}. \tag{4.16}$$

The model (4.1) is mathematically and epidemiologically meaningful, therefore we can consider the flow generated by the model for analysis.

4.3.2 Positivity of Solutions

Theorem 5: *Let the initial values for the state variables for the model* (4.1) *be* $S_h(0) > 0, E_h(0) \geq 0, I_h(0) \geq 0, S_a(0) > 0, E_a(0) \geq 0, I_a(0) \geq 0$ *and* $D \geq 0$ *then the solutions of the model* (4.1) *are positive* $\forall t \geq 0$.

Proof: Let's consider the equations (3.64a) of the model system (4.1) which is:

$$\frac{dS_h}{dt} = \Lambda_h + \Pi_h I_h - \left(\frac{\beta_1(1-\tau_h)I_h + \beta_2(1-\tau_a)I_a + \beta_3(1-\varepsilon)D}{N_h} \right) S_h - \mu_h S_h, \tag{4.17}$$

From (4.17) we get the inequality:

$$\frac{dS_h}{dt} \geq - \left(\frac{\beta_1(1-\tau_h)I_h + \beta_2(1-\tau_a)I_a + \beta_3(1-\varepsilon)D}{N_h} \right) S_h - \mu_h S_h. \tag{4.18}$$

Solving differential equation (4.18) and apply initial condition, we get:

$$S_h(t) \geq S_h(0) e^{\int_0^t - \left(\frac{\beta_1(1-\tau_h)I_h(s) + \beta_2(1-\tau_a)I_a(s) + \beta_3(1-\varepsilon)D}{N_h(s)} + \mu_h \right) ds}. \tag{4.19}$$

Then $S_h(t) \geq 0, \forall\, t \geq 0$.

From equation (3.64b) of the model (4.1) we have:

$$\frac{dE_h}{dt} = \left(\frac{\beta_1(1-\tau_h)I_h + \beta_2(1-\tau_a)I_a + \beta_3(1-\varepsilon)D}{N_h} \right) S_h - (\gamma_h + \mu_h)E_h, \tag{4.20}$$

Equation (4.20) gives the inequality

$$\frac{dE_h}{dt} \geq -(\gamma_h + \mu_h)E_h. \tag{4.21}$$

Separating variables, integration and application of initial condition, equation (4.21) gives:

$$E_h(t) \geq E_h(0) e^{-(\gamma_h + \mu_h)t}. \tag{4.22}$$

Then $E_h \geq 0\ \forall\, t \geq 0$.

From equation (3.64c) of the model (4.1) we have:

$$\frac{dI_h}{dt} = \gamma_h E_h - (\mu_h + \alpha_h + \tau_h)I_h, \tag{4.23}$$

whose inequality is:

$$\frac{dI_h}{dt} \geq -(\mu_h + \alpha_h + \tau_h)I_h. \tag{4.24}$$

By separating variable and solving equation, (4.24) we get:

$$I_h(t) \geq I_h(0) e^{(\mu_h + \alpha_h + \tau_h)t}. \tag{4.25}$$

Then, $I_h \geq 0 \; \forall \; t \geq 0$

Consider model equation (3.64d) from the model system (4.1):

$$\frac{dS_a}{dt} = \Lambda_a - \left(\frac{\beta_4(1-\tau_h)I_h + \beta_5(1-\tau_a)I_a + \beta_6(1-\varepsilon)D}{N_a} + \mu_a \right) S_a, \tag{4.26}$$

from the equation (4.26) we get the inequality:

$$\frac{dS_a}{dt} \geq - \left(\frac{\beta_4(1-\tau_h)I_h + \beta_5(1-\tau_a)I_a + \beta_6(1-\varepsilon)D}{N_a} + \mu_a \right) S_a. \tag{4.27}$$

Separate variable and integrate equation (4.27) and apply initial conditions to get:

$$S_a(t) \geq S_a(0) e^{\int_0^t - \left(\frac{\beta_4(1-\tau_h)I_h(s) + \beta_5(1-\tau_a)I_a(s) + \beta_6(1-\varepsilon)D(s)}{N_a(s)} + \mu_a \right) ds}. \tag{4.28}$$

So, $S_a \geq 0 \; \forall \; t \geq 0$.

Consider equation (3.64e) of the model system (4.1) which is:

$$\frac{dE_a}{dt} = \left(\frac{\beta_4(1-\tau_h)I_h + \beta_5(1-\tau_a)I_a + \beta_6(1-\varepsilon)D}{N_a} \right) S_a - (\gamma_a + \mu_a)E_a, \tag{4.29}$$

which gives the inequality:

$$\frac{dE_a}{dt} \geq -(\gamma_a + \mu_a)E_a. \tag{4.30}$$

Separating variables, integrating and applying initial condition (4.30) gives:

$$E_a(t) \geq E_a(0) e^{-(\gamma_a + \mu_a)t}. \tag{4.31}$$

Hence $E_a \geq 0 \; \forall \; t \geq 0$.

Again from the model equation (3.64f) of the model (4.1) we have the inequality:

$$\frac{dI_a}{dt} \geq -(\mu_a + \alpha_a + \tau_a)I_a. \tag{4.32}$$

By solving the differential equation (4.32) we get:

$$I_a(t) \geq I_a(0) e^{-(\mu_a + \alpha_a + \tau_a)t}. \tag{4.33}$$

Then, $I_a \geq 0 \; \forall \; t \geq 0$.

Lastly from the equation (3.64g) of the model (4.1) we have the inequality:

$$\frac{dD}{dt} \geq -(\omega + \theta)D. \tag{4.34}$$

By solving the equation (4.34) we get:

$$\begin{aligned} D &\geq Ce^{-(\omega+\theta)t}. \\ D(t) &\geq D(0)e^{-(\omega+\theta)t}. \end{aligned} \tag{4.35}$$

Then, $D \geq 0 \; \forall \; t \geq 0$.

Therefore solutions of the model system (4.1) are positive and bounded since $S_h(t) > 0, E_h(t) > 0, I_h(t) > 0, S_a(t) > 0, E_a(t) > 0, I_a(t) > 0, D(t) > 0 \; \forall t > 0$.

4.3.3 Effective Reproduction Number R_e

To determine effective reproduction number if control parameters are administered, we use next generation approach by (Van, 2002). The control strategies are effective when the effective reproduction number $R_e < 1$ and they are ineffective if reproduction number $R_e > 1$. If new infections and transfer terms are denoted by H_i and P_i respectively, then the effective reproduction number R_e is given as the maximum eigenvalue. That is:

$$R_e = \rho(HP^{-1}), \tag{4.36}$$

where

$$H = \frac{\partial H_i}{\partial X_j}(DF^0) \;\; and \;\; P = \frac{\partial P_i}{\partial X_j}(DF^0).$$

From the model system (4.1) H_i and P_i are:

$$H_i = \begin{bmatrix} \dfrac{((1-\tau_h)\beta_1 I_h + (1-\tau_a)\beta_2 I_a + \beta_3(1-\varepsilon)(D))S_h}{N_h} \\ 0 \\ \dfrac{(1-\tau_h)\beta_4 I_h + (1-\tau_a)\beta_5 I_a + (1-\varepsilon)\beta_6(D)}{N_a} \\ 0 \\ 0 \end{bmatrix}, \tag{4.37}$$

and

$$P_i = \begin{bmatrix} (\mu_h+\gamma_h)E_h \\ \gamma_h E_h - (\mu_h+\alpha_h+\tau_h)I_h \\ (\gamma_a+\mu_a)I_a \\ \gamma_a E_a - (\mu_a+\alpha_a+\tau_a)I_a \\ \rho\,(1-\varepsilon)I_a - (\omega+\theta)(D) \end{bmatrix}. \tag{4.38}$$

Jacobian of matrices H_i and P_i are therefore given by:

$$H = \begin{bmatrix} 0 & (1-\tau_h)\beta_1 & 0 & (1-\tau_a)\beta_2 & (1-\varepsilon)\beta_3 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & (1-\tau_h)\beta_4 & 0 & (1-\tau_a)\beta_5 & (1-\varepsilon)\beta_6 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}. \tag{4.39}$$

and

$$P=\begin{bmatrix} \mu_h+\gamma_h & 0 & 0 & 0 & 0 \\ -\gamma_h & \mu_h+\alpha_{h+\tau_h} & 0 & 0 & 0 \\ 0 & 0 & \gamma_a+\mu_a & 0 & 0 \\ 0 & 0 & -\gamma_a & \mu_a+\alpha_a+\tau_a & 0 \\ 0 & 0 & 0 & \rho\,(1-\varepsilon) & \omega+\theta \end{bmatrix}. \tag{4.40}$$

The inverse of the matrix V is:

$$P^{-}1=\begin{bmatrix} \frac{1}{(\mu_h+\gamma_h)} & 0 & 0 & 0 & 0 \\ K_1 & \frac{1}{(\mu_h+\alpha_{h+\tau_h})} & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{(\gamma_a+\mu_a)} & 0 & 0 \\ 0 & 0 & K_2 & \frac{1}{(\mu_a+\alpha_a+\tau_a)} & 0 \\ 0 & 0 & K_3 & K_4 & \frac{1}{(\omega+\theta)} \end{bmatrix}, \tag{4.41}$$

where,

$$K_1=\frac{\gamma_h}{(\mu_h+\gamma_h)(\mu_h+\alpha_{h+\tau_h})},\ K_2=\frac{\gamma_a}{(\gamma_a+\mu_a)(\mu_a+\alpha_a+\tau_a)},$$
$$K_3=\frac{\rho\,(1-\varepsilon)\gamma_a}{(\mu_a+\alpha_a+\tau_a)(\gamma_a+\mu_a)(\omega+\theta)},\ K_4=\frac{\rho\,(1-\varepsilon)}{(\mu_a+\alpha_a+\tau_a)(\omega+\theta)}.$$

The product HP^{-1} is given by:

$$HP^{-1}=\begin{bmatrix} n & \frac{(1-\tau_h)\beta_1}{\mu_h+\alpha_h+\tau_a} & t & \frac{(1-\tau_h)\beta_2\gamma_a}{\mu_a+\alpha_a+\tau_a}-\frac{(1-\varepsilon)^2\beta_3\rho\,\gamma_a}{(\mu_a+\alpha_a+\tau_a)(\omega+\theta)} & \frac{(1-\varepsilon)\beta_3}{\omega+\theta} \\ 0 & 0 & 0 & 0 & 0 \\ r & \frac{(1-\tau_h)\beta_4}{\mu_h+\alpha_h+\tau_h} & m & \frac{(1-\tau_a)\beta_5}{\mu_a+\alpha_a+\tau_a}-\frac{\rho\,(1-\varepsilon)^2\beta_6}{(\mu_a+\alpha_a+\tau_a)(\omega+\theta)} & \frac{(1-\varepsilon)\beta_6}{\omega+\theta} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}. \tag{4.42}$$

The effective reproductive number R_e is given by:

$$R_e=\frac{1}{2}\left(\frac{(1-\tau_a)(\omega+\theta)\beta_5\gamma_a+(1-\varepsilon)^2\beta_6\rho\,\gamma_a}{(\gamma_a+\mu_a)(\mu_a+\alpha_a+\tau_a)(\omega+\theta)}+\frac{\beta_1(1-\tau_h)\gamma_h}{(\mu_h+\gamma_h)(\mu_h+\alpha_h+\tau_h)}\right)$$
$$+\frac{1}{2}\left(\sqrt{\left(\frac{(1-\tau_a)(\omega+\theta)\beta_5\gamma_a+(1-\varepsilon)^2\beta_6\rho\,\gamma_a}{(\gamma_a+\mu_a)(\mu_a+\alpha_a+\tau_a)(\omega+\theta)}-\frac{\beta_1(1-\tau_h)\gamma_h}{(\mu_h+\gamma_h)(\mu_h+\alpha_h+\tau_h)}\right)^2+4\,rt}\right). \tag{4.43}$$

where,

$$n = \frac{(1-\tau)\,\beta_1\gamma_h}{(\mu_h+\gamma_h)\,(\mu_h+\alpha_h+\tau)},$$
$$r = \frac{(1-\tau_h)\,\beta_4\gamma_h}{(\mu_h+\gamma_h)\,(\mu_h+\alpha_h+\tau_h)},$$
$$t = \frac{(1-\tau_a)\,(\omega+\theta)\,\beta_2\gamma_a+\beta_3\,(1-\varepsilon)^2\,\rho\,\gamma_a}{(\gamma_a+\mu_a)\,(\mu_a+\alpha_a+\tau_a)\,(\omega+\theta)},$$
$$m = \frac{(1-\tau_a)\,(\omega+\theta)\,\beta_5\gamma_a+(1-\varepsilon)^2\,\beta_6\rho\,\gamma_a}{(\gamma_a+\mu_a)\,(\mu_a+\alpha_a+\tau_a)\,(\omega+\theta)},$$
$$rt = \frac{(1-\tau_a)\,(\omega+\theta)\,(1-\tau_h)\,\beta_2\beta_4\gamma_h\gamma_a+(1-\tau_h)\,(1-\varepsilon)^2\,\beta_3\beta_4\gamma_h\rho\,\gamma_a}{(\mu_h+\gamma_h)\,(\mu_h+\alpha_h+\tau_h)\,(\gamma_a+\mu_a)\,(\mu_a+\alpha_a+\tau_a)\,(\omega+\theta)}.$$

The effective reproduction number R_e decrease as we increase human treatment, quarantine of infected animals and inspection of dairy products. Bovine tuberculosis contained if infected humans are diagnosed and treated, infected animals quarantined and dairy products inspected.

