MODELLING THE TRANSMISSION DYNAMICS AND CONTROL OF TAENIASIS AND CYSTICERCOSIS IN HUMANS, PIGS AND CATTLE

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A Dissertation Submitted in Partial Fulfilment of the Requirements for the Degree of Doctor of Philosophy in Mathematical and Computer Sciences and Engineering of the Nelson Mandela African Institution of Science and Technology

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ABSTRACT

Taeniasis and cysticercosis are neglected food-borne diseases that pose challenges to food safety, human health and livelihood of rural livestock farmers. Cysticercosis reduces the market value for pigs and cattle by making pork and beef unsafe for consumption. In this research, deterministic and continuous time Markov chain (CTMC) models are formulated and analyzed to study the transmission dynamics of taeniasis and cysticercosis in humans, pigs and cattle. To study the dynamics of the diseases, we derived the basic reproduction numbers $\mathscr{R}_{01}$ for *T. saginata* and R_0 for *T. saginata* and *T. solium* parasitic infections by next generation matrix method. Global stability for models' equilibria are established by Lyapunov functions, Metzler matrix approach and cooperative systems theory whereas the normalized forward sensitivity index is employed to determine the sensitive parameters. The multitype branching process is adopted to compute the probability of diseases' extinction or outbreak. The optimal control and cost-effectiveness analyses are used to determine the disease's optimal strategy and most cost-effective strategy for disease's control respectively. Analysis shows that both the disease free and endemic equilibria exist. The disease free equilibria are globally asymptotically stable when $\mathscr{R}_{01} < 1$ and $R_0 < 1$ whereas the endemic equilibria are globally asymptotically stable when $\mathscr{R}_{01} > 1$ and $R_0 > 1$. Sensitivity analysis for *T. saginata* bovine cysticercosis and human taeniasis model shows that *T. saginata* eggs to cattle transmission coefficient, human and cattle recruitment rates, the probability of humans to contract taeniasis, the open defecation rate by humans with taeniasis and the rate of eating raw or undercooked infected beef are the most positive sensitive parameters whereas the natural mortality rates for humans, cattle and *T. saginata* eggs, and the proportion of unconsumed infected beef are the most negative sensitive parameters. Sensitivity results for taeniasis and cysticercosis transmission dynamics model due to *T. solium* and *T. saginata* tapeworms in humans, pigs and cattle show that human's recruitment, the probability of humans to acquire taeniasis and the open defecation rate by humans with taeniasis are the most positive sensitive parameters whereas the *T. saginata* natural mortality rate is the most negative sensitive parameter. CTMC model results for *T. saginata* bovine cysticercosis and human taeniasis show that the probability of diseases' extinction is high when the diseases emerge from a small number of *T. saginata* eggs or from infected cattle and there is major diseases' outbreak when the diseases emerge from infectious beef. On the other hand, CTMC model results for taeniasis and cysticercosis transmission dynamics due to *T. solium* and *T. saginata* tapeworms in humans, pigs and cattle indicate that the probability of disease's extinction is high when the diseases emerge from humans with cysticercosis or a small number of taenia eggs in the environment. However, there is a major outbreak when the diseases emerge from humans with taeniasis or from infectious pork and beef. Basing on sensitivity analysis, various control strategies were designed. The *T. saginata* optimal control results indicate that

a strategy which targets improving meat cooking and treatment of infected humans is the optimal strategy. The *T. solium* and *T. saginata* model results show that a strategy which combines meat inspection, treatment of humans with taeniasis and improving hygiene and sanitation is the most effective strategy for diseases' control. Thus, to control the diseases at minimal cost, we recommend that more efforts be directed to treat humans with taeniasis and improve meat inspection, meat cooking, and hygiene and sanitation.

DECLARATION

I, **Joshua Angolwisye Mwasunda** 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 being concurrently submitted for degree award in any other institution.

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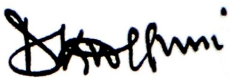
Dr. Damian Kajunguri  01.08.2022

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CERTIFICATION

The undersigned certify that they have read and found the dissertation acceptable by the Nelson Mandela African Institution of Science and Technology.

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DEDICATION

This work is dedicated to my entire family, especially to my parents: Pastor Angolwisye Mwasunda and Seliva Sichembe, my lovely wife Yolanda Mlaki, my son Gaddiel and my daughters Jackline, Jocelyn and Gaynelle for their love, patience, encouragement, prayers as well as moral and spiritual support.

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ABBREVIATIONS

WHO	World Health Organization
USA	United State of America
CT	Computed Tomography
MRI	Magnetic Resonance Imaging
CNS	Central Nervous System
URT	United Republic of Tanzania
NTDs	Neglected Tropical Diseases
FAO	Food and Agriculture Organization
COSTECH	Commission for Science and Technology
NIMR	National Institute of Medical Research
EE	Endemic Equilibrium
DFE	Disease Free Equilibrium
MLE	Maximum Likelihood Estimation
ICER	Incremental Cost Effectiveness Ratio
IAR	Infection Averted Ratio
CTMC	Continuous Time Markov Chain
ANOVA	Analysis of Variance
ODE	Ordinary Differential Equations
RK4	Runge Kutta Order 4

CHAPTER ONE

INTRODUCTION

1.1 Background of the Problem

Taeniasis refers to the dwelling of adult tapeworms in the human's small intestine while cysticercosis is the tissue infection by larval form of taenia tapeworms due to ingestion of raw or inadequately cooked meat (Symeonidou *et al.*, 2018). Taeniasis and cysticercosis are parasitic food-borne diseases that pose challenges to food safety, affecting human health and livelihood of rural livestock farmers. Cysticercosis affects pigs' and cattle's market value by making pork and beef unsafe for consumption (WHO, 2005; Winskill *et al.*, 2017). *Taenia solium*, *Taenia saginata* and *Taenia asiatica* are tapeworm species which cause taeniasis and cysticercosis (WHO, 2021). *Taenia asiatica* is dominant in Asia while other tapeworm species are globally distributed (WHO, 2005). *Taenia solium* and *Taenia saginata* tapeworm species cause human taeniasis and cysticercosis in cattle and pigs. *Taenia solium* also causes cysticercosis in human.

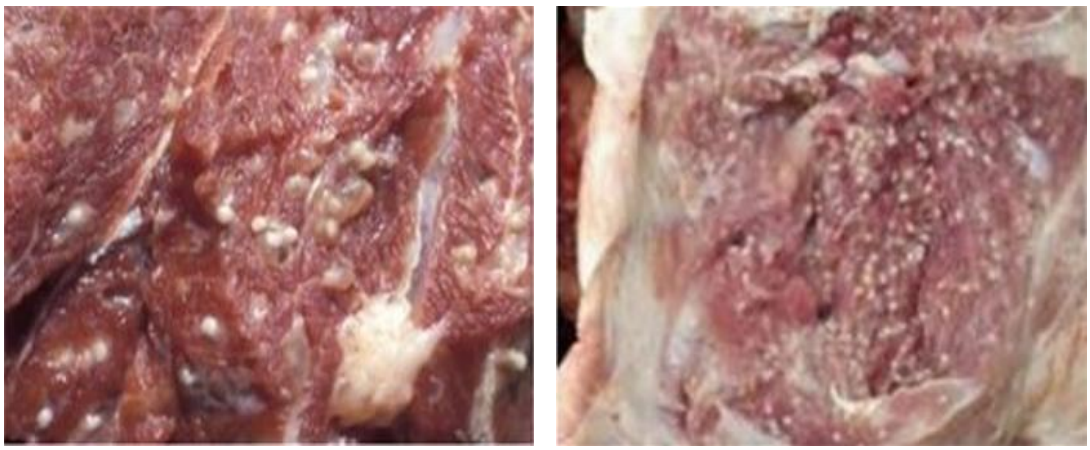
In transmission of taeniasis and cysticercosis, humans are the mandatory hosts of the adult stage tapeworms whereas pigs and cattle are the intermediate hosts for the larval stage parasites (Schantz *et al.*, 1993). Taeniasis and cysticercosis are among the major parasitic food-borne diseases that have been neglected for so long in many developing countries of Latin America, Africa and Asia (WHO, 2021; Ngowi *et al.*, 2019). They are common in rural areas that are characterized by poor sanitation, open defecation by humans with taeniasis, pigs' and cattle's free range farming system, low standard of slaughter facilities, inadequate or no meat inspection, poverty and lack of treatment for the diseases (Mkupasi *et al.*, 2011; Alemneh *et al.*, 2017; Flisser *et al.*, 2006; WHO, 2005). The diseases mainly affect pastoral and poor rural communities that depend on pork and beef as sources of food (WHO, 2021; Trevisan *et al.*, 2017).

The recurrence of taeniasis and cysticercosis in non-endemic area such as Australia, Canada, USA and Europe is due to the growing popularity of pork consumption, travel, commerce and immigrant workforce (Symeonidou *et al.*, 2018). For instance, in Western Europe, a total of 751 infected individuals were reported from 1985-2011 (Symeonidou *et al.*, 2018).

1.1.1 Modes of Transmission

Humans acquire intestinal tapeworm infection (taeniasis) when they eat raw or undercooked beef or pork infected with tapeworm larval cysts, which develop into adult tapeworms that live in the small intestine (WHO, 2021). Figure 1 shows beef and pork that contain larval cysts. The

tapeworm eggs are passed out with faeces when humans with taeniasis defecate in the fields and spread over the environment through winds, water, insects and animals feet thus contaminating pastures, folder, soil and water sources (Dermauw *et al.*, 2018).



(a) Beef **(b) Pork**

Figure 1: Larval cysts in beef and pork

Cattle acquire cysticercosis when they consume *Taenia saginata* eggs from the contaminated environment while pigs acquire cysticercosis through direct consumption of human feaces or indirectly when they ingest *Taenia solium* eggs from the contaminated environment (Symeonidou *et al.*, 2018). Figure 2 shows how humans, cattle and pigs can acquire cysticercosis from the contaminated environment. When taenia eggs are consumed by cattle and pigs, they hatch, penetrate the intestinal wall and reach the blood circulation system, where they spread throughout the body tissues and organs such as heart, diaphragm, kidney, lungs, liver and tongue, and develop into cysticerci (Ito *et al.*, 2004; WHO, 2005).

Taenia saginata tapeworm causes few symptoms in both humans and cattle. In humans symptoms include anal pruritus, mild abdominal pain, distress and some people suffer from a pathological fear of tapeworms (Dermauw *et al.*, 2018; Tembo & Craig, 2015). In cattle, the infection is asymptomatic but may cause greater losses in economy for the meat industry due to cost of meat inspection, carcass condemnation and treatment upon detection of larval cysts (Jansen *et al.*, 2018; Alemneh *et al.*, 2017; Grindle, 1978).

Figure 2: Transmission of infection from the contaminated environment (WHO, 2021)

Humans contract cysticercosis when they consume *Taenia solium* eggs from the contaminated environment via contaminated water, fruits and vegetables, or by putting contaminated fingers in the mouth (Del Brutto, 2013). Once the tapeworm eggs are ingested, they hatch in the small intestine, and develop into larvae that penetrate the intestinal wall and migrate to various parts of the body such as eyes, muscles, skin and the central nervous system through blood circulatory system, where they form cysts. Figure 3 shows transmission of taeniasis and cysticercosis in humans, pigs and cattle.

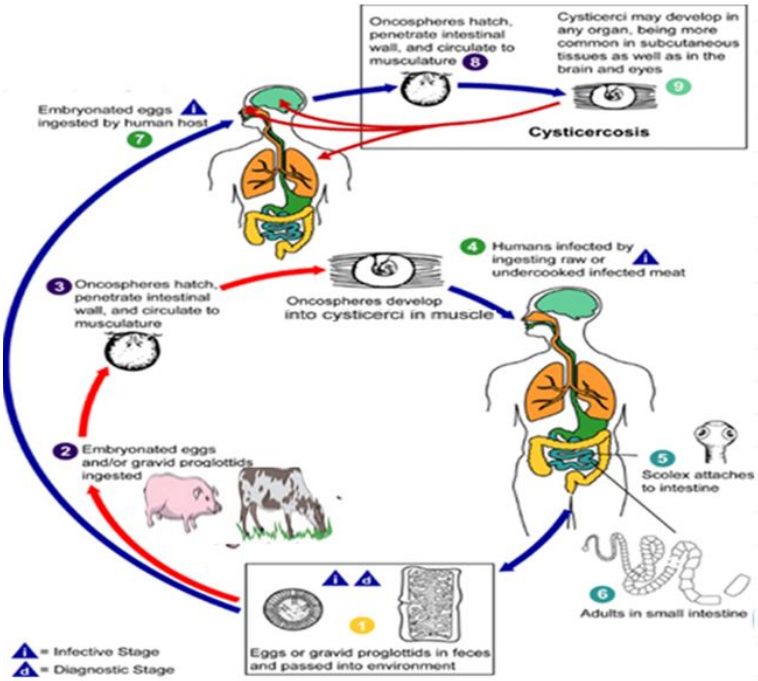


Figure 3: Taeniasis and cysticercosis transmission cycle (Ndimubanzi *et al.*, 2010)

In humans, cysticercosis is responsible for different clinical manifestations that can be very severe and even cause death. Symptoms of the disease include severe headache, blindness, convulsions and epileptic seizure (Sorvillo *et al.*, 2007). When the cysts reach and infect the brain, they cause neurocysticercosis which is the most severe form of tapeworm infection of the central nervous system and it is the major cause of epilepsy worldwide (WHO, 2005, 2021). In countries where human cysticercosis is endemic, it is responsible for about 30% of all epileptic cases. Gobally, about 50 million people suffer from epilepsy and 80% of them are from developing countries (WHO, 2021).

1.1.2 Diagnosis

The diagnosis of human taeniasis is done by examination of stool samples that are collected and examined in the laboratory using a microscope to observe the existence of proglottids or eggs in the patient's stool, while the diagnosis of human cysticercosis involve asking a patient about the symptoms, doing a biopsy of subcutaneous cysts, immunodiagnosis, radiography, using computed tomography (CT) scan and magnetic resonance imaging (MRI). To diagnosis neurocysticercosis it requires both central nervous system (CNS) imaging with CT brain scans or MRI and serological tests. Diagnosis of cysticercosis in pigs is done through tongue inspection, meat inspection and serological tests while diagnosis of cysticercosis in cattle is through meat inspection and serological tests (WHO, 2005, 2021).

1.1.3 Treatment

The treatment of human taeniasis is done after accurate diagnosis, which involves administration of prescribed medication of praziquantel, niclosamide, nitazoxanide or albendazole which is taken orally. The treatment of human cysticercosis is done by taking doses of albendazole and plaziquantel depending on the number and stage of cysticerci, location and symptoms observed by the physician. However, the treatment of neurocysticercosis is more difficult with varying success and it may involve prolonged doses of albendazole and (or) praziquantel with supporting therapy such as anti-epileptic drugs, corticosteroids, and surgery for some cases (WHO, 2005, 2021).

1.1.4 Prevention and Control

To prevent and control taeniasis and cysticercosis in humans, pigs and cattle, various interventions are required. These include treatment of infected humans, vaccination and (or) treatment of infected pigs and cattle, improving standards of meat inspection, proper handling of pork or beef at both household and community level, improving pork and beef cooking, good sanitary and hygiene conditions, community health education on hygiene and food safety, and improv-

ing animal husbandry. Vaccines for pigs include S3Pvac and TSOL18 whereas cattle vaccines are TSA-18 and TSA-9. Anthelmintic pigs' and cattle's treatment involves the use of drugs such as flubendazole, fenbendazole, oxfendazole, praziquantel and nitazoxanide (Lightowlers, 2010; Lightowlers *et al.*, 1996; Mkupasi *et al.*, 2013).

1.2 Statement of the Problem

Taeniasis and cysticercosis are serious public health problems that have been neglected for too long, especially in developing countries (Winskill *et al.*, 2017). The diseases threaten human and animal health, and the livelihood of rural subsistence farming communities. Cysticercosis reduces the market value for pigs and cattle, making pork and beef unsafe for consumption. For example, the value loss due to bovine cysticercosis in Belgium was estimated to be 696 631 pounds per year and other costs related to meat inspection, destruction of carcasses and insurance were estimated to reach 597 856, 2107 and 4 250 985 pounds per year respectively (Jansen *et al.*, 2018). In the same country, the estimated annual value losses to the animal owner, slaughter houses and insurance companies were approximately 453 024, 34 848 and 209 088 pounds respectively (Jansen *et al.*, 2018). In 2012, Tanzania had 17 853 human cysticercosis cases, 212 epilepsy induced deaths and 183 927 (11.7% of all pig population) porcine cysticercosis cases with cysticercosis induced economic burden of US$ 7.9 million (Trevisan *et al.*, 2017).

Various studies have reported that cysticercosis is a serious public health problem in many regions of Tanzania. For example, the studies have shown that during the survey 26 out of 282 pigs were infected with *T. solium* parasite in Mbeya rural and Mbozi districts (Kabululu *et al.*, 2020); 110 out 330 pigs were infected in Nyasa district (Shonyela *et al.*, 2017), 23 out 308 pigs were infected with *T. Solium* whereas 155 out of 4020 people were infected with *T. solium* parasite in Iringa rural district (Yohana *et al.*, 2013). Generally, taeniasis and cysticercoisi are endemic in Northern, Central and Southern regions of Tanzania particularly in rural areas where people depend on pig and cattle farming to sustain their life. The districts that are more affected include Nyasa, Mbinga, Mbozi, Mbeya Rural, Mbeya City, Chunya, Mbulu, Babati, Kongwa, Morogoro, Iringa, Moshi and Arusha (Ngowi *et al.*, 2019). Taeniasis and cysticercosis have high prevalence rates in Latin America, Africa and Asia (Dermauw et al., 2018).

Tanzania is the second country in Africa with the largest livestock population after Ethiopia (URT, 2016; Michael *et al.*, 2018). Tanzania's human population has grown rapidly from 12.3 million in 1967 to 43.6 million in 2012 and it is projected to reach 70.1 million in 2025 (Agwanda & Amani, 2014). The study by Wilson (2015) has shown that the demand for meat has increased particularly in urban areas whereby meat consumed is mainly produced by rural small scale farmers who keep pigs and cattle under free range system. For example, the meat demand

reached 160 000 tonnes in 2010 and is projected to be 500 000 tonnes in 2030 thus exceeding the demand for other African countries. Thus, owing to the rate at which taeniasis and cyticercosis spread and human population growth, urgent measures are needed to control the spread of these diseases.

In 2010, the World Health Organization (WHO) listed taeniasis and cysticercosis as major NTDs that need an effective strategy to be implemented to control and eliminate taeniasis and cysticercosis in selected countries by 2020 (WHO & FAO, 2014). Cysticercosis has been identified by WHO and the Food and Agriculture Organization (FAO) as the leading cause of all deaths due to foodborne diseases (WHO & FAO, 2014). Globally, the disease affects approximately 50 million people and nearly 50,000 people die annually due to cysticercosis (Aung & Spelman, 2016). Neurocysticercosis cases are estimated to be between 2.56 and 8.30 million worldwide (WHO, 2021). In Africa, taeniasis and cysticercosis are widely distributed with high prevalence rates in Central and Eastern African countries, including Tanzania. Human and porcine cysticercosis is highly endemic in Tanzania with porcine cysticercosis prevalence rates 0.3-17.4% and 5.5-16.9% in Southern and Northern parts of the country respectively (Kavishe *et al.*, 2017; Mwanjali *et al.*, 2013). Taeniasis and cysticercosis are among of the Tanzania's important health research priorities which were added to the list of the country's health research priorities in 2013–2018 and 2015–2020 by Tanzania Commission for Science and Technology (COSTECH) and the National Institute for Medical Research (NIMR) (Ngowi *et al.*, 2018).

Mathematical modelling has become an important tool in analyzing the transmission dynamics and control of infectious diseases. Currently, there are only few statistical and deterministic mathematical models that have been formulated and analyzed to study the dynamics and control of taeniasis and cysticercosis in humans and pigs (Gonzalez *et al.*, 2002; Kyvsgaard *et al.*, 2007; Braae *et al.*, 2016; Winskill *et al.*, 2017; José *et al.*, 2018; Sánchez-Torres *et al.*, 2019). None of these studies have considered cattle population in studying the dynamics and control of taeniasis and cysticercosis. Also, previous studies have not considered the role of infected pork and beef in the spread of taeniasis and cysticercosis. Additionally, previous studies have not performed optimal control and cost effective analysis for the control of taeniasis and cysticercosis. Moreover, most of the previous mathematical studies have used deterministic modelling approach which do not address some uncertainties that might happen such as the possibility of an infective to die, recover or being identified before it causes infection and thus the possibility of diseases extinction or outbreak. The mathematical models which can address such uncertainties are continuous time Markov chain (CTMC) stochastic models. In this study, we formulate and analyze both deterministic and CTMC stochastic models by incorporating cattle population, and infectious pork and beef on the dynamics of taeniasis and cysticercosis in humans, pigs and cattle, and suggest the optimal diseases' control measures.

1.3 Research Objectives

1.3.1 Main Objective

The main objective of the study is to develop and analyze mathematical models for the transmission dynamics and control of taeniasis and cysticercosis in humans, pigs and cattle.

1.3.2 Specific Objectives

The specific objectives of the study are:

(i) To formulate and analyze deterministic and continuous time Markov chain stochastic models for taeniasis and cysticercosis transmission dynamics.

(ii) To determine parameters that drive taeniasis and cysticercosis.

(iii) To compute the probability of taeniasis and cysticercosis extinction or outbreak.

(iv) To determine the optimal control strategy for the control of taeniasis and cysticercosis in humans, pigs and cattle.

(v) To determine the most cost-effectiveness strategy for diseases' control.

1.4 Research Questions

The following fundamental questions guide our study:

(i) How can deterministic and continuous time Markov chain stochastic models for the dynamics and control of taeniasis and cysticercosis be formulated and analyzed?

(ii) How can parameters that drive taeniasis and cysticercosis be determined?

(iii) How can the probability of extinction or outbreak of taeniasis and cysticercosis in humans, pigs and cattle be determined?

(iv) Are there any optimal control options that can be considered to minimize the transmission of taeniasis and cysticercosis in humans, pigs and cattle?

(v) What is the most cost-effective optimal control strategy for the control of taeniasis and cysticercosis?

1.5 Rationale of the Study

Most of rural farmers earn their incomes through livestock keeping. Apart from that, also rural farmers keep livestock as their sources of food and for the sake of draft power and obtaining manure for their farms (Swanepoel *et al.*, 2010). However, the animals they keep are faced by parasitic diseases such as bovine and porcine cysticercosis that affect cattle and pigs respectively. Porcine cysticercosis and bovine cysticercosis affect human health through consumption of raw or under-cooked beef or pork infected with larval cysts and ingestion of T. solium eggs that are shed by humans with taeniasis in the open fields. Thus, understanding how these disease are transmitted is very essential for designing appropraite measures to curb the spread of these diseases. Mathematical modelling is one of the important tools which helps in understanding and analyzing the transmission dynamics of these diseases for designing appropriate control measures.

1.6 Significance of Study

The knowledge obtained from this study will be important to individuals, policy makers, researchers, government, educators and various organizations in the following ways:

(i) The study will give insight to people in understanding the parameters that drive taeniasis and cysticercosis among human, pig and cattle populations, which in turn will protect them from being infected by taking appropriate disease's control measures.

(ii) Through optimal control and cost effectivenss analyses, the study will provide knowledge to the government and policy makers that will help them to establish policies and design effective programmes to prevent and control the spread of these diseases at minimal costs.

(iii) The knowledge gained from this study will help livestock farmers to take appropriate measures to protect their animals from being infected and therefore improve their economic wellbeing through promoting health of their animals which in turn will lead into food security.

(iv) The study will help educators to raise awareness among people on the transmission and control of taeniasis and cysticercosis through training programmes, workshops, information campaigns, media, workshops and educational seminars.

(v) To researchers, the study will add up the knowledge to the existing literature and provide a platform for further research on taeniasis and cycticercosis.

1.7 Delineation of Study

This study aims at formulating and analyzing mathematical models for the dynamics and control of taeniasis in humans, pigs and cattle. The research work is divided into five chapters. Chapter One is devoted to general introduction of the research topic, describing in detail how taeniasis and cysticercosis are transmitted in humans, pigs and cattle. In Chapter Two deals with the literature review on mathematical and non-mathematical studies for the transmission dynamics and control of taeniasis and cysticercosis. In Chapter Three, both deterministic and continuous time Markov chain models for the dynamics of taeniasis and cysticercosis in humans, pigs and cattle have been formulated and analyzed. Furthermore, the optimal control and cost-effectiveness analyses has been carried out. Chapter Four deals with numerical simulation of the previously established mathematical models whereby the results are presented graphically and in tabular form, followed by their detail discussions. Chapter Five provides conclusion remarks and recommendations following the results obtained in Chapters Three and Four.

However, our study is limited to the dynamics of taeniasis and cysticercosis in areas where pigs and cattle are kept under free range system and where there is no migration of humans, pigs and cattle. Due to lack of taeniasis and cysticercosis data in humans, pigs and cattle from the Ministries of Health, Livestock and Fisheries, some parameter values have been assumed and other have been obtained from literature. For the case of *T. saginata* parasitic infection, we have used human infection cases in Debre Birhan City of Ethiopia (2013-2017) to fit model parameter values. To provide more insights on the disease dynamics and assess the impact of various control strategies, this study can be extended by studying the role of migration of susceptible humans, pigs and cattle in the dynamics and control of taeniasis and cysticercosis; modelling humans with cysticercosis and neurocysticercosis as two different entities; studying the role of seasonal variation on the dynamics of taeniasis and cyticercosis in humans, pigs and cattle; use of delay differential equations to address the influence of latent period on diseases' dynamics; implementation of fractional order differential equations in studying the dynamics of the diseases and applying discrete time Markov chain and stochastic differential equations epidemic models to study the dynamics of these diseases.

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction

Mathematical models play a significant role in understanding the transmission dynamics and control of infectious diseases and they are useful tools in decision making process. They are also helpful on the choice of the best strategy to be considered for disease control, the optimal allocation of the available scarce resources, or the best combination of strategies that can be used to control the spread of the disease. The optimal control theory together with cost effective analysis are very crucial in analyzing and choosing effective strategies to control the disease in a given population at a minimal cost. There are various mathematical models that have been formulated and analyzed to study the transmission dynamics and control of infectious food-borne diseases. In this chapter, we review some key mathematical models for transmission dynamics of taeniasis and cysticercosis in humans and pigs, continuous time Markov chain stochastic models for some infectious diseases, mathematical models for optimal control and cost-effectiveness analyses of environmentally transmitted diseases and non-mathematical studies on taeniasis and cysticercosis.

2.2 Mathematical Models for Taenia Solium Taeniasis and Cysticercosis

Gonzalez *et al.* (2002) developed and analyzed a dynamic–stochastic model to assess the control of porcine cysticercosis. The developed model was simulated to study the dynamics of pig population, the infection in both human and pig populations, the impact of various control strategies on *Taenia solium* parasite and the financial benefit of various strategies on the control of the disease. In model simulations, they evaluated a number of control strategies with various combinations and durations of human and porcine treatment. The simulation results indicated that the treatment of both infected humans and pigs are more effective for the control of the diseases. However, the study did not capture the dyamics of taeniasis and cysticercosis due to both *Taenia solium* and *Taenia saginata* tapeworms and did not consider the continuous time Markov chain stochastic model to study the diseases' dynamics. In addition, the study did not perform the optimal control and cost effectiveness analysis to determine the most effective strategy in diseases' control.

Kyvsgaard *et al.* (2007) formulated and analyzed a SIR deterministic and stochastic version of Reed-Frost (a chain binomial) model to simulate the dynamics and control of *Taenia solium* tapeworm parasite. In the model, the transmission of infections was modeled as a function

of probability of disease transmission which depend on each contact made between susceptible and infected populations during a specific time increment. The disease transmission from humans to pigs was considered as susceptible pigs feeding on human feaces infected with tapeworm eggs while human infection was modeled as susceptible humans feeding on undercooked pork infected with tapeworm larval cysts. In assessing the possibility of eliminating the infections in small populations, they first simulated the deterministic model followed by stochastic version of the model for each case. Three interventions that were considered in the model are cooking habits, meat inspection and the use of latrines; rapid detection, human treatment and pigs vaccination; and treatment of either pig or human populations. The results showed that mass-treatment is effective in disease control and can lead into reduction of taeniasis and cysticercosis in short time. However, the study did not consider the continuous time Markov chain stochastic model which predicts the likelihood of diseases' extinction or outbreak depending on initial number of infectives at the beginning of the diseases' outbreak. Also, they did not consider the dynamics of taeniasis and cysticercosis due *T. saginata* tapeworms and did not implement optimal control and cost effectiveness analyses.

Braae *et al.* (2016) developed and analyzed a cystiSim agent-based (statistical) model to study the dynamics and control of *Taenia solium* tapeworms. They used the field data from Tanzania to develop a model in statistical R programme. Three interventions: pig vaccination, pig treatment, and human treatment were implemented singularly or in combination to assess the effect of each strategy on disease control and they allowed customary coverage and settings. The results indicated that all interventions targeting on pig population were effective provided that the coverage and efficacy was sufficiently high. Small coverage interventions on pig populations were effective if additional human host interventions were implemented. Generally, cystiSim agent based model revealed that the combination of interventions in both human and pig hosts are more effective for disease control in humans and pigs if the coverage of 75% is maintained over at least for the duration of four years. However, the model was statistical focusing only on human and pig populations to study disease control measures. The study failed to address the role of infectious beef in addressing the dynamics and control of taeniasis and cysticercosis. The study also has not considered the continuous time Markov chain stochastic model which is more realistic and helps to determine the probability of disease extinction or outbreak. In addition, the study has not applied the optimal control and cost effectiveness analyses to decide on the most effective disease control strategy.

Winskill *et al.* (2017) formulated and analyzed a deterministic mathematical model to study the impacts of various strategies on the control of *Taenia solium* taeniasis and cysticercosis in humans and pigs by using the EPICYST transmission model. The pig population was divided into susceptible, vaccinated, pigs harboring low cyst burdens, pigs harboring high cyst burdens and recovered pigs while human population was divided into susceptible, humans with taeniasis, humans with cysticercosis and humans with both taeniasis and cysticercosis. Intervention strategies that were implemented singly or in combination include pig vaccination, treatment of infected pigs, improved animal husbandry, improved sanitation, improved meat inspection and treatment of humans with taeniasis. The results showed that treatment interventions which focus on human or pig population are more effective in disease control when used singly, with annual treatment of pigs and humans. However, the study ignored the role of pork and beef infected with tapeworm larval cysts in the dynamics of taeniasis and cysticercosis in humans, pigs and cattle. Also, the study did not consider the continuous time Markov chain stochastic model which captures the uncertainties that are inherent to disease transmission and helps in predicting the likelihood of disease extinction or outbreak. Furthermore, the study did not implement the optimal control and cost effectiveness analysis which are essential to decide the most effective strategy for disease control.

José *et al.* (2018) formulated and analyzed a density dependent mathematical model for the transmission dynamics of taeniasis and cysticercosis with chemotherapy in humans and pigs based on the life cycle of *Taenia solium* tapeworm. The formulated model comprised deterministic equations with stochastic elements that describe changes in the mean parasite burden and incorporates the overall pattern of the parasites' distribution. The results indicated that chemotherapeutic interventions that focus on infected pigs or humans with taeniasis are effective in reducing the mean intensity of human taeniasis, porcine cysticercosis and human cysticercosis. The treatment of humans with cysticercosis alone has no impact on the control of human teniasis and porcine cysticercosis since human cysticercosis has no influence on value of the basic reproductive number. However, the study did not consider taeniasis and cysticercosis due to consumption of infected beef and did not consider the continuous time Markov chain stochastic model to study the disease dynamics. In addition, the study did not implement the optimal control and cost effectiveness analysis to decide on the effective strategy which can be implemented to control taeniasis and cysticercosis at minimal cost.

Sánchez-Torres *et al.* (2019) developed and analyzed a deterministic mathematical model to assess the dynamics and control of taeniasis and cysticercosis in humans and pigs based on the life cycle of *Taenia solium* tapeworm. The developed model was an extension of the SI model in José *et al.* (2018). Two control strategies that were considered in model simulations are vaccination of pigs and treatment of humans with taeniasis which were implemented concurrently,

successively or in any order. Numerical simulations were done to assess the impact of vaccination of pigs using different schedules, and the combined human treatment and pig vaccination strategy. The results showed that vaccination of pigs has influence on the transmission dynamics among the vaccinated population and other hosts. For the case when the coverage rate and/or the protective efficacy is below 100%, then a combination of vaccinating pigs twice with treatment of humans with taeniasis is effective in disease control in human and pig populations. However, as in José *et al.* (2018), the study did not capture the dynamics of taeniasis and cysticercosis due ingestion of raw or undercooked beef that is infected with tapeworm larval cycts and did not consider the continuous time Markov chain model to study the diseases' dynamics. Also, the study did not apply the optimal control and cost effectiveness analysis to suggest the most effective strategy for diseases' control.

2.3 Continuous Time Markov Chain Stochastic Models for Infectious Diseases

The continuous time Markov chain (CTMC) stochastic models play a significant role in analyzing the dynamics of infectious diseases as they capture uncertainties that are inherent to disease transmission. Unlike deterministic and other stochastic models, the CTMC models are useful in computing the probabilities of disease extinction depending on number of infectives at the beginning of the disease outbreak (Allen & Lahodny Jr, 2012; Lahodny Jr *et al.*, 2015; Maliyoni, 2021). Generally in CTMC stochastic models, all state variables are considered to be discrete and time is continuous. Thus, CTMC models are more realistic than deterministic models as they describe a discrete movement of individuals between classes and not average rates as the case of deterministic models (Maliyoni, 2021). Recently, there are only few continuous time Markov chain stochastic models that have been formulated to analyze the dynamics of various infectious diseases.

Lahodny Jr *et al.* (2015) formulated and analyzed a continuous time Markov Chain (CTMC) stochastic model which is a version of the corresponding deterministic model, to estimate the probability of extinction or major outbreak for an environmentally transmitted infectious disease. They applied multitype branching process to find the probability generating functions that were used to compute the probability of disease extinction P_0 in human population which was then compared with the estimate probability P_a that were obtained from the proportion of 10,000 sample paths of the CTMC model using different initial values of infected classes. The results showed that the solutions of CTMC stochastic model are not far from the solutions of the deterministic model. They fluctuate within the solution of their corresponding deterministic equations. The analytical and numerical results showed that P_a is a good estimate of P_0 for the probability of disease extinction.

A study by Zevika & Soewono (2018) was carried out to study the transmission dynamics of Zika disease using deterministic and continuous time Markov chain (CTMC) models. The study applied a multitype branching process to derive expression for probability generating functions that were used to compute the probabilities of disease extinction and the stochastic extinction threshold. The results showed that CTMC solutions are relatively close to the corresponding deterministic solutions. The analytical probabilities of disease extinction P_0 are closely related to the approximate probabilities, P_a from numerical simulations. The probability of disease extinction was high if the disease emerged from either infectious humans or infected congenital infants. The probability of disease extinction declined if the disease emerged from infectious mosquitoes and became smaller if the disease emerged from all infected classes.

Marwa *et al.* (2019) formulated and analyzed a deterministic model with its corresponding continuous time Markov chain (CTMC) model for cholera epidemic to assess its transmission dynamics. They used the multitype branching process to compute the probability of disease extinction. Numerical results showed that solution of the CTMC model are close to deterministic model solutions. That is, CTMC model solutions fluctuates within the solution of the deterministic model.

Maliyoni *et al.* (2019) formulated and analyzed a continuous time Markov Chain (CTMC) stochastic model for the dynamics of two pathogens in a single tick population based on a deterministic tick-borne disease model. The multitype branching process was applied to compute the probability generating functions that were used to derive the stochastic threshold for pathogen extinction or outbreak. The results showed that the approximate probability of pathogen extinction P_a computed from numerical simulations was in good agreement with the probability of extinction that was derived from the branching process P_0. The probability of disease extinction was high if a tick infected with *Rickettsia parkeri* species introduced the pathogen at the beginning of disease outbreak unlike if the pathogens were introduced by a tick infected with *Rickettsia amblyommii* species or a co-infected tick. Moreover, the probability of disease extinction declined if the pathogens were introduced by all three infected classes at the beginning of the epidemic.

Maliyoni (2021) formulated and analyzed a continuous time Markov Chain (CTMC) stochastic model to estimate the probability of West Nile Virus disease extinction or outbreak in birds. The multitype branching process was applied to derive the stochastic extinction threshold for the CTMC model. The analytical results showed that the probability of disease extinction was in good agreement with the approximate probability of disease extinction from numerical simulations. Moreover, the results show that the probability of disease extinction was high if initially there was only an exposed mosquito at the beginning of disease outbreak unlike if the disease

emerged from infectious mosquitoes and birds. The epidemic duration was very short if the disease was introduced by exposed mosquitoes.

2.4 Mathematical Models for the Optimal Control and Cost-effectiveness Analysis of Environmentally Transmitted Diseases

Okosun *et al.* (2016) formulated the optimal control and cost effectiveness analysis model for leptospirosis by incorporating human and livestock (vectors). They used strategies which involve vector vaccination, and treatment of infected humans and vectors. The Pontryagin's maximum principle was used to derive necessary conditions for the optimal control of the disease. The cost-effectiveness analysis was carried out by using ANOVA and Incremental Cost-Effectiveness Ratio (ICER). The results revealed that the most cost-effective strategy for the control of leptospirosis was the combination of the vector vaccination and treatment of infective livestocks.

Tilahun *et al.* (2017) developed and analyzed a nonlinear deterministic model to study the dynamics and optimal control of typhoid fever disease with cost-effective strategies in communities where the population is varying. The Pontryagin's maximum principle was applied using three control strategies which were implemented singularly or in combination to determine the optimal control strategy. These strategies were treatment of infected individuals; proper hygiene, adequate sanitation and vaccination; and the screening of carriers. The Incremental Cost Effectiveness Ratio was used to determine the most cost-effective strategy. Numerical results revealed that a strategy that combines improved hygiene and sanitation, vaccination and treatment of infected individuals was the best cost-effective strategy to eradicate the disease.

Berhe *et al.* (2018) formulated and analyzed a mathematical model for the optimal control and cost effectiveness analysis of dysentery diarrhea epidemic. They used the Pontryagin's maximum principle to derive the conditions that are necessary for the optimal controls to exist. Three intervention strategies that were used in the optimal control problem are health education and hygiene, treatment of infected individuals, and improved sanitation. They applied the Incremental Cost Effectiveness Ratio to determine the most cost-effective strategy for disease control. The results showed that the strategy that combined health education campaign with improved sanitation was most efficient and cost-effective for the control of the disease.

Panja (2019) established a mathematical model for optimal control and cost-effectiveness analysis of a cholera epidemic model. Three intervention strategies which are treatment, vaccination and awareness programs were implemented in combination to study their impacts on the disease control. The Incremental Cost Effectiveness Ratio (ICER) was applied to determine the most cost-effective strategy. The results indicated that a strategy which target on treatment of infected people and awareness program was the most cost-effective in the control of cholera disease.

Nyerere *et al.* (2020) formulated a mathematical model for optimal control and cost-effectiveness analysis for the infectiology of brucellosis. The Pontryagin's maximum principle was implemented using various intervention strategies in combination to determine their impacts on the control of the disease in humans, livestock and small ruminants. These interventions included livestock vaccination, gradual culling of cattle and small ruminants, personal protection, and environmental hygiene and sanitation. The Incremental Cost Effective Ratio was used to determine the most cost-effective strategy. Numerical results showed that a strategy which combines livestock vaccination, gradual culling of cattle and small ruminants through slaughter, environmental sanitation, and personal protection is the most cost-effective strategy for disease control.

The optimal control mathematical model with cost effectiveness analysis for the control of anthrax in human and animal populations was done by Osman *et al.* (2020). In their study, they used four intervention strategies which are human preventive control measures, treatment of infected humans, vaccination of animals and treatment of infected animals that were implemented in combination of two strategies. The Infection Averted Ratio (IAR) was used to determine the most cost-effective strategy for disease control. The results showed that a strategy which combined vaccination of animals and prevention of humans was the most effective strategy in combating the anthrax epidemics in humans and animals.

2.5 Non-Mathematical Studies for Taeniasis and Cysticercosis

Non-mathematical studies have also been done on the epidemiology, prevalence and distribution of taeniasis and cysticercosis in different regions within and outside Tanzania. Various associated risk factors for transmission of these diseases have been indicted including the knowledge of farmers about the diseases, livestock management systems, availability of water sources, consumption of infected meat and absence of latrines (Mwanjali *et al.*, 2013; Ngowi *et al.*, 2004; Yohana *et al.*, 2013; Kabululu *et al.*, 2015; Mkupasi *et al.*, 2011; Dermauw *et al.*, 2018; Yimer & Gebrmedehan, 2019; Shonyela *et al.*, 2017; Saratsis *et al.*, 2019; Kabululu *et al.*, 2018; Kumar & Tadesse, 2011). Control measures that have been suggested include chemotherapy, meat

inspection, use of vaccines and the use of latrines. However, these studies have failed to describe the parameters that drive the diseases, show optimal control measures and the most cost effective strategy for diseases' control. Also, non-mathematical studies have not shown the possibility of diseases outbreak or extinction if the diseases emerge from which class of infectives. Thus, mathematical modelling approach will fill this gap by describing all these aspects.

CHAPTER THREE

MATERIALS AND METHODS

3.1 Introduction

In this chapter, we formulate and analyze both deterministic and continuous time Markov chain (CTMC) stochastic models for transmission dynamics of taeniasis and cysticercosis in humans, pigs and cattle. The deterministic models are formulated using non-linear ordinary differential equations. These equations lead into a dynamical systems that govern the transmission dynamics of taeniasis and cysticercosis in humans, pigs and cattle. The resulting models are tested for positivity and boundedness of their solutions. The models are extended to include various interventions such as vaccination, pigs' and cattle indoor keeping, improved meat (pork and beef) cooking, treatment and improved hygiene and sanitation to assess their impacts in diseases' control.

The disease free and endemic equilibria are computed and their stability are analyzed. The linearization method in combination with trace and determinant method is used to analyze the local stability of disease free equilibrium whereas Lyapunov functions and Metzler matrix method are used to analyze global stability of disease free equilibria. The theory of cooperative systems and Lyapunov functions in combination with the LaSalle's invariant principle are used to analyze the stability of the endemic equilibria. The basic reproduction numbers $\mathscr{R}_{01}$ and R_0 which determines whether the disease clears or persists are computed by the next generation matrix method as described by Diekmann *et al.* (1990). Sensitivity indices of parameters in the basic reproduction numbers $\mathscr{R}_{01}$ and R_0 are derived by the normalized forward sensitivity index as described by Chitnis *et al.* (2008).

Basing on the deterministic models, the continuous time Markov chain (CTMC) stochastic models are formulated and analyzed to study the dynamics of taeniasis and cysticercosis in humans, pigs and cattle. The multitype branching process is applied to compute the probability of disease extinction or outbreak. The Pontryagin's maximum principle is adopted to determine the most optimal strategy for diseases' control whereas the incremental cost-effectiveness ratio (ICER) is used to determine the most cost-effective strategy for the diseases' control.

3.2 Dynamics of Taenia Saginata Bovine Cysticercosis and Human Taeniasis

Bovine cysticercosis is an infection of cattle caused by the larval stage of *Taenia saginata* (beef tapeworm) (Kumar & Tadesse, 2011). Humans acquire infection when they consume raw or undercooked beef infected with larval cysts which mature to adult tapeworms in the

human small intestine, a condition which is called human taeniasis (Symeonidou *et al.*, 2018). When infected humans defecate in the fields, they release tapeworm eggs which contaminate pastures, fodder and water sources (Dermauw *et al.*, 2018; Symeonidou *et al.*, 2018). Cattle acquire cysticercosis when they ingest *T. saginata* eggs that were shed in human feaces during grazing, or when they ingest contaminated fodder or water (Symeonidou *et al.*, 2018). When *T. saginata* eggs are ingested by cattle, they hatch, penetrate the intestinal wall and reach the blood circulation system, there being taken to various tissues and organs such as the heart, diaphragm, kidney, lungs, liver and tongue where they develop into cysticerci (Ito *et al.*, 2004; WHO, 2005). Human taeniasis can be controlled when infected beef is cooked to a sufficient internal temperature of 56°C to 65°C, or freezed at the temperature of -10°C for five days to ensure that all cysts are killed (Grindle, 1978; Lesh & Brady, 2019).

Bovine cysticercosis and human taeniasis are common in areas where people defecate in open spaces, particularly in rural areas where people keep their cattle under free range system (Alemneh *et al.*, 2017; Flisser *et al.*, 2006; Trevisan *et al.*, 2017). Such areas are highly characterized by poor sanitation, low standard of slaughter facilities, inadequate or no meat inspection and the treatment for these diseases is not readily available (Mkupasi *et al.*, 2011). The life cycle of *T. saginata* is presented in Fig. 4.

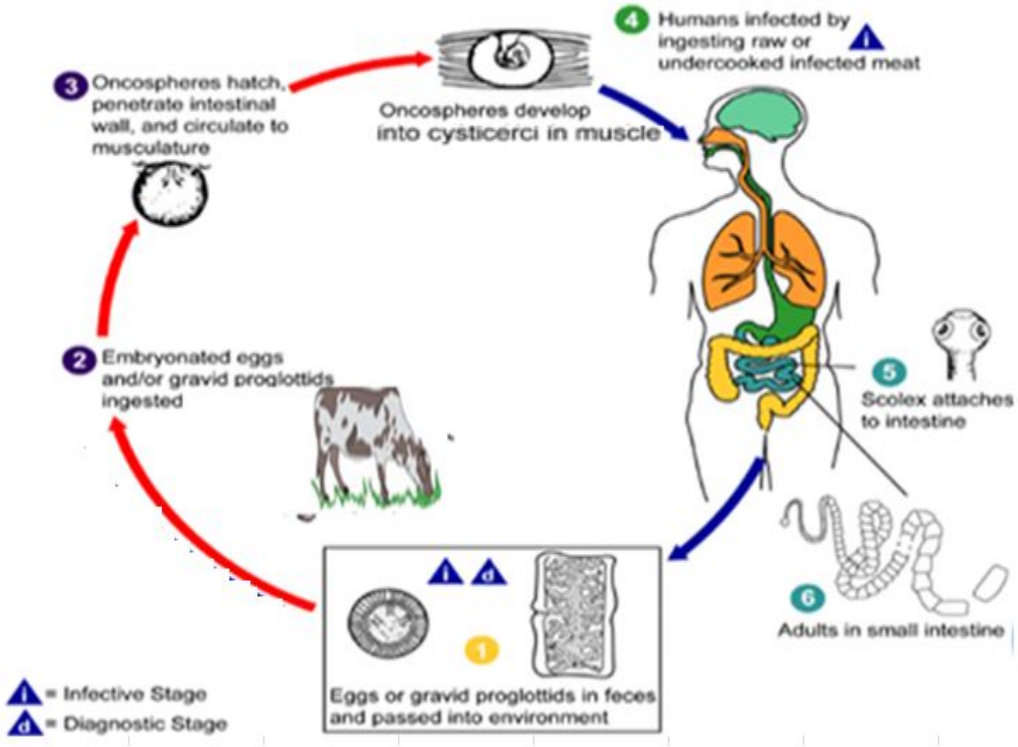


Figure 4: The Life Cycle of *Taenia saginata* (CDC, 2013)

Human taeniasis due to *T. saginata* affects people who depend on beef as an important food component of their diet and consume raw or undercooked beef infected with *T. saginata* tapeworm larval cysts (Kumar & Tadesse, 2011). In developing countries, most rural farmers keep cattle in large numbers, that are usually kept under free range system (URT, 2016; Michael *et al.*, 2018).

Taenia saginata bovine cysticercosis and human taeniasis are globally distributed, occuring in both developed and developing countries. In developed countries, the disease prevalence is very low and it is reemerging in previous disease free areas due to migration of infected humans and cattle exchange (Yimer & Gebrmedehan, 2019). The prevalence rates are higher in developing countries of Latin America, Africa and some parts of Asia (Braae *et al.*, 2018). In Africa, *T. saginata* parasite is prevalent in almost all regions with higher rates in Eastern and Southern Africa including Ethiopia, Sudan, Tanzania, Kenya, South Africa, Botswana, Zimbabwe and Zambia whereby Ethiopia has the highest parasite prevalence rates (Dermauw *et al.*, 2018). In such countries, most people keep cattle for their livelihoods, serving as a source of food, manure, draft power and income for challenging times (Dermauw *et al.*, 2018; Swanepoel *et al.*, 2010). *T. saginata* parasite poses a threat to the economy as most people keep catttle for their livelihood. The parasite causes few symptoms in humans, including anal pruritus, mild abdominal pain, distress and some people even suffer from a pathological fear of tapeworms (Dermauw *et al.*, 2018). In cattle, the infection is asymptomatic but may cause considerable economic losses for the meat sector due to carcass condemnation or treatment upon detection of tapeworm larval cysts during meat inspection and related insurance costs (Dermauw *et al.*, 2018). Economic losses from *T. saginata* bovine cysticercosis and human taeniasis are measured in terms of disease prevalence, potential market price of cattle, grade of infected cattle, treatment cost for detained carcasses and medical costs for infected humans (Alemneh *et al.*, 2017; Grindle, 1978). In Botswana and Kenya, the annual losses due to *T. saginata* bovine cysticercosis and human taeniasis approach 0.5 and 1 million pounds respectively while the loss per animal slaughtered is 2.25 and 1 million pounds respectively. In Ethiopia, the average annual loss due to taenicidal drugs for treatment is about 4 937 583.21 Ethiopian Birr (Alemneh *et al.*, 2017).

Taenia saginata bovine cysticercosis and human taeniasis have been neglected for long time in developing countries due to the absence of symptoms in cattle, lack of data on its economic impact, and because human taeniasis is considered as a minor health problem. However, the prevalence of these diseases is a clear sign of inadequate hygiene and sanitation, consumption of raw or undercooked beef and insufficient meat inspection, which accelerate disease transmission (Dermauw *et al.*, 2018). The diagnosis of human taeniasis is usually done by examination of stool samples while bovine cysticercosis diagnosis involves meat inspection and serologi-

cal tests (Okello & Thomas, 2017; Winskill *et al.*, 2017). The treatment of human taeniasis is through administration of prescribed medication of praziquantel, tribendimidine, albendazole and niclosamide (Okello & Thomas, 2017). Intervention strategies in cattle involve the use of TSA-9 and TSA-18 vaccines (Kumar & Tadesse, 2011; Lightowlers *et al.*, 1996), and anthelmintic treatment with flubendazole, fenbendazole, oxfendazole, praziquantel and nitazoxanide (WHO, 2005; Winskill *et al.*, 2017).

Understanding well how *Taenia saginata* bovine cysticercosis and human taeniasis are transmitted is useful in designing appropriate diseases' control strategies. Mathematical modeling has become an important tool in studying and analyzing the transmission dynamics and control of infectious diseases. Over the past two decades, only few mathematical models have been formulated and analyzed to study the dynamics and control of human taeniasis and cysticercosis in humans and pigs due to *Taenia solium* tapeworm parasite (Gonzalez *et al.*, 2002; Kyvsgaard *et al.*, 2007; Braae *et al.*, 2016; Winskill *et al.*, 2017; José *et al.*, 2018; Sánchez-Torres *et al.*, 2019). None of these studies have considered the dynamics and control of bovine cysticercosis and human taeniasis due to *Taenia saginata* tapeworm parasite. In addition, these studies have not applied continuous time Markov chain (CTMC) stochastic models to study the dynamics of taeniasis and cysticercosis. In this chapter, we develop and analyze both deterministic and CTMC stochastic models for the transmission dynamics of *T. saginata* bovine cysticercosis and human taeniasis by including the compartment of infected beef. Furthermore, some control strategies are implemented in the deterministic model to study their impacts in diseases' control.

3.3 Taenia Saginata Deterministic Model

The mathematical model for transmission dynamics and control of *Taenia saginata* bovine cysticercosis and human taeniasis is formulated by considering the model flow generated by Kyvsgaard *et al.* (2007) that studied the dynamics and control of *Taenia solium* parasitic infection in human and pig populations using a Reed-Frost stochastic model. Human taeniasis and bovine cysticercosis utilize humans as definitive hosts and cattle as the most intermediate hosts (Dermauw *et al.*, 2018). The model divides human population into S_H and I_{HT} classes that represent susceptible humans and humans with taeniasis respectively. The cattle population is divided into S_C, V_C, I_C and R_C that represent susceptible cattle, vaccinated cattle, cattle infected with cysticercosis and cattle that recover from cysticercosis respectively. The compartments B_I and E_T represent infected beef and the number of *Taenia saginata* eggs in the environment respectively.

Susceptibe humans are recruited through birth at a rate Λ_H and increase at a rate χ when infected individuals recover from taeniasis. However, they diminish through consumption of raw or insufficiently cooked infected beef at a rate $\alpha_b(1-\kappa)$ where κ is the cooking rate of infected beef. Humans with taeniasis increase at rate $\alpha_b(1-\kappa)$ when susceptible humans consume raw or undercooked infected beef. The parameter β_T denotes the probability of susceptible humans to acquire taeniasis due to consumption of an infected beef, that is, the per capita rate at which susceptible individuals acquire taeniasis. All human compartments suffer natural death at a rate μ_h. The number of *Taenia saginata* eggs in the environment grow at a rate ν as a result of open defecation by humans with taeniasis and decrease at rates τ and μ_e due to improved hygiene and sanitation, and natural death respectively. They are further reduced at a rate c_h due to humans' efforts to spray chemicals for killing *T. saginata* eggs from the contaminated environment.

Susceptible cattle are recruited at a rate Λ_C due to birth. They increase at rates ψ_v and π due to waning of vaccine and when recovered cattle loose their immunity respectively. They diminish at a rate γ_b when they acquire cysticercosis from the contaminated environment, at a rate ψ_s when they are vaccinated and at a rate σ_s when they are slaughtered. Vaccinated cattle increase at a rate ψ_s when susceptible cattle are vaccinated and decrease at rates σ_s and ψ_v when they are slaughtered and when the vaccine wanes respectively. They further diminish at a rate $\rho_b\gamma_b$ when vaccinated cattle acquire infections from the contaminated environment due to ineffectiveness of vaccines. Infected cattle increase at rates γ_b and $\rho_b\gamma_b$ as a consequence of susceptible and vaccinated cattle to feed on contaminated environment. Recovered cattle increase at a rate λ due to treatment of cattle infected with cysticercosis and decrease at a rate σ_r when they are slaughtered. All cattle classes suffer natural death at a rate μ_b. The infected beef increases when infected cattle are slaughtered at a rate η and decreases when consumed by humans at a rate α_b. The parameter ε represents proportion of an infected beef which is not consumed by susceptible humans.

In model formulation, we consider the free range farming system for cattle and we do not consider migration. We assume that the number of *Taenia saginata* eggs consumed by cattle have negligible effect on the total number of eggs in the environment and that both infected humans and cattle cannot recover from infections without treatment. Also, we assume that the cattle vaccines are not 100% effective and hence they wane after sometime. We further assume that cattle and humans do not suffer disease induced mortality, they become carriers for their life and that the rate at which susceptible humans consume raw or undercooked infected beef depend on the amount of infected beef that is present. The quantity of beef is measured in terms of number of infected cattle that are slaughtered for consumption. Furthermore, cattle contact rate with *Taenia saginata* eggs in the environment is assumed to follow the principle of mass action. The model for the transmission dynamics of *Taenia saginata* bovine cysticercosis and

human taeniasis with control measures is summarized by the flow diagram in Fig. 5 and model parameters are summarized in Table 1.