When $\tau_h=\tau_a=\varepsilon=0$, effective reproduction R_e becomes basic reproduction number R_0, which is:

$$\begin{aligned} R_0 =& \frac{1}{2}\left(\frac{\gamma_a\,(\omega\,\beta_5+\rho\,\beta_6)}{(\gamma_a+\mu_a)\,(\mu_a+\alpha_a)\,\omega}+\frac{\beta_1\gamma_h}{(\mu_h+\gamma_h)\,(\mu_h+\alpha_h)}\right) \\ &+\frac{1}{2}\left(\sqrt{\left(\frac{\gamma_a\,(\omega\,\beta_5+\rho\,\beta_6)}{(\gamma_a+\mu_a)\,(\mu_a+\alpha_a)\,\omega}-\frac{\beta_1\gamma_h}{(\mu_h+\gamma_h)\,(\mu_h+\alpha_h)}\right)^2+4ce}\right). \end{aligned} \tag{4.44}$$

where $ce = \dfrac{\gamma_h\gamma_a(\omega\beta_2+\rho\beta_3)}{\omega(\gamma_h+\mu_h)(\alpha_h+\mu_h)(\gamma_a+\mu_a)}$.

Basic and effective reproduction numbers (R_0 and R_e) are plotted on the same graph in Figure 10 to assess the effect of control strategies.

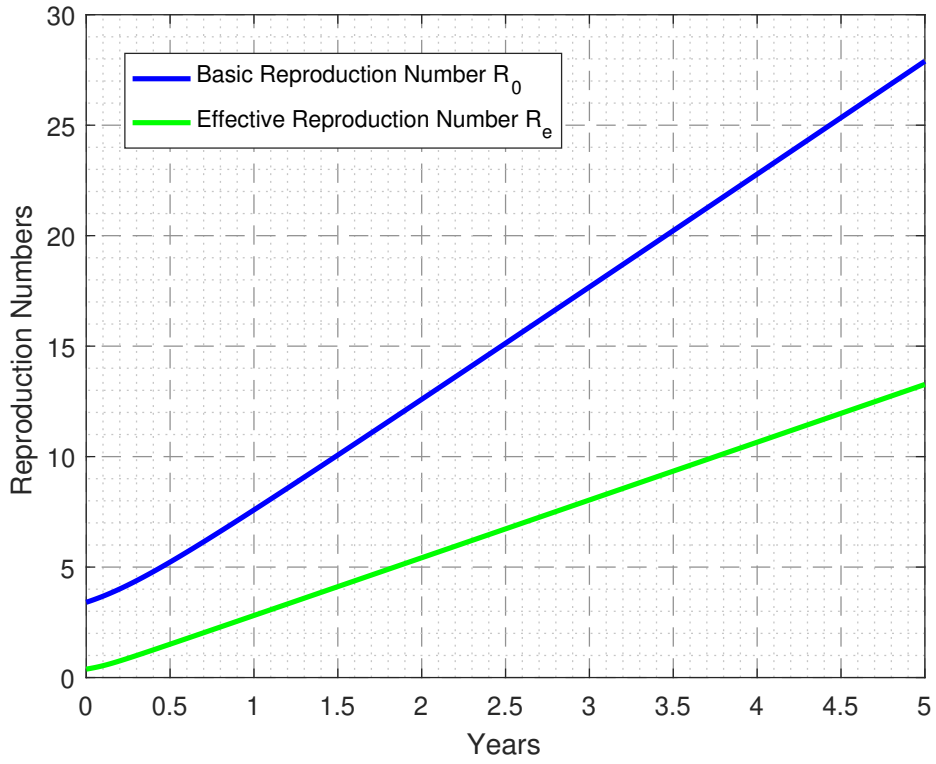


Figure 10: Reproduction number without and with controls

The graph in blue represents new infections before control strategies are applied and green graph when control strategies are applied. Results show that new infections decrease proportionally as control strategies administered.

4.3.4 Stability Analysis

Local stability of disease-free equilibrium investigated by Linearization method. Disease-free equilibrium is locally asymptotically stable if the matrix of a linearized system has negative eigenvalues. The Jacobian of the system (4.1) at DFE is given by:

$$J_c = \begin{bmatrix} -\mu_h & 0 & -(1-\tau_h)\beta_1 & 0 & 0 & -(1-\tau_a)\beta_2 & -(1-\varepsilon)\beta_3 \\ 0 & -\mu_h-\gamma_h & (1-\tau_h)\beta_1 & 0 & 0 & (1-\tau_a)\beta_2 & (1-\varepsilon)\beta_3 \\ 0 & \gamma_h & -\mu_h-\alpha_h-\tau_h & 0 & 0 & 0 & 0 \\ 0 & 0 & -(1-\tau_h)\beta_4 & -\mu_a & 0 & -(1-\tau_a)\beta_5 & -(1-\varepsilon)\beta_6 \\ 0 & 0 & (1-\tau_h)\beta_4 & 0 & -\mu_a-\gamma_a & (1-\tau_a)\beta_5 & (1-\varepsilon)\beta_6 \\ 0 & 0 & 0 & 0 & \gamma_a & -\mu_a-\alpha_a-\tau_a & 0 \\ 0 & 0 & 0 & 0 & 0 & \rho(1-\varepsilon) & -(\omega+\theta) \end{bmatrix}. \tag{4.45}$$

From the first and the fourth columns, eigenvalues are $-\mu_h$ and $-\mu_a$. Matrix (4.45) is then reduced into

$$K_c = \begin{bmatrix} -\mu_h-\gamma_h & (1-\tau_h)\beta_1 & 0 & (1-\tau_a)\beta_2 & (1-\varepsilon)\beta_3 \\ \gamma_h & -\alpha_h-\mu_h-\tau_h & 0 & 0 & 0 \\ 0 & (1-\tau_h)\beta_4 & -\mu_a-\gamma_a & (1-\tau_a)\beta_5 & (1-\varepsilon)\beta_6 \\ 0 & 0 & \gamma_a & -\alpha_a-\mu_a-\tau_a & 0 \\ 0 & 0 & 0 & \rho(1-\varepsilon) & -\omega-\theta. \end{bmatrix}. \tag{4.46}$$

Trace $tr(K_c)$ of the matrix (4.46) is:

$$tr(K_c) = -\left((\gamma_h+\mu_h)+(\alpha_h+\mu_h+\tau_h)+(\gamma_a+\mu_a)+(\alpha_a+\mu_a+\tau_a)+(\omega+\theta)\right) < 0. \tag{4.47}$$

Determinant of the matrix (4.46) is given by:

$$\begin{aligned} \det(K_c) =& \frac{\gamma_h(1-\tau_h)(\gamma_a+\mu_a)(\alpha_a+\mu_a+\tau_a)\beta_1+\gamma_a\beta_5(1-\tau_a)(\gamma_h+\mu_h)(\alpha_h+\mu_h+\tau_h)}{\gamma_a\beta_5(1-\tau_a)(\gamma_h+\mu_h)(\alpha_h+\mu_h+\tau_h)} \\ &+\frac{(1-\tau_h)\gamma_h\gamma_a\beta_4\left(\beta_2(1-\tau_a)(\omega+\theta)+(1-\varepsilon)(1-\varepsilon)\beta_3\rho\right)}{(\gamma_h+\mu_h)(\alpha_h+\mu_h+\tau_h)(\gamma_a+\mu_a)(\alpha_a+\mu_a+\tau_a)} \\ &-\left(\frac{\gamma_h\gamma_a\beta_1(\beta_5(1-\tau_a)+\beta_6(1-\varepsilon)\rho(1-\varepsilon))}{(\gamma_h+\mu_h)(\alpha_h+\mu_h+\tau_h)(\gamma_a+\mu_a)(\alpha_a+\mu_a+\tau_a)(\omega+\theta)}\right). \end{aligned} \tag{4.48}$$

Determinant $\det(K_c) > 0$ if

$$\begin{aligned} &\frac{\gamma_h(1-\tau_h)(\gamma_a+\mu_a)(\alpha_a+\mu_a+\tau_a)\beta_1+\gamma_a\beta_5(1-\tau_a)(\gamma_h+\mu_h)(\alpha_h+\mu_h+\tau_h)}{\gamma_a\beta_5(1-\tau_a)(\gamma_h+\mu_h)(\alpha_h+\mu_h+\tau_h)} \\ &+\frac{(1-\tau_h)\gamma_h\gamma_a\beta_4\left(\beta_2(1-\tau_a)(\omega+\theta)+(1-\varepsilon)(1-\varepsilon)\beta_3\rho\right)}{(\gamma_h+\mu_h)(\alpha_h+\mu_h+\tau_h)(\gamma_a+\mu_a)(\alpha_a+\mu_a+\tau_a)} \\ &-\left(\frac{\gamma_h\gamma_a\beta_1(\beta_5(1-\tau_a)+\beta_6(1-\varepsilon)\rho(1-\varepsilon))}{(\gamma_h+\mu_h)(\alpha_h+\mu_h+\tau_h)(\gamma_a+\mu_a)(\alpha_a+\mu_a+\tau_a)(\omega+\theta)}\right) > 1. \end{aligned} \tag{4.49}$$

The disease free equilibrium is asymptotically stable if condition (4.49) holds.