Table 1: Description of model parameters

Parameter	Description
Λ_H	Recruitment rate of human population
μ_h	Per capita natural death rate of humans
α_b	Rate of eating raw or undercooked infected beef
Λ_C	Recruitment rate of cattle
γ_b	*T. saginata* eggs to susceptible cattle transmission coefficient
ρ_b	Reduction rate of *T. saginata* eggs transfer to vaccinated cattle
η	Slaughter rate of infected cattle
μ_b	Per capita natural death rate of cattle
ε	Proportion of unconsumed infected beef
β_T	Probability of human infection with taeniasis
ν	Defecation rate by humans who are infected with taeniasis
μ_e	Per capita natural death rate of *Taenia saginata* eggs
σ_s	Harvesting rate of susceptible cattle
σ_v	Harvesting rate of vaccinated cattle
σ_r	Harvesting rate of recovered cattle
λ	Proportion of infected cattle recovered from cysticercosis
π	Proportion of recovered cattle that becomes susceptible
ψ_s	Proportion of susceptible cattle that is vaccinated
ψ_v	Vaccine waning rate
τ	Rate of improvement in sanitation (use of toilets)
c_h	The rate of killing *T. saginata* eggs on the contaminated environment
κ	Cooking rate (% to sufficient internal temperature) of infected beef
χ	Recovery rate of humans with taeniasis

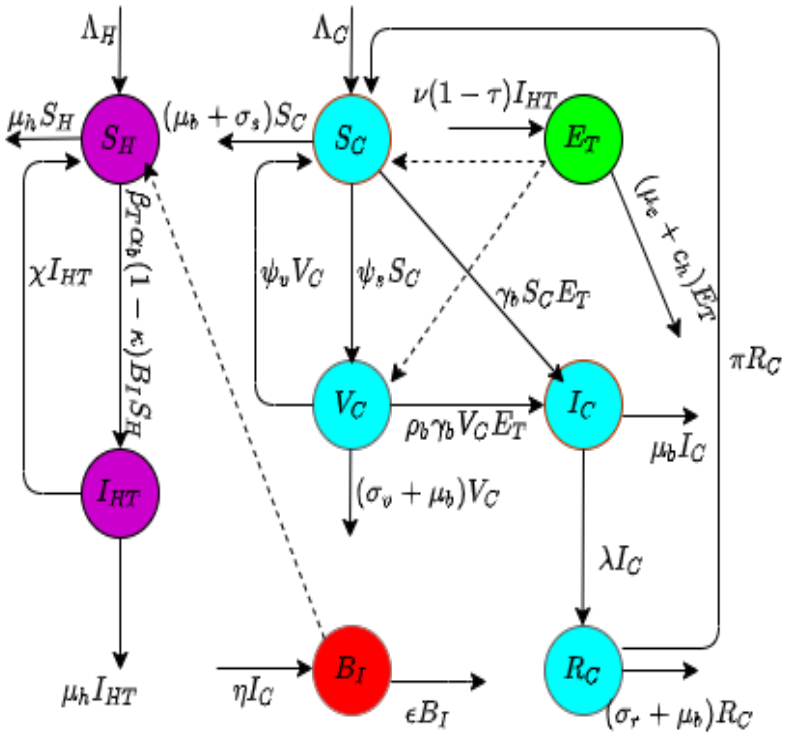


Figure 5: The model flow diagram for *T. saginata* infection

The model that describes the transmission dynamics and control of *Taenia saginata* bovine cysticercosis and human taeniasis is given by the model system (3.1).

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H + \chi I_{HT} - \beta_T \alpha_b (1-\kappa) B_I S_H - \mu_h S_H, \\
\frac{dI_{HT}}{dt} &= \beta_T \alpha_b (1-\kappa) B_I S_H - (\mu_h + \chi) I_{HT}, \\
\frac{dS_C}{dt} &= \Lambda_C + \psi_v V_C + \pi R_C - \gamma_b S_C E_T - (\sigma_s + \psi_s + \mu_b) S_C, \\
\frac{dV_C}{dt} &= \psi_s S_C - \rho_b \gamma_b V_C E_T - (\sigma_v + \psi_v + \mu_b) V_C, \\
\frac{dI_C}{dt} &= \gamma_b S_C E_T + \rho_b \gamma_b V_C E_T - (\eta + \lambda + \mu_b) I_C, \\
\frac{dR_C}{dt} &= \lambda I_C - (\pi + \sigma_r + \mu_b) R_C, \\
\frac{dB_I}{dt} &= \eta I_C - (\varepsilon + \alpha_b) B_I, \\
\frac{dE_T}{dt} &= \nu(1-\tau) I_{HT} - (\mu_e + c_h) E_T,
\end{aligned} \tag{3.1}$$

with initial conditions:

$S_H(0) > 0; I_{HT}(0) \geq 0; S_C(0) > 0; V_C(0) > 0; I_C(0) \geq 0; R_C(0) \geq 0; B_I(0) \geq 0$ and $E_T(0) \geq 0$.

3.4 Positivity and Boundedness of Model Solutions

For the model system (3.1) to be biologically and epidemiologically meaningful, we need to prove that the model solutions are positive and bounded. Given the model system (3.1), there exists a region

$$\Omega = \left\{ (S_H, I_{HT}, S_C, V_C, I_C, R_C, B_I, E_T) \in \mathbb{R}^8_+ \right\} \tag{3.2}$$

such that the model solutions are positive and bounded.

3.4.1 Positivity of Model Solutions

Theorem 3.1

Let the initial conditions for the model system (3.1) be:

$$S_H(0) > 0; I_{HT}(0) \geq 0; S_C(0) > 0; V_C(0) > 0; I_C(0) \geq 0; R_C(0) \geq 0; B_I(0) \geq 0, E_T(0) \geq 0.$$

Then the solutions $(S_H, I_{HT}, S_C, V_C, I_C, R_C, B_I, E_T)$ of the model with positive initial conditions, will remain non-negative for all time $t > 0$.

Proof.

$$\text{Let } t_1 = sup\{t > 0 : S_H > 0, I_{HT} > 0, S_C > 0, V_C > 0, I_C > 0, R_C > 0, B_I > 0, E_T > 0 \in [0,t]\},$$

so that $t_1 > 0$. Then, from the first equation in the model system (3.1) for susceptible humans, we have:

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H + \chi I_{HT} - \beta_T \alpha_b (1-\kappa) B_I S_H - \mu_h S_H, \\
\frac{dS_H}{dt} &\geq -\left(\beta_T \alpha_b (1-\kappa) B_I + \mu_h\right) S_H, \\
\frac{dS_H}{S_H} &\geq -\left(\beta_T \alpha_b (1-\kappa) B_I + \mu_h\right) dt, \\
S_H(t_1) &\geq S_H(0) e^{\int_0^{t_1} -\left(\beta_T \alpha_b (1-\kappa) B_I(s) + \mu_h\right) ds} \geq 0.
\end{aligned} \tag{3.3}$$

Using the same approach, it can be shown that:

$$I_{HT}(t) \geq 0; S_C(t) \geq 0; V_C(t) \geq 0; I_C(t) \geq 0; R_C(t) \geq 0; B_I(t) \geq 0; E_T(t) \geq 0, \forall t \geq 0. \tag{3.4}$$

Therefore, all solutions of the Model (3.1) will remain positive for all non-negative initial conditions.

3.4.2 Boundedness of Model Solutions

To prove the boundedness of solutions, we let the total populations for humans and cattle be $H = S_H + I_{HT}$ and $T_C = S_C + V_C + I_C + R_C$ respectively. Considering the human population, we have:

$$\begin{aligned} \frac{dH}{dt} &= \Lambda_H - \mu_h H, \\ \frac{dH}{dt} &+ \mu_h H = \Lambda_H. \end{aligned} \tag{3.5}$$

Using integration factor, we obtain:

$$H(t) = \frac{\Lambda_H}{\mu_h} + \left(H(0) - \frac{\Lambda_H}{\mu_h} \right) e^{-\mu_h t}, \tag{3.6}$$

where $H(0) = S_H(0) + I_{HT}(0)$. Following the same procedures, it can be shown that the total cattle population is given by:

$$T_C(t) \leq \frac{\Lambda_C}{\mu_b} + \left(T_C(0) - \frac{\Lambda_C}{\mu_b} \right) e^{-\mu_b t} \tag{3.7}$$

where $T_C(0) = S_C(0) + V_C(0) + I_C(0) + R_C(0)$. We consider two cases to analyze solutions (3.6) and (3.7): when $H(0) > \frac{\Lambda_H}{\mu_h}$, $T_C(0) > \frac{\Lambda_C}{\mu_b}$ and when $H(0) < \frac{\Lambda_H}{\mu_h}$, $T_C(0) < \frac{\Lambda_C}{\mu_b}$. Both cases give:

$$\begin{aligned} H(t) &\leq \Phi_t = \max\left\{ \frac{\Lambda_H}{\mu_h}, H(0) \right\}, \\ T_C(t) &\leq \Psi_t = \max\left\{ \frac{\Lambda_C}{\mu_b}, T_C(0) \right\}. \end{aligned} \tag{3.8}$$

Since $H = S_H + I_{HT} \leq \Phi_t$, it follows that

$$I_{HT} \leq \Phi_t. \tag{3.9}$$

We need to show that E_T is also bounded. The last equation for *T. saginata* eggs in model system (3.1), can be written as:

$$\frac{dE_T}{dt} + (\mu_e + c_h) E_T = \nu(1-\tau) I_{HT}. \tag{3.10}$$

Using equation (3.9), equation (3.10) can be written as:

$$\frac{dE_T}{dt} + (\mu_e + c_h) E_T \leq \nu(1-\tau) \Phi_t. \tag{3.11}$$

Applying integration factor, we have:

$$E_T(t) \leq \frac{\nu(1-\tau)\Phi_t}{(\mu_e + c_h)} + \left(E_T(0) - \frac{\nu(1-\tau)\Phi_t}{(\mu_e + c_h)} \right) e^{-(\mu_e + c_h)t}, \tag{3.12}$$

from which we get:

$$E_T(t) \leq \Gamma_t = \max \left\{ \frac{\nu(1-\tau)\Phi_t}{(\mu_e + c_h)}, E_T(0) \right\}. \tag{3.13}$$

Using the same approach for infected beef equation in model system (3.1), we get:

$$B_I(t) \leq \xi_t = \max \left\{ \frac{\eta \Psi_t}{(\varepsilon + \alpha_b)}, B_I(0) \right\}. \tag{3.14}$$

The solutions of the model system (3.1) enter the region:

$$\begin{aligned} \Omega = \Big\{ & (S_H, I_{HT}, S_C, V_C, I_C, R_C, B_I, E_T) \in \mathbb{R}_+^8 : 0 \leq H(t) \leq \Phi_t; 0 \leq T_C(t) \leq \Psi_t; \\ & 0 \leq E_T(t) \leq \Gamma_t; 0 \leq B_I(t) \leq \xi_t \Big\}. \end{aligned} \tag{3.15}$$

Therefore, all solutions of the model (3.1) are positive invariant in the region:

$$\begin{aligned} \Omega = \Big\{ & (S_H, I_{HT}, S_C, V_C, I_C, R_C, B_I, E_T) \in \mathbb{R}_+^8 : 0 \leq H \leq \Phi_t; 0 \leq T_C \leq \Psi_t; 0 \leq E_T \leq \Gamma_t; \\ & 0 \leq B_I \leq \xi_t \Big\}. \end{aligned} \tag{3.16}$$

where

$$\begin{aligned} \Phi_t &= \max \left\{ \frac{\Lambda_H}{\mu_h}, H(0) \right\}, \Gamma_t = \max \left\{ \frac{\nu(1-\tau)\Phi_t}{(\mu_e + c_h)}, E_T(0) \right\}, \\ \Psi_t &= \max \left\{ \frac{\Lambda_B}{\mu_b}, T_C(0) \right\}, \xi_t = \max \left\{ \frac{\eta \Psi_t}{(\varepsilon + \alpha_b)}, B_I(0) \right\}, \end{aligned} \tag{3.17}$$

3.5 The Basic Model

When control measures are not implemented, the model system (3.1) reduces to the basic model which is represented by the following system of differential equations:

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H - \beta_T \alpha_b B_I S_H - \mu_h S_H, \\
\frac{dI_{HT}}{dt} &= \beta_T \alpha_b B_I S_H - \mu_h I_{HT}, \\
\frac{dS_C}{dt} &= \Lambda_C - \gamma_b S_C E_T - (\sigma_s + \mu_b) S_C, \\
\frac{dI_C}{dt} &= \gamma_b S_C E_T - (\eta + \mu_b) I_C, \\
\frac{dB_I}{dt} &= \eta I_C - (\varepsilon + \alpha_b) B_I, \\
\frac{dE_T}{dt} &= \nu I_{HT} - \mu_e E_T,
\end{aligned} \tag{3.18}$$

with initial conditions: $S_H(0) > 0; I_{HT}(0) \geq 0; S_C(0) > 0; I_C(0) \geq 0; B_I(0) \geq 0$ and $E_T(0) \geq 0$.

Using similar approach as in Section 3.2.2, it can be shown that all solutions of the Model (3.18) are positive invariant in the region:

$$\Pi = \left\{ (S_H, I_{HT}, S_C, I_C, B_I, E_T) \in \mathbb{R}^6_+ : 0 \leq H \leq \phi_t; 0 \leq T_C \leq \psi_t; 0 \leq E_T \leq \gamma_t; 0 \leq B_I \leq \zeta_t \right\}. \tag{3.19}$$

where $H = S_H + I_{HT}, T_C = S_C + I_C$ and

$$\begin{aligned}
\phi_t &= \max\left\{ \frac{\Lambda_H}{\mu_h}, H(0) \right\}, \quad \psi_t = \max\left\{ \frac{\Lambda_C}{\mu_b}, T_C(0) \right\}, \\
\gamma_t &= \max\left\{ \frac{\nu \phi_t}{\mu_e}, E_T(0) \right\}, \quad \zeta_t = \max\left\{ \frac{\eta \psi_t}{(\varepsilon + \alpha_b)}, B_I(0) \right\}.
\end{aligned} \tag{3.20}$$

The model systems (3.1) and (3.18) are biologically and epidemiologically meaningful. Therefore, we can consider the flow generated by these models for analysis. These results are summarized in Theorem 3.2.

Theorem 3.2

The solutions of the model systems (3.1) and (3.18) are positive and bounded in the regions Ω and Π respectively.

3.5.1 Analysis of the Basic Model

In this section, we analyze the basic model (3.18), so as to identify the parameters that drive *T. saginata* bovine cysticercosis and human taeniasis. This analysis is useful in making decision on the appropriate strategies to control the diseases.

3.5.2 Disease Free Equilibrium and Basic Reproduction Number R_0

In absence of *T. saginata* bovine cysticercosis and human taeniasis, we obtain the disease free equilibrium E^0 which is given by:

$$E^0\left(S_H, I_{HT}, S_C, I_C, B_I, E_T\right) = \left(\frac{\Lambda_H}{\mu_h}, 0, \frac{\Lambda_C}{\sigma_s + \mu_b}, 0, 0, 0\right). \tag{3.21}$$

The basic reproduction number $\mathscr{R}_{01}$ is the expected number of secondary infections that may arise as a result of introducing one infected individual in a fully susceptible population (Diekmann *et al.*, 1990). When $\mathscr{R}_{01} < 1$, the disease clears whereas when $\mathscr{R}_{01} > 1$, the disease persists within the population. In computing $\mathscr{R}_{01}$, we adopt the next generation matrix method (Van den Driessche & Watmough, 2002). Let $\mathscr{F}_i$ be the new infections in compartment i and $\mathscr{V}_i^+$ and $\mathscr{V}_i^-$ be the transfer terms in and out of the compartment i respectively, then the infected classes can be written as:

$$\frac{dx_i}{dt} = \mathscr{F}_i(x) - \mathscr{V}_i^+(x) - \mathscr{V}_i^-(x). \tag{3.22}$$

Using the next generation matrix method, we define $\mathscr{F}_i$ and $\mathscr{V}_i$ by:

$$\mathscr{F}_i = \begin{pmatrix} \beta_T \alpha_b B_I S_H \\ \gamma_b S_C E_T \\ 0 \\ 0 \end{pmatrix}, \quad \mathscr{V}_i = \begin{pmatrix} \mu_h I_{HT} \\ (\eta + \mu_b) I_C \\ -\eta I_C + (\varepsilon + \alpha_b) B_I \\ -\nu I_{HT} + \mu_e E_T \end{pmatrix}. \tag{3.23}$$

The Jacobian matrices F and V at the disease free equilibrium E^0 are given by:

$$F = \frac{\partial \mathscr{F}_i}{\partial x_j}\left(E^0\right), \quad V = \frac{\partial \mathscr{V}_i}{\partial x_j}\left(E^0\right). \tag{3.24}$$

The basic reproduction number $\mathscr{R}_{01}$ is the largest eigenvalue of the next generation matrix given by:

$$\mathscr{R}_{01} = \rho\left(FV^{-1}\right). \tag{3.25}$$

From equation (3.24), F and V are:

$$F = \begin{pmatrix} 0 & 0 & \dfrac{\beta_T \alpha_b \Lambda_H}{\mu_h} & 0 \\ 0 & 0 & 0 & \dfrac{\gamma_b \Lambda_C}{(\sigma_s + \mu_b)} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} \mu_h & 0 & 0 & 0 \\ 0 & \eta + \mu_b & 0 & 0 \\ 0 & -\eta & (\varepsilon + \alpha_b) & 0 \\ -\nu & 0 & 0 & \mu_e \end{pmatrix}. \tag{3.26}$$

The next generation matrix is given by:

$$FV^{-1} = \begin{pmatrix} 0 & \dfrac{\beta_T \eta \alpha_b \Lambda_H}{\mu_h(\eta + \mu_b)(\varepsilon + \alpha_b)} & \dfrac{\beta_T \alpha_b \Lambda_H}{\mu_h(\varepsilon + \alpha_b)} & 0 \\ \dfrac{\nu \gamma_b \Lambda_B}{\mu_e \mu_h(\sigma_s + \mu_b)} & 0 & 0 & \dfrac{\gamma_b \Lambda_B}{\mu_e(\sigma_s + \mu_b)} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}. \tag{3.27}$$

Using definition (3.25), the basic reproduction number $\mathscr{R}_{01}$ is:

$$\mathscr{R}_{01} = \sqrt{\frac{\beta_T \nu \alpha_b \gamma_b \eta \Lambda_H \Lambda_C}{\mu_h^2 \mu_e (\eta + \mu_b)(\alpha_b + \varepsilon)(\mu_b + \sigma_s)}}. \tag{3.28}$$

To interpret the basic reproduction number, we rewrite $\mathscr{R}_{01}$ in the form:

$$\mathscr{R}_{01} = \sqrt{\beta_T \gamma_b \frac{\Lambda_H}{\mu_h} \frac{\nu}{\mu_e} \frac{1}{\mu_h} \frac{\Lambda_C}{(\mu_b + \sigma_s)} \frac{\eta}{(\eta + \mu_b)} \frac{\alpha_b}{(\alpha_b + \varepsilon)}}. \tag{3.29}$$

The terms in Equation (3.29) can be interpreted as follows: ν/μ_e is the density of *T. saginata* eggs released by humans with taeniasis, $1/\mu_h$ is the human life expectancy and β_T is the probability of humans to be infected with taeniasis due to consumption of raw or insufficiently cooked beef that is infected with tapeworm larval cysts. The terms Λ_H/μ_h and $\Lambda_C/(\mu_b + \sigma_s)$ are initial populations of susceptible humans and susceptible cattle respectively; $1/(\mu_b + \sigma_s)$ is the average time the cattle spends in susceptible class; $1/(\eta + \mu_b)$ is the infectious period of infected cattle; $1/(\alpha_b + \varepsilon)$ is the average infectious period for infected beef whereas $\eta/(\eta + \mu_b)$ is the proportion of infected cattle that are slaughtered for consumption. The term $\alpha_b/(\alpha_b + \varepsilon)$ is the proportion of infected beef that is eaten by susceptible humans whereas γ_b is the rate at which *T. saginata* eggs are consumed by cattle from the contaminated environment. Generally, the basic reproduction number $\mathscr{R}_{01}$ will increase in proportion to slaughter rate of infected cattle, the rate of eating undercooked infected beef, probability of human to acquire taeniasis, human and cattle recruitment rates, defecation rate by humans with taeniasis and the rate at which *T. saginata* eggs are consumed whereas $\mathscr{R}_{01}$ decreases when the slaughter rate of sus-

ceptible cattle, rate of unconsumed infected beef and the mortality rates for humans, cattle and *T. saginata* eggs are increased.

3.5.3 Parameter Estimation for the Basic Taenia Saginata Model

In this section, we estimate parameter values for the *Taenia saginata* basic model by linking the formulated model with human taeniasis cases in Debre Birhan City of Central Ethiopia from the year 2013 to 2017 (Yimer & Gebrmedehan, 2019) as shown in Table 2. The method of maximum likelihood estimator (MLE) is implemented to estimate model parameters. In this method, we define the likelihood function $f(\theta)$ which gives the sum of squares of residual by:

$$f(\theta) = \sum_{j=1}^{n} \left(y_j - y_j^{est} \right)^2, \tag{3.30}$$

where y_j and y_j^{est} are the real data and the solution of model equations (3.1) respectively for $j = 1, 2, .., n$.

Table 2: Humans with *T. saginata* infection in Debre Birhan City, Ethiopia (2013-2017)

Year	No. of Stool Examined	No. of *T. saginata* Positive
2013	852	37
2014	656	24
2015	525	19
2016	232	9
2017	219	8

The MLE approach is uses to fit data in Table 2 into the model system (3.1). The graphical representation of fitted data is shown in Fig. 6 and the corresponding fitted parameter values are indicated in Table 3.

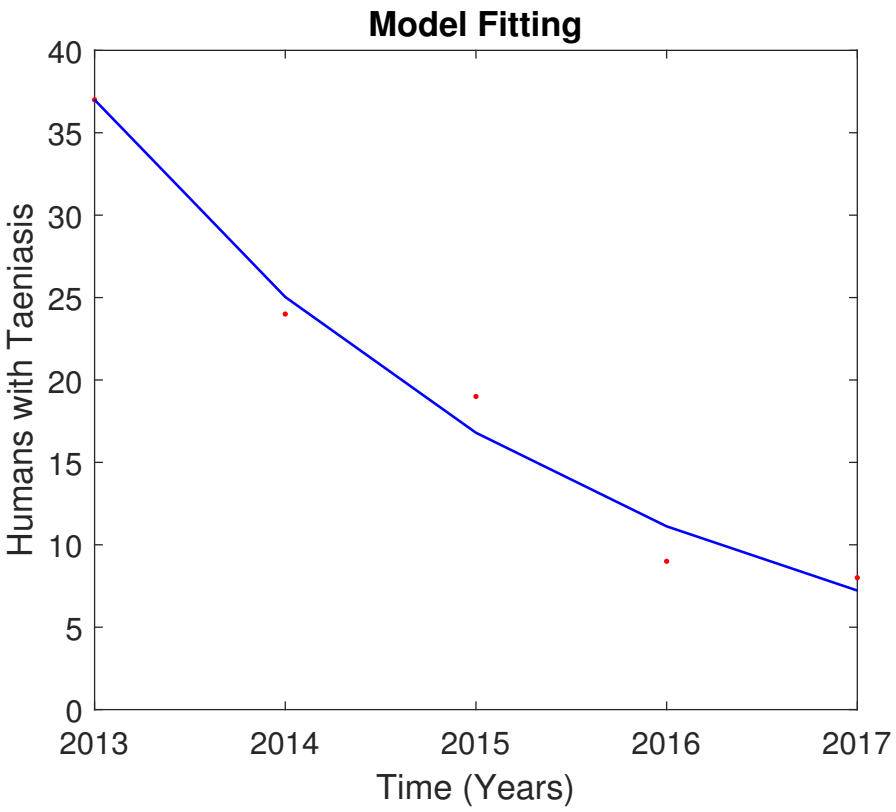


Figure 6: Fitted data for humans with *T. saginata* infection in Debre Birhan City, Ethiopia

Table 3: Parameter values

Parameter	Value	Unit	Source	Parameter	Value	Unit	Source
Λ_H	4,627	yr^{-1}	Fitted	Λ_C	562	yr^{-1}	Fitted
μ_h	0.0126	yr^{-1}	Fitted	γ_b	0.0053	yr^{-1}	Fitted
α_b	0.2300	yr^{-1}	Assumed	ρ_b	0.2177	yr^{-1}	Fitted
η	0.0871	yr^{-1}	Fitted	μ_b	0.4491	yr^{-1}	Fitted
β_T	0.0930	-	Assumed	ν	0.1777	yr^{-1}	Fitted
μ_e	9.0762	yr^{-1}	Fitted	σ_s	0.1959	yr^{-1}	Fitted
σ_v	0.1761	yr^{-1}	Fitted	σ_r	0.2180	yr^{-1}	Fitted
λ	0.1481	yr^{-1}	Fitted	π	0.3098	yr^{-1}	Fitted
ψ_s	0.1244	yr^{-1}	Fitted	ψ_v	0.2577	yr^{-1}	Fitted
τ	0.3470	yr^{-1}	Fitted	c_h	0.0669	yr^{-1}	Fitted
κ	0.5416	-	Fitted	χ	0.3756	yr^{-1}	Fitted
ε	0.3158	yr^{-1}	Fitted				

3.5.4 Sensitivity Analysis of the Basic Reproduction Number $\mathscr{R}_{01}$

The normalized forward sensitivity index method as described by Chitnis *et al.* (2008), is adopted to determine the most sensitive parameters. Suppose δ is a parameter in $\mathscr{R}_{01}$ then its sensitivity index is given by:

$$\Gamma_{\delta}^{\mathscr{R}_{01}} = \frac{\partial \mathscr{R}_{01}}{\partial \delta} \times \frac{\delta}{\mathscr{R}_{01}}. \tag{3.31}$$

Using Equation (3.31) and parameter values in Table 3, we obtain sensivity indices for each parameter as shown in Table 4. The positive sign of sensitivity index indicates that an increase (decrease) of parameter value while keeping other parameters constant increases (decreases) the magnitude of expected new infections whereas the negative sign indicates that an increase (decrease) of parameter value causes a decrease (increase) in expected new infections.

Table 4: Sensitivity indices

Parameter	Sensitivity Index	Parameter	Sensitivity Index
Λ_H	+ 0.5000	Λ_C	+ 0.5000
β_T	+ 0.5000	μ_e	- 0.5000
γ_b	+ 0.5000	η	+ 0.3956
ν	+ 0.5000	σ_s	- 0.1863
α_b	+ 0.2893	ε	- 0.2893

Since it is not biologically feasible and not practical to increase the natural mortality rates of populations, sensitivity indices for natural deaths of human and cattle populations have not been included in Table 4. The most positive sensitive parameters in the model system (3.18) are *T. saginata* eggs to cattle transmission coefficient (γ_b), human and cattle recruitment rates (Λ_H and Λ_C respectively), the probability of humans infection with taeniasis (β_T) and the open defecation rate by humans who are infected with taeniasis (ν) whereas the most negative sensitive parameter is the mortality rate of *T. saginata* eggs (μ_e) in the environment. Therefore, to control *Taenia saginata* bovine cysticercosis and human taeniasis, more efforts should be directed to the construction and use of toilets, meat inspection and killing *T. saginata* eggs from contaminated environment through spraying chemicals.