4.3.5 Endemic Equilibrium for the Model with Controls

Endemic equilibrium of the model with control are computed in terms of the force of infection due to the complexity of the model equations. To compute endemic equilibrium, we set the right part of the model system (4.1) equal to zero.

$$\Lambda_h + \Pi_h - X_h S_h - \mu_h S_h = 0. \tag{4.50a}$$

$$X_h S_h - (\gamma_h + \mu_h) E_h - 0. \tag{4.50b}$$

$$\gamma_h E_h - (\mu_h + \alpha_h + \tau_h) I_h = 0. \tag{4.50c}$$

$$\Lambda_a + \Pi_a - X_a S_a - \mu_a S_a - 0. \tag{4.50d}$$

$$X_a S_a - (\gamma_a + \mu_a) E_a = 0. \tag{4.50e}$$

$$\gamma_a E_a - (\mu_a + \alpha_a + \tau_a) I_a = 0. \tag{4.50f}$$

$$\rho(1-\varepsilon) I_a - (\omega + \theta) D = 0. \tag{4.50g}$$

Using the forces of infection for humans X_h and animals X_a and solve simultaneous the model system equations we get

$$\begin{aligned}
S_h^* &= \frac{\Lambda_h (\mu_h + \gamma_h)(\tau_h + \alpha_h + \mu_h)}{(\mu_h + \gamma_h)(\tau_h + \alpha_h + \mu_h)\left(X_h^* + \mu_h\right) - \Pi_h X_h^* \gamma_h}, \\
E_h^* &= \frac{\Lambda_h X_h^* (\tau_h + \alpha_h + \mu_h)}{(\mu_h + \gamma_h)(\tau_h + \alpha_h + \mu_h)\left(X_h^* + \mu_h\right) - \Pi_h X_h^* \gamma_h}, \\
I_h^* &= \frac{\Lambda_h \gamma_h X_h^*}{(\mu_h + \gamma_h)(\tau_h + \alpha_h + \mu_h)\left(X_h^* + \mu_h\right) - \Pi_h X_h^* \gamma_h}, \\
S_a^* &= \frac{\Lambda_a (\mu_a + \gamma_a)(\tau_a + \alpha_a + \mu_a)}{(\mu_a + \gamma_a)(\tau_a + \alpha_a + \mu_a)(X_a^* + \mu_a) - \Pi_a X_a^* \gamma_a}, \\
E_a^* &= \frac{(\tau_a + \alpha_a + \mu_a) \Lambda_a X_a}{(\mu_a + \gamma_a)(\tau_a + \alpha_a + \mu_a)(X_a^* + \mu_a) - \Pi_a X_a^* \gamma_a}, \\
I_a^* &= \frac{\Lambda_a \gamma_a X_a^*}{(\mu_a + \gamma_a)(\tau_a + \alpha_a + \mu_a)(X_a^* + \mu_a) - \Pi_a X_a^* \gamma_a}, \\
D^* &= \frac{(1-\varepsilon) \Lambda_a \gamma_a X_a^*}{((\mu_a + \gamma_a)(\tau_a + \alpha_a + \mu_a)(X_a^* + \mu_a) - \pi_a X_a^* \gamma_a)(\omega + \theta)}.
\end{aligned} \tag{4.51}$$

However, the model system (4.1) has the possibility to undergo backward bifurcation when $R_e = 1$

4.3.6 Bifurcation Analysis for the Model with Controls

In this section, bifurcation analysis was performed to determine whether the model (4.1) undergoes backward bifurcation when $R_e = 1$. If we rename the state variables $S_H, E_h, I_h, S_a, E_a, I_a, D$ to be $y_1, y_2, y_3, y_4, y_5, y_6, y_7$ and introduce the vector notations $Y = (y_1, y_2, y_3, y_4, y_5, y_6, y_7)^T$, then the model system (4.1) can be written as $\frac{dY}{dt} = G(Y)$, where $G(Y) = (g_1, g_2, g_3, g_4, g_5, g_6, g_7)^T$ as follows:

$$
\begin{aligned}
\frac{dy_1}{dt} &= g_1 = \Lambda_h + \Pi_h y_6 - \left(\frac{\beta_1(1-\tau_h)y_3 + \beta_2(1-\tau_a)y_6 + \beta_3(1-\varepsilon)y_7}{y_1+y_2+y_3}\right) y_1 - \mu_h y_1.\\
\frac{dy_2}{dt} &= g_2 = \left(\frac{\beta_1(1-\tau_h)y_3 + \beta_2(1-\tau_a)y_6 + \beta_3(1-\varepsilon)y_7}{y_1+y_2+y_3}\right) y_1 - (\gamma_h+\mu_h)y_2.\\
\frac{dy_3}{dt} &= g_3 = \gamma_h y_2 - (\mu_h+\alpha_h+\tau_h)y_3.\\
\frac{dy_4}{dt} &= g_4 = \Lambda_a - \left(\frac{\beta_4(1-\tau_h)y_3 + \beta_5(1-\tau_a)y_6 + \beta_6(1-\varepsilon)y_7}{y_4+y_5+y_6}\right) y_4 - \mu_a y_4.\\
\frac{dy_5}{dt} &= g_5 = \left(\frac{\beta_4(1-\tau_h)y_3 + \beta_5(1-\tau_a)y_6 + \beta_6(1-\varepsilon)y_7}{y_4+y_5+y_6}\right) y_4 - (\gamma_a+\mu_a)y_5.\\
\frac{dy_6}{dt} &= g_6 = \gamma_a y_5 - (\mu_a+\alpha_a+\tau_a)y_6.\\
\frac{dy_7}{dt} &= g_7 = \rho(1-\varepsilon)y_6 - (\omega+\theta)y_7.
\end{aligned}
\tag{4.52}
$$

The Jacobian of the system (4.52) at DFE is:

$$
J = \begin{bmatrix}
-\mu_h & 0 & -\beta_1(1-\tau_h) & 0 & 0 & -\beta_2(1-\tau_a) & -\beta_3(1-\varepsilon)\\
0 & -\mu_h-\gamma_h & \beta_1(1-\tau_h) & 0 & 0 & \beta_2(1-\tau_a) & \beta_3(1-\varepsilon)\\
0 & \gamma_h & -\mu_h-\alpha_h-\tau_h & 0 & 0 & 0 & 0\\
0 & 0 & -\beta_4(1-\tau_h) & -\mu_a & 0 & -\beta_5(1-\tau_a) & -\beta_6(1-\varepsilon)\\
0 & 0 & \beta_4(1-\tau_h) & 0 & -\mu_a-\gamma_a & \beta_5(1-\tau_a) & \beta_6(1-\varepsilon)\\
0 & 0 & 0 & 0 & \gamma_a & -\mu_a-\alpha_a-\tau_a & 0\\
0 & 0 & 0 & 0 & 0 & \rho(1-\varepsilon) & -(\omega+\theta)
\end{bmatrix}.
\tag{4.53}
$$

Suppose we choose the bifurcation parameter to be $\beta_3 = \beta^{**}$ when $R_e = 1$. Now, solving for $\beta_3 = \beta^{**}$ when $R_e = 1$ we get:

$$
(2-p-q)^2 = (p-q)^2 + 4\left(\frac{(1-\tau_a)(\omega+\theta)(1-\tau_h)\beta_2\beta_4\gamma_h\gamma_a + (1-\tau_h)(1-\varepsilon)^2\beta_3\beta_4\gamma_h\rho\,\gamma_a}{(\mu_h+\gamma_h)(\mu_h+\alpha_h+\tau_h)(\gamma_a+\mu_a)(\mu_a+\alpha_a+\tau_a)(\omega+\theta)}\right)
$$

Through simplifications we get:

$$
\beta_3 = ((1-s-(p+q)-pq)\left(\frac{(\mu_h+\gamma_h)(\mu_h+\alpha_h+\tau_h)(\gamma_a+\mu_a)(\mu_a+\alpha_a+\tau_a)(\omega+\theta)}{(1-\tau_a)(\omega+\theta)(1-\tau_h)\beta_2\beta_4\gamma_h\gamma_a}\right)
\tag{4.54}
$$

where

$$q = \frac{\beta_1 (1-\tau_h)\gamma_h}{(\mu_h+\gamma_h)(\mu_h+\alpha_h+\tau_h)}$$
$$p = \frac{(1-\tau_a)(\omega+\theta)\beta_5\gamma_a + (1-\varepsilon)^2\beta_6\rho\,\gamma_a}{(\gamma_a+\mu_a)(\mu_a+\alpha_a+\tau_a)(\omega+\theta)}$$
$$s = \frac{(1-\tau_a)(\omega+\theta)(1-\tau_h)\beta_2\beta_4\gamma_h\gamma_a}{(\mu_h+\gamma_h)(\mu_h+\alpha_h+\tau_h)(\gamma_a+\mu_a)(\mu_a+\alpha_a+\tau_a)(\omega+\theta)}$$

From (4.53) we can compute right and left eigenvectors. Beginning with right eigenvectors which are given by $(r_i)^T$ where $i = 1,2,...7$ we have:

$$\begin{aligned}
r_2 &= \frac{\alpha_h+\mu_h+\tau_h}{\gamma_a\beta_4(1-\tau_a)}\left(\frac{N_1-\gamma_a\beta_5(1-\tau_a)(\omega+\theta)-\rho\gamma_a\beta_6(1-\varepsilon)^2}{\rho(1-\varepsilon)}\right)r_7.\\
r_3 &= \left(\frac{N_1-\gamma_a\beta_5(1-\tau_a)(\omega+\theta)-\rho\gamma_a\beta_6(1-\varepsilon)^2}{\gamma_a\rho\beta_4(1-\varepsilon)(1-\tau_h)}\right)r_7,\\
r_4 &= \left(\frac{(\omega+\theta)(\gamma_a+\mu_a)(\alpha_a+\mu_a+\tau_a)}{\gamma_a\mu_a\rho(1-\tau_a)}\right)r_7,\\
r_5 &= \left(\frac{(\omega+\theta)(\alpha_a+\mu_a+\tau_a)}{\rho\gamma_a(1-\varepsilon)}\right)r_7,\\
r_6 &= \left(\frac{\omega+\theta}{\rho(1-\varepsilon)}\right)r_7,\\
r_1 &= \frac{r_3\beta_1(1-\tau_h)-r_6\beta_2(1-\tau_a)-r_7\beta_3(1-\varepsilon)}{\mu_h},\\
&\text{where } r_7>0 \text{ is free right eigenvector}\\
&\text{and } N_1=(\omega+\theta)(\gamma_a+\mu_a)(\alpha_a+\mu_a+\tau_a).
\end{aligned} \tag{4.55}$$

Left eigenvectors is given by $(L_i)^T$ where $i = 1,2,...7$

$$\begin{aligned}
L_1 = L_4 = 0.\quad L_3 &= \left(\frac{\gamma_h+\mu_h}{\gamma_h}\right)L_2,\\
L_5 &= \left(\frac{(\gamma_h+\mu_h)(\alpha_h+\mu_h+\tau_h)-\gamma_h\beta_1(1-\tau_h)}{\gamma_h\beta_4(1-\tau_h)}\right)L_2,\\
L_6 &= \left(\frac{(\gamma_a+\mu_a)((\gamma_h+\mu_h)(\alpha_h+\mu_h+\tau_h)-\gamma_h\beta_1(1-\tau_h))}{\gamma_h\gamma_a\beta_4(1-\tau_h)}\right)L_2,\\
L_7 &= \left(\frac{(\gamma_h\beta_3\beta_4N_2+\beta_6(1-\varepsilon)((\gamma_h+\mu_h)(\alpha_h+\mu_h+\tau)-\gamma_h\beta_1(1-\kappa))}{\gamma_h\beta_4(\omega+\theta)(1-\tau_h)}\right)L_2.\\
&\text{where } L_2>0 \text{ is free left eigenvector, and } N_2=(1-\varepsilon)(1-\tau_h).
\end{aligned} \tag{4.56}$$

Computation of a_c

From the model system (4.52) the associated non-zero partial derivatives of G at disease free

equilibrium are given by:

$$\begin{aligned}
&\frac{\partial^2 g_2}{\partial y_3^2} = -\frac{2\beta_1(1-\tau_h)\mu_h}{\Lambda_h}, \frac{\partial^2 g_2}{\partial y_2 \partial y_3} = -\frac{\beta_1(1-\tau_h)\mu_h}{\Lambda_h},\\
&\frac{\partial^2 g_2}{\partial y_2 \partial y_6} = -\frac{\beta_2(1-\tau_a)\mu_h}{\Lambda_h}, \frac{\partial^2 g_2}{\partial y_2 \partial y_7} = \frac{-\beta^*(1-\varepsilon)\mu_h}{\Lambda_h},\\
&\frac{\partial^2 g_2}{\partial y_3 \partial y_6} = -\frac{\beta_2(1-\tau_a)\mu_h}{\Lambda_h}, \frac{\partial^2 g_2}{\partial y_3 \partial y_7} = \frac{-\beta^*(1-\varepsilon)\mu_h}{\Lambda_h},\\
&\frac{\partial^2 g_5}{\partial y_6^2} = -\frac{2\beta_5(1-\tau_a)\mu_a}{\Lambda_a}, \frac{\partial^2 g_5}{\partial y_3 \partial y_5} = -\frac{2\beta_4(1-\tau_h)\mu_a}{\Lambda_a},\\
&\frac{\partial^2 g_5}{\partial y_3 \partial y_6} = -\frac{\beta_4(1-\tau_h)\mu_a}{\Lambda_a}, \frac{\partial^2 g_5}{\partial y_5 \partial y_6} = \frac{-\beta_5(1-\tau_a)\mu_a}{\Lambda_a},\\
&\frac{\partial^2 g_5}{\partial y_5 \partial y_7} = \frac{-\beta_6(1-\varepsilon)\mu_a}{\Lambda_a}, \frac{\partial^2 g_5}{\partial y_6 \partial y_7} = \frac{-\beta_6(1-\varepsilon)\mu_a}{\Lambda_a}.
\end{aligned} \tag{4.57}$$

Since $L_1 = L_4 = 0$ it follows that,

$$a_c = L_2 \sum_{i,j=1}^{n} r_i r_j \frac{\partial^2 g_2}{\partial y_i \partial y_j} + L_5 \sum_{i,j=1}^{n} r_i r_j \frac{\partial^2 g_5}{\partial y_i \partial y_j}. \tag{4.58}$$

To compute the values of a_c we substitute the partial derivatives from (4.57) into (4.58) to get:

$$\begin{aligned}
a_c = &\left(\frac{2\mu_h X_h r_3(\alpha_h+\mu_h+\gamma_h)\beta_1(1-\tau_h)\gamma_a((\omega+\theta)\beta_5(1-\tau_a)+\rho\beta_6(1-\varepsilon))}{X_h\gamma_h X_a\gamma_a\rho\beta_4(1-\tau_h)(1-\varepsilon)}\right) r_7 L_2\\
&-\left(\frac{2\mu_h X_a r_3 \Psi_1(\beta_1(1-\tau_h)(\omega+\theta)\Psi_1+\gamma_a\beta_4(1-\tau_h)(\Psi_2+\rho\beta^{**}(1-\varepsilon)))}{X_h\gamma_h X_a\gamma_a\rho\beta_4(1-\tau_h)(1-\varepsilon)}\right) r_7 L_2\\
&-\left(\frac{2X_h\gamma_h(\omega+\theta)\beta_4(1-\tau_h)\mu_a(r_5+r_6)\Psi_1}{X_h\gamma_h X_a\gamma_a\rho\beta_4(1-\tau_h)(1-\varepsilon)}\right) r_7 L_5.
\end{aligned}$$

where $\Psi_1 = (\gamma_a+\mu_a)(\alpha_a+\mu_a+\tau_a)$, $\Psi_2 = \beta_2(\omega+\theta)(1-\tau_a)$.

To analyze the sign of a_c we consider two cases.

Case I:

$a_c < 0$ if

$$\begin{aligned}
&\frac{\gamma_a((1-\tau_a)(\omega+\theta)\beta_5+\rho\beta_6(1-\varepsilon)^2)}{(\omega+\theta)(\gamma_a+\mu_a)(\alpha_a+\mu_a+\tau_a)} < 1\\
\text{and } &\frac{\Psi^2\beta_1(1-\tau_h)(\omega+\theta)+\gamma_a\beta_4(1+\tau_h)(\Psi_2+\rho\beta^{**}(1-\varepsilon))}{(\alpha_h+\mu_h+\tau_h)\beta_1(1-\tau_h)\gamma_a(\omega+\theta)\beta_5(1-\tau_a)+\rho\beta_6(1-\varepsilon)}\\
&+\frac{\gamma_a(\omega+\theta)\beta_4(1-\tau_h)\mu_a(r_5+r_6)}{r_3(\alpha_h+\mu_h+\tau_h)\beta_1(1-\tau_h)\gamma_a(\omega+\theta)\beta_5(1-\tau_a)+\rho\beta_6(1-\varepsilon)} > 1
\end{aligned} \tag{4.59}$$

Case II:

$a_c > 0$ if

$$\frac{\gamma_a((1-\tau_a)(\omega+\theta)\beta_5+\rho\beta_6(1-\varepsilon)^2)}{(\omega+\theta)(\gamma_a+\mu_a)(\alpha_a+\mu_a+\tau_a)} > 1$$

$$\text{and } \frac{\Psi^2\beta_1(1-\tau_h)(\omega+\theta)+\gamma_a\beta_4(1+\tau_h)(\Psi_2+\rho\beta^{**}(1-\varepsilon))}{(\alpha_h+\mu_h+\tau_h)\beta_1(1-\tau_h)\gamma_a(\omega+\theta)\beta_5(1-\tau_a)+\rho\beta_6(1-\varepsilon)} + \frac{\gamma_a(\omega+\theta)\beta_4(1-\tau_h)\mu_a(r_5+r_6)}{r_3(\alpha_h+\mu_h+\tau_h)\beta_1(1-\tau_h)\gamma_a(\omega+\theta)\beta_5(1-\tau_a)+\rho\beta_6(1-\varepsilon)} < 1 \tag{4.60}$$

Computation of b_c

Recall from (4.56) since $L_1 = L_4 = 0$, $r_7 > 0$ is free right eigenvector and $L_2 > 0$ is free left eigenvector then, b_c becomes:

$$\begin{aligned} b_c &= L_2 \sum_{i=1}^{n} r_i \frac{\partial^2 g_k}{\partial y_i \partial \beta^{**}}(0,0), \\ b_c &= L_2 r_7 \frac{\partial^2 g_2}{\partial y_7 \partial \beta^{**}}, \\ b_c &= (1-\varepsilon) L_2 r_7 > 0. \end{aligned} \tag{4.61}$$

From the computation of a_c and b_c we can establish the following results.

If $a_c < 0, b_c > 0$ when β^{**} changes from negative to positive, disease-free equilibrium changes its stability from stable to unstable. correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.

Theorem 6: *If*

$$\frac{\gamma_a((1-\tau_a)(\omega+\theta)\beta_5+\rho\beta_6(1-\varepsilon)^2)}{(\omega+\theta)(\gamma_a+\mu_a)(\alpha_a+\mu_a+\tau_a)} > 1$$

and

$$\frac{\Psi^2\beta_1(1-\tau_h)(\omega+\theta)+\gamma_a\beta_4(1+\tau_h)(\Psi_2+\rho\beta^{**}(1-\varepsilon))}{(\alpha_h+\mu_h+\tau_h)\beta_1(1-\tau_h)\gamma_a(\omega+\theta)\beta_5(1-\tau_a)+\rho\beta_6(1-\varepsilon)} + \frac{\gamma_a(\omega+\theta)\beta_4(1-\tau_h)\mu_a(r_5+r_6)}{r_3(\alpha_h+\mu_h+\tau_h)\beta_1(1-\tau_h)\gamma_a(\omega+\theta)\beta_5(1-\tau_a)+\rho\beta_6(1-\varepsilon)} < 1.$$

the model system (4.1) *undergoes backward bifurcation when* $R_e = 1$.

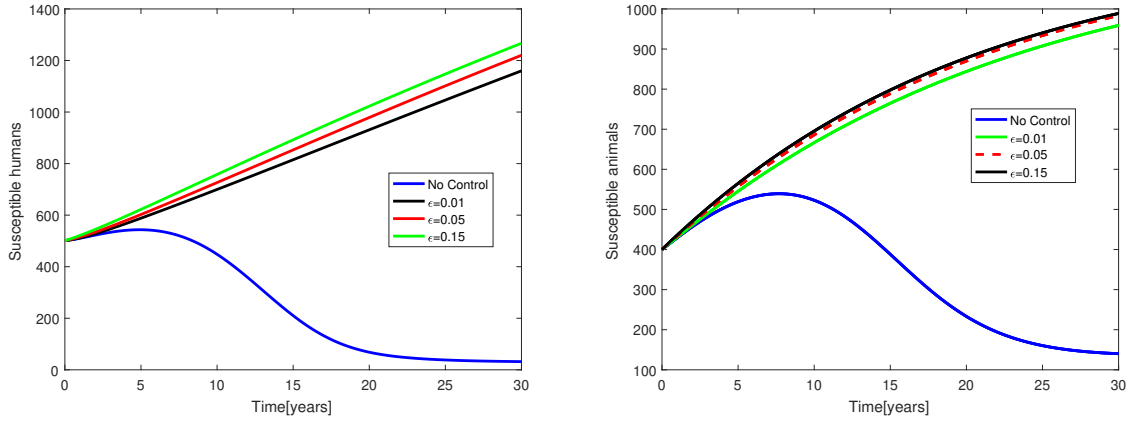
4.4 Numerical Simulation for the Model with Controls

Numerical simulations are performed to discuss how bTB can be eliminated from the population using treatment of infected humans, quarantine of infected animals, and inspection of dairy products. The initial condition we assumed to be $S_h = 530; E_h = 15; I_h = 4; S_a = 500; E_a = 25; I_a = 10$ and $D = 13$. Some of the parameters are estimated because the disease is neglected and there is no surveillance data. The parameters are summarized in Table 7 as follows:

Table 7: Parameter Values of the Model system (4.1)

Parameter	Interpretation	Value yr^{-1}	Source.
γ_a	Progression rate from E_a to I_a	0.18	Ssematimba *et al.* (2015).
Π_h	Human recovery rate	0.00271	Hassan *et al.* (2014).
μ_h	Human natural death rate	0.01	Liu *et al.* (2016)
β_5	Rate of cow infected via animal	0.6	Agusto *et al.* (2011).
μ_h	Human natural death rate	0.01	Liu *et al.* (2016)
α_h	Human death rate due to disease induced	0.139	Liu *et al.* (2016).
τ_h	Treatment rate for infected human	0.58	Liu *et al.* (2016) .
ρ	Rate of producing infected dairy products	0.69	Estimated.
β_2	Human infection rate from infected animals	0.55	Hassan *et al.* (2014).
ω	rate of decaying dairy products	0.4	Estimated.
β_3	Human infection rate from infected dairy products	0.999	Estimated.
β_4	rate of cow infected via human	0.25	Estimated.
μ_a	Animal natural death rate	0.1	Mariner *et al.* (2006).
β_6	rate of animals infected via dairy products	0.34	Estimated.

Parameter	Interpretation	Value yr^{-1}	Source.
γ_h	Progression rate from E_h to I_h	0.15	Dye and Williams (2008).
β_1	Human infection rate from infected human	0.35	Estimated.
α_a	Animal death due to disease induced	0.0304	Agusto *et al.* (2011).
τ_a	Quarantine rate for infected animals	0.85	Liu *et al.* (2016).
ε	dairy products inspection rate	0.5	Estimated.

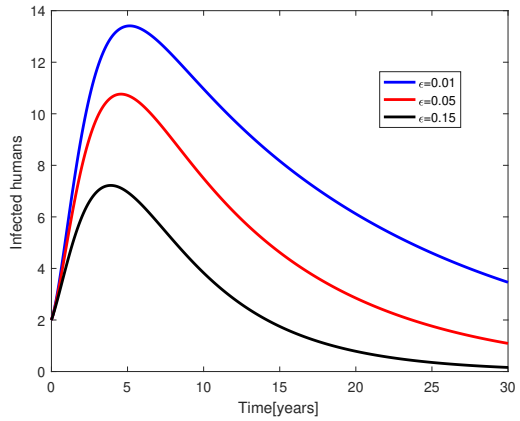


(a) Susceptible humans after inspection of dairy products

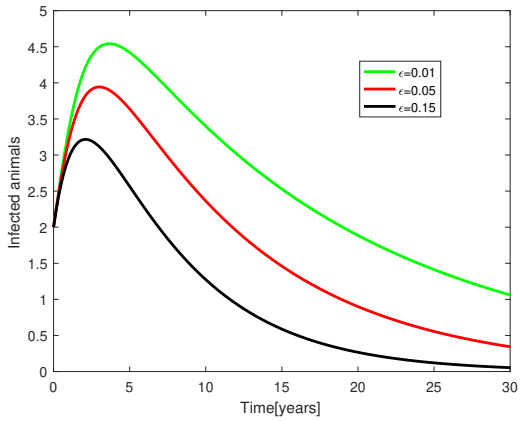
(b) Susceptible animals after inspection of dairy products

Figure 11: The impacts of control parameters on S_h and S_a

Figure 11 shows the impacts of dairy products inspection on the transmission of bTB in humans and animals. The results show that inspection of dairy products helps to reduce the spread of the disease to both humans and animals. The blue line graph indicated susceptible humans and susceptible animals before controls of bTB. Susceptible human and animal class increases as inspection of dairy products increase from 1% to 15%.