3.5.5 The Endemic Equilibrium

The endemic equilibrium is a point at which human taeniasis and bovine cysticercosis exist in humans and cattle respectively. It is given by $E^* = (S_H^*, I_{HT}^*, S_C^*, I_C^*, B_I^*, E_T^*)$, where:

$$\begin{aligned}
S_H^* &= \frac{\Lambda_H(\eta+\mu_b)(\varepsilon+\alpha_b)(\gamma_b E_T^* + \sigma_s + \mu_b)}{\beta_T \eta \alpha_b \gamma_b \Lambda_C E_T^* + \mu_h(\eta+\mu_b)(\varepsilon+\alpha_b)(\gamma_b E_T^* + \sigma_s + \mu_b)}, \\
I_{HT}^* &= \frac{\beta_T \eta \alpha_b \gamma_b \Lambda_C \Lambda_H E_T^*}{\mu_h \beta_T \eta \alpha_b \gamma_b \Lambda_C + \mu_h(\eta+\mu_b)(\varepsilon+\alpha_b)(\gamma_b E_T^* + \sigma_s + \mu_b)}, \\
S_C^* &= \frac{\Lambda_C}{(\gamma_b E_T^* + \sigma_s + \mu_b)}, \\
I_C^* &= \frac{\gamma_b \Lambda_B E_T^*}{(\eta+\mu_b)(\gamma_b E_T^* + \sigma_s + \mu_b)}, \\
B_I^* &= \frac{\eta \gamma_b \Lambda_C E_T^*}{(\eta+\mu_b)(\varepsilon+\alpha_b)(\gamma_b E_T^* + \sigma_s + \mu_b)}, \\
E_T^* &= \frac{\nu \Lambda_H (\sigma_s+\mu_b)(\mathscr{R}_{01}+1)(\mathscr{R}_{01}-1)}{\mathscr{R}_{01}^2 \mu_e \mu_h (\sigma_s+\mu_b) + \gamma_b \nu \Lambda_H}.
\end{aligned} \tag{3.32}$$

The model system (3.18) has a unique endemic equilibrium whenever $\mathscr{R}_{01} > 1$. This result is summarized in Theorem 3.3.

Theorem 3.3
The model system of Equations (3.18) has a unique endemic equilibrium when the basic reproduction number $\mathscr{R}_{01} > 1$.

3.6 Stability Analysis of Equilibrium Points

3.6.1 The Global Stability of Disease Free Equilibrium E^0

Theorem 3.4
The disease free equilibrium E^0 of the model system (3.18) is globally asymptotically stable whenever $\mathscr{R}_{01} < 1$.

Proof. Let $\mathscr{L}$ be the Lyapunov function given by:

$$\mathscr{L} = I_{HT} + \frac{\mu_h}{\nu} E_T + \frac{\beta_T \alpha_b \eta \Lambda_H}{\mu_h(\varepsilon+\mu_b)(\eta+\mu_b)} I_C + \frac{\beta_T \alpha_b \Lambda_H}{\mu_h(\varepsilon+\mu_b)} B_I, \tag{3.33}$$

The time derivative of Lyapunov function $\mathscr{L}$ is:

$$\begin{aligned}
\frac{d\mathscr{L}}{dt} &= \frac{dI_{HT}}{dt} + \frac{\mu_h}{\nu}\frac{dE_T}{dt} + \frac{\beta_T\alpha_b\eta\Lambda_H}{\mu_h(\varepsilon+\mu_b)(\eta+\mu_b)}\frac{dI_C}{dt} + \frac{\beta_T\alpha_b\Lambda_H}{\mu_h(\varepsilon+\mu_b)}\frac{dB_I}{dt}, \\
&= \beta_T\alpha_b B_I S_H - \mu_h I_{HT} + \frac{\mu_h}{\nu}\Big(\nu I_{HT} - \mu_e E_T\Big) \\
&\quad + \frac{\beta_T\alpha_b\eta\Lambda_H}{\mu_h(\varepsilon+\mu_b)(\eta+\mu_b)}\Big(\gamma_b S_C E_T - (\eta+\mu_b)I_C\Big) + \frac{\beta_T\alpha_b\Lambda_H}{\mu_h(\varepsilon+\mu_b)}\Big(\eta I_C - (\varepsilon+\alpha_b)B_I\Big), \\
&\leq \frac{\beta_T\alpha_b\Lambda_H}{\mu_h}B_I - \mu_h I_{HT} + \mu_h I_{HT} - \frac{\mu_h\mu_e}{\nu}E_T + \frac{\gamma_b\beta_T\alpha_b\eta\Lambda_H\Lambda_C}{\mu_h(\varepsilon+\mu_b)(\eta+\mu_b)(\sigma_s+\mu_b)}E_T \\
&\quad - \frac{\beta_T\alpha_b\eta\Lambda_H}{\mu_h(\varepsilon+\mu_b)}I_C + \frac{\beta_T\alpha_b\eta\Lambda_H}{\mu_h(\varepsilon+\mu_b)}I_C - \frac{\beta_T\alpha_b\Lambda_H}{\mu_h}B_I, \\
&= -\frac{\mu_h\mu_e}{\nu}E_T + \frac{\gamma_b\beta_T\alpha_b\eta\Lambda_H\Lambda_C}{\mu_h(\varepsilon+\mu_b)(\eta+\mu_b)(\sigma_s+\mu_b)}E_T, \\
&= -\frac{\mu_h\mu_e}{\nu}(1-R_0^2)E_T, \\
\frac{d\mathscr{L}}{dt} &= -\frac{\mu_h\mu_e}{\nu}(1-\mathscr{R}_{01})(1+\mathscr{R}_{01})E_T.
\end{aligned} \tag{3.34}$$

Thus, $\frac{d\mathscr{L}}{dt} < 0$ when $\mathscr{R}_{01} < 1$ and $E_T > 0$. However, $\frac{d\mathscr{L}}{dt} = 0$ whenever $\mathscr{R}_{01} = 1$ or $E_T = 0$. Thus, the largest invariant set $(S_H, I_{HT}, S_C, I_C, B_I, E_T) \in \Pi$, whenever $\mathscr{R}_{01} \leq 1$, is the singleton disease free equilibrium E^0 where Π is given in Equation (3.19). Therefore, from the LaSalle's invariance principle (LaSalle, 1968), the disease free equilibrium E^0 is globally asymptotically stable in the region Π. □

3.6.2 Global Stability of Endemic Equilibrium E^*

Theorem 3.5

The endemic equilibrium E^* of the model system (3.18) is globally asymptotically stable whenever $\mathscr{R}_{01} > 1$.

Proof. We apply the theory of cooperate systems to prove the global stability of endemic equilibrium E^* (Zhao & Jing, 1996; Wang *et al.*, 2013). Assuming that $H(0) < \Lambda_H/\mu_h$ and $T_C(0) < \Lambda_C/(\sigma_s+\mu_b)$, it can be observed from the model system (3.18) that $H = S_H + I_{HT} \to \Lambda_H/\mu_h$ and $T_C = S_C + I_C \to \Lambda_C/(\sigma_s+\mu_b)$ respectively as $t \to \infty$. Therefore S_H and S_C can be expressed by $\Lambda_H/\mu_h - I_{HT}$ and $\Lambda_B/(\sigma_s+\mu_b) - I_C$ respectively. Then, the model system (3.18) reduces to system:

$$
\begin{aligned}
\frac{dI_{HT}}{dt} &= \beta_T \alpha_b B_I \Big(\frac{\Lambda_H}{\mu_h} - I_{HT}\Big) - \mu_h I_{HT},\\
\frac{dI_C}{dt} &= \gamma_b \Big(\frac{\Lambda_H}{\sigma_s + \mu_b} - I_C\Big) E_T - (\eta + \mu_b) I_C,\\
\frac{dB_I}{dt} &= \eta I_C - (\varepsilon + \alpha_b) B_I,\\
\frac{dE_T}{dt} &= \nu I_{HT} - \mu_e E_T.
\end{aligned}
\tag{3.35}
$$

Thus, the dynamics of system (3.35) can be analyzed in the region:

$$
\begin{aligned}
\Pi = \Big\{ (I_{HT}, I_C, B_I, E_T) \in \mathbb{R}^4_+ : 0 \le I_{HT} \le \frac{\Lambda_H}{\mu_h}; 0 \le I_B \le \frac{\Lambda_C}{(\sigma_s + \mu_b)}; 0 \le E_T \le \frac{\nu \Lambda_H}{\mu_h \mu_e};\\
0 \le B_I \le \frac{\eta \Lambda_C}{(\varepsilon + \alpha_b)(\sigma_s + \mu_b)} \Big\}.
\end{aligned}
\tag{3.36}
$$

Therefore, we only need to prove the assumption in Corollary 3.2 (Zhao & Jing, 1996) for system (3.35).

Let

$$
f(u) = \begin{pmatrix} f_1(u_1,u_2,u_3,u_4) \\ f_2(u_1,u_2,u_3,u_4) \\ f_3(u_1,u_2,u_3,u_4) \\ f_4(u_1,u_2,u_3,u_4) \end{pmatrix} = \begin{pmatrix} \beta_T \alpha_b \Big(\frac{\Lambda_H}{\mu_h} - u_1\Big) u_3 - \mu_h u_1 \\ \gamma_b \Big(\frac{\Lambda_C}{\sigma_s + \mu_b} - u_2\Big) u_4 - (\eta + \mu_b) u_2 \\ \eta u_2 - (\varepsilon + \alpha_b) u_3 \\ \nu u_1 - \mu_e u_4 \end{pmatrix},
\tag{3.37}
$$

then $f : \mathbb{R}^4_+ \to \mathbb{R}^4_+$ is a mapping which is continuously differentiable in the region Π. It can be observed that $f(0) = 0$ and $f_i(u) \ge 0$ for all $u \in \Pi$ with $u_i = 0$, $i = 1,2,3,4$. Therefore, f is a cooperative system since $\partial f_i / \partial u_j \ge 0$, $(i \ne j)$ for $u \in \Pi$. Thus, for every $\alpha \in (0,1)$ and $u \in \Pi$, we have:

$$
\begin{aligned}
f_1(\alpha u_1, \alpha u_2, \alpha u_3, \alpha u_4) &= \beta_T \alpha_b \Big(\frac{\Lambda_H}{\mu_h} - \alpha u_1\Big) \alpha u_3 - \mu_h \alpha u_1,\\
&\ge \beta_T \alpha_b \Big(\frac{\Lambda_H}{\mu_h} - u_1\Big) \alpha u_3 - \mu_h \alpha u_1,\\
&= \alpha f_1(u_1,u_2,u_3,u_4).
\end{aligned}
\tag{3.38}
$$

With the same argument, we can verify that:

$$
\begin{aligned}
f_2(\alpha u_1, \alpha u_2, \alpha u_3, \alpha u_4) &\ge \alpha f_2(u_1,u_2,u_3,u_4),\\
f_3(\alpha u_1, \alpha u_2, \alpha u_3, \alpha u_4) &\ge \alpha f_3(u_1,u_2,u_3,u_4),\\
f_4(\alpha u_1, \alpha u_2, \alpha u_3, \alpha u_4) &\ge \alpha f_4(u_1,u_2,u_3,u_4).
\end{aligned}
\tag{3.39}
$$

Therefore, f is strictly sublinear on Π. Computing $Df(u)$, we obtain:

$$Df(u) = \begin{pmatrix} -\mu_h - \beta_T \alpha_b u_3 & 0 & \beta_T \alpha_b \left(\frac{\Lambda_H}{\mu_h} - u_1\right) & 0 \\ 0 & -(\eta + \mu_b) - \gamma_b u_4 & 0 & \gamma_b \left(\frac{\Lambda_C}{\sigma_s + \mu_b} - u_2\right) \\ 0 & \eta & -(\varepsilon + \alpha_b) & 0 \\ \nu & 0 & 0 & -\mu_e \end{pmatrix}, \tag{3.40}$$

where $Df(u) = \left(\partial f_i / \partial u_j\right)$ for $1 \leq i, j \leq 4$. Thus, $Df(u)$ is irreducible for $u \in \Pi$.

From equation (3.40), we obtain:

$$Df(0) = \begin{pmatrix} -\mu_h & 0 & \frac{\beta_T \alpha_b \Lambda_H}{\mu_h} & 0 \\ 0 & -(\eta + \mu_b) & 0 & \frac{\gamma_b \Lambda_C}{\sigma_s + \mu_b} \\ 0 & \eta & -(\varepsilon + \alpha_b) & 0 \\ \nu & 0 & 0 & -\mu_e \end{pmatrix}, \tag{3.41}$$

whose characteristic equation is:

$$\lambda^4 + c_3 \lambda^3 + c_2 \lambda^2 + c_1 \lambda + c_0 = 0, \tag{3.42}$$

whereby the coefficients c_i for $i = 0, 1, 2, 3$ are given as follows:

$$\begin{aligned} c_0 &= \mu_e \mu_h (\varepsilon + \alpha_b)(\eta + \mu_b)(1 + \mathscr{R}_{01})(1 - \mathscr{R}_{01}), \\ c_1 &= \mu_e (\varepsilon + \alpha_b)(\eta + \mu_b + \mu_h) + \mu_h (\varepsilon + \alpha_b + \mu_e)(\eta + \mu_b), \\ c_2 &= \mu_e (\varepsilon + \alpha_b) + (\eta + \mu_b + \mu_h)(\varepsilon + \alpha_b + \mu_e), \\ c_3 &= (\eta + \mu_b + \mu_h) + (\varepsilon + \alpha_b + \mu_e). \end{aligned} \tag{3.43}$$

It can be observed that $c_0 < 0$ when $\mathscr{R}_{01} > 1$. Hence, the spectrum of $Df(0)$ is given as $s(Df(0)) := max\left\{Re\ \lambda : det(\lambda I - Df(0))\right\} > 0$. Therefore, basing on the Corollary 3.2 (Zhao & Jing, 1996), we conclude that the equilibrium $(I^*_{HT}, I^*_C, B^*_I, E^*_T)$ of system (3.35) is globally asymptotically stable. Applying the theory of asymptotic autonomous systems in Thieme (1992), we further obtain that the endemic equilibrium $(S^*_H, I^*_{HT}, S^*_C, I^*_C, B^*_I, E^*_T)$ for model system (3.18) is globally asymptotically stable. □

3.7 Analysis of the Model with Interventions

In this section, we analyze the model system (3.1) that includes interventions with the aim of assessing the impact of these control measures on disease elimination or reduction. To assess the effectiveness of these control measures, we therefore, compare the effective reproduction number $\mathscr{R}_{e1}$ with the basic reproduction number $\mathscr{R}_{01}$.

3.7.1 Disease Free Equilibrium and Effective Reproduction Number $\mathscr{R}_{e1}$

When there is no taeniasis and cysticercosis in humans and cattle respectively, we obtain the disease free equilibrium P^0 for the model system (3.1) given by:

$$P^0\left(S_H, I_{HT}, S_C, V_C, I_C, R_C, B_I, E_T\right) = \left(\frac{\Lambda_H}{\mu_h}, 0, \frac{c_0\Lambda_C}{K_0}, \frac{\psi_s\Lambda_C}{K_0}, 0, 0, 0, 0\right), \tag{3.44}$$

where

$$c_0 = (\sigma_v + \psi_v + \mu_b) \text{ and } K_0 = c_0(\sigma_s + \mu_b) + \psi_s(\sigma_v + \mu_b). \tag{3.45}$$

The effective reproduction number $\mathscr{R}_{e1}$ is the expected number of secondary infections that may occur as a result of introducing one infected individual in a susceptible population when some interventions are implemented to control the spread of the disease (Diekmann *et al.*, 1990). When $\mathscr{R}_{e1} < 1$, the control measures are effective whereas when $\mathscr{R}_{e1} > 1$, then the control measures becomes less effective in diseases' control. The effective reproduction number $\mathscr{R}_{e1}$ for model system (3.1) given by:

$$\mathscr{R}_{e1} = \sqrt{\frac{(\sigma_v + \psi_v + \mu_b + \rho_b\psi_s)(1-\kappa)(1-\tau)\beta_T \nu \alpha_b \gamma_b \eta \Lambda_H \Lambda_C}{\Big((\sigma_v + \psi_v + \mu_b)(\sigma_s + \mu_b) + \psi_s(\sigma_v + \mu_b)\Big)\mu_h(\mu_h + \chi)(\mu_e + c_h)(\eta + \lambda + \mu_b)(\alpha_b + \varepsilon)}}. \tag{3.46}$$

It can be seen from Equation (3.46), that the implementation of control measures leads into the decrease of effective reproduction number $\mathscr{R}_{e1}$ and thus the possibility of controlling the diseases within short time. When there are no controls (that is $\tau = \kappa = \psi_s = \psi_v = \lambda = \chi = c_h = 0$), $\mathscr{R}_{e1}$ reduces to $\mathscr{R}_{01}$ where $\mathscr{R}_{01}$ is given by (3.28). Furthermore, it can be observed that the basic and the effective reproduction numbers ($\mathscr{R}_{01}$ and $\mathscr{R}_{e1}$) are closely related and their relationship is given by:

$$\mathscr{R}_{e1} = \mathscr{R}_{01}\sqrt{\frac{(\sigma_v + \psi_v + \mu_b + \rho_b\psi_s)\mu_e\mu_h(1-\kappa)(1-\tau)(\sigma_s + \mu_b)(\eta + \mu_b)}{\Big((\sigma_v + \psi_v + \mu_b)(\sigma_s + \mu_b) + \psi_s(\sigma_v + \mu_b)\Big)(\chi + \mu_h)(c_h + \mu_e)(\eta + \lambda + \mu_b)}}. \tag{3.47}$$

Since the implementation of control measures reduces number of secondary infections, thus it is can be concluded that

$$\sqrt{\frac{(\sigma_v+\psi_v+\mu_b+\rho_b\psi_s)\mu_e\mu_h(1-\kappa)(1-\tau)(\sigma_s+\mu_b)(\eta+\mu_b)}{\Big((\sigma_v+\psi_v+\mu_b)(\sigma_s+\mu_b)+\psi_s(\sigma_v+\mu_b)\Big)(\chi+\mu_h)(c_h+\mu_e)(\eta+\lambda+\mu_b)}}<1, \tag{3.48}$$

therefore, when control measures are implemented the basic reproduction $\mathscr{R}_{01}$ is reduced by the factor in (3.47). This is due to the fact that increasing the efforts to control human taeniasis and bovine cysticercosis result into the decrease in the magnitude of secondary infections. This situation is also well supported by simulation results in Chapter 4.

The Global Stability of the Disease Free Equilibrium P^0

Theorem 3.6

The disease free equilibrium P^0 of the model system (3.1) is globally asymptotically stable if $\mathscr{R}_{e1}<1$ and unstable otherwise.

Proof. Let $\mathscr{G}$ be the Lyapunov function given by:

$$\mathscr{G}=I_{HT}+\frac{(\mu_h+\chi)}{\nu(1-\tau)}E_T+\frac{\beta_T\alpha_b(1-k)\eta\Lambda_H}{\mu_h(\varepsilon+\mu_b)(\eta+\lambda+\mu_b)}I_C+\frac{\beta_T\alpha_b(1-k)\Lambda_H}{\mu_h(\varepsilon+\mu_b)}B_I. \tag{3.49}$$

The time derivative of the Lyapunov function $\mathscr{G}$ is:

$$\begin{aligned}\frac{d\mathscr{G}}{dt}&=\frac{dI_{HT}}{dt}+\frac{(\mu_h+\chi)}{(\nu(1-\tau))}\frac{dE_T}{dt}+\frac{\beta_T\alpha_b(1-k)\eta\Lambda_H}{\mu_h(\varepsilon+\mu_b)(\eta+\lambda+\mu_b)}\frac{dI_C}{dt}+\frac{\beta_T\alpha_b(1-k)\Lambda_H}{\mu_h(\varepsilon+\mu_b)}\frac{dB_I}{dt}\\&=\beta_T\alpha_b(1-\kappa)B_IS_H-(\mu_h+\chi)I_{HT}+\frac{(\mu_h+\chi)}{\nu(1-\tau)}\Big(\nu(1-\tau)I_{HT}-(\mu_e+c_h)E_T\Big)\\&\quad+\frac{\beta_T\alpha_b(1-k)\eta\Lambda_H}{\mu_h(\varepsilon+\mu_b)(\eta+\lambda+\mu_b)}\Big(\gamma_bS_CE_T+\rho_b\gamma_bV_CE_T-(\eta+\lambda+\mu_b)I_C\Big)\\&\quad+\frac{\beta_T\alpha_b(1-k)\Lambda_H}{\mu_h(\varepsilon+\mu_b)}\Big(\eta I_C-(\varepsilon+\alpha_b)B_I\Big),\end{aligned} \tag{3.50}$$

$$
\begin{aligned}
\frac{d\mathscr{G}}{dt} &\leq \frac{\beta_T\alpha_b(1-\kappa)\Lambda_H}{\mu_h}B_I-(\mu_h+\chi)I_{HT}+(\mu_h+\chi)I_{HT}-\frac{(\mu_h+\chi)(\mu_e+c_h)}{\nu(1-\tau)}E_T\\
&\quad+\frac{(c_0+\rho_b\psi_s)\gamma_b\beta_T\alpha_b(1-k)\eta\Lambda_H\Lambda_C}{K_0\mu_h(\varepsilon+\mu_b)(\eta+\lambda+\mu_b)}E_T-\frac{\beta_T\alpha_b(1-k)\eta\Lambda_H}{\mu_h(\varepsilon+\mu_b)}I_C+\frac{\beta_T\alpha_b(1-k)\eta\Lambda_H}{\mu_h(\varepsilon+\mu_b)}I_C\\
&\quad-\frac{\beta_T\alpha_b(1-k)\Lambda_H}{\mu_h}B_I,\\
&=-\frac{(\mu_h+\chi)(\mu_e+c_h)}{\nu(1-\tau)}E_T+\frac{(c_0+\rho_b\psi_s)\gamma_b\beta_T\alpha_b(1-k)\eta\Lambda_H\Lambda_C}{K_0\mu_h(\varepsilon+\mu_b)(\eta+\lambda+\mu_b)}E_T,\\
&=-\frac{(\mu_h+\chi)(\mu_e+c_h)}{\nu(1-\tau)}(1-\mathscr{R}_{e1}^2)E_T,\\
\frac{d\mathscr{G}}{dt} &=-\frac{(\mu_h+\chi)(\mu_e+c_h)}{\nu(1-\tau)}(1-\mathscr{R}_{e1})(1+\mathscr{R}_{e1})E_T.
\end{aligned}
\tag{3.51}
$$

Thus, $\frac{d\mathscr{G}}{dt}<0$ when $\mathscr{R}_{e1}<1$ and $E_T>0$. The derivative $\frac{d\mathscr{G}}{dt}=0$ when $\mathscr{R}_{e1}=1$ or $E_T=0$. Thus, the largest invariant set $(S_H,I_{HT},S_C,V_C,I_C,R_C,B_I,E_T)\in\Omega$, whenever $\mathscr{R}_{e1}\leq 1$, is the singleton disease free equilibrium P^0 where Ω is given in equation (3.16). Therefore, from the LaSalle's invariance principle (LaSalle, 1968), the disease free equilibrium P^0 is globally asymptotically stable in the region Ω. □

3.7.2 The Endemic Equilibrium P^*

In the presence of taeniasis and cysticercosis in humans and cattle respectively, we obtain the endemic equilibrium $P^*=(S_H^*,I_{HT}^*,S_C^*,V_C^*,I_C^*,R_C^*,B_I^*,E_T^*)$, for the model system (3.1) with:

$$
\begin{aligned}
S_H^* &= \frac{(\chi I_{HT}^*+\Lambda_H)(\varepsilon+\alpha_b)(h_2+\gamma_bE_T^*(h_1E_T^*+h_3))}{\mu_h(\varepsilon+\alpha_b)(h_2+\gamma_bE_T^*(h_1E_T^*+h_3))+h_0E_T^*(c_0+\rho_b\psi_s+\rho_b\gamma_bE_T^*)},\\
I_{HT}^* &= \frac{h_0\Lambda_HE_T^*(c_0+\rho_b\psi_s+\rho_b\gamma_bE_T^*)}{\mu_h\Big((\varepsilon+\alpha_b)(\chi+\mu_h)\Big(h_2+\gamma_bE_T^*(h_3+h_1\gamma_bE_T^*)\Big)+h_0E_T^*(c_0+\rho_b\psi_s+\rho_b\gamma_bE_T^*)\Big)},\\
S_C^* &= \frac{\Lambda_C(\eta+\lambda+\mu_b)(\pi+\sigma_r+\mu_b)(\rho_b\gamma_bE_T^*+c_0)}{h_2+\gamma_bE_T^*(h_3+h_1\gamma_bE_T^*)},\\
V_C^* &= \frac{\psi_s\Lambda_B(\eta+\lambda+\mu_b)(\pi+\sigma_r+\mu_b)}{h_2+\gamma_bE_T^*(h_3+h_1\gamma_bE_T^*)},\\
I_C^* &= \frac{\gamma_b\Lambda_BE_T^*(\pi+\sigma_r+\mu_b)(\rho_b\gamma_bE_T^*+c_0+\rho_b\psi_s)}{h_2+\gamma_bE_T^*(h_3+h_1\gamma_bE_T^*)},\\
R_C^* &= \frac{\lambda\gamma_b\Lambda_BE_T^*(\rho_b\gamma_bE_T^*+c_0+\rho_b\psi_s)}{h_2+\gamma_bE_T^*(h_3+h_1\gamma_bE_T^*)},\\
B_I^* &= \frac{\eta\gamma_b\Lambda_BE_T^*(\pi+\sigma_r+\mu_b)(\rho_b\gamma_bE_T^*+c_0+\rho_b\psi_s)}{(h_2+\gamma_bE_T^*(h_3+h_1\gamma_bE_T^*)(\varepsilon+\alpha_b)},
\end{aligned}
\tag{3.52}
$$

where

$$
\begin{aligned}
c_0 &= (\sigma_v + \psi_v + \mu_b) > 0,\\
h_0 &= (1-\tau)\eta\alpha_b\beta_T\gamma_b\Lambda_C(\pi + \mu_b + \sigma_r) > 0,\\
h_1 &= \rho_b(\pi\eta + \mu_b(\eta + \pi + \lambda + \mu_b) + (\eta + \lambda + \mu_b)\sigma_r) > 0,\\
h_2 &= (\eta + \lambda + \mu_b)(\pi + \mu_b + \sigma_r)((\mu_b + \sigma_v)(\mu_b + \sigma_s + \psi_s) + (\mu_b + \sigma_s)\psi_v) > 0,\\
h_3 &= \mu_b^3(1-\rho_b) + \rho_b((\eta + \lambda)(\pi + \sigma_r)\sigma_s + (\eta\pi + (\eta + \lambda)\sigma_r)\psi_s) + (\eta\pi + (\eta + \lambda)\sigma_r)(\sigma_v + \psi_v)\\
&\quad + \mu_b^2(\eta + \pi + \lambda + \sigma_r + \sigma_v + \rho_b(\eta + \pi + \lambda + \sigma_r + \sigma_s + \psi_s) + \psi_v) + \mu_b(\mathscr{D} + \mathscr{O}) > 0,\\
\mathscr{D} &= \rho_b(\pi(\eta + \lambda) + (\eta + \pi + \lambda)(\sigma_s + \psi_s) + \sigma_r(\eta + \lambda + \sigma_s + \psi_s)) > 0,\\
\mathscr{O} &= \pi\eta + (\eta + \pi + \lambda) + (\sigma_v + \psi_v) + \sigma_r(\eta + \lambda + \sigma_v + \psi_v) > 0.
\end{aligned}
\tag{3.53}
$$

To obtain E_T^*, we solve for the positive real roots of the quadratic equation:

$$
\mathscr{A}E_T^{*2} + \mathscr{B}E_T^* + \mathscr{C} = 0, \tag{3.54}
$$

where

$$
\begin{aligned}
\mathscr{A} &= (c_h + \mu_e)\mu_h\gamma_b(\rho_b h_0 + \gamma_b h_1(\varepsilon + \alpha_b)(\chi + \mu_h)) > 0,\\
\mathscr{B} &= \mu_h(c_h + \mu_e)\Big((c_0 + \rho_b\psi_s)h_0 + \gamma_b(\varepsilon + \alpha_b)(\chi + \mu_h)h_3\Big) - h_0(1-\tau)\nu\rho_b\psi_s\Lambda_H,\\
\mathscr{C} &= h_2(\varepsilon + \alpha_b)(\chi + \mu_h)(1 + \mathscr{R}_{e1})(1 - \mathscr{R}_{e1}).
\end{aligned}
\tag{3.55}
$$

We apply the approach in Levin (2002), Madubueze *et al.* (2015) and Wu *et al.* (2013) to determine the existence of positive real roots for Equation (3.54) depending on the signs of $\mathscr{B}$ and $\mathscr{C}$, since $\mathscr{A}$ is always positive.

(i) If $\mathscr{B} < 0$, $\mathscr{C} = 0$ or $\mathscr{B}^2 - 4\mathscr{A}\mathscr{C} = 0$, then Equation (3.54) has a unique positive root, which provides an opportunity for existence of a unique endemic equilibrium for the model system (3.1).

(ii) If $\mathscr{C} < 0$ and $\mathscr{B}^2 - 4\mathscr{A}\mathscr{C} > 0$, then there exists one positive real root for equation (3.54) which leads to emergence of a unique endemic equilibrium for the model system (3.1).

(iii) If $\mathscr{C} > 0$, $\mathscr{B} < 0$ and $\mathscr{B}^2 - 4\mathscr{A}\mathscr{C} > 0$, then Equation (3.54) has two positive real root, leading into the existence of two endemic equilibria for the model system (3.1). This may result into the emergence of backward bifurcation. However, forward bifurcation may occur depending on the conditions outlined in Theorem 3.7.

Theorem 3.7
The model system of equations (3.1) has a unique endemic equilibria when the effective reproduction number $\mathscr{R}_{e1} > 1$.

Cases (i) - (iii) above indicate that there exist an interval of $\mathscr{R}_{e1}$ for which equation (3.54) has two positive equilibria:

$$E^*_{T_1} = \frac{-\mathscr{B} - \sqrt{\mathscr{B}^2 - 4\mathscr{A}\mathscr{C}}}{2\mathscr{A}}\ ,\ E^*_{T_2} = \frac{-\mathscr{B} + \sqrt{\mathscr{B}^2 - 4\mathscr{A}\mathscr{C}}}{2\mathscr{A}}. \tag{3.56}$$

If we set the discriminant $\mathscr{B}^2 - 4\mathscr{A}\mathscr{C} = 0$ and solve for the critical points of R_e, we obtain:

$$R_c = \sqrt{1 - \frac{\mathscr{B}^2}{4\mathscr{A}h_2(\varepsilon + \alpha_b)(\chi + \mu_h)}}. \tag{3.57}$$

When the discriminant $\mathscr{B}^2 - 4\mathscr{A}\mathscr{C} > 0$, then $R_c < R_e$. Therefore, there is a possibility of emergence of backward bifurcation when $R_c < \mathscr{R}_{e1} < 1$.

3.7.3 Bifurcation Analysis

The presence of vaccination or immunity in a population can cause some epidemiological models to become bi-stable such that $\mathscr{R}_{e1} < 1$ may not be sufficient for elimination of the disease (Garba *et al.*, 2008). However, $\mathscr{R}_{e1} < 1$ is necessary for avoiding an epidemic due to few infectives that were initially introduced in the population (Chitnis *et al.*, 2006). Other models may become bi-stable due to change of stability at the bifurcation point that occurs when $\mathscr{R}_{e1} = 1$ (Madubueze *et al.*, 2015). Bifurcation analysis is therefore a very useful tool for proving the bi-stability of model equilibria. Generally, the bifurcation can be forward or backward depending on the direction of the bifurcation parameter. When the bifurcation is forward then the disease free equilibrium is locally asymptotically stable whenever $\mathscr{R}_{e1} < 1$, such that the disease does not persist in the population and the endemic equilibrium becomes locally asymptotically stable whenever $\mathscr{R}_{e1} > 1$ (Madubueze *et al.*, 2015). When the bifurcation is backward, then the endemic equilibrium occurs when $\mathscr{R}_{e1} < 1$ and the disease free equilibrium may exist whenever $R_{e1} > 1$. Therefore, we apply the center manifold theory as used in Castillo-Chavez & Song (2004) to analyze the bifurcation condition for the dynamics of *Taenia saginata* bovine cysticercosis and human taeniasis, and the local stability of the endemic equilibrium around $\mathscr{R}_{e1} = 1$.

Theorem 3.8

Consider a system of ordinary differential equations (Castillo-Chavez & Song, 2004):

$$\frac{dx}{dt} = f(x,\beta), f : \mathbb{R}^n \mapsto \mathbb{R}^n \text{ and } f \in C^2(\mathbb{R}^n \times \mathbb{R}), \tag{3.58}$$

where β is a bifurcation parameter.

1. Let $D_x f(0,0) = \dfrac{\partial f_i}{\partial x_j}(0,0)$ be the linearization matrix of system (3.1) around the equilibrium 0 with β evaluated at 0. Zero is a simple eigenvalue of $D_x f(0,0)$ and all other eigenvalues have negative real parts.

2. The linearization matrix $D_x f(0,0)$ has a right eigenvector ω and a left eigenvector v corresponding to the zero eigenvalue.

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

$$a = \sum_{k,i,j=1}^{n} v_k \omega_i \omega_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0); \;\; b = \sum_{k,i=1}^{n} v_k \omega_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}(0,0). \tag{3.59}$$

i. If $a > 0$ and $b > 0$, then equilibrium 0 is locally asymptotically stable, and there exists unstable positive equilibrium when $\beta < 0$ with $|\beta| << 1$. In this case, the direction of bifurcation at $\beta = 0$ is backward.

ii. If $a < 0$, $b > 0$ and $\beta > 0$ with $|\beta| << 1$, then equilibrium 0 becomes unstable, and there exists a stable positive equilibrium which is locally asymptotically stable. In this case, the direction of bifurcation at $\beta = 0$ is forward.

The linearization matrix $D_x f(0,0)$ of model system (3.1) at the disease free equilibrium P^0 is:

$$\begin{pmatrix}
-\mu_h & \chi & 0 & 0 & 0 & 0 & -\beta_T \alpha_b (1-\kappa)\Lambda_H/\mu_h & 0 \\
0 & -(\mu_h+\chi) & 0 & 0 & 0 & 0 & \beta_T \alpha_b (1-\kappa)\Lambda_H/\mu_h & 0 \\
0 & 0 & -N_1 & \psi_v & 0 & \pi & 0 & -\gamma_b c_0 \Lambda_C/K_0 \\
0 & 0 & \psi_s & -c_0 & 0 & 0 & 0 & -\rho_b \gamma_b \Lambda_C/K_0 \\
0 & 0 & 0 & 0 & -N_2 & 0 & 0 & \gamma_b \Lambda_C (c_0 + \rho_b \psi_s)/K_0 \\
0 & 0 & 0 & 0 & \lambda & -N_3 & 0 & 0 \\
0 & 0 & 0 & 0 & \eta & 0 & -(\varepsilon+\alpha_b) & 0 \\
0 & \nu(1-\tau) & 0 & 0 & 0 & 0 & 0 & -(\mu_e + c_h)
\end{pmatrix}, \tag{3.60}$$

where $N_1 = (\sigma_s + \psi_s + \mu_b),\ N_2 = (\eta + \lambda + \mu_b),\ N_3 = (\pi + \sigma_r + \mu_b)$. By choosing γ_b as the bifurcation parameter that occurs when $\mathscr{R}_{e1} = 1$ and solving for γ_b, we have:

$$\mathscr{R}_{e1}^2 = \frac{(c_0 + \rho_b\psi_s)(1-\kappa)(1-\tau)\beta_T \nu \alpha_b \gamma_b \eta \Lambda_H \Lambda_C}{K_0 \mu_h (\mu_h + \chi)(\mu_e + c_h)(\eta + \lambda + \mu_b)(\alpha_b + \varepsilon)} = 1, \tag{3.61}$$

Implying that:

$$\gamma_b = \frac{K_0 \mu_h (\mu_h + \chi)(\mu_e + c_h)(\eta + \lambda + \mu_b)(\alpha_b + \varepsilon)}{(c_0 + \rho_b\psi_s)(1-\kappa)(1-\tau)\beta_T \nu \alpha_b \eta \Lambda_H \Lambda_C}. \tag{3.62}$$

Thus, from Theorem 3.8, the linearization matrix (3.60) with γ_b evaluated at 0, has a simple zero eigenvalue and all other eigenvalues have negative real parts. Therefore, we are required to compute the quantities a and b by using the definition in (3.59).

The right and left eigenvectors (ω and v) of linearization matrix $D_x f(0,0)$ associated with zero eigenvalue are found when $D_x f(0,0)\ \omega = 0$ and $D_x f(0,0)^T v = 0$, where:

$$\omega = (\omega_1,\ \omega_2,\ \omega_3,\ \omega_4,\ \omega_5,\ \omega_6,\ \omega_7,\ \omega_8)^T \text{ and } v = (v_1,\ v_2,\ v_3,\ v_4,\ v_5,\ v_6,\ v_7,\ v_8)^T. \tag{3.63}$$

In so doing, we obtain:

$$\begin{aligned}
&\omega_1 = -\frac{(\mu_e + c_h)\omega_8}{\nu(1-\tau)},\ \omega_2 = \frac{(\mu_e + c_h)\omega_8}{\nu(1-\tau)},\ \omega_3 = \frac{(\sigma_v + \psi_v + \mu_b)\omega_4}{\psi_s},\\
&\omega_4 = \frac{\pi\psi_s\omega_5}{\psi_v\psi_s - c_0(\sigma_s + \psi_s + \mu_b)},\ \omega_5 = \frac{\gamma_b\Lambda_C(c_0 + \rho_b\psi_s)\omega_8}{K_0(\eta + \lambda + \mu_b)},\\
&\omega_6 = \frac{\lambda(\varepsilon + \alpha_b)\omega_7}{\eta(\pi + \sigma_r + \mu_b)},\ \omega_7 = \frac{\eta\,\omega_5}{(\varepsilon + \alpha_b)},\ \omega_8 > 0,\\
&v_1 = 0,\ v_2 = \frac{\nu(1-\tau)v_8}{(\mu_h + \chi)},\ v_3 = 0,\ v_4 = 0,\ v_5 = \frac{\eta v_7}{(\eta + \lambda + \mu_b)},\\
&v_6 = 0,\ v_7 = \frac{\beta_T\alpha_b(1-\kappa)\Lambda_H v_2}{\mu_h(\varepsilon + \alpha_b)},\ v_8 = \frac{\gamma_b\Lambda_C(c_0 + \rho_b\psi_s)v_5}{K_0(\mu_e + c_h)}.
\end{aligned} \tag{3.64}$$

The non-zero second partial derivatives of f_k at the disease free equilibrium P^0 are given as follows:

$$\begin{aligned}
&\frac{\partial^2 f_1}{\partial S_H \partial B_I} = -\beta_T\alpha_b(1-\kappa),\ \frac{\partial^2 f_2}{\partial S_H \partial B_I} = \beta_T\alpha_b(1-\kappa),\ \frac{\partial^2 f_3}{\partial S_C \partial E_T} = -\gamma_b,\\
&\frac{\partial^2 f_4}{\partial V_C \partial E_T} = -\rho_b\gamma_b,\ \frac{\partial^2 f_5}{\partial S_C \partial E_T} = \gamma_b,\ \frac{\partial^2 f_5}{\partial V_C \partial E_T} = \rho_b\gamma_b,\ \frac{\partial^2 f_3}{\partial E_T \partial \gamma_b} = \frac{c_0\Lambda_C}{K_0},\\
&\frac{\partial^2 f_4}{\partial E_T \partial \gamma_b} = -\frac{\rho_b\psi_s\Lambda_C}{K_0},\ \frac{\partial^2 f_5}{\partial E_T \partial \gamma_b} = \frac{(c_0 + \rho_b\psi_s)\Lambda_C}{K_0}.
\end{aligned} \tag{3.65}$$

Thus, we obtain:

$$
\begin{aligned}
a =& v_1\omega_1\omega_7\frac{\partial^2 f_1}{\partial S_H\partial B_I}+v_2\omega_1\omega_7\frac{\partial^2 f_2}{\partial S_H\partial B_I}+v_3\omega_3\omega_8\frac{\partial^2 f_3}{\partial S_C\partial E_T}+v_4\omega_4\omega_8\frac{\partial^2 f_4}{\partial V_C\partial E_T}\\
&+v_5\omega_3\omega_8\frac{\partial^2 f_5}{\partial S_C\partial E_T}+v_5\omega_4\omega_8\frac{\partial^2 f_5}{\partial V_C\partial E_T},\\
=& v_2\omega_1\omega_7\frac{\partial^2 f_2}{\partial S_H\partial B_I}+v_5\omega_3\omega_8\frac{\partial^2 f_5}{\partial S_C\partial E_T}+v_5\omega_4\omega_8\frac{\partial^2 f_5}{\partial V_C\partial E_T},\\
=& -\frac{\beta_T\alpha_b\eta(1-\kappa)(\mu_e+c_h)}{(\mu_h+\chi)(\varepsilon+\alpha_b)}v_8\omega_5\omega_8+\frac{\gamma_b(\sigma_v+\psi_v+\mu_b)\eta}{(\eta+\lambda+\mu_b)\psi_s}v_7\omega_4\omega_8+\frac{\rho_b\gamma_b\eta}{(\eta+\lambda+\mu_b)}v_7\omega_4\omega_8\\
=& -\frac{\beta_T\alpha_b\eta(1-\kappa)(\mu_e+c_h)}{(\mu_h+\chi)(\varepsilon+\alpha_b)}v_8\omega_5\omega_8+\frac{\gamma_b\eta(c_0+\rho_b\psi_s)}{(\eta+\lambda+\mu_b)\psi_s}v_7\omega_4\omega_8,\\
=& \frac{\beta_T\alpha_b\eta(1-\kappa)}{(\mu_h+\chi)(\varepsilon+\alpha_b)}\Big[\frac{\pi\nu(1-\tau)\Lambda_H\gamma_b(c_0+\rho_b\psi_s)}{\mu_h(\eta+\lambda+\mu_b)\Big(\psi_v\psi_s-c_0(\sigma_s+\psi_s+\mu_b)\Big)}-(\mu_e+c_h)\Big]v_8\omega_5\omega_8,\\
=& \frac{-\beta_T\alpha_b\eta(1-\kappa)}{(\mu_h+\chi)(\varepsilon+\alpha_b)}\Big[\frac{\pi\nu(1-\tau)\Lambda_H\gamma_b(c_0+\rho_b\psi_s)}{\mu_h(\eta+\lambda+\mu_b)m_0}+(\mu_e+c_h)\Big]v_8\omega_5\omega_8<0,\\
b =& v_3\omega_8\frac{\partial^2 f_3}{\partial E_T\partial\gamma_b}+v_4\omega_8\frac{\partial^2 f_4}{\partial E_T\partial\gamma_b}+v_5\omega_8\frac{\partial^2 f_5}{\partial E_T\partial\gamma_b}=\frac{\eta(c_0+\rho_b\psi_s)\Lambda_C}{K_0(\eta+\lambda+\mu_b)}v_7\omega_8>0,
\end{aligned}
\tag{3.66}
$$

where $m_0=(\sigma_v+\mu_b)(\sigma_s+\psi_s+\mu_b)+\psi_v(\sigma_s+\mu_b)>0$.

3.7.4 Bifurcation Diagram

Theorem 3.9

The model system (3.1) undergoes a forward bifurcation at $\mathscr{R}_{e1}=1$, where $\mathscr{R}_{e1}$ is the effective reproduction number. Figure 7 indicates the existence of forward bifurcation in model system (3.1). When $\gamma_b>0$ and $|\gamma_b|<<1$, then there exist unstable disease free equilibrium and the endemic equilibrium P^* which is locally asymptotically stable for $\mathscr{R}_{e1}>1$ near 1.

Forward bifurcation occurs when the disease free equilibrium looses its stability and a stable endemic equilibrium occurs as the effective reproduction number $\mathscr{R}_{e1}$ increases through one (Gumel, 2012). In other words, if the effective reproduction number $\mathscr{R}_{e1}<1$, then the disease free equilibrium exists and it is stable, meaning that human taeniasis and bovine cysticercosis will die out. When $\mathscr{R}_{e1}>1$, then the free equilibrium becomes unstable resulting in an endemic equilibrium, which indicates that human taeniasis and bovine cysticercosis will persist. Usually when forward bifurcation occurs, then the requirement $\mathscr{R}_{e1}<1$ is necessary and sufficient for effective disease control (Akrami & Atabaigi, 2020).

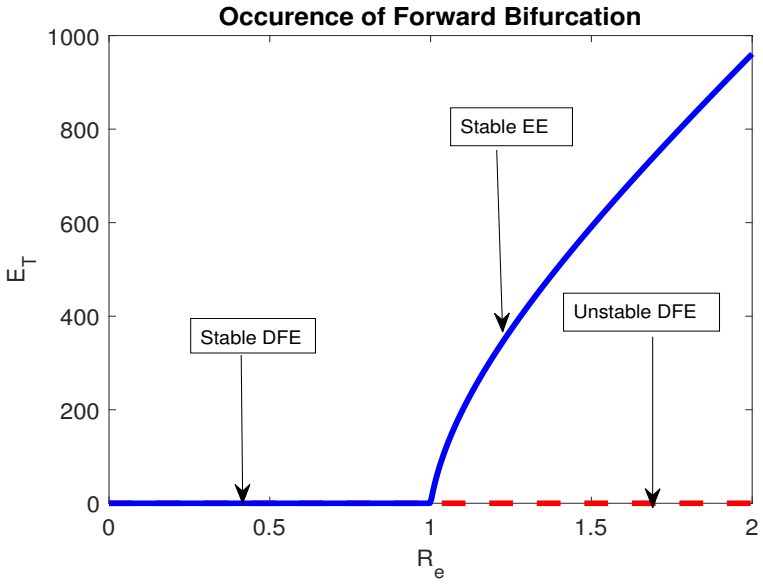


Figure 7: The plot of *T. saginata* eggs (E_T) against effective reproduction number $(\mathscr{R}_{e1})$

3.7.5 Global Stability of the Endemic Equilibrium P^*

Definition 3.1

A function $V(x)$ is called a Lyapunov function if it is continuous and differentiable for all values of $x \in \mathbb{U}$, satisfying the conditions:

(i) $V(0) = 0$,

(ii) $V(x) > 0 \; \forall x \in \mathbb{U} - \{0\}$,

(iii) $V'(x) \leq 0 \; \forall x \in \mathbb{U} - \{0\}$.

Theorem 3.10

The endemic equilibrium P^* of the model system (3.1) is globally asymptotically stable if the effective reproduction number $\mathscr{R}_{e1} > 1$.

Proof. To prove Theorem 3.10, we adopt the approach in Osman *et al.* (2020). Consider the non-linear function:

$$\begin{aligned}\mathscr{F} =& S_H^*\left(\frac{S_H}{S_H^*} - \ln\frac{S_H}{S_H^*}\right) + I_{HT}^*\left(\frac{I_{HT}}{I_{HT}^*} - \ln\frac{I_{HT}}{I_{HT}^*}\right) + S_C^*\left(\frac{S_C}{S_C^*} - \ln\frac{S_C}{S_C^*}\right) + V_C^*\left(\frac{V_C}{V_C^*} - \ln\frac{V_C}{V_C^*}\right) \\ &+ I_C^*\left(\frac{I_C}{I_C^*} - \ln\frac{I_C}{I_C^*}\right) + R_C^*\left(\frac{R_C}{R_C^*} - \ln\frac{R_C}{R_C^*}\right) + B_I^*\left(\frac{B_I}{B_I^*} - \ln\frac{B_I}{B_I^*}\right) + E_T^*\left(\frac{E_T}{E_T^*} - \ln\frac{E_T}{E_T^*}\right).\end{aligned} \tag{3.67}$$

It can be observed that the function $\mathscr{F}$ in (3.67) satisfies conditions (i) and (ii) in the Definition 3.1 for all $x \geq 0$ where $x = \{S_H, I_{HT}, S_C, V_C, I_C, R_C, B_I, E_T\}$ and $x*$ is the endemic equilibrium.

Hence a function $\mathscr{F}$ is a Lyapunov function. To prove if condition (iii) holds, we differentiate $\mathscr{F}$ along the solution of the model system (3.1) to obtain:

$$\begin{aligned}\frac{d\mathscr{F}}{dt} &= \left(1-\frac{S_H^*}{S_H}\right)\frac{dS_H}{dt} + \left(1-\frac{I_{HT}^*}{I_{HT}}\right)\frac{dI_{HT}}{dt} + \left(1-\frac{S_C^*}{S_C}\right)\frac{dS_C}{dt} + \left(1-\frac{V_C^*}{V_C}\right)\frac{dV_C}{dt} \\ &\quad + \left(1-\frac{I_C^*}{I_C}\right)\frac{dI_C}{dt} + \left(1-\frac{R_C^*}{R_C}\right)\frac{dR_C}{dt} + \left(1-\frac{B_I^*}{B_I}\right)\frac{dB_I}{dt} + \left(1-\frac{E_T^*}{E_T}\right)\frac{dE_T}{dt}.\end{aligned} \tag{3.68}$$

Substituting equations of model syatem (3.1) into equation (3.68), we have:

$$\begin{aligned}\frac{d\mathscr{F}}{dt} &= \left(1-\frac{S_H^*}{S_H}\right)\left(\Lambda_H + \chi I_{HT} - pB_I S_H - \mu_h S_H\right) + \left(1-\frac{I_{HT}^*}{I_{HT}}\right)\left(pB_I S_H - (\mu_h+\chi)I_{HT}\right) \\ &\quad + \left(1-\frac{S_C^*}{S_C}\right)\left(\Lambda_C + \psi_v V_C + \pi R_C - \gamma_b(1-\tau)S_C E_T - d_1 S_C\right) + \left(1-\frac{R_C^*}{R_C}\right)\left(\lambda I_C - d_3 R_C\right) \\ &\quad + \left(1-\frac{V_C^*}{V_C}\right)\left(\psi_s S_C - \rho\gamma_b(1-\tau)V_C E_T - d_2 V_C\right) + \left(1-\frac{B_I^*}{B_I}\right)\left(\eta I_B - (\varepsilon+\alpha_b)B_I\right) \\ &\quad + \left(1-\frac{I_C^*}{I_C}\right)\left(\gamma_b(1-\tau)S_C E_T + \rho\gamma_b(1-\tau)V_C E_T - d_4 I_C\right) \\ &\quad + \left(1-\frac{E_T^*}{E_T}\right)\left(\nu(1-\tau)I_{HT} - (\mu_e+c_h)E_T\right).\end{aligned} \tag{3.69}$$

If we let $p = \beta_T\alpha_b(1-\kappa), d_1 = (\sigma_s+\psi_s+\mu_b), d_2 = (\sigma_v+\psi_v+\mu_b), d_3 = (\pi+\sigma_r+\mu_b)$, $d_4 = (\eta+\lambda+\mu_b)$, then:

$$\begin{aligned}\frac{d\mathscr{F}}{dt} &= \Lambda_H + \chi I_{HT} - pB_I S_H - \mu_h S_H + pB_I S_H - (\mu_h+\chi)I_{HT} + \Lambda_C + \psi_v V_C + \pi R_C \\ &\quad - \gamma_b(1-\tau)S_C E_T - d_1 S_C + \psi_s S_C - \rho\gamma_b(1-\tau)V_C E_T - d_2 V_C + \gamma_b(1-\tau)S_C E_T \\ &\quad + \rho\gamma_b(1-\tau)V_C E_T - d_4 I_C + \lambda I_C - d_3 R_C + \eta I_B - (\varepsilon+\alpha_b)B_I + \nu(1-\tau)I_{HT} \\ &\quad - \frac{\Lambda_C S_C^*}{S_C} - (\mu_e+c_h)E_T - \frac{\Lambda_H S_H^*}{S_H} - \frac{\chi I_{HT} S_H^*}{S_H} + pB_I S_H^* + \mu_h S_H^* - \frac{pB_I S_H I_{HT}^*}{I_{HT}} \\ &\quad + (\mu_h+\chi)I_{HT}^* - \frac{\psi_v V_C S_C^*}{S_C} - \frac{\pi R_C S_C^*}{S_C} + \gamma_b(1-\tau)S_C^* E_T + d_1 S_C^* - \frac{\psi_s S_C V_C^*}{V_C} \\ &\quad + \rho\gamma_b(1-\tau)V_C^* E_T + d_2 V_C^* - \frac{\gamma_b(1-\tau)S_C E_T I_C^*}{I_C} - \frac{\rho\gamma_b(1-\tau)V_C E_T I_C^*}{I_C} + d_4 I_C^* \\ &\quad - \frac{\lambda I_C R_C^*}{R_C} + d_3 R_C^* - \frac{\eta I_B B_I^*}{B_I} + (\varepsilon+\alpha_b)B_I^* - \frac{\nu(1-\tau)I_{HT}E_T^*}{E_T} + (\mu_e+c_h)E_T^*.\end{aligned} \tag{3.70}$$

Equation (3.70) can be written as:

$$\frac{d\mathscr{F}}{dt} = \mathscr{M} - \mathscr{N}, \tag{3.71}$$

where

$$\begin{aligned}\mathscr{M} =&\Lambda_H + \chi I_{HT} + pB_I S_H + \Lambda_C + \psi_v V_C + \pi R_C + \psi_s S_C + \gamma_b(1-\tau)S_C E_T + \rho\gamma_b(1-\tau)V_C E_T \\ &+ \lambda I_C + \eta I_B + \nu(1-\tau)I_{HT} + pB_I S_H^* + \mu_h S_H^* + (\mu_h+\chi)I_{HT}^* + \gamma_b(1-\tau)S_C^* E_T + d_1 S_C^* \\ &+ \rho\gamma_b(1-\tau)V_C^* E_T + d_2 V_C^* + d_4 I_C^* + d_3 R_C^* + (\varepsilon+\alpha_b)B_I^* + (\mu_e+c_h)E_T^*, \end{aligned} \tag{3.72}$$

$$\begin{aligned}\mathscr{N} =&pB_I S_H + \mu_h S_H + (\mu_h+\chi)I_{HT} + \gamma_b(1-\tau)S_C E_T + d_1 S_C + \rho\gamma_b(1-\tau)V_C E_T + d_2 V_C \\ &+ d_4 I_C + d_3 R_C + (\varepsilon+\alpha_b)B_I + (\mu_e+c_h)E_T + \frac{\Lambda_H S_H^*}{S_H} + \frac{\chi I_{HT} S_H^*}{S_H} + \frac{pB_I S_H I_{HT}^*}{I_{HT}} + \frac{\Lambda_C S_C^*}{S_C} \\ &+ \frac{\psi_v V_C S_C^*}{S_C} + \frac{\pi R_C S_C^*}{S_C} + \frac{\psi_s S_C V_C^*}{V_C} + \frac{\gamma_b(1-\tau)S_C E_T I_C^*}{I_C} + \frac{\rho\gamma_b(1-\tau)V_C E_T I_C^*}{I_C} + \frac{\lambda I_C R_C^*}{R_C} \\ &+ \frac{\eta I_B B_I^*}{B_I} + \frac{\nu(1-\tau)I_{HT}E_T^*}{E_T}. \end{aligned} \tag{3.73}$$

It can be seen from Equation (3.71) that if $\mathscr{M} < \mathscr{N}$ then $\frac{d\mathscr{F}}{dt} < 0$ and if $\Omega = \Omega^*$ then $\frac{d\mathscr{F}}{dt} = 0$, where Ω is given in equation (3.2) and $\Omega^*$is the endemic equilibrium. Thus, the largest invariant set in Ω is the endemic equilibrium. Hence from the LaSalle's invariant principle (LaSalle, 1976), we can conclude that as $t \to \infty$, the solution of the model system (3.1) approaches the endemic equilibrium P^* when $\mathscr{R}_{e1} > 1$. Therefore, the endemic equilibrium P^* is globally asymptotically stable in the invariant set Ω if $\mathscr{M} < \mathscr{N}$.