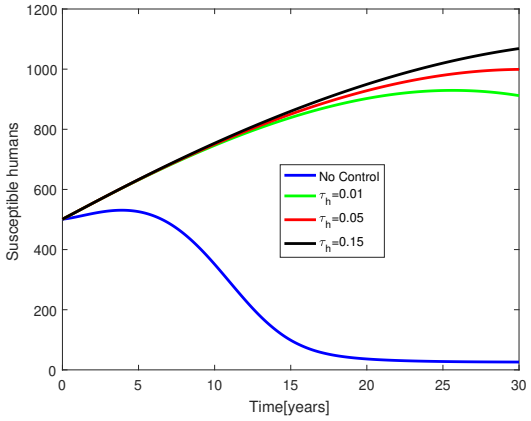
(a) Infected humans after dairy products inspection



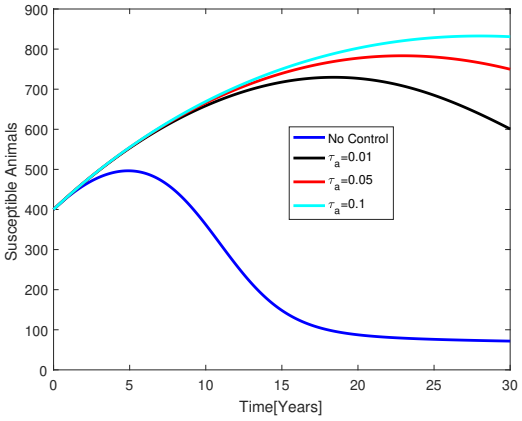
(b) Infected animals after dairy products inspection

Figure 12: Infected humans and animals after controls

Infected humans and animals classes decrease when we introduce dairy products inspection as a control for the transmission of the disease, as shown in Fig. 12 (a) and (b), respectively. If inspection of dairy product is applied effectively the infection of bTB from these products can be reduced; hence infected classes decrease as indicated in Fig. 12.



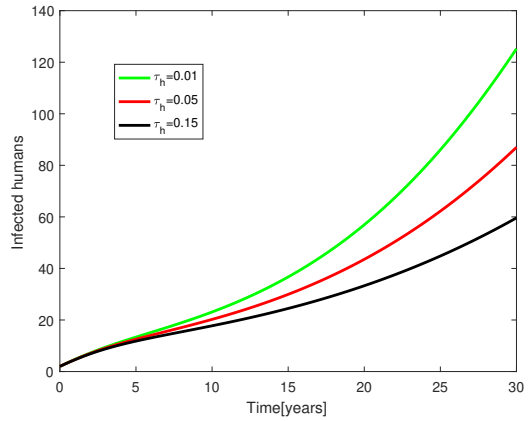
(a) Effect of human treatment on S_h



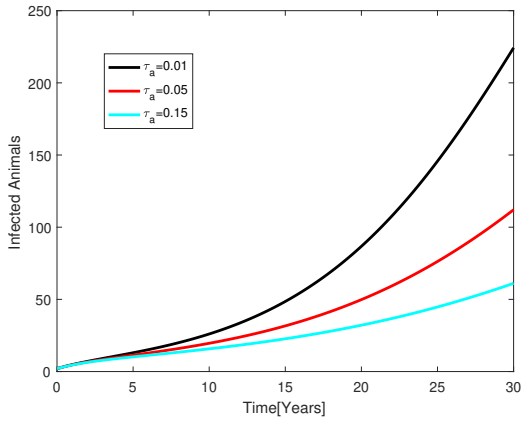
(b) Effect of animals quarantine on S_a

Figure 13: S_h and S_a after treatment of I_h and quarantine of I_a

Figure 13 (a) shows the effects of human treatment, and Fig. 13 (b) shows the effects of animals quarantine on susceptible humans and animals classes, respectively. Simulation shows that if infected humans are treated and infected animals are quarantined once they diagnosed with bTB, the transmission of the disease from infected classes decrease. For example, susceptible classes increase when human treatment rate increases from 1 to 15% as shown in Fig. 13(a).



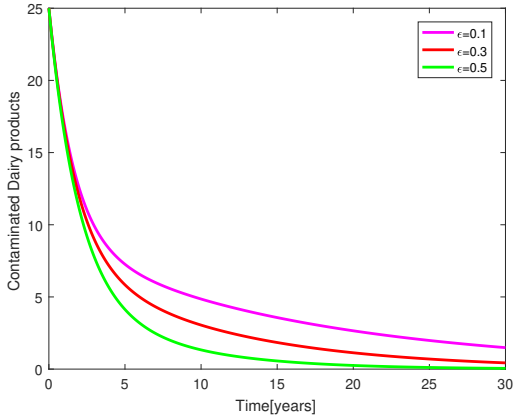
(a) Treatment on infected humans



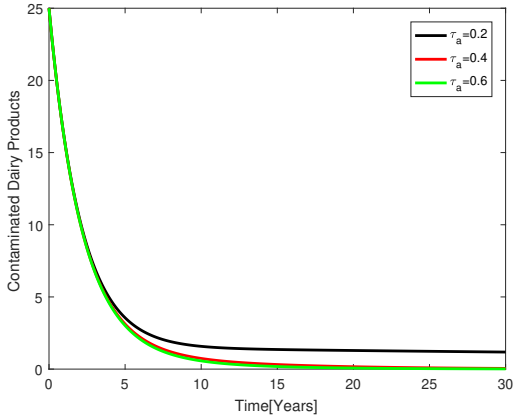
(b) Quarantine on infected animals

Figure 14: I_h and I_a after treatment of I_h and quarantine of I_a

The infected human class tends to decrease as infected human treatment rate increases from 1% to 15% as shown in Fig. 14(a) and (b). Furthermore, the quarantine of infectious animals from 1% to 15% lead to the decreases of infectious animal class as shown in Fig. 14(b).



(a) Influence of dairy products inspection (ε) on the production of dairy products



(b) Influence of quarantine of infected animals (τ_a) on the production of dairy products

Figure 15: The impacts of control parameters on the production of dairy products

Figure 15 shows that quarantine of infected animals and dairy products inspection. As infected animals quarantined for about 20% to 60%, and dairy products inspected for about 10% to 50%, the rate of producing infectious dairy products decreases. So the strict inspection of dairy products and quarantine of infected animals helps to reduce the production of contaminated products as show on Fig. 15.

4.4.1 Conclusion

Bovine Tuberculosis model was formulated and analyzed to determine the proposed controls can help to contain the disease. Disease-free is asymptotically stable when the effective re-

production number is less than unit $R_e < 1$ and unstable otherwise. The bTB model undergoes backward bifurcation when effective reproduction number $R_e = 1$. In the numerical simulation, when we increase the rates of treatment of infected humans, quarantine of infected animals and inspection of dairy product, the effective reproduction number R_e decreases proportionally.

CHAPTER FIVE

CONCLUSION AND RECOMMENDATIONS

5.1 Conclusion

This study used mathematical modelling to investigate the transmission dynamics of bTB in human and animals before and after including controls. The basic reproduction number R_0 computed and analyzed. The disease-free equilibrium was proved to be locally asymptotically stable when $R_0 < 1$ and endemic equilibrium is stable when $R_0 > 1$. However, disease-free and endemic equilibria coexist when $R_0 = 1$. Sensitivity analysis shows that, production of dairy products ρ, the animal infection rate from infected animals β_5, infection rate due to the consumption of dairy products to animals β_6, the human infection rate from the consumption of infectious dairy products β_3 and human infection rate due to the interaction with infected animals β_2 all drives the spread of bTB. Numerical simulations show that before the introduction of the controls, susceptible individuals decreased with an increase in infectious individuals. However, after introducing control strategies, the number of susceptible humans and animals increases while the number of infected humans and animals diminishes. Therefore the proposed control parameters can help to contain bTB if they implemented effectively.

5.2 Recommendations

In this work, we have accomplished our objectives. However, the work is not exhaustive hence the study can be extended by including the following:

(i) Role of the environment on transmission dynamics of bovine tuberculosis between wild animals and domestic animals.

(ii) Impacts of weather condition on the persistence of bovine tuberculosis.

(iii) Optimal control for bovine tuberculosis on human treatment, animals quarantine, an inspection of dairy products and education campaign.

REFERENCES

Agusto, F. B., Lenhart, S., Gumel, A. B. and Odoi, A. (2011). Mathematical Analysis of a Model for the Transmission Dynamics of Bovine Tuberculosis. *Mathematical Methods in the Applied Sciences*. **34**(15): 1873–1887.

Allen, A., Skuce, R. and Byrne, A. (2018). Bovine Tuberculosis in Britain and Ireland–A Perfect Storm? the Confluence of Potential Ecological and Epidemiological Impediments to Controlling a Chronic Infectious Disease. *Frontiers in Veterinary Science*. **5**: 109.

Ameni, G., Aseffa, A., Engers, H., Young, D., Gordon, S., Hewinson, G. and Vordermeier, M. (2007). High prevalence and increased severity of pathology of bovine tuberculosis in holsteins compared to zebu breeds under field cattle husbandry in central ethiopia. *Clin. Vaccine Immunol.*. **14**(10): 1356–1361.

Ameni, G., Miörner, H., Roger, F. and Tibbo, M. (2000). Comparison between comparative tuberculin and gamma-interferon tests for the diagnosis of bovine tuberculosis in ethiopia. *Tropical animal health and production*. **32**(5): 267–276.

Assembly, W. (2009). Bovine Tuberculosis. *Techniques*. (May): 1–16.

Biet, F., Boschiroli, M. L., Thorel, M. F. and Guilloteau, L. A. (2005). Zoonotic Aspects of Mycobacterium Bovis and Mycobacterium Avium-Intracellulare Complex (mac). *Veterinary Research*. **36**(3): 411–436.

Bonsu, O., Laing, E. and Akanmori, B. (2000). Prevalence of Tuberculosis in Cattle in the Dangme-West District of Ghana, Public Health Implications. *Acta Tropica*. **76**(1): 9–14.

Bowong, S. (2010). Optimal Control of the Transmission Dynamics of Tuberculosis. *Nonlinear Dynamics*. **61**(4): 729–748.

Brooks-Pollock, E. and Danon, L. (2017). Defining the Population Attributable Fraction for Infectious Diseases. *International Journal of Epidemiology*. **46**(3): 976–982.

Centers for Disease Control & Prevention (2011). Mycobacterium Bovis (Bovine Tuberculosis) in Humans. *Mycobacterium Bovis (bovine tuberculosis) in humans*. p. 2.
URL: *http://www.cdc.gov/tb/publications/factsheets/general/mbovis.pdf*

Chitnis, N., Hyman, J. M. and Cushing, J. M. (2008). Determining Important Parameters in the Spread of Malaria Through the Sensitivity Analysis of a Mathematical Model. *Bulletin of Mathematical Biology*. **70**(5): 1272.

Cousins, D. and Roberts, J. (2001). Australia's Campaign to Eradicate Bovine Buberculosis: the Battle for Freedom and Beyond. *Tuberculosis*. **81**(1-2): 5–15.