3.8 Taenia Saginata CTMC Stochastic Model

To analyze the dynamics of Taenia saginata bovine cysticercosis and human taeniasis, it is important to predict whether the presence of a small number of infectious hosts or *T. saginata* eggs can result into the extinction or outbreak of cysticercosis in cattle and taeniasis in humans. In the deterministic model, the basic reproduction number $\mathscr{R}_{01}$ is very useful in determining whether the disease will die or persist in the population. If $\mathscr{R}_{01} > 1$ then the disease outbreak occurs whereas if $\mathscr{R}_{01} < 1$ the disease dies in the population. However in stochastic models, this is not realistic because if a small number of infectives is introduced in a fully susceptible population they may die, recover or be removed before transmitting the disease (Lahodny Jr *et al.*, 2015). To determine the probability of diseases' extinction or outbreak, it is important to consider a stochastic epidemic model. The best stochastic version of a deterministic model is the continuous time Markov chain (CTMC) model where time is continuous and the random variables are discrete. In CTMC model, it is possible to determine the stochastic threshold which is similar to the basic reproduction number for the deterministic model, however the stochastic threshold depends on initial number of infectives introduced in the susceptible populations.

A stochastic model plays a great role to determine the probability of disease extinction or outbreak. In deterministic model, the basic reproduction number is usually used to determine on average whether the presence of small number of infectious hosts will result into disease extinction or outbreak (Lahodny Jr *et al.*, 2015). Since the CTM model indicates the possibility of disease outbreak if the disease emerges from a certain infectious class, thus it gives useful information on the intervention measures to be considered for controlling the spread of the disease. A CTMC stochastic model is applied due to the fact it capture uncertinities that are inherent to disease transmission (Allen & Burgin, 1999). Generally, a CTMC stochastic model is more realistic as it describes a discrete movement of individuals between classes and not average rates as in a deterministic model (Maliyoni, 2021). In a CTMC stochastic model, the numbers are usually integers while a deterministic model assumes that there is a continuously changes in the values. In this section, we develop a continuous time markov chain (CTMC) model and apply multitype branching process to compute the probability of disease outbreak or extinction (Maliyoni, 2021).

3.8.1 Taenia Saginata CTMC Model Formulation

The continuous time Markov chain (CTMC) stochastic model is developed based on assumptions used in deterministic model (3.18). For simplicity, the same notations and parameters from deterministic model are used. Let S_H, I_{HT}, S_C and I_C denote the discrete random variables for susceptible humans, humans with taeniasis, susceptible cattle and the infected cattle respectively. The discrete random variables B_I and E_T represent infectious beef and number of *T. saginata* eggs in the environment respectively.

Let $\vec{Z}(t) = [S_H, I_{HT}, S_C, I_C, B_I, E_T]^T$ be the associated random vector for all discrete random variables. The CTMC model is assumed to be time-homogeneous and satisfies the Markov property that the future state of the process at time $(t + \Delta t)$ depends on the current state at time t (Allen, 2010; Maliyoni *et al.*, 2017). From the Markov assumptions, the time between events is exponentially distributed with parameter (Lahodny Jr *et al.*, 2015; Maliyoni *et al.*, 2017):

$$\begin{aligned}\psi(\vec{Z}) =& \Lambda_H + \mu_h N_H + \Lambda_C + \mu_b N_C + \nu I_{HT} + \sigma_s S_C + \eta I_C \\ &+ (\varepsilon + \alpha_b) B_I + \mu_e E_T + \beta_T \alpha_b B_I S_H + \gamma_b S_C E_T .\end{aligned} \tag{3.74}$$

In CTMC model, the events are their corresponding transition rates are usually derived from the deterministic model. The transitions of events are obtained by considering the recruitment and movement of an individual from one compartment to another when initially there is only one individual while other sub-populations do not exist.The events and transition rates are summarized in Table 5.

Table 5: State transitions and rates for the CTMC model

Event	Transition $\Delta\vec{Z}(t)$	Transition rate, p
Recruitment of S_H	$(1,0,0,0,0,0)$	Λ_H
Infection of S_H	$(-1,1,0,0,0,0)$	$\beta_T \alpha_b B_I S_H$
Natural death of S_H	$(-1,0,0,0,0,0)$	$\mu_h S_H$
Natural death of I_{TH}	$(0,-1,0,0,0,0)$	$\mu_h I_{TH}$
Shedding of E_T	$(0,0,0,0,0,1)$	νI_{TH}
Recruitment of S_C	$(0,0,1,0,0,0)$	Λ_C
Slaughter of S_C	$(0,0,-1,0,0,0)$	$\sigma_s S_C$
Infection of S_C	$(0,0,-1,1,0,0)$	$\gamma_b S_C E_T$
Natural death of S_C	$(0,0,-1,0,0,0)$	$\mu_b S_C$
Slaughter of I_C	$(0,0,0,-1,1,0)$	ηI_C
Natural death of I_C	$(0,0,0,-1,0,0)$	$\mu_b I_C$
Throwing of infectious beef B_I	$(0,0,0,0,-1,0)$	εB_I
Natural death of E_T	$(0,0,0,0,0,-1)$	$\mu_e E_T$

The value $1(-1)$ and 0 are respectively, an increase (decrease) by 1 and no change in state for the variable from time t to $(t+\Delta t)$.

3.8.2 The Multitype Branching Process

The theory of multitype branching process is used to approximate the dynamics of the nonlinear CTMC near the disease free equilibrium E^0 (Allen & Lahodny Jr, 2012; Allen & van den Driessche, 2013; Maliyoni *et al.*, 2017). The theory is useful in determining the probability of disease extinction or outbreak. If initially there are few infectives that are introduced in a susceptible population, the branching process either grows exponentially or hits zero (Allen, 2017). The multitype branching process is applied to infectious classes (infected humans, cattle, beef and the *T. saginata* eggs in the environment) which are the sources of infections. Both susceptible humans and cattle are assumed to be at the disease free equilibrium (DFE), that is $S_H^0 = \Lambda_H/\mu_h$ and $S_C^0 = \Lambda_C/(\sigma_s+\mu_b)$ (Lahodny & Allen, 2013). The term 'offspring' or 'birth' is used to describe susceptible humans and cattle that become infectious after consuming raw or undercooked infectious beef and *T. saginata* eggs in the contaminated environment respectively. We assume that infectious hosts of type i, I_i give birth to infectious individuals of type j, I_j and that the number of offspring produced by an individual of type i is independent of the number of offspring produced by either type i or type j, $j \neq i$ (Allen & van den Driessche, 2013; Maliyoni *et al.*, 2017). Furthermore, we assume that the initial susceptible humans and cattle are sufficiently large, that is $S_H(0) \approx N_H(0) = S_H^0$ and $S_C(0) \approx N_C(0) = S_C^0$ where $N_H(0)$ and $N_C(0)$ are the total human and cattle initial populations respectively. Since the multitype branching process is linear near the disease free equilibrium, then the number of births and deaths are

independent. The offspring probability generating functions (pgfs) for the birth and death of infectious humans, cattle, *T. saginata* eggs and infectious beef are defined. These offspring pgfs are then used to determine the probability of disease extinction or outbreak (Lahodny & Allen, 2013).

Let $\{Z_{ji}\}_{j=1}^{n}$ be the offspring random variable for type $i, i = 1,2,...,n$ infectious hosts where Z_{ji} is the number of offspring of type j produced by an infective of type i. The offspring pgf for infectious population I_i is defined if there is initially one infectious host at the beginning of the disease outbreak, say, $I(0) = 1$ and all other types are zero, $I_j = 0$. The offspring pgf $f_i : [0,1]^n \longrightarrow [0,1]$ for type i individuals given $I_i(0) = 1$ and $I_j(0) = 0, j \neq i$ is given as (Allen, 2010; Lahodny & Allen, 2013; Maliyoni, 2021):

$$f_i(u_1,u_2,...,u_n) = \sum_{k_n=0}^{\infty} ... \sum_{k_1=0}^{\infty} P_i(k_1,k_2,...,k_n)u_1^{k_1}...u_n^{k_n}, \tag{3.75}$$

where

$$P_i(k_1,k_2,...,k_n) = Prob\{Z_{1j} = k_1, Z_{2j} = k_2, ..., Z_{nj} = k_n\} \tag{3.76}$$

is the probability that one infectious individual of type i gives birth to k_j individuals of type j. We use equation (3.75) to define an $n \times n$ non-negative and irreducible expectation matrix $\mathbf{M}_1 = [m_{ji}]$ where m_{ji} is the expected number of offsprings for individuals of type j produced by an infective of type i. The elements of matrix $\mathbf{M_1}$ are determined by differentiating f_i with respect to u_j and then evaluating all the u variables at 1 (Lahodny Jr *et al.*, 2015; Maliyoni, 2021), that is:

$$m_{ji} = \left.\frac{\partial f_i}{\partial u_j}\right|_{\mathbf{u}=1} < \infty. \tag{3.77}$$

The probability of disease extinction or outbreak is determined by the size of the specrtal radius of the expectation matrix $\mathbf{M}_1$, $\rho(\mathbf{M}_1)$. If $\rho(\mathbf{M}_1) \leq 1$, then the probability of disease extinction is one, that is:

$$P_0 = \lim_{t \longrightarrow \infty} Prob\{\vec{I}(t) = \vec{0}\} = 1, \tag{3.78}$$

and if $\rho(\mathbf{M}_1) > 1$ then there exist a positive probability such that the probability of diseases' extinction is given by:

$$P_0 = \lim_{t \longrightarrow \infty} Prob\{\vec{I}(t) = \vec{0}\} = q_1^{i1} q_2^{i2} ... q_k^{ik} < 1, \tag{3.79}$$

where $(q_1,q_2,...,q_k)$ is the unique fixed point of the k offspring pgf, $f_i(q_1,q_2,...,q_k) = q_i$ and $0 < q_i < 1, i = 1,2,...,k$ (Lahodny & Allen, 2013; Lahodny Jr *et al.*, 2015; Maliyoni *et al.*,

2017). The probability of diseases' outbreak is (Maliyoni, 2021; Maliyoni *et al.*, 2017):

$$1 - P_0 = 1 - q_1^{i1} q_2^{i2} ... q_k^{ik}. \tag{3.80}$$

3.8.3 The Stochastic Threshold for Taenia Saginata CTMC Model

We apply the multitype branching process to the CTMC stochastic model by defining the probability generating functions for all infectious classes. The model assumes that the initial susceptible humans and cattle are at disease free equilibrium and sufficiently large such that $S_H(0) = \Lambda_H/\mu_h = S_H^0$ and $S_C(0) = \Lambda_C/(\sigma + \mu_b) = S_C^0$. If initially there was only one infectious human with taeniasis ($I_{HT}(0) = 1$) and no infectious cattle, beef and *T. saginata* eggs in the environment ($I_C(0) = 0, B_I(0) = 0$ and $E_T(0) = 0$ respectively), we define the offspring probability generating function (pgf) by using equation (3.75). Thus, the offspring probability generating function for I_{TH} is given by:

$$f_1(u_1, u_2, u_3, u_4) = \frac{\nu u_1 u_4 + \mu_h}{\nu + \mu_h}. \tag{3.81}$$

The term $\nu/(\nu + \mu_h)$ represents the probability that the initial infectious human sheds a *T. saginata* egg in the environment and the initial host does not die resulting into one infectious human and one *T. saginata* egg in the environment whereas $\mu_h/(\nu + \mu_h)$ is the probability that an initial infectious human dies naturally before shedding a *T. saginata* egg resulting to zero infectious humans and therefore no *T. saginata* eggs in the environment.

The offspring pgf for I_C given that $I_{HT}(0) = 0, I_C(0) = 1, B_I(0) = 0, E_T(0) = 0$ is:

$$f_2(u_1, u_2, u_3, u_4) = \frac{\eta u_3 + \mu_b}{\eta + \mu_b}. \tag{3.82}$$

The term $\eta/(\eta + \mu_b)$ is the probability that the initial infectious cattle is slaughtered for consumption resulting to zero infectious cattle whereas the term $\mu_b/(\eta + \mu_b)$ represents the probability that the initial infectious cattle may die before being slaughtered leading to zero infectious cattle and consequently no infectious beef.

The offspring pgf for B_I given that $I_{HT}(0) = 0, I_C(0) = 0, B_I(0) = 1, E_T(0) = 0$ is:

$$f_3(u_1, u_2, u_3, u_4) = \frac{\beta_T \alpha_b S_H^0 u_1 u_3 + \varepsilon}{\beta_T \alpha_b S_H^0 + \varepsilon}. \tag{3.83}$$

The term $\beta\alpha S_H^0/(\beta\alpha S_H^0 + \varepsilon)$ represents the probability that a proportion of initial infectious beef is lost due to consumption by susceptible humans resulting to one infectious human. The

term $\varepsilon/(\beta\alpha S_H^0+\varepsilon)$ is the probability that the initial infectious beef is thrown leading to zero infectious beef and therefore no infectious human.

The offspring pgf for E_T given that $I_{HT}(0)=0, I_C(0)=0, B_I(0)=0, E_T(0)=1$ is:

$$f_4(u_1,u_2,u_3,u_4)=\frac{\gamma_b S_C^0 u_2 u_4+\mu_e}{\gamma_b S_C^0+\mu_e}. \tag{3.84}$$

The term $\gamma S_C^0/(\gamma S_C^0+\mu_e)$ represents the probability that the initial *T. saginata* egg infects a susceptible cattle leading to one infectious cattle and *T. saginata* egg while $\mu_e/(\gamma S_C^0+\mu_e)$ is the probability that a *T. saginata* egg is lost due to natural death leading to zero *T. saginata* egg and zero infectious cattle.

The expectation matrix $\mathbf{M_1}$ of the offspring probability generating function (pgf) is given by:

$$\mathbf{M_1}=\begin{pmatrix} \frac{\nu}{\nu+\mu_h} & 0 & \frac{\beta_T\alpha_b S_H^0}{\beta_T\alpha_b S_H^0+\varepsilon} & 0 \\ 0 & 0 & 0 & \frac{\gamma_b S_C^0}{\gamma_b S_C^0+\mu_e} \\ 0 & \frac{\eta}{\eta+\mu_b} & \frac{\beta_T\alpha_b S_H^0}{\beta_T\alpha_b S_H^0+\varepsilon} & 0 \\ \frac{\nu}{\nu+\mu_h} & 0 & 0 & \frac{\gamma_b S_C^0}{\gamma_b S_C^0+\mu_e} \end{pmatrix}. \tag{3.85}$$

The stochastic threshold for disease extinction or outbreak in human and cattle populations for the CTMC epidemic model is the spectral radiusof the expectation matrix, $\rho(\mathbf{M_1})$. The threshold $\rho(\mathbf{M_1})$ for CTMC model and the basic reproduction number $\mathscr{R}_{01}$ for deterministic model are closely related. The diseases die in human and cattle populations if $\rho(\mathbf{M_1})\leq 1$ or $\mathscr{R}_{01}\leq 1$. In deterministic models, the diseases persist in the population if $\mathscr{R}_{01}>1$. However in stochastic models, if $\rho(\mathbf{M_1})>1$ there is a possibility for the diseases to die or persist depending on the type and number of an infectives that were initially introduced in a susceptible population (Allen & van den Driessche, 2013; Lahodny & Allen, 2013; Maliyoni, 2021). Thus, if $\rho(\mathbf{M_1})>1$, there exist a fixed point $(q_1,q_2,q_3,q_4)\in(0,1)^4$ of the offspring generating functions (3.81)-(3.84) that is used in writing the probability of disease extinction (Allen & Lahodny Jr, 2012; Maliyoni *et al.*, 2017). To get the fixed point, we set $f_i(q_1,q_2,q_3,q_4)=q_i$ for $i=1,2,3,4$. That is:

$$
\begin{aligned}
q_1 &= \frac{\nu q_1 q_4 + \mu_h}{\nu + \mu_h}, \\
q_2 &= \frac{\eta q_3 + \mu_b}{\eta + \mu_b}, \\
q_3 &= \frac{\beta_T \alpha_b S_H^0 q_1 q_3 + \varepsilon}{\beta_T \alpha_b S_H^0 + \varepsilon}, \\
q_4 &= \frac{\gamma_b S_C^0 q_2 q_4 + \mu_e}{\gamma_b S_C^0 + \mu_e}.
\end{aligned} \tag{3.86}
$$

Since $S_H^0 = \Lambda_H/\mu_h$ and $S_C^0 = \Lambda_C/(\sigma_b + \mu_b)$, then from (3.86) we have:

$$
\begin{aligned}
q_1 &= \frac{\mu_h}{\nu(1-q_4) + \mu_h}, \\
q_2 &= \frac{\eta q_3 + \mu_b}{\eta + \mu_b}, \\
q_3 &= \frac{\varepsilon \mu_h}{\beta_T \alpha_b \Lambda_H (1-q_1) + \varepsilon \mu_h}, \\
q_4 &= \frac{\mu_e(\sigma_s + \mu_b)}{\gamma_b \Lambda_C (1-q_2) + \mu_e(\sigma_s + \mu_b)},
\end{aligned} \tag{3.87}
$$

from which we get;

$$
q_2 = \frac{\varepsilon \alpha_b \beta_T \eta \nu \Lambda_H}{(\varepsilon + \alpha_b)(\alpha_b \beta_T \nu \Lambda_H + g_0)} \left(\frac{1}{\mathscr{R}_{01}^2}\right) + \frac{\alpha_b \beta_T \nu \Lambda_H \mu_b + g_0}{\alpha_b \beta_T \nu \Lambda_H (\eta + \mu_b) + g_0}, \tag{3.88}
$$

where $g_0 = \varepsilon \mu_h (\eta + \mu_b)(\nu + \mu_h)$. Thus, other expressions for the rest of q_i can be easily obtained. Therefore, the probability of disease outbreak for infectives of type i is:

$$
1 - P_0 = 1 - q_1^{i1} q_2^{i2} q_3^{i3} q_4^{i4}, \tag{3.89}
$$

where ik are initial values of infectious humans, cattle, beef and *Taenia saginata* eggs that were introduced in a susceptible population for $k = 1, 2, 3, 4$ respectively.

3.9 The Optimal Control Model for Bovine Cysticercosis and Human Taeniasis

Based on sensitivity analysis results for the basic reproduction number $\mathscr{R}_{01}$ for model system (3.18) which are presented in Table 4, we focus on the time dependent control variable $u_1(t)$ which measures the effect of improving beef cooking for reducing the possibility of human infection with taeniasis, $u_2(t)$ that measures the control efforts due to cattle vaccination and $u_3(t)$ that measures treatment efforts for humans who are infected with taeniasis. Thus, incoporating

these control variables in the model system (3.1), we obtain:

$$\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H + u_3(t)I_{HT} - \beta_T\alpha_b(1-u_1(t))B_I S_H - \mu_h S_H,\\
\frac{dI_{HT}}{dt} &= \beta_T\alpha_b(1-u_1(t))B_I S_H - (\mu_h + u_3(t))I_{HT},\\
\frac{dS_C}{dt} &= \Lambda_C + \psi_v V_C + \pi R_C - \gamma_b S_C E_T - (\sigma_s + u_2(t) + \mu_b)S_C,\\
\frac{dV_C}{dt} &= u_2(t)S_C - \rho_b\gamma_b V_C E_T - (\sigma_v + \psi_v + \mu_b)V_C,\\
\frac{dI_C}{dt} &= \gamma_b S_C E_T + \rho_b\gamma_b V_C E_T - (\eta + \lambda + \mu_b)I_C,\\
\frac{dR_C}{dt} &= \lambda I_C - (\pi + \sigma_r + \mu_b)R_C,\\
\frac{dB_I}{dt} &= \eta I_C - (\varepsilon + \alpha_b)B_I,\\
\frac{dE_T}{dt} &= \nu I_{HT} - \mu_e E_T,
\end{aligned} \tag{3.90}$$

The aim is to minimize the number of infected humans, cattle and the cost associated with implementation of these controls. The objective function that minimizes the cost for administering these controls is given as:

$$J = \int_0^{T_f} \Big(C_1 I_{HT} + C_2 I_C + C_3 u_2 S_C + \frac{1}{2}\sum_{i=1}^{i=3} A_i u_i^2 \Big) dt \tag{3.91}$$

subject to system of differential equations (3.90), where C_1 and C_2 are the constants for minimizing prevalence of humans who are infected with taeniasis and infected cattle respectively while C_3 is the constant for minimizing the number of vaccines used to vaccinate cattle. The coefficients A_1, A_2 and A_3 are relative cost weights for each individual control measure that are used to transform the integral into cost expended over a period of T_f years which is the time period for applying the control strategy (Rong *et al.*, 2021; Osman, Otoo & Makinde, 2020). The initial values are chosen to be 1800, 1500, 340, 130, 250, 90, 83 and 100 for $S_H, I_{HT}, I_{HC}, S_C, V_C, I_C, R_C, B_I$ and E_T classes respectively. Therefore, we seek to find the optimal controls u_1^*, u_2^* and u_3^* such that:

$$J(u_1^*, u_2^*, u_3^*) = \min_U J(u_1, u_2, u_3), \tag{3.92}$$

where $U = \{u : u \text{ is measurable and } 0 \leq u_i(t) \leq 1 \text{ for } t \in [0, T_f]\}$ is the control set.

3.9.1 Characterization of the Optimal Control Problem

We apply Pontryagin's maximum principle (Biswas *et al.*, 2017; Pontryagin, 1962) which provides the necessary conditions that an optimal control problem must satisfy. This principle converts the system of differential equations (3.90) and equation (3.91) into minimization problem point-wise Hamiltonian ($\mathscr{H}$), with respect to control variables (u_1, u_2, u_3).

If we defined a Lagrangian $\mathscr{L}$ for the control problem by:

$$\mathscr{L} = C_1 I_{HT} + C_2 I_C + C_3 u_2 S_C + \frac{1}{2}\sum_{i=1}^{i=3} A_i u_i^2, \tag{3.93}$$

then the Hamiltonian function $\mathscr{H}$ for the control problem is given as:

$$\mathscr{H} = \mathscr{L} + \lambda_1 \frac{dS_H}{dt} + \lambda_2 \frac{dI_{HT}}{dt} + \lambda_3 \frac{dS_C}{dt} + \lambda_4 \frac{dV_C}{dt} + \lambda_5 \frac{dI_C}{dt} + \lambda_6 \frac{dR_C}{dt} + \lambda_7 \frac{dB_I}{dt} + \lambda_8 \frac{dE_T}{dt}, \tag{3.94}$$

where λ_k, for $k = 1,2,3,...,8$ are the adjoint variables associated with state variables $S_H, I_{HT}, S_C, V_C, I_C, R_C, B_I$ and E_T respectively.

If we let $k_1 = (\sigma_s + \mu_b + u_2)$ and $k_2 = (\eta + \lambda_b + \mu_b)$, the Hamiltonian $\mathscr{H}$ becomes:

$$\begin{aligned}
\mathscr{H} = {} & C_1 I_{HT} + C_2 I_C + C_3 u_2 S_C + \frac{1}{2}\sum_{i=1}^{i=3} A_i u_i^2 + \lambda_1 \Big(\Lambda_H + u_3 I_{HT} - \beta_T (1-u_1)\alpha_b B_I S_H - \mu_h S_H \Big) \\
& + \lambda_2 \Big(\beta_T (1-u_1)\alpha_b B_I S_H - (u_3 + \mu_h) I_{HT} \Big) + \lambda_3 \Big(\Lambda_C + \pi_b R_C + \psi_v V_C - \gamma_b S_C E_T - k_1 S_C \Big) \\
& + \lambda_4 \Big(u_2 S_C - \rho_b \gamma_b V_C E_T - (\sigma_v + \mu_b + \psi_v) V_C \Big) + \lambda_5 \Big(\gamma_b S_C E_T + \rho_b \gamma_b V_C E_T - k_2 I_C \Big) \\
& + \lambda_6 \Big(\lambda_b I_C - (\sigma_b + \pi_b + \mu_b) R_C \Big) + \lambda_7 \Big(\eta I_C - (\varepsilon + \alpha_b) B_I \Big) + \lambda_8 \Big(\nu I_{HT} - \mu_e E_T \Big).
\end{aligned} \tag{3.95}$$

Using the Pontryagin's maximum principle (Pontryagin *et al.*, 1962; Pontryagin, 2018), there exists adjoint variables that satisfy:

$$\begin{aligned}
&\frac{d\lambda_1}{dt} = -\frac{\partial \mathscr{H}}{\partial S_H}, \ \frac{d\lambda_2}{dt} = -\frac{\partial \mathscr{H}}{\partial I_{HT}}, \ \frac{d\lambda_3}{dt} = -\frac{\partial \mathscr{H}}{\partial S_C}, \ \frac{d\lambda_4}{dt} = -\frac{\partial \mathscr{H}}{\partial V_C}, \\
&\frac{d\lambda_5}{dt} = -\frac{\partial \mathscr{H}}{\partial I_C}, \ \frac{d\lambda_6}{dt} = -\frac{\partial \mathscr{H}}{\partial R_C}, \ \frac{d\lambda_7}{dt} = -\frac{\partial \mathscr{H}}{\partial B_I}, \ \frac{d\lambda_8}{dt} = -\frac{\partial \mathscr{H}}{\partial E_T},
\end{aligned} \tag{3.96}$$

with transversality condition:

$$\lambda_k(T_f) = 0. \tag{3.97}$$

Therefore, the adjoint system is given as:

$$\begin{aligned}
\frac{d\lambda_1}{dt} &= \beta_T(1-u_1)(\lambda_1-\lambda_2)\alpha_b B_I + \mu_h\lambda_1,\\
\frac{d\lambda_2}{dt} &= (u_3+\mu_h)\lambda_2 - C_1 - u_3\lambda_1 - \nu\lambda_8,\\
\frac{d\lambda_3}{dt} &= \gamma_b(\lambda_3-\lambda_5)E_T + (\sigma_s+\mu_b+u_2)\lambda_3 - u_2\lambda_4 - u_2C_3,\\
\frac{d\lambda_4}{dt} &= \rho_b\gamma_b(\lambda_4-\lambda_5)E_T + (\sigma_v+\mu_b+\psi_v)\lambda_4 - \psi_v\lambda_3,\\
\frac{d\lambda_5}{dt} &= (\eta+\lambda_b+\mu_b)\lambda_5 - \lambda_b\lambda_6 - \eta\lambda_7 - C_2,\\
\frac{d\lambda_6}{dt} &= (\sigma_b+\pi_b+\mu_b)\lambda_6 - \pi_b\lambda_3,\\
\frac{d\lambda_7}{dt} &= \beta_T(1-u_1)(\lambda_1-\lambda_2)\alpha_b S_H + (\varepsilon+\alpha_b)\lambda_7,\\
\frac{d\lambda_8}{dt} &= \gamma_b(\lambda_3-\lambda_5)S_C + \rho_b\gamma_b(\lambda_4-\lambda_5)V_C + \mu_e\lambda_8.
\end{aligned} \tag{3.98}$$

To obtain the optimality conditions, we differentiate the Hamiltonian function (3.95) with respect to the control variables and solve when derivative is zero, that is:

$$\begin{aligned}
\frac{\partial\mathscr{H}}{\partial u_1} &= A_1u_1 - (\lambda_2-\lambda_1)\beta_T\alpha_b B_I S_H = 0,\\
\frac{\partial\mathscr{H}}{\partial u_2} &= A_2u_2 - (\lambda_3-\lambda_4-C_3)S_C = 0,\\
\frac{\partial\mathscr{H}}{\partial u_3} &= A_3u_3 - (\lambda_2-\lambda_1)I_{HT} = 0.
\end{aligned} \tag{3.99}$$

Since the characterization of the optimal control problem holds on the interior of the control set U, thus we have:

$$\begin{aligned}
u_1^* &= max\Big\{0, min\Big(1, \frac{(\lambda_2-\lambda_1)\beta_T\alpha_b B_I S_H}{A_1}\Big)\Big\},\\
u_2^* &= max\Big\{0, min\Big(1, \frac{(\lambda_3-\lambda_4-C_3)S_C}{A_2}\Big)\Big\},\\
u_3^* &= max\Big\{0, min\Big(1, \frac{(\lambda_2-\lambda_1)I_{HT}}{A_3}\Big)\Big\}.
\end{aligned} \tag{3.100}$$

where λ_k are solutions of the system of equations (3.98).

3.10 Dynamics of Taeniasis and Cysticercosis in Humans, Pigs and Cattle

Taeniasis and cysticercosis are foodborne infections which are caused by the adult and larval form tapeworms respectively (Symeonidou *et al.*, 2018). Three tapeworm species that cause taeniasis in humans and cysticercosis in humans, pigs and cattle are *Taenia solium*, *Taenia saginata* and *Taenia asiatica* (WHO, 2021). While *T. asiatica* is endemic only in Asia, other tapeworm species are distributed worldwide (WHO, 2005). Taeniasis and cysticercosis are associated with poor hygiene and sanitation, open human defecation, free range farming system for pigs and cattle, and poverty (Flisser *et al.*, 2006; WHO, 2005). They affect mainly poor and marginalized communities who always have close contact with pigs and cattle, and depend on pork and beef as their sources of food (WHO, 2021; Trevisan *et al.*, 2017).

Humans acquire taeniasis when they consume raw or undercooked beef or pork that contains the tapeworm larval cysts, which develop into adult tapeworms in the human intestine (WHO, 2021). When humans who are infected with taeniasis defecate in the fields, the tapeworm eggs that are passed out with human feaces may spread over the environment by several means, such as water, winds, animal feet and insects, and contaminate the soil, fodder, pastures and water sources (Dermauw *et al.*, 2018). Domestic cattle get cysticercosis infection when they feed on the contaminated environment while pigs acquire cysticercosis through direct consumption of human feaces or indirectly when they ingest taenia eggs from the contaminated environment (Symeonidou *et al.*, 2018). When taenia eggs are consumed by cattle and pigs, they hatch, penetrate the intestinal wall and reach the blood circulation system, where they spread throughout the body tissues and organs such as heart, diaphragm, kidney, lungs, liver and tongue, and develop into cysticerci (Ito *et al.*, 2004; WHO, 2005). Human cysticercosis is a result of consuming *T. solium* eggs from the contaminated environment via contaminated water, fruits and vegetables, or by putting contaminated fingers in the mouth (Del Brutto, 2013). Once the eggs are ingested, they hatch in the small intestine, and develop into larvae which penetrate the intestinal wall and migrate to various parts of the body such as eyes, muscles, skin and the central nervous system through the blood circulatory system, where they form larval cysts (WHO, 2005). When the cysts reach and infect the brain, they cause neurocysticercosis which is the most severe form of tapeworm infection of the central nervous system and is the major cause of epilepsy worldwide (WHO, 2021).

Taeniasis and cysticercosis are endemic in many developing countries of Latin America, Africa and Asia (Symeonidou *et al.*, 2018). The diseases threaten people's health and livelihood of subsistence farming communities, posing considerable challenges in food safety and reducing the market value of pigs and cattle by making pork and beef unsafe for consumption (WHO, 2005; Winskill *et al.*, 2017). Globally, taeniasis and cysticercosis affect approximately 50 million

people and nearly 50 000 people die annually due to cysticercosis (Aung & Spelman, 2016). In 2010, the World Health Organization (WHO) listed cysticercosis as a major neglected tropical disease and later in 2015, it was identified as a leading cause of deaths from foodborne diseases. In Tanzania, cysticercosis has been reported to be a serious health problem whereby the parasite is spread in almost all regions (WHO, 2020). Porcine cysticercosis has been reported to be highly endemic in southern, central and northern regions of Tanzania with the prevalence rate of 0.3–17.4%, 14.9% and 5.5–16.9% respectively (Mkupasi *et al.*, 2018; Trevisan *et al.*, 2017). Human infections have also been reported in the country (Kavishe *et al.*, 2017; Mwanjali *et al.*, 2013). For example, in 2012 Tanzania had 17 853 cases and 212 deaths due to epilepsy while porcine cysticercosis cases were 183 927 and the economic burden for cysticercosis was estimated to be US$ 7.9 million (Trevisan *et al.*, 2017).

The diagnosis of human taeniasis is done by examination of stool samples while diagnosis of human cysticercosis involves doing a biopsy of subcutaneous cysts, immunodiagnosis, radiography, computed tomography (CT) scan and magnetic resonance imaging (MRI). The diagnosis of neurocysticercosis requires both central nervous system imaging with CT brain scans or MRI and serological testing (WHO, 2005). The diagnosis of cysticercosis in pigs and cattle involves meat inspection, serological tests and tongue inspection (Winskill *et al.*, 2017). The treatment of human taeniasis and cysticercosis is through administration of prescribed medication of praziquantel, niclosamide, nitazoxanide or albendazole (WHO, 2005). The treatment of neurocysticercosis may involve prolonged doses of albendazole and/or praziquantel with supporting therapy such as anti-epileptic drugs, corticosteroids, and possibly surgery for some cases (WHO, 2005). Intervention strategies in pigs and cattle involve using vaccines such as S3Pvac & TSOL18 for pigs and TSA-18 & TSA-9 for cattle (Lightowlers *et al.*, 1996; Lightowlers, 2010), and anthelmintic treatment with flubendazole, fenbendazole, oxfendazole, praziquantel and nitazoxanide (WHO, 2005; Winskill *et al.*, 2017).

Mathematical modelling plays an important role in understanding the dynamics of infectious diseases and it is useful in designing appropriate disease control strategies. Currently, there are several mathematical models that have been formulated and analyzed to study the dynamics and control of parasitic food-borne diseases such as cholera, brucellosis and echinococcus (Cui *et al.*, 2014; Lolika & Mushayabasa, 2018; Nyabadza *et al.*, 2019; Sun *et al.*, 2017; Wang *et al.*, 2013; Wu *et al.*, 2013; Yang *et al.*, 2012). In particular, some statistical and deterministic models with some stochastic elements have been formulated and analyzed to study the transmission dynamics and control of taeniasis and cysticercosis in humans and pigs. These include studies by Gonzalez *et al.* (2002), Kyvsgaard *et al.* (2007), Braae *et al.* (2016), Winskill *et al.* (2017), José *et al.* (2018) and Sánchez-Torres *et al.* (2019). Most of deterministic models in previous studies have considered human infection with taeniasis as a consequence of susceptible humans

interacting with pigs that are infected with cysticercosis something that is practically wrong. Moreover, previous studies did not include the cattle population to study the transmission dynamics of taeniasis and cysticercosis in humans, pigs and cattle. In addition, the studies did not apply continuous time Markov chain models to study the disease dynamics. To get new insights on the dynamics of taeniasis and cysticercosis, we formulate and analyze both deterministic and continuous time Markov chain stochastic models for the transmission dynamics of taeniasis and cysticercosis by including cattle population and incorporating compartments of infected pork and beef.

3.11 Deterministic Model Formulation

The basic model for the dynamics of taeniasis and cysticercosis is formulated by modifying the work by Winskill *et al.* (2017) that studied intervention strategies against *T. solium* cysticercosis, by including cattle population and incorporating compartments of infected pork and beef as described in Kyvsgaard *et al.* (2007). Taeniasis and cysticercosis use humans as the definitive hosts, and pigs and cattle as the intermediate hosts (Dermauw *et al.*, 2018).

The model divides the human population into S_H, I_{HT} and I_{HC} classes that represent susceptible humans, humans who are infected with taeniasis and humans who are infected with cysticercosis respectively. The pig population is divided into S_P and I_P classes that represent susceptible pigs and pigs that are infected with cysticercosis respectively while the cattle population is divided into S_C and I_C that represent susceptible cattle and cattle that are infected with cysticercosis respectively. The compartments P_I and B_I represent infected pork and beef respectively, and E_T represents the number of taenia eggs in the environment.

Susceptibe humans are recruited through birth at per capita rate ψ and diminish through consumption of raw or insufficiently cooked infected pork or beef at rates α_p and α_b respectively. They also diminish by acquiring cysticercosis through consumption of *T. solium* eggs from the contaminated environment at a rate θ either in contaminated water, fruits, vegetables or by putting contaminated fingers in the mouth (Del Brutto, 2013). Humans who are infected with cysticercosis increase at a rate θ when susceptible humans consume *T. solium* eggs from the contaminated environment and they reduce due to disease induced death at a rate μ_d. Humans who are infected with taeniasis increase at rates α_P and α_b when susceptible humans consume raw or undecooked infected pork and beef respectively. The parameter β_T denotes the probability of susceptible humans to contract taeniasis following consumption of raw or undercooked infected pork or beef. All humans suffer natural death at a rate μ_h. The number of taenia eggs in the environment grow as a result of open defecation by humans who are infected with taeniasis at a rate ν and decrease due to natural death at a rate μ_e.

Susceptible pigs and cattle are recruited at per capita rates Λ and ϕ due to birth and reduce at rates γ_p and γ_c respectively when they acquire cysticercosis from the contaminated environment. Both pigs and cattle are further slaughtered for consumption at rates ρ and σ respectively. Infected pigs and cattle increase at rates γ_p and γ_c respectively as a consequence of susceptible pigs and cattle feeding on contaminated environment. All pigs and cattle classes suffer natural death at rates μ_p and μ_c respectively. Infected pork increases when infected pigs are slaughtered at a rate ω and decreases when consumed by humans at a rate α_p whereas infected beef increases when infected cattle are slaughtered for consumption at a rate η and decreases when consumed by humans at a rate α_b. The parameters δ and ε are respectively the proportions of infected pork and beef unconsumed by susceptible humans.

In model formulation we consider the free range farming system for both pig and cattle populations, and we do not consider migration. We assume that humans can be infected by either taeniasis or cysticercosis; the number of taenia eggs consumed by humans, pigs and cattle has negligible effect on the total number of eggs in the environment and infected humans, pigs and cattle cannot recover naturally without treatment. We further assume that pigs and cattle do not suffer disease induced mortality, they become carriers for their life and the rate at which susceptible humans consume infected raw or undercooked pork or beef depend on the amount of infected pork or beef which is present. Humans, pigs and cattle contact rates with taenia eggs in the environment are assumed to follow the principle of mass action. The model for the transmission dynamics of taeniasis and cysticercosis in humans, pigs and cattle is summarized by using the flow diagram in Fig. 8. The state variables and parameters are summarized in Tables 6 and 7 respectively.

Table 6: Description of the state variables

Variable	Description	Variable	Description
S_H	Susceptible humans	P_I	Cysticercosis infected pork
I_{HT}	Humans infected with taeniasis	S_C	Susceptible cattle
I_{HC}	Humans infected with cysticercosis	I_C	Cattle infected with cysticercosis
S_P	Susceptible pigs	B_I	Cysticercosis infected beef
I_P	Pigs infected with cysticercosis	E_T	Taenia eggs in the environment

Table 7: Parameters' description and their values (unit: yr^{-1})

Parameter	Description	Value	Source
ψ	Per capita humans' recruitment rate	2247	Wu *et al.* (2013)
μ_h	Per capita natural death rate of humans	0.0141	Wang *et al.* (2013)
μ_d	Human cysticercosis induced death rate	0.0925	Wang *et al.* (2013)
Λ	Per capita pigs' recruitment rate	1450	Assumed
γ_p	*T. solium* eggs to pig infection rate	0.01	Kyvsgaard *et al.* (2007)
α_p	Rate of eating raw/undercooked pork	0.012	Assumed
ρ	Harvesting rate of susceptible pigs	0.252	Assumed
ω	Slaughter rate of infected pigs	0.083×4	Kyvsgaard *et al.* (2007)
μ_p	Per capita natural death rate of pigs	0.083×12	Winskill *et al.* (2017)
δ	Proportion of unconsumed infected pork	0.358	Assumed
ϕ	Per capita calle's recruitment rate	750	Assumed
γ_c	*T. saginata* to cattle transmission rate	0.00625	Assumed
α_b	Rate of eating raw/undercooked beef	0.023	Assumed
σ	Harvesting rate of susceptible cattle	0.213	Assumed
η	Slaughter rate of infected cattle	0.235	Assumed
μ_c	Per capita natural death rate of cattle	0.33	Wang *et al.* (2013)
ε	Proportion of unconsumed infected beef	0.225	Assumed
β_T	Probability of humans getting taeniasis	0.093	Assumed
ν	Defecation rate by humans with taeniasis	0.150	Assumed
θ	*T. solium* to human transmission rate	0.00523	Assumed
μ_e	Per capita death rate of taenia eggs	10.42	Wang *et al.* (2013)

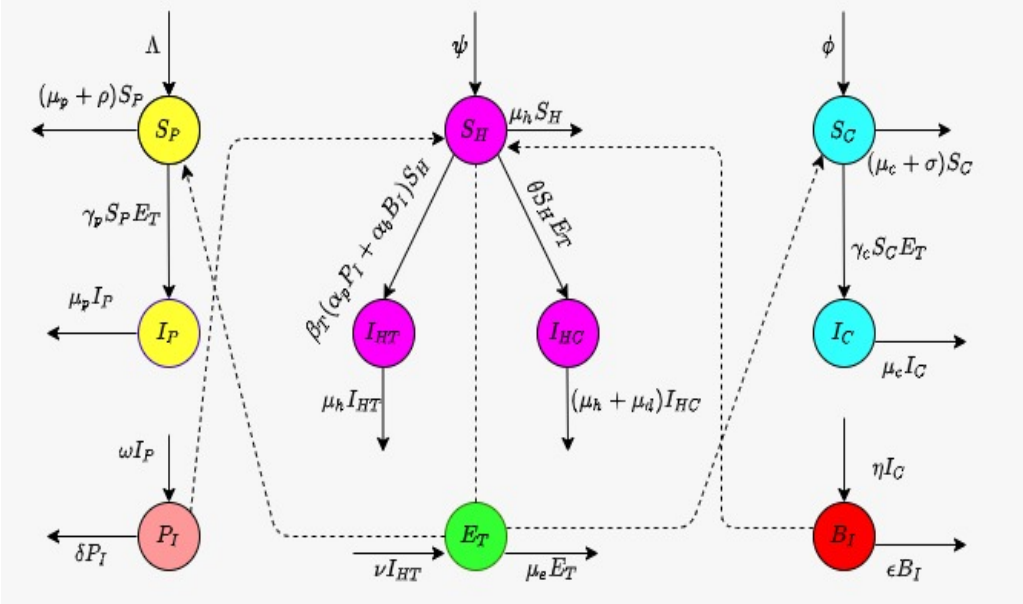


Figure 8: The model flow diagram for the dynamics of taeniasis and cysticercosis in humans, pigs and cattle

Some parameter values in this paper are assumed within realistic ranges due to the fact that only little has been done on this area, the diseases being common in rural areas where there is inadequate or no meat inspection and the treatment is not readily available (Mkupasi *et al.*, 2011).

The model that describes the transmission dynamics of taeniasis and cysticercosis in humans, pigs and cattle is governed by the following system of differential equations:

$$\begin{aligned}
\frac{dS_H}{dt} &= \psi - \beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \theta S_H E_T - \mu_h S_H,\\
\frac{dI_{HT}}{dt} &= \beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \mu_h I_{HT},\\
\frac{dI_{HC}}{dt} &= \theta S_H E_T - (\mu_h + \mu_d)I_{HC},\\
\frac{dS_P}{dt} &= \Lambda - \gamma_p S_P E_T - (\rho + \mu_p)S_P,\\
\frac{dI_P}{dt} &= \gamma_p S_P E_T - (\omega + \mu_p)I_P,\\
\frac{dP_I}{dt} &= \omega I_P - (\delta + \alpha_P)P_I,\\
\frac{dS_C}{dt} &= \phi - \gamma_c S_C E_T - (\sigma + \mu_c)S_C,\\
\frac{dI_C}{dt} &= \gamma_c S_C E_T - (\eta + \mu_c)I_C,\\
\frac{dB_I}{dt} &= \eta I_C - (\varepsilon + \alpha_b)B_I,\\
\frac{dE_T}{dt} &= \nu I_{HT} - \mu_e E_T,
\end{aligned} \tag{3.101}$$

with initial conditions:

$$\begin{aligned}
&S_H(0) > 0; I_{HT}(0) \geq 0; I_{HC}(0) \geq 0; S_P(0) > 0; I_P(0) \geq 0; P_I(0) \geq 0;\\
&S_C(0) > 0; I_C(0) \geq 0; B_I(0) \geq 0 \text{ and } E_T(0) \geq 0.
\end{aligned}$$

3.12 Positivity of Solutions and Invariant Region

For the model system (3.101) to be biologically and epidemiologically meaningful, we need to show that the model solutions are positive and bounded. Given the model system (3.101), there exixts a region

$$\Omega_1 = \Big\{(S_H, I_{HT}, I_{HC}, S_P, I_P, P_I, S_C, I_C, B_I, E_T) \in \mathbb{R}_+^{10}\Big\}. \tag{3.102}$$

such that the model solutions are positive and bounded.

3.12.1 Positivity of Solutions

Theorem 3.11

Let the initial conditions for the model system (3.101) be:

$$\begin{aligned} &S_H(0) > 0; I_{HT}(0) \geq 0; I_{HC}(0) \geq 0; S_P(0) > 0; I_P(0) \geq 0; P_I(0) \geq 0; \\ &S_C(0) > 0; I_C(0) \geq 0; B_I(0) \geq 0; E_T(0) \geq 0. \end{aligned}$$

Then the solutions $(S_H, I_{HT}, S_P, I_P, P_I, S_C, I_C, B_I, E_T)$ of the model with positive initial conditions, will remain non-negative for all time $t > 0$.

Proof.

$$\begin{aligned} \text{Let } t_1 = &sup\{t > 0 : S_H > 0, I_{HT} > 0, I_{HC} > 0, S_P > 0, I_P > 0, P_I > 0, S_C > 0, I_C > 0, \\ &B_I > 0, E_T > 0 \in [0,t]\}, \end{aligned}$$

so that $t_1 > 0$. Then, from the first equation in the model system (3.101) for susceptible humans, we have:

$$\begin{aligned} \frac{dS_H}{dt} &= \psi - \beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \theta S_H E_T - \mu_h S_H, \\ \frac{dS_H}{dt} &\geq -(\beta_T(\alpha_p P_I + \alpha_b B_I) + \theta E_T + \mu_h) S_H, \\ \frac{dS_H}{S_H} &\geq -(\beta_T(\alpha_p P_I + \alpha_b B_I) + \theta E_T + \mu_h)\, dt, \\ S_H(t_1) \geq S_H(0) e^{\int_0^{t_1} -(\beta_T(\alpha_p P_I(s) + \alpha_b B_I(s)) + \theta E_T(s) + \mu_h)\, ds} &\geq 0. \end{aligned} \tag{3.103}$$

Using the same approach, it can be shown that:

$$\begin{aligned} &I_{HT}(t) \geq 0; I_{HC}(t) \geq 0; S_P(t) \geq 0; I_P(t) \geq 0; P_I(t) \geq 0; S_C(t) \geq 0; \\ &I_C(t) \geq 0; B_I(t) \geq 0; E_T(t) \geq 0, \forall t \geq 0. \end{aligned} \tag{3.104}$$

Therefore, all solutions of the model system (3.101) will remain positive for all non-negative initial conditions.

3.12.2 Invariant Region

To show that the model solutions are bounded, we let the total populations for humans, pigs and cattle be $H = S_H + I_{HT} + I_{HC}$, $T_P = S_P + I_P$ and $T_C = S_C + I_C$ respectively. By considering the

human population, we have:

$$\begin{aligned} \frac{dH}{dt} &= \psi - \mu_h H - \mu_d I_{HC}, \\ \frac{dH}{dt} &+ \mu_h H \leq \psi. \end{aligned} \tag{3.105}$$

Integrating throughout, we obtain:

$$H(t) \leq \frac{\psi}{\mu_h} + \left(H(0) - \frac{\psi}{\mu_h} \right) e^{-\mu_h t}, \tag{3.106}$$

where $H(0) = S_H(0) + I_{HT}(0) + I_{HC}(0)$. Following the same procedures, it can be shown that the populations for pigs and cattle are given by:

$$T_P(t) \leq \frac{\Lambda}{\mu_p} + \left(T_P(0) - \frac{\Lambda}{\mu_p} \right) e^{-\mu_p t} \text{ and } T_C(t) \leq \frac{\phi}{\mu_c} + \left(T_C(0) - \frac{\phi}{\mu_c} \right) e^{-\mu_c t} \tag{3.107}$$

respectively, where $T_P(0) = S_P(0) + I_P(0)$ and $T_C(0) = S_C(0) + I_C(0)$. The analysis of the solutions (3.106) and (3.107) considers two cases: when $H(0) > \frac{\psi}{\mu_h}$, $T_P(0) > \frac{\Lambda}{\mu_p}$, $T_C(0) > \frac{\phi}{\mu_c}$ and when $H(0) < \frac{\psi}{\mu_h}$, $T_P(0) < \frac{\Lambda}{\mu_p}$, $T_C(0) < \frac{\phi}{\mu_c}$ that give:

$$\begin{aligned} H(t) &\leq \Phi_t = \max\left\{ \frac{\psi}{\mu_h}, H(0) \right\}, \\ T_P(t) &\leq \Pi_t = \max\left\{ \frac{\Lambda}{\mu_p}, T_P(0) \right\}, \\ T_C(t) &\leq \Psi_t = \max\left\{ \frac{\phi}{\mu_c}, T_C(0) \right\}. \end{aligned} \tag{3.108}$$

Since $H(t) = S_H + I_{HT} + I_{HC} \leq \Phi_t$, it follows that

$$I_{HT} \leq \Phi_t. \tag{3.109}$$

We need to show that E_T is also bounded. The last equation in model system (3.101) for taenia eggs in the environment, can be written as:

$$\frac{dE_T}{dt} + \mu_e E_T = \nu I_{HT}. \tag{3.110}$$

Using equation (3.109), equation (3.110) becomes:

$$\frac{dE_T}{dt} + \mu_e E_T \leq \nu \Phi_t. \tag{3.111}$$

Integrating throughout, we obtain:

$$E_T(t) \leq \frac{\nu\Phi_t}{\mu_e} + \left(E_T(0) - \frac{\nu\Phi_t}{\mu_e}\right)e^{-\mu_e t}, \tag{3.112}$$

from which we get:

$$E_T(t) \leq \Gamma_t = \max\left\{\frac{\nu\Phi_t}{\mu_e}, E_T(0)\right\}, \tag{3.113}$$

Using the same approach for the sixth and ninth equations for infected pork and beef respectively in the model system (3.101), we get:

$$P_I(t) \leq \Theta_t = \max\left\{\frac{\omega\Pi_t}{(\alpha_b+\delta)}, P_I(0)\right\} \text{and } B_I(t) \leq \xi_t = \max\left\{\frac{\eta\Psi_t}{(\varepsilon+\alpha_b)}, B_I(0)\right\}. \tag{3.114}$$

Therefore, all solutions of the model system (3.101) are positive invariant in the region:

$$\begin{aligned}\Omega_1 = \Big\{&(S_H, I_{HT}, I_{HC}, S_P, I_P, P_I, S_C, I_C, B_I, E_T) \in \mathbb{R}_+^{10} : 0 \leq H(t) \leq \Phi_t; \\ &0 \leq T_p(t) \leq \Pi_t; 0 \leq T_C(t) \leq \Psi_t; 0 \leq E_T(t) \leq \Gamma_t; 0 \leq P_I(t) \leq \Theta_t; 0 \leq B_I(t) \leq \xi_t\Big\}.\end{aligned} \tag{3.115}$$

All solutions which start at the boundary of region Ω_1 converge to this region. The model system (3.101) is biologically and epidemiologically meaningful and therefore we can consider the flow generated by the model for analysis.

3.13 Model Equilibria and Basic Reproduction Number R_0

3.13.1 The Disease Free Equilibrium E_1^0

When there are no infections in humans, pigs and cattle, we obtain the disease free equilibrium E_1^0 which is given by:

$$E_1^0\,(S_H, I_{HT}, I_{HC}, S_P, I_P, P_I, S_C, I_C, B_I, E_T) = \left(\frac{\psi}{\mu_h}, 0, 0, \frac{\Lambda}{(\rho+\mu_p)}, 0, 0, \frac{\phi}{(\sigma+\mu_c)}, 0, 0, 0\right). \tag{3.116}$$

3.13.2 The Basic Reproduction Number R_0

The basic reproduction number R_0 is the expected number of secondary infections that may occur as a result of introducing one infected individual in a fully susceptible population (Diekmann *et al.*, 1990). When $R_0 < 1$, the disease clears whereas when $R_0 > 1$, the disease persists within the population. In computing R_0, we adopt the next generation matrix method as used by (Van den Driessche & Watmough, 2002). Let $\mathscr{F}_i$ be the new infections in compartment i and

$\mathscr{V}_i^+$ and $\mathscr{V}_i^-$ be the transfer terms in and out of the compartment i respectively, then the infected classes can be written as:

$$\frac{dx_i}{dt} = \mathscr{F}_i(x) - \mathscr{V}_i^+(x) - \mathscr{V}_i^-(x). \tag{3.117}$$

Using the next generation matrix method, we define $\mathscr{F}_i$ and $\mathscr{V}_i$ by:

$$\mathscr{F}_i = \begin{pmatrix} \beta_T(\alpha_p P_I + \alpha_b B_I)S_H \\ \theta S_H E_T \\ \gamma_p S_P E_T \\ 0 \\ \gamma_c S_C E_T \\ 0 \\ 0 \end{pmatrix}, \quad \mathscr{V}_i = \begin{pmatrix} \mu_h I_{HT} \\ (\mu_h + \mu_d) I_{HC} \\ (\omega + \mu_p) I_P \\ -\omega I_P + (\delta + \alpha_p) P_I \\ (\eta + \mu_c) I_C \\ -\eta I_C + (\varepsilon + \alpha_b) B_I \\ -\nu I_{HT} + \mu_e E_T \end{pmatrix}. \tag{3.118}$$

The Jacobian matrices F and V at the disease free equilibrium E_1^0 are given by:

$$F = \frac{\partial \mathscr{F}_i}{\partial x_j}\left(E_1^0\right), \quad V = \frac{\partial \mathscr{V}_i}{\partial x_j}\left(E_1^0\right). \tag{3.119}$$

The basic reproduction number R_0 is the spectral radius of the next generation matrix given by:

$$R_0 = \rho\left(FV^{-1}\right). \tag{3.120}$$

Using the definitions in (3.119), we have:

$$F = \begin{pmatrix} 0 & 0 & 0 & \frac{\beta_T \alpha_p \psi}{\mu_h} & 0 & \frac{\beta_T \alpha_b \psi}{\mu_h} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\theta \psi}{\mu_h} \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\gamma_p \Lambda}{(\rho + \mu_p)} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\gamma_c \phi}{(\sigma + \mu_c)} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{3.121}$$

and

$$V = \begin{pmatrix} \mu_h & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & (\mu_h+\mu_d) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (\omega+\mu_p) & 0 & 0 & 0 & 0 \\ 0 & 0 & -\omega & \delta+\alpha_p & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \eta+\mu_c & 0 & 0 \\ 0 & 0 & 0 & 0 & -\eta & (\varepsilon+\alpha_b) & 0 \\ -\nu & 0 & 0 & 0 & 0 & 0 & \mu_e \end{pmatrix}. \tag{3.122}$$

The next generation matrix is given by:

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta_T\omega\alpha_p S_H^0}{C_1} & \frac{\beta_T\alpha_p S_H^0}{(\delta+\alpha_p)} & \frac{\beta_T\eta\alpha_b S_H^0}{C_2} & \frac{\beta_T\alpha_b S_H^0}{(\varepsilon+\alpha_b)} & 0 \\ \frac{\nu\theta S_H^0}{\mu_e\mu_h} & 0 & 0 & 0 & 0 & 0 & \frac{\theta S_H^0}{\mu_e} \\ \frac{\nu\gamma_p S_P^0}{\mu_e\mu_h} & 0 & 0 & 0 & 0 & 0 & \frac{\gamma_p S_P^0}{\mu_e} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\nu\gamma_c S_C^0}{\mu_e\mu_h} & 0 & 0 & 0 & 0 & 0 & \frac{\gamma_c S_C^0}{\mu_e} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \tag{3.123}$$

where $C_1 = (\omega+\mu_p)(\delta+\alpha_p), C_2 = (\eta+\mu_c)(\varepsilon+\alpha_b), S_H^0 = \frac{\psi}{\mu_h}, S_P^0 = \frac{\Lambda}{(\rho+\mu_p)}$ and $S_C^0 = \frac{\phi}{(\sigma+\mu_c)}$. Using the definition in (3.120), the basic reproduction number R_0 is given by:

$$R_0 = \sqrt{R_{HP}+R_{HC}}, \tag{3.124}$$

where

$$\begin{aligned} R_{HP} &= \frac{\beta_T\nu\alpha_p\gamma_p\omega\psi\Lambda}{\mu_h^2\mu_e(\omega+\mu_p)(\alpha_p+\delta)(\mu_p+\rho)}, \\ R_{HC} &= \frac{\beta_T\nu\alpha_b\gamma_c\eta\psi\phi}{\mu_h^2\mu_e(\eta+\mu_c)(\alpha_b+\varepsilon)(\mu_c+\sigma)}. \end{aligned} \tag{3.125}$$

R_{HP} is the partial reproduction number due to interaction of humans and pigs whereas R_{HC} is the partial reproduction number due to interaction of humans and cattle. To give the biological meaning for R_0, we rewrite R_{HP} and R_{HC} in the forms:

$$R_{HP} = \beta_T \gamma_p \frac{\psi}{\mu_h} \frac{\nu}{\mu_e} \frac{1}{\mu_h} \frac{\Lambda}{(\mu_p + \rho)} \frac{\omega}{(\omega + \mu_p)} \frac{\alpha_p}{(\alpha_p + \delta)},$$
$$R_{HC} = \beta_T \gamma_c \frac{\psi}{\mu_h} \frac{\nu}{\mu_e} \frac{1}{\mu_h} \frac{\phi}{(\mu_c + \sigma)} \frac{\eta}{(\eta + \mu_c)} \frac{\alpha_b}{(\alpha_b + \varepsilon)}. \tag{3.126}$$

The terms in (3.126) can be interpreted as follows: ν/μ_e is the density of taenia eggs released by humans who are infected with taeniasis, $1/\mu_h$ is the human life expectancy and β_T is the probability of humans to be infected with taeniasis due to consumption of raw or insufficiently cooked pork or beef infected with tapeworm larval cysts. The terms $\psi/\mu_h, \Lambda/(\mu_p + \rho)$ and $\phi/(\mu_c + \sigma)$ are initial populations for susceptible humans, pigs and cattle respectively; $1/(\mu_p + \rho)$ and $1/(\mu_c + \sigma)$ are the average times pigs and cattle spend in susceptible classes respectively; $1/(\omega + \mu_p)$ and $1/(\eta + \mu_c)$ are the infectious periods of infected pigs and cattle respectively; $1/(\alpha_p + \delta)$ and $1/(\alpha_b + \varepsilon)$ are the average infectious period for infected pork and beef respectively whereas $\omega/(\omega + \mu_p)$ and $\eta/(\eta + \mu_c)$ are the proportions of infected pigs and cattle that are slaughtered for consumption respectively. The terms $\alpha_p/(\alpha_p + \delta)$ and $\alpha_b/(\alpha_b + \varepsilon)$ are the proportions of infected pork and beef that are eaten by susceptible humans respectively whereas γ_p and γ_c are the rates at which *T. solium* eggs and *T. saginata* eggs are consumed by pigs and cattle respectively from the contaminated environment.