Davidson, J. A., Loutet, M. G., Connor, C., Kearns, C., Smith, R. M., Lalor, M. K., Thomas, H. L., Abubakar, I. and Zenner, D. (2017). Epidemiology of mycobacterium bovis disease in humans in england, wales, and northern ireland, 2002–2014. *Emerging infectious diseases*. **23**(3): 377.

De Garine-Wichatitsky, M., Caron, A., Kock, R., Tschopp, R., Munyeme, M., Hofmeyr, M. and Michel, A. (2013). A Review of Bovine Tuberculosis at the Wildlife-Livestock-Human Interface in Sub-Saharan Africa. *Epidemiology and Infection*. **141**(7): 1342–1356.

Dejene, S. W., Heitkönig, I. M., Prins, H. H., Lemma, F. A., Mekonnen, D. A., Alemu, Z. E., Kelkay, T. Z. and De Boer, W. F. (2016). Risk Factors for Bovine Tuberculosis (bTB) in Cattle in Ethiopia. *PLoS ONE*. **11**(7): 1–16.

Demelash, B., Inangolet, F., Oloya, J., Asseged, B., Badaso, M., Yilkal, A. and Skjerve, E. (2009). Prevalence of bovine tuberculosis in ethiopian slaughter cattle based on postmortem examination. *Tropical animal health and production*. **41**(5): 755–765.

Diekmann, O., Heesterbeek, J. A. P. and Metz, J. A. (1990). On the Definition and the Computation of the Basic Reproduction Ratio r 0 in Models for Infectious Diseases in Heterogeneous Populations. *Journal of Mathematical Biology*. **28**(4): 365–382.

Durnez, L., Katakweba, A., Sadiki, H., Katholi, C. R., Kazwala, R. R., MacHang'U, R. R., Portaels, F. and Leirs, H. (2011). Mycobacteria in Terrestrial Small Mammals on Cattle Farms in Tanzania. *Veterinary Medicine International*. **2011**.

Dye, C. and Williams, B. G. (2008). Eliminating Human Tuberculosis in the Twenty-First Century. *Journal of the Royal Society Interface*. **5**(23): 653–662.

Fellin, W., Lessmann, H., Oberguggenberger, M. and Vieider, R. (2005). Analyzing Uncertainty in Civil Engineering. Springer.

Griffin, J., Williams, D. and Collins, J. (2000). a Compartmental Model for the Within-Herd Spread of M. Bovis in Irish Cattle Herds. *Proceedings of the 9th International Symposium on Veterinary Epidemiology and Economics*. pp. 1997–1999.

Gumel, A. B. and Song, B. (2008). Existence of multiple-Stable equilibria for a multi-Drug-resistant model of mycobacterium tuberculosis. *Mathematical Biosciences and Engineering: MBE*. **5**(3): 437–455.

Gumi, B., Schelling, E., Firdessa, R., Aseffa, A., Tschopp, R., Yamuah, L., Young, D. and Zinsstag, J. (2011). Prevalence of bovine tuberculosis in pastoral cattle herds in the oromia region, southern ethiopia. *Tropical animal health and production*. **43**(6): 1081.

Hassan, A. S., Garba, S. M., Gumel, A. B. and Lubuma, J.-S. (2014). Dynamics of Mycobacterium and Bovine Tuberculosis in a Human-Buffalo Population. *Computational and Mathematical Methods in Medicine*. **2014**.

Jemal, A. M. (2016). Review on Zoonotic Importance of Bovine Tuberculosis and its Control. *Open Access Library Journal*. **3**(03): 1.

Katale, B. Z., Mbugi, E. V., Karimuribo, E. D., Keyyu, J. D., Kendall, S., Kibiki, G. S., Godfrey-Faussett, P., Michel, A. L., Kazwala, R. R., van Helden, P. and Matee, M. I. (2013). Prevalence and risk factors for infection of bovine tuberculosis in indigenous cattle in the Serengeti ecosystem, Tanzania. *BMC Veterinary Research*. **9**.

Katale, B. Z., Mbugi, E. V., Kendal, S., Fyumagwa, R. D., Kibiki, G. S., Godfrey-Faussett, P., Keyyu, J. D., Van Helden, P. and Matee, M. I. (2012). Bovine Tuberculosis at the Human-Livestock-Wildlife Interface: Is it a Public Health Problem in Tanzania? A Review. *Onderstepoort J Vet Res*. **79**(2): 1–8.
URL: *http://ojvr.org/index.php/ojvr/article/view/463*

Kilale, A. M. (2016). Mycobacteria in northern tanzania: Exposure and risk of disease among agropastoralists and programmatic challenges in investigation of re-treatment cases. *The Veterinary Journal*. **11**(7): 1–16.

Leo, G. A. D. and Natalini, S. (2015). Epidemiological modeling for the assessment of bovine tuberculosis surveillance in Emilia-Romagna Italy. *The Veterinary Journal*. **11**: 5.

Liu, S., Li, A., Feng, X., Zhang, X. and Wang, K. (2016). A Dynamic Model of Human and Livestock Tuberculosis Spread and Control in Urumqi, Xinjiang, China. *Computational and Mathematical Methods in Medicine*. **2016**.

Mariner, J. C., McDermott, J., Heesterbeek, J., Thomson, G. and Martin, S. (2006). A model of contagious bovine pleuropneumonia transmission dynamics in east africa. *Preventive Veterinary Medicine*. **73**(1): 55–74.

Mathews, F., Macdonald, D. W., Taylor, G. M., Gelling, M., Norman, R. A., Honess, P. E., Foster, R., Gower, C. M., Varley, S., Harris, A., Palmer, S., Hewinson, G. and Webster, J. P. (2006). Bovine tuberculosis (Mycobacterium bovis) in British farmland wildlife: The importance to agriculture. *Proceedings of the Royal Society B: Biological Sciences*. **273**(1584): 357–365.

McCulloch, Steven P & Reiss, M. J. (2017). Bovine tuberculosis and badger control in britain: Science, policy and politics. *Journal of Agricultural and Environmental Ethics*. **30**(4): 469–484.

Menzies, FD & Neill, S. (2000). Cattle-to-cattle transmission of bovine tuberculosis. *The Veterinary Journal*. **160**(2): 92–106.

Michel, A. L., Bengis, R. G., Keet, D., Hofmeyr, M., De Klerk, L., Cross, P., Jolles, A. E., Cooper, D., Whyte, I., Buss, P. *et al.* (2006). Wildlife tuberculosis in south african conservation areas: implications and challenges. *Veterinary Microbiology*. **112**(2-4): 91–100.

OHagan, M., Courcier, E., Drewe, J., Gordon, A., McNair, J. and Abernethy, D. (2015). Risk factors for visible lesions or positive laboratory tests in bovine tuberculosis reactor cattle in northern ireland. *Preventive Veterinary Medicine*. **120**(3-4): 283–290.

OIE (2016). Bovine tuberculosis. *OIE Terrestrial Maual 2015*. (September): 1–72.
URL: *http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.04.06_BOVINE_TB.pdf%0Aht*
standard-setting/terrestrial-manual/access-online/

Palmer, M. V., Thacker, T. C., Waters, W. R., Gortázar, C. and Corner, L. A. (2012). Mycobacterium bovis: A model pathogen at the interface of livestock, wildlife, and humans. *Veterinary Medicine International*. **2012**.

Pavlik, I. (2006). The experience of new european union member states concerning the control of bovine tuberculosis. *Veterinary microbiology*. **112**(2-4): 221–230.

Perez, A. M., Ward, M. P., Charmandarián, A. and Ritacco, V. (2002). Simulation model of within-herd transmission of bovine tuberculosis in argentine dairy herds. *Preventive Veterinary Medicine*. **54**(4): 361–372.

Phepa, P. B., Chirove, F. and Govinder, K. S. (2016). Modelling the role of multi-transmission routes in the epidemiology of bovine tuberculosis in cattle and buffalo populations. *Mathematical Biosciences*. **277**: 47–58.

Picasso, C., Alvarez, J., VanderWaal, K. L., Fernandez, F., Gil, A., Wells, S. J. and Perez, A. (2017). Epidemiological investigation of bovine tuberculosis outbreaks in uruguay (2011–2013). *Preventive Veterinary Medicine*. **138**: 156–161.

Ramos, D. F., Silva, P. E. A. and Dellagostin, O. A. (2015). Diagnosis of bovine tuberculosis: review of main techniques. *Braz. J. Biol*. **75**(4): 830–837.
URL: *http://dx.doi.org/*

Regassa, A., Medhin, G. and Ameni, G. (2008). Bovine tuberculosis is more prevalent in cattle owned by farmers with active tuberculosis in central ethiopia. *The Veterinary Journal*. **178**(1): 119–125.

Shirima, G., Kazwala, R. and Kambarage, D. (2003). Prevalence of bovine tuberculosis in cattle in different farming systems in the eastern zone of Tanzania. *Preventive Veterinary Medicine*. **57**(3): 167–172.

Silva, C. J. and Torres, D. F. (2013). Optimal control for a tuberculosis model with reinfection and post-exposure interventions. *Mathematical Biosciences*. **244**(2): 154–164.

Ssematimba, A., Jores, J. and Mariner, J. C. (2015). Mathematical modelling of the transmission dynamics of contagious bovine pleuropneumonia reveals minimal target profiles for improved vaccines and diagnostic assays. *PLoS ONE*. **10**(2): 1–15.

Tschopp, R., Schelling, E., Hattendorf, J., Aseffa, A. and Zinsstag, J. (2009). Risk factors of bovine tuberculosis in cattle in rural livestock production systems of ethiopia. *Preventive Veterinary Medicine*. **89**(3-4): 205–211.

Van, Pauline & Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences*. **180**(1-2): 29–48.

WHO (2016). Global tuberculosis report 2017: World health organization; 2017. *Back to Cited Text*. (1).

WHO (2018). Global tuberculosis report 2018. World Health Organization.

Wilkinson, D., Smith, G., Delahay, R. and Cheeseman, C. (2004). A model of bovine tuberculosis in the badger Meles meles: an evaluation of different vaccination strategies. *Journal of Applied Ecology*. **41**: 492–501.

APPENDICES

Appendix 1: MATLAB CODES FOR CHAPTER THREE

A.1 MATLAB codes for Figure 2

```
%Defining function 'Teddy.m' and it's corresponding equations as follows
function dy=Teddy_1(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;Lambda_h=60;beta_3=0.999;gamma_h=0.18; mu_h=0.01;alpha_h=0.139;omega=0.4;
beta_4=0.25;beta_5=0.6;beta_6=0.34;gamma_a=0.18;Lambda_a=65; mu_a=0.05;alpha_a=0.12;rho=0.69;

  %Variable description
Sh=y(1);Eh=y(2);Ih=y(3);Sa=y(4);Ea=y(5);Ia=y(6);D=y(7);
Nh=y(1)+y(2)+y(3);
Na=y(4)+y(5)+y(6);

  %Equation of the model
dy(1)=Lambda_h -((beta_1*Ih+beta_2*Ia+beta_3*D)*Sh)/Nh-mu_h*Sh ;
dy(2)=((beta_1*Ih+beta_2*Ia+beta_3*D)*Sh)/Nh- (gamma_h+mu_h)* Eh;
dy(3)=gamma_h*Eh-(alpha_h+mu_h)*Ih;
dy(4)=Lambda_a-((beta_4*Ih+beta_5*Ia+beta_6*D)*Sa)/Na-mu_a*Sa;
dy(5)=((beta_4*Ih+beta_5*Ia+beta_6*D)*Sa)/Na-(mu_a+gamma_a)*Ea ;
dy(6)=gamma_a*Ea-(alpha_a+mu_a)*Ia;
dy(7)=rho*Ia-omega*D;

RUNNING FILE
clear all
clc
tspan =0:0.1:30 ; %Time in yrs ,
y0=[500, 10, 3, 500, 11, 5, 5];
[t,y]=ode45(@Teddy_1,tspan,y0);

figure(2a)
set(gca,'FontSize',10)
set(legend,'FontSize',10)
plot(t,y(:,1),'g',t,y(:,2),'b',t,y(:,3),'r',t,y(:,7),'y',' LineWidth',1.5);
ylim([0 1000])
legend('S_h','E_h','I_H','D');
xlabel('Time[years]');
```

```
ylabel('Human Populations');
%  title('Human Population Vs Time');
grid off
hold off
hold on

figure(2b)
set(gca,'FontSize',10)
set(legend,'FontSize',10)
plot(t,y(:,4),'k',t,y(:,5),'m',t,y(:,6),'c',t,y(:,7),'y','
   LineWidth',1.5);
ylim([0 1000])
legend('S_a','E_a','I_a','D');
xlabel('Time[years]');
ylabel('Animal Populations');
 %title('Animal Population Vs Time');
grid off
hold off
hold on
```

A.2 MATLAB codes for Figure 3 and 4

```
%Defining function 'Teddy_D1.m' and it's corresponding
   equations as follows
function dy=Teddy_D1(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.999;Lambda_h=60;gamma_h=0.18;
   mu_h=0.018;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;Lambda_a=65;beta_6=0.34;gamma_a=0.195;
   mu_a=0.05;alpha_a=0.12;
rho=0.1;omega=0.4;

  %Defining function 'Teddy_D2.m' and it's corresponding
     equations as follows
function dy=Teddy_D2(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.999;Lambda_h=60;gamma_h=0.18;
   mu_h=0.018;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;Lambda_a=65;beta_6=0.34;gamma_a=0.195;
   mu_a=0.05;alpha_a=0.12;
rho=0.2;omega=0.4;

  %Defining function 'Teddy_D3.m' and it's corresponding
     equations as follows
```

```
function dy=Teddy_D3(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.999;Lambda_h=60;gamma_h=0.18;
   mu_h=0.018;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;beta_6=0.34;gamma_a=0.195;Lambda_a=65;
   mu_a=0.05;alpha_a=0.12;
rho=0.3;omega=0.4;

  %Defining function 'Teddy_D4.m' and it's corresponding
     equations as follows
function dy=Teddy_D4(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.999;Lambda_h=60;gamma_h=0.18;
   mu_h=0.018;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;beta_6=0.34;gamma_a=0.195;Lambda_a=65;
   mu_a=0.05;alpha_a=0.12;
rho=0.5;omega=0.4;

%Defining function 'Teddy_D5.m' and it's corresponding
   equations as follows
function dy=Teddy_D5(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;Lambda_h=60;beta_3=0.999;gamma_h=0.18;
   mu_h=0.018;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;beta_6=0.34;Lambda_a=65;gamma_a=0.195;
   mu_a=0.05;alpha_a=0.12;
rho=0.6;omega=0.4;

  %Variable description
Sh=y(1);Eh=y(2);Ih=y(3);Sa=y(4);Ea=y(5);Ia=y(6);D=y(7);
Nh=y(1)+y(2)+y(3);
Na=y(4)+y(5)+y(6);
  %Equation of the model
dy(1)=Lambda_h -((beta_1*Ih+beta_2*Ia+beta_3*D)*Sh)/Nh-mu_h*Sh
   ;
dy(2)=((beta_1*Ih+beta_2*Ia+beta_3*D)*Sh)/Nh- (gamma_h+mu_h)*
   Eh;
dy(3)=gamma_h*Eh-(alpha_h+mu_h)*Ih;
dy(4)=Lambda_a-((beta_4*Ih+beta_5*Ia+beta_6*D)*Sa)/Na-mu_a*Sa;
dy(5)=((beta_4*Ih+beta_5*Ia+beta_6*D)*Sa)/Na-(mu_a+gamma_a)*Ea
   ;
dy(6)=gamma_a*Ea-(alpha_a+mu_a)*Ia;
```

```
dy(7)=rho*Ia-omega*D;

%RUNNING FILE
tspan =0:0.1:30 ; %Time in yrs ,
y0=[530, 9, 2, 500, 10, 2, 5];
[t1,y1]=ode45(@Teddy_D1,tspan,y0);
[t2,y2]=ode45(@Teddy_D2,tspan,y0);
[t3,y3]=ode45(@Teddy_D3,tspan,y0);
[t4,y4]=ode45(@Teddy_D4,tspan,y0);
[t5,y5]=ode45(@Teddy_D5,tspan,y0);

figure(3a)
plot(t1,y1(:,1),'g',t2,y2(:,1),'b',t3,y3(:,1),'r',t4,y4(:,1),'
   k',t5,y5(:,1),'m','LineWidth',2)
legend('\rho=0.1','\rho=0.2','\rho=0.3','\rho=0.4','\rho=0.5')
xlabel('Time[years]')
ylabel('Susceptible humans')
hold on

figure(3b)
plot(t1,y1(:,4),'g',t2,y2(:,4),'b--',t3,y3(:,4),'r',t4,y4(:,4)
   ,'k--',t5,y5(:,4),'m','LineWidth',3)
legend('\rho=0.1','\rho=0.2','\rho=0.3','\rho=0.4','\rho=0.5')
xlabel('Time[years]')
ylabel('Susceptible animals')
hold on

figure(4a)
plot(t1,y1(:,3),'g',t2,y2(:,3),'b',t3,y3(:,3),'r',t4,y4(:,3),'
   k',t5,y5(:,3),'m','LineWidth',2)
legend('\rho=0.1','\rho=0.2','\rho=0.3','\rho=0.4','\rho=0.5')
xlabel('Time[years]')
ylabel('Infected humans')
hold on

figure(4b)
plot(t1,y1(:,6),'g',t2,y2(:,6),'b--',t3,y3(:,6),'r',t4,y4(:,6)
   ,'k--',t5,y5(:,6),'m','LineWidth',3)
legend('\rho=0.1','\rho=0.2','\rho=0.3','\rho=0.4','\rho=0.5')
xlabel('Time[years]')
ylabel('Infected animals')
hold off
```

A.4 MATLAB codes for Figure 7 and 8

```
%Defining function 'Teddy_B31.m' and it's corresponding
   equations as follows
function dy=Teddy_B31(~,y)
```

```
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.1;Lambda_h=60;gamma_h=0.18;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;beta_6=0.34;Lambda_a=65;gamma_a=0.18;
   mu_a=0.05;alpha_a=0.12;
rho=0.69;omega=0.4;

%Defining function 'Teddy_B32.m' and it's corresponding
   equations as follows
function dy=Teddy_B32(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.3;Lambda_h=60;gamma_h=0.18;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;Lambda_a=65;beta_6=0.34;gamma_a=0.18;
   mu_a=0.05;alpha_a=0.12;
rho=0.69;omega=0.4;

%Defining function 'Teddy_B33.m' and it's corresponding
   equations as follows
function dy=Teddy_B33(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.5;Lambda_h=60;gamma_h=0.18;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;Lambda_a=65;beta_6=0.34;gamma_a=0.18;
   mu_a=0.05;alpha_a=0.12;
rho=0.69;omega=0.4;

%Defining function 'Teddy_B34.m' and it's corresponding
   equations as follows
function dy=Teddy_B34(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;Lambda_h=60;beta_3=0.7;gamma_h=0.18;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;Lambda_a=65;beta_6=0.34;gamma_a=0.18;
   mu_a=0.05;alpha_a=0.12;
rho=0.69;omega=0.4;

%Defining function 'Teddy_B35.m' and it's corresponding
   equations as follows
function dy=Teddy_B35(~,y)
```

```
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;Lambda_h=60;beta_3=0.9;gamma_h=0.18;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;beta_6=0.34;Lambda_a=65;gamma_a=0.18;
   mu_a=0.05;alpha_a=0.12;
rho=0.69;omega=0.4;

%Defining function 'Teddy_B61.m' and it's corresponding
   equations as follows
function dy=Teddy_B61(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.999;Lambda_h=60;gamma_h=0.18;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;Lambda_a=65;beta_6=0.1;gamma_a=0.18;
   mu_a=0.05;alpha_a=0.12;
rho=0.69;omega=0.4;

%Defining function 'Teddy_B62.m' and it's corresponding
   equations as follows
function dy=Teddy_B62(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;Lambda_h=60;beta_3=0.999;gamma_h=0.18;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;beta_6=0.3;Lambda_a=65;gamma_a=0.18;
   mu_a=0.05;alpha_a=0.12;
rho=0.69;omega=0.4;

%Defining function 'Teddy_B63.m' and it's corresponding
   equations as follows
function dy=Teddy_B63(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.999;Lambda_h=60;gamma_h=0.18;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;beta_6=0.5;Lambda_a=65;gamma_a=0.18;
   mu_a=0.05;alpha_a=0.12;
rho=0.69;omega=0.4;

%Defining function 'Teddy_B64.m' and it's corresponding
   equations as follows
function dy=Teddy_B64(~,y)
```

```
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.999;Lambda_h=60;gamma_h=0.18;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;Lambda_a=65;beta_6=0.7;gamma_a=0.18;
   mu_a=0.05;alpha_a=0.12;
rho=0.69;omega=0.4;

%Defining function 'Teddy_B65.m' and it's corresponding
   equations as follows
function dy=Teddy_B65(~,y)
dy=zeros(size(y));

  %parameter declaration
beta_1=0.35;beta_2=0.55;Lambda_h=60;beta_3=0.999;gamma_h=0.18;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.35;beta_5=0.59;beta_6=0.9;Lambda_a=65;gamma_a=0.18;
   mu_a=0.05;alpha_a=0.12;
  rho=0.69;omega=0.4;

  %Variable description
Sh=y(1);Eh=y(2);Ih=y(3);Sa=y(4);Ea=y(5);Ia=y(6);D=y(7);
Nh=y(1)+y(2)+y(3);
Na=y(4)+y(5)+y(6);

  %Equation of the model
dy(1)=Lambda_h -((beta_1*Ih+beta_2*Ia+beta_3*D)*Sh)/Nh-mu_h*Sh
   ;
dy(2)=((beta_1*Ih+beta_2*Ia+beta_3*D)*Sh)/Nh- (gamma_h+mu_h)*
   Eh;
dy(3)=gamma_h*Eh-(alpha_h+mu_h)*Ih;
dy(4)=Lambda_a-((beta_4*Ih+beta_5*Ia+beta_6*D)*Sa)/Na-mu_a*Sa;
dy(5)=((beta_4*Ih+beta_5*Ia+beta_6*D)*Sa)/Na-(mu_a+gamma_a)*Ea
   ;
dy(6)=gamma_a*Ea-(alpha_a+mu_a)*Ia;
dy(7)=rho*Ia-omega*D;

%RUNNING FILES
clc
clear all
tspan =0:0.1:30 ; %Time in yrs ,
%y0=[200, 30, 90, 8000, 5500, 4500, 200];
y0=[530, 9, 2, 500, 10, 2, 5];
[t1,y1]=ode45(@Teddy_B31,tspan,y0);
[t2,y2]=ode45(@Teddy_B32,tspan,y0);
[t3,y3]=ode45(@Teddy_B33,tspan,y0);
[t4,y4]=ode45(@Teddy_B34,tspan,y0);
```

```
[t5,y5]=ode45(@Teddy_B35,tspan,y0);

figure(7a)
plot(t1,y1(:,1),'g',t2,y2(:,1),'b',t3,y3(:,1),'r',t4,y4(:,1),'
   k',t5,y5(:,1),'m','LineWidth',2)
legend('\beta_3=0.1','\beta_3=0.3','\beta_3=0.5','\beta_3=0.7'
   ,'\beta_3=0.8')
xlabel('Time[years]')
ylabel('Susceptible humans')
hold on

figure(8a)
plot(t1,y1(:,3),'g',t2,y2(:,3),'b',t3,y3(:,3),'r',t4,y4(:,3),'
   k',t5,y5(:,3),'m','LineWidth',2)
legend('\beta_3=0.1','\beta_3=0.3','\beta_3=0.5','\beta_3=0.7'
   ,'\beta_3=0.9')
xlabel('Time[years]')
ylabel('Infected humans')
hold on
hold off

clc
clear all
tspan =0:0.1:30 ; %Time in yrs ,
y0=[530, 9, 2, 500, 10, 2, 5];
[t1,y1]=ode45(@Teddy_B61,tspan,y0);
[t2,y2]=ode45(@Teddy_B62,tspan,y0);
[t3,y3]=ode45(@Teddy_B63,tspan,y0);
[t4,y4]=ode45(@Teddy_B64,tspan,y0);
[t5,y5]=ode45(@Teddy_B65,tspan,y0);

figure(7b)
plot(t1,y1(:,4),'g',t2,y2(:,4),'b--',t3,y3(:,4),'r',t4,y4(:,4)
   ,'k--',t5,y5(:,4),'m','LineWidth',3)
legend('\beta_6=0.1','\beta_6=0.3','\beta_6=0.5','\beta_6=0.7'
   ,'\beta_9=0.9')
xlabel('Time[years]')
ylabel('Susceptible animals')
hold on

figure(8b)
plot(t1,y1(:,6),'g',t2,y2(:,6),'b--',t3,y3(:,6),'r',t4,y4(:,6)
   ,'k--',t5,y5(:,6),'m','LineWidth',3)
legend('\beta_6=0.01','\beta_6=0.3','\beta_6=0.5','\beta_6=0.7
   ','\beta_9=0.9')
xlabel('Time[years]')
ylabel('Infected animals')
hold on
```

```
hold off
```

Appendix B: MATLAB CODES FOR CHAPTER FOUR

B.1 MATLAB codes for Figure 10

```
close all
Lambda_H=36;beta_1=0.35;beta_2=0.55;beta_3=0.999;gamma_H=0.35;
   mu_H=0.01;alpha_H=0.139;
Lambda_a=58;gamma_a=0.34;mu_a=0.05;alpha_a=0.12;omega=0.1;rho
   =0.569;
beta_6=0.134;beta_4=0.25;%;beta_5=0.6

Lambda_h=36;beta_1=0.35;beta_2=0.035;beta_3=0.0999;gamma_h
   =0.35;mu_h=0.01;alpha_h=0.139;
Lambda_a=58;beta_4=0.25;beta_6=0.34;gamma_a=0.34;mu_a=0.05;
   alpha_a=0.12;
rho=0.569;omega=0.45;epsilon=0.75;theta=0;Pi_h=0.000271;tau_a
   =0.22;tau_h=0.58;%beta_5=0.6;

beta_5=0:0.01:5;
beta_5=0:0.01:5;
R_0=((beta_1 * gamma_H)/((gamma_H+mu_H)*(alpha_H+mu_H))+(
   gamma_a*(omega*beta_5+rho*beta_6))/((gamma_a+mu_a)*(alpha_a+
   mu_a)*omega)+sqrt(((gamma_a*(omega*beta_5+rho*beta_6))/((
   gamma_a+mu_a)*(alpha_a+mu_a)*omega)-(beta_1 * gamma_H)/((
   gamma_H+mu_H)*(alpha_H+mu_H))).^2+4*(omega*beta_2+rho*beta_3
   )*(gamma_a*beta_4*gamma_H)/((gamma_a+mu_a)*(alpha_a+mu_a)*(
   gamma_H+mu_H)*(alpha_H+mu_H)*omega))).*2;

R_e=((1-tau_a).*(omega+theta).*beta_5.*gamma_a+(1-epsilon)
   .^2.*beta_6.*rho.*gamma_a)./((gamma_a+mu_a).*(mu_a+alpha_a+
   tau_a).*(omega+theta))+beta_1.*(1-tau_h).*gamma_h./((mu_h+
   gamma_h).*(mu_h+alpha_h+tau_h))+sqrt((((1-tau_a).*(omega+
   theta).*beta_5.*gamma_a+(1-epsilon).^2.*beta_6.*rho.*gamma_a
   )./((gamma_a+mu_a).*(mu_a+alpha_a+tau_a)*(omega+theta))-
   beta_1.*(1-tau_h).*gamma_h./((mu_H+gamma_h).*(mu_h+alpha_h+
   tau_h))).^2+(4.*((1-tau_a).*(omega+theta).*(1-tau_h).*beta_2
   .*beta_4.*gamma_h.*gamma_a+(1-tau_h).*(1-epsilon).^2.*beta_3
   .*beta_4.*gamma_h.*rho.*gamma_a))./((mu_h+gamma_h).*(mu_h+
   alpha_h+tau_h).*(gamma_a+mu_a).*(mu_a+alpha_a+tau_a).*(omega
   +theta))).*2;

plot(beta_5,R_0,'b',beta_5,R_e,'g','lineWidth',2);
grid on
grid minor
ax = gca;
%ax.GridColor = [.5 .5 .5];
ax.GridLineStyle = '--';
```

```
ax.GridAlpha = 0.5;
xlabel('Years')
ylabel('Reproduction Numbers')
legend('Basic Reproduction Number R_0','Effective Reproduction
    Number R_e')
```

B.2 MATLAB codes for Figure 11, 12 and 15

```
%Defining function 'Co_Teddy_E1.m' and it's corresponding
   equations as follows
function dy=Co_Teddy_E1(~,y)
dy=zeros(size(y));

   %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.999;Lambda_h=60;gamma_h=0.35;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.25;beta_5=0.6;Lambda_a=65;beta_6=0.34;gamma_a=0.34;
   mu_a=0.05;alpha_a=0.12;
rho=0.569;omega=0.459;epsilon=0.1;tau_a=0.79;theta=0;tau_h
   =0.58;Pi_h=0.01;Pi_a_a=0.03;

%Defining function 'Co_Teddy_E2.m' and it's corresponding
   equations as follows
function dy=Co_Teddy_E2(~,y)
dy=zeros(size(y));

   %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.999;Lambda_h=60;gamma_h=0.35;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.25;beta_5=0.6;Lambda_a=65;beta_6=0.34;gamma_a=0.34;
   mu_a=0.05;alpha_a=0.12;
rho=0.569;omega=0.459;epsilon=0.3;tau_a=0.79;theta=0;tau_h
   =0.58;Pi_h=0.01;Pi_a_a=0.03;

%Defining function 'Co_Teddy_E3.m' and it's corresponding
   equations as follows
function dy=Co_Teddy_E3(~,y)
dy=zeros(size(y));

   %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.999;Lambda_h=65;gamma_h=0.35;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.25;beta_5=0.6;Lambda_a=65;beta_6=0.34;gamma_a=0.34;
   mu_a=0.05;alpha_a=0.12;
rho=0.569;omega=0.459;epsilon=0.5;tau_a=0.79;theta=0;tau_h
   =0.58;Pi_h=0.01;Pi_a_a=0.03;

%Defining function 'Co_Teddy_E4.m' and it's corresponding
```

```
   equations as follows
function dy=Co_Teddy_E4(~,y)
dy=zeros(size(y));

   %parameter declaration
beta_1=0.35;beta_2=0.55;beta_3=0.999;Lambda_h=60;gamma_h=0.35;
   mu_h=0.01;alpha_h=0.139;
beta_4=0.25;beta_5=0.6;Lambda_a=65;beta_6=0.34;gamma_a=0.34;
   mu_a=0.05;alpha_a=0.12;
rho=0.569;omega=0.459;epsilon=0.7;tau_a=0.79;theta=0;tau_h
   =0.58;Pi_h=0.01;Pi_a_a=0.03;

%Defining function 'Teddy_ENOCO.m' and it's corresponding
   equations as follows
function dy=Teddy_ENOCO(~,y)
dy=zeros(size(y));

  %parameter declaration
Lambda_H=36;beta_1=0.35;beta_2=0.55;beta_3=0.999;gamma_H=0.35;
   mu_H=0.01;alpha_H=0.139;
Lambda_a=58;beta_4=0.25;beta_5=0.6;beta_6=0.34;gamma_a=0.34;
   mu_a=0.05;alpha_a=0.12;
  rho=0.569;omega=0.45;

  %Variable description
Sh=y(1);Eh=y(2);Ih=y(3);Sa=y(4);Ea=y(5);Ia=y(6);D=y(7);
Nh=y(1)+y(2)+y(3);
Na=y(4)+y(5)+y(6);

  %Equation of the model
dy(1)=Lambda_h+Pi_h*Ih -((beta_1*(1-tau_h)*Ih+beta_2*(1-tau_a)
   *Ia+beta_3*(1-epsilon)*D)*Sh)/Nh-mu_h*Sh;
dy(2)=((beta_1*(1-tau_h)*Ih+beta_2*(1-tau_a)*Ia+beta_3*(1-
   epsilon)*D)*Sh)/Nh- (gamma_h+mu_h)*Eh;
dy(3)=gamma_h*Eh-(alpha_h+mu_h+tau_h)*Ih;
dy(4)=Lambda_a+Pi_a_a*Ia-((beta_4*(1-tau_h)*Ih+beta_5*(1-tau_a
   )*Ia+beta_6*(1-epsilon)*D)*Sa)/Na-mu_a*Sa;
dy(5)=((beta_4*(1-tau_h)*Ih+beta_5*(1-tau_a)*Ia+beta_6*(1-
   epsilon)*D)*Sa)/Na-(mu_a+gamma_a)*Ea;
dy(6)=gamma_a*Ea-(tau_a+alpha_a+mu_a)*Ia;
dy(7)=rho*(1-epsilon)*Ia-(omega+theta)*D;

%RUNNING FILE
tspan =0:0.1:30 ; %Time in yrs ,
y0=[501, 9, 2, 400, 10, 2, 25];
[t,y]=ode45(@Ma_Teddy_2ENOCO,tspan,y0);
[t1,y1]=ode45(@Co_Teddy_E1,tspan,y0);
[t2,y2]=ode45(@Co_Teddy_E2,tspan,y0);
```

```
[t3,y3]=ode45(@Co_Teddy_E3,tspan,y0);
[t4,y4]=ode45(@Co_Teddy_E4,tspan,y0);
[t5,y5]=ode45(@Co_Teddy_E5,tspan,y0);

figure(11a)
plot(t,y(:,1),'b', t1,y1(:,1),'k',t2,y2(:,1),'r',t3,y3(:,1),'g
   ','LineWidth',2)
%ylim([0 1000])
legend('No Control','\epsilon=0.1','\epsilon=0.3','\epsilon
   =0.5')
xlabel('Time[years]')
ylabel('Susceptible humans')
hold on
hold off

figure(11b)
plot(t,y(:,4),'b',t1,y1(:,4),'g',t3,y3(:,4),'r--',t5,y5(:,4),'
   k','LineWidth',2)
legend('No Control','\epsilon=0.1','\epsilon=0.3','\epsilon
   =0.9')
xlabel('Time[years]')
ylabel('Susceptible animals')
hold on

figure(12a)
plot(t1,y1(:,3),'b',t2,y2(:,3),'r',t3,y3(:,3),'k','LineWidth'
   ,2)
%ylim([0 1000])
legend('\epsilon=0.1','\epsilon=0.3','\epsilon=0.5')
xlabel('Time[years]')
ylabel('Infected humans')
hold on
hold off

figure(12b)
plot(t1,y1(:,6),'g',t2,y2(:,6),'r',t3,y3(:,6),'k','LineWidth'
   ,2)
%ylim([0 1000])
legend('\epsilon=0.1','\epsilon=0.3','\epsilon=0.5')
xlabel('Time[years]')
ylabel('Infected animals')
hold on
hold off

figure(15a)
plot(t1,y1(:,7),'m',t2,y2(:,7),'r',t3,y3(:,7),'g','LineWidth'
   ,2)
%ylim([0 1000])
```

```
107 legend('\epsilon=0.1','\epsilon=0.3','\epsilon=0.5')
108 xlabel('Time[years]')
109 ylabel('Dairy products')
110 hold on
111 hold off
```