3.13.3 Sensitivity Analysis

To determine how sensitive the model parameters are to the diseases' transmission, we adopt the normalized forward sensitivity index approach as used in Chitnis *et al.* (2008). If ψ is a parameter in the basic reproduction number R_0, then its sensitivity index is given by:

$$\Gamma_{\psi}^{R_0} = \frac{\partial R_0}{\partial \psi} \times \frac{\psi}{R_0}. \tag{3.127}$$

Using equation (3.127) and parameter values in Table 7, we obtain sensitivity indices for each parameter as shown in Table 8.

Table 8: Sensitivity indices of model parameters

Parameter	Sensitivity Index	Parameter	Sensitivity Index
Λ	+0.1421	ω	+0.1066
ϕ	+0.3579	η	+0.2090
ψ	+0.5000	ν	+0.5000
γ_p	+0.1421	δ	- 0.1375
γ_c	+0.3579	ε	- 0.3343
α_p	+0.1375	ρ	- 0.0287
α_b	+0.3343	σ	- 0.1404
β_T	+0.5000	μ_e	- 0.5000

Since it does not make biological sense and not practical to increase the natural death of populations, sensitivity indices for natural mortality rates of humans, pigs and cattle have not been included in Table 8. The positive sign of sensitivity index indicates that an increase or decrease of parameter value while keeping other parameters constant increases or decreases the basic reproduction number R_0. The negative sign indicates that an increase or decrease of parameter value causes a decrease or increase in expected new average infection R_0. For instance, $\gamma_p = +0.1421$ means that an increase in γ_p by 10%, increases R_0 by 1.421% and hence the disease transmission; and $\delta = -0.1375$ means that an increase in δ by 20% causes a decrease in R_0 by 2.75% and thus decrease the disease transmission.

The most positive sensitive parameters in the model are human's recruitment (ψ), the probability of humans infection with taeniasis (β_T) and the defecation rate by humans who are infected with taeniasis (ν) whereas the most negative sensitive parameter is the natural mortality rate of taenia eggs (μ_e) and the proportion of unconsumed infectious pork and beef (ε).

3.13.4 The Endemic Equilibrium E_1^*

The endemic equilibrium is a point at which taeniasis and cysticercosis exist in humans, pigs and cattle. It is obtained by setting the derivatives in model system (3.101) equal to zero and solving for state variables. The endemic equilibrium $E_1^* = (S_H^*, I_{HT}^*, I_{HC}^*, S_P^*, I_P^*, P_I^*, S_C^*, I_C^*, B_I^*, E_T^*)$ where:

$$
\begin{aligned}
S_H^* &= \frac{\psi}{(\beta_T F_0 E_T^* + \theta E_T^* + \mu_h)}, \\
I_{HT}^* &= \frac{\beta_T F_0 E_T^* S_H^*}{\mu_h}, \\
I_{HC}^* &= \frac{\theta S_H^* E_T^*}{(\mu_h + \mu_d)}, \\
S_P^* &= \frac{\Lambda}{(\gamma_p E_T^* + \rho + \mu_p)}, \\
I_P^* &= \frac{\gamma_p \Lambda E_T^*}{(\gamma_p E_T^* + \rho + \mu_p)(\omega + \mu_p)}, \\
P_I^* &= \frac{\omega \gamma_p \Lambda E_T^*}{(\gamma_p E_T^* + \rho + \mu_p)(\omega + \mu_p)(\delta + \alpha_p)}, \\
S_C^* &= \frac{\phi}{(\gamma_c E_T^* + \sigma + \mu_c)}, \\
I_C^* &= \frac{\gamma_c \phi E_T^*}{(\gamma_c E_T^* + \sigma + \mu_c)(\eta + \mu_c)}, \\
B_I^* &= \frac{\eta \gamma_c \phi E_T^*}{(\gamma_c E_T^* + \sigma + \mu_c)(\eta + \mu_c)(\varepsilon + \alpha_b)},
\end{aligned}
\tag{3.128}
$$

with

$$F_0 = \frac{\alpha_p \omega \gamma_p \Lambda}{(\gamma_p E_T^* + \rho + \mu_p)(\omega + \mu_p)(\alpha_p + \delta)} + \frac{\alpha_b \eta \gamma_c \phi}{(\gamma_c E_T^* + \sigma + \mu_c)(\eta + \mu_c)(\alpha_b + \varepsilon)}. \tag{3.129}$$

To obtain E_T^*, we solve for the real roots of the polynomial:

$$a_0 {E_T^*}^3 + a_1 {E_T^*}^2 + a_2 E_T^* + a_3 = 0, \tag{3.130}$$

where

$$\begin{aligned}
a_0 =& 1 > 0, \\
a_1 =& \frac{\alpha_p \omega \Lambda \beta_T}{\theta(\mu_p + \omega)(\alpha_p + \delta)} + \frac{\alpha_b \eta \phi \beta_T}{\theta(\mu_c + \eta)(\alpha_p + \varepsilon)} + \frac{(\mu_c + \sigma)}{\gamma_c} + \frac{(\mu_p + \rho)}{\gamma_p} + \frac{\mu_h}{\theta} > 0, \\
a_2 =& \frac{(\rho + \mu_p)(\sigma + \mu_c)}{\gamma_c \gamma_p} + \frac{\mu_h(\sigma + \mu_c)}{\theta \gamma_c} + \frac{\mu_h(\rho + \mu_p)}{\theta \gamma_p} + \frac{\alpha_p \omega \Lambda \beta_T (\sigma + \mu_c)}{\theta \gamma_c (\mu_p + \omega)(\alpha_p + \delta)} + \\
& \frac{\alpha_b \eta \phi \beta_T (\rho + \mu_p)}{\theta \gamma_p (\mu_c + \eta)(\alpha_p + \varepsilon)} - \left(\frac{\nu \psi \beta_T \alpha_p \omega \Lambda \gamma_c}{\mu_h \mu_e \gamma_c \theta (\mu_p + \omega)(\alpha_p + \delta)} + \frac{\nu \psi \beta_T \alpha_b \eta \phi \gamma_p}{\mu_h \mu_e \gamma_p \theta (\mu_c + \eta)(\alpha_p + \varepsilon)} \right), \\
a_3 =& \frac{\mu_h(\sigma + \mu_c)(\rho + \mu_p)(1 + R_0)(1 - R_0)}{\theta \gamma_c \gamma_p}.
\end{aligned} \tag{3.131}$$

To analyze the possible number of positive real roots of polynomial (3.130) when $R_0 < 1$ and $R_0 > 1$, we adopt the approach in Okosun *et al.* (2016). Using this approach, the number of possible real roots when $R_0 < 1$ and $R_0 > 1$ are summarized in Table 9.

Table 9: Number of possible positive real roots when $R_0 < 1$ and $R_0 > 1$

Cases	a_0	a_1	a_2	a_3	R_0	No. of Sign Change	No. of +ve Real Roots
1	+	+	+	+	$R_0 < 1$	0	0
2	+	+	+	-	$R_0 > 1$	1	1
3	+	+	-	+	$R_0 < 1$	2	0,2
4	+	+	-	-	$R_0 > 1$	1	1

Therefore, the model system (3.101) has a unique endemic equilibrium when $R_0 > 1$ as shown in cases 2 and 4. However, when $R_0 < 1$ there is a possibility of occurence of multiple endemic equilibria, a case where there may be a backward bifurcation. Hence we state the following theorem:

Theorem 3.12
The model system (3.101) has a unique endemic equilibrium when the basic reproduction number $R_0 > 1$ and there is a possibility of backward bifurcation when $R_0 < 1$.

3.14 Stability Analysis of Model Equilibria

3.14.1 Global Stability of the Disease Free Equilibrium E_1^0

Theorem 3.13
The disease free equilibrium E_1^0 of the model system (3.101) is globally asymptotically stable when $R_0 < 1$.

Proof. To analyze the global stability of the disease free equilibrium E_1^0, we adopt the approach used in Castillo-Chavez & Song (2004) and Dumont *et al.* (2008). Using this method, the model system (3.101) is written as:

$$\begin{aligned} \frac{dX_r}{dt} &= B(X_r - X_{DFE}) + B_1 X_n, \\ \frac{dX_n}{dt} &= B_2 X_n, \end{aligned} \tag{3.132}$$

where X_r and X_n are the non-transmitting and transmitting classes respectively, X_{DFE} is the disease free equilibrium, whereas B, B_1 and B_2 are the matrices to be computed. Here, we have:

$$\begin{aligned} X_r &= (S_H, S_P, S_C)^T, \\ X_{DFE} &= \left(\frac{\psi}{\mu_h}, \frac{\Lambda}{\mu_p + \rho}, \frac{\phi}{\mu_c + \sigma} \right)^T. \end{aligned} \tag{3.133}$$

Thus,

$$X_r - X_{DFE} = \left(S_H - \frac{\psi}{\mu_h}, S_P - \frac{\Lambda}{\mu_p + \rho}, S_C - \frac{\phi}{\mu_c + \sigma} \right)^T. \tag{3.134}$$

Therefore, from Equation (3.132) we have:

$$\begin{pmatrix} \psi - \beta_T(\alpha_p P_I + \alpha_b B_I) S_H - \theta S_H E_T - \mu_h S_H \\ \Lambda - \gamma_p S_P E_T - (\rho + \mu_p) S_P \\ \phi - \gamma_c S_C E_T - (\sigma + \mu_c) S_P \end{pmatrix} = B \begin{pmatrix} S_H - \dfrac{\psi}{\mu_h} \\ S_P - \dfrac{\Lambda}{\mu_p + \rho} \\ S_C - \dfrac{\phi}{\mu_c + \sigma} \end{pmatrix} + B_1 \begin{pmatrix} I_{HT} \\ I_{HC} \\ I_P \\ P_I \\ I_C \\ B_I \\ E_T \end{pmatrix} \tag{3.135}$$

and

$$\begin{pmatrix} \beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \mu_h I_{HT} \\ \theta S_H E_T - (\mu_h + \mu_d) I_{HC} \\ \gamma_p S_P E_T - (\omega + \mu_p) I_P \\ \omega I_P - (\delta + \alpha_p) P_I \\ \gamma_c S_C E_T - (\eta + \mu_c) I_C \\ \eta I_C - (\varepsilon + \alpha_b) B_I \\ \nu I_{HT} - \mu_e E_T \end{pmatrix} = B_2 \begin{pmatrix} I_{HT} \\ I_{HC} \\ I_P \\ P_I \\ I_C \\ B_I \\ E_T \end{pmatrix}. \tag{3.136}$$

From (3.135) and (3.136), matrices B, B_1 and B_2 are given by:

$$B = \begin{pmatrix} -\mu_h & 0 & 0 \\ 0 & -(\rho + \mu_p) & 0 \\ 0 & 0 & -(\sigma + \mu_c) \end{pmatrix},$$

$$B_1 = \begin{pmatrix} 0 & 0 & 0 & -\beta_T \alpha_p S_H & 0 & -\beta_T \alpha_b S_H & -\theta S_H \\ 0 & 0 & 0 & 0 & 0 & 0 & \gamma_p S_P \\ 0 & 0 & 0 & 0 & 0 & 0 & \gamma_c S_C \end{pmatrix},$$

and

$$B_2 = \begin{pmatrix} -\mu_h & 0 & 0 & \beta_T \alpha_p S_H & 0 & \beta_T \alpha_b S_H & 0 \\ 0 & -(\mu_h + \mu_d) & 0 & 0 & 0 & 0 & \theta S_H \\ 0 & 0 & -(\omega + \mu_p) & 0 & 0 & 0 & \gamma_p S_P \\ 0 & 0 & \omega & -(\delta + \alpha_p) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\eta + \mu_c) & 0 & \gamma_c S_C \\ 0 & 0 & 0 & 0 & \eta & -(\varepsilon + \alpha_b) & 0 \\ \nu & 0 & 0 & 0 & 0 & 0 & -\mu_e \end{pmatrix}.$$

It can be observed that matrix B has real and negative eigenvalues. Thus, the system:

$$\frac{dX_r}{dt} = B(X_r - X_{DFE}) + B_1 X_n \tag{3.137}$$

is globally asymptotically stable at X_{DFE}.

To prove the stability of B_2, we adopt Metzler matrix approach and apply the lemma in Dumont *et al.* (2008). A Metzler matrix is a matrix whose off diagonal elements are non-negative, denoted by $B_2(i, j) \geq 0$, for all $i \neq j$. Thus, it can be observed that B_2 is a Metzler matrix.

Lemma 3.1

Let M be a square Metzler matrix written in block form:

$$M = \begin{pmatrix} P & Q \\ R & S \end{pmatrix}, \tag{3.138}$$

where P and S are square matrices. Matrix M is Metzler stable if and only if matrices P and $S - RP^{-1}Q$ are Metzler stable.

Proof. Comparing Metzler matrix B_2 with a square Metzler matrix M, matrices P, Q, R and S are defined as:

$$P = \begin{pmatrix} -\mu_h & 0 & 0 \\ 0 & -(\mu_h + \mu_d) & 0 \\ 0 & 0 & -(\omega + \mu_p) \end{pmatrix}, \; Q = \begin{pmatrix} \beta_T \alpha_p S_H & 0 & \beta_T \alpha_b S_H & 0 \\ 0 & 0 & 0 & \theta S_H \\ 0 & 0 & 0 & \gamma_p S_P \end{pmatrix}, \tag{3.139}$$

$$R = \begin{pmatrix} 0 & 0 & \omega \\ 0 & 0 & 0 \\ 0 & 0 & 0 \\ \nu & 0 & 0 \end{pmatrix}, \; S = \begin{pmatrix} -(\delta + \alpha_p) & 0 & 0 & 0 \\ 0 & -(\eta + \mu_c) & 0 & \gamma_c S_C \\ 0 & \eta & -(\varepsilon + \alpha_b) & 0 \\ 0 & 0 & 0 & -\mu_e \end{pmatrix}. \tag{3.140}$$

Clearly, P is a stable Metzler matrix. After some computations, we obtain:

$$S - RP^{-1}Q = \begin{pmatrix} -(\delta + \alpha_p) & 0 & 0 & \dfrac{\omega \gamma_p S_P}{(\omega + \mu_p)} \\ 0 & -(\eta + \mu_c) & 0 & \gamma_c S_C \\ 0 & \eta & -(\varepsilon + \alpha_b) & 0 \\ \dfrac{\nu \beta_T \alpha_p S_H}{\mu_h} & 0 & \dfrac{\nu \beta_T \alpha_b S_H}{\mu_h} & -\mu_e \end{pmatrix}. \tag{3.141}$$

Thus, $S - RP^{-1}Q$ is Metzler stable if $Det\ (S - RP^{-1}Q) > 0$. That is:

$$\mathscr{W}(\delta + \alpha_p)\mu_e - \frac{\nu \beta_T S_H \Big(\omega \alpha_p \gamma_p \mathscr{W} S_P + \eta \alpha_b \gamma_c (\delta + \alpha_b)(\omega + \mu_p) S_C \Big)}{\mu_h (\omega + \mu_p)} > 0, \tag{3.142}$$

where $\mathscr{W} = (\varepsilon + \alpha_c)(\eta + \mu_c)$. Substituting the values for S_H, S_P and S_C at disease free equilibrium E_1^0 into (3.142) and simplifying the expression, we obtain $1 - R_0^2 > 0$, where R_0 is given in (3.124). Therefore, the disease free equilibrium E_1^0 is globally asymptotically stable when $R_0 < 1$. □

3.14.2 Global Stability of the Endemic Equilibrium E_1^*

Theorem 3.14

The endemic equilibrium (E_1^*) for the model system (3.101) is globally asymptotically stable when $R_0 > 1$ and unstable otherwise.

Proof. Since the endemic equilibrium exists if and only if $R_0 > 1$, we adopt the approach in Osman, Otoo & Sebil (2020) to prove global stability of endemic equilibrium E_1^*. Consider the non-linear function:

$$\begin{aligned}\mathscr{L} = {} & S_H^*\left(\frac{S_H}{S_H^*} - \ln\frac{S_H}{S_H^*}\right) + I_{HT}^*\left(\frac{I_{HT}}{I_{HT}^*} - \ln\frac{I_{HT}}{I_{HT}^*}\right) + I_{HC}^*\left(\frac{I_{HC}}{I_{HC}^*} - \ln\frac{I_{HC}}{I_{HC}^*}\right) \\ & + S_P^*\left(\frac{S_P}{S_P^*} - \ln\frac{S_P}{S_P^*}\right) + I_P^*\left(\frac{I_P}{I_P^*} - \ln\frac{I_P}{I_P^*}\right) + P_I^*\left(\frac{P_I}{P_I^*} - \ln\frac{P_I}{P_I^*}\right) \\ & + S_C^*\left(\frac{S_C}{S_C^*} - \ln\frac{S_C}{S_C^*}\right) + I_C^*\left(\frac{I_C}{I_C^*} - \ln\frac{I_C}{I_C^*}\right) + B_I^*\left(\frac{B_I}{B_I^*} - \ln\frac{B_I}{B_I^*}\right) \\ & + E_T^*\left(\frac{E_T}{E_T^*} - \ln\frac{E_T}{E_T^*}\right).\end{aligned} \tag{3.143}$$

It can be observed that the function $\mathscr{L}$ in (3.143) satisfies conditions (i) and (ii) in the Definition 3.1 for all $x \geq 0$ where $x = \{S_H, I_{HT}, I_{HC}, S_P, I_P, P_I, S_C, I_C, B_I, E_T\}$ and $x*$ is the endemic equilibrium. Hence a function $\mathscr{L}$ is a Lyapunov function. To prove if condition (iii) holds, the time derivative of $\mathscr{L}$ is found which is given by:

$$\begin{aligned}\frac{d\mathscr{L}}{dt} = {} & \left(1 - \frac{S_H^*}{S_H}\right)\frac{dS_H}{dt} + \left(1 - \frac{I_{HT}^*}{I_{HT}}\right)\frac{dI_{HT}}{dt} + \left(1 - \frac{I_{HC}^*}{I_{HC}}\right)\frac{dI_{HC}}{dt} + \left(1 - \frac{S_P^*}{S_P}\right)\frac{dS_P}{dt} \\ & + \left(1 - \frac{I_P^*}{I_P}\right)\frac{dI_P}{dt} + \left(1 - \frac{P_I^*}{P_I}\right)\frac{dP_I}{dt} + \left(1 - \frac{S_C^*}{S_C}\right)\frac{dS_C}{dt} + \left(1 - \frac{I_C^*}{I_C}\right)\frac{dI_C}{dt} \\ & + \left(1 - \frac{B_I^*}{B_I}\right)\frac{dB_I}{dt} + \left(1 - \frac{E_T^*}{E_T}\right)\frac{dE_T}{dt},\end{aligned} \tag{3.144}$$

Substituting equations of model system (3.101) into Equation (3.144), we have:

$$\begin{aligned}\frac{d\mathscr{L}}{dt} &= \left(1-\frac{S_H^*}{S_H}\right)(\psi-\beta_T\alpha_p P_I S_H-\beta_T\alpha_b B_I S_H-\theta S_H E_T-\mu_h S_H)\\ &+\left(1-\frac{I_{HT}^*}{I_{HT}}\right)(\beta_T(\alpha_p P_I+\alpha_b B_I)S_H-\mu_h I_{HT})+\left(1-\frac{I_{HC}^*}{I_{HC}}\right)(\theta S_H E_T-(\mu_h+\mu_d)I_{HC})\\ &+\left(1-\frac{S_P^*}{S_P}\right)(\Lambda-\gamma_p S_P E_T-(\rho+\mu_p)S_P)+\left(1-\frac{I_P^*}{I_P}\right)(\gamma_p S_P E_T-(\omega+\mu_p)I_P)\\ &+\left(1-\frac{P_I^*}{P_I}\right)(\omega I_P-(\delta+\alpha_p)P_I)+\left(1-\frac{S_C^*}{S_C}\right)(\phi-\gamma_c S_C E_T-(\sigma+\mu_c)S_C)\\ &+\left(1-\frac{I_C^*}{I_C}\right)(\gamma_c S_C E_T-(\eta+\mu_c)I_C)+\left(1-\frac{B_I^*}{B_I}\right)(\eta I_C-(\varepsilon+\alpha_b)B_I)\\ &+\left(1-\frac{E_T^*}{E_T}\right)(\nu I_{HT}-\mu_e E_T).\end{aligned} \tag{3.145}$$

Equation (3.145) can be written as:

$$\begin{aligned}\frac{d\mathscr{L}}{dt} &= \psi-\beta_T\alpha_p P_I S_H-\beta_T\alpha_b B_I S_H-\theta S_H E_T-\mu_h S_H-\frac{\psi S_H^*}{S_H}+\beta_T\alpha_p P_I S_H^*\\ &+\beta_T\alpha_b B_I S_H^*+\theta E_T S_H^*+\mu_h S_H^*+\beta_T\alpha_p P_I S_H+\beta_T\alpha_b B_I S_H-\mu_h I_{HT}\\ &-\frac{\beta_T\alpha_p P_I S_H I_{HT}^*}{I_{HT}}-\frac{\beta_T\alpha_b B_I S_H I_{HT}^*}{I_{HT}}+\mu_h I_{HT}^*+\theta S_H E_T-(\mu_h+\mu_d)I_{HC}\\ &-\frac{\theta S_H E_T I_{HC}^*}{I_{HC}}+(\mu_h+\mu_d)I_{HC}^*+\Lambda-\gamma_p S_P E_T-(\rho+\mu_p)S_P-\frac{\Lambda S_P^*}{S_P}\\ &+\gamma_p E_T S_P^*+(\rho+\mu_p)S_P^*+\gamma_p S_P E_T-(\omega+\mu_p)I_P-\frac{\gamma_p S_P E_T I_P^*}{I_P}+(\omega+\mu_p)I_P^*\\ &+\omega I_P-(\delta+\alpha_p)P_I-\frac{\omega I_P P_I^*}{P_I}+(\delta+\alpha_p)P_I^*+\phi-\gamma_c S_C E_T-(\sigma+\mu_c)S_C-\frac{\phi S_C^*}{S_C}\\ &+\gamma_c E_T S_C^*+(\sigma+\mu_c)S_C^*+\gamma_c S_C E_T-(\eta+\mu_c)I_C-\frac{\gamma_c S_C E_T I_C^*}{I_C}+(\eta+\mu_c)I_C^*\\ &+\eta I_C-(\varepsilon+\alpha_b)B_I-\frac{\eta I_C B_I^*}{B_I}+(\varepsilon+\alpha_b)B_I^*+\nu I_{HT}-\mu_e E_T-\frac{\nu I_{HT}E_T^*}{E_T}+\mu_e E_T^*.\end{aligned} \tag{3.146}$$

After rearrangement, Equation (3.146) can be written as:

$$\frac{d\mathscr{L}}{dt} = \mathscr{J}-\mathscr{P}, \tag{3.147}$$

where

$$\begin{aligned}\mathscr{J} &= \psi + \beta_T\alpha_p P_I S_H^* + \beta_T\alpha_b B_I S_H^* + \theta E_T S_H^* + \mu_h S_H^* + \beta_T\alpha_p P_I S_H + \beta_T\alpha_b B_I S_H + \mu_h I_{HT}^* \\ &+ \theta S_H E_T + (\mu_h+\mu_d) I_{HC}^* + \Lambda + \gamma_p E_T S_P^* + (\rho+\mu_p) S_P^* + \gamma_p S_P E_T + (\omega+\mu_p) I_P^* \\ &+ \omega I_P + (\delta+\alpha_p) P_I^* + \phi + \gamma_c E_T S_C^* + (\sigma+\mu_c) S_C^* + \gamma_c S_C E_T + (\eta+\mu_c) I_C^* + \eta I_C \\ &+ (\varepsilon+\alpha_b) B_I^* + \nu I_{HT} + \mu_e E_T^*,\end{aligned} \tag{3.148}$$

$$\begin{aligned}\mathscr{P} &= \beta_T\alpha_p P_I S_H + \beta_T\alpha_b B_I S_H + \theta S_H E_T + \mu_h S_H + \frac{\psi S_H^*}{S_H} + \mu_h I_{HT} + \frac{\beta_T\alpha_p P_I S_H I_{HT}^*}{I_{HT}} \\ &+ \frac{\beta_T\alpha_b B_I S_H I_{HT}^*}{I_{HT}} + (\mu_h+\mu_d) I_{HC} + \frac{\theta S_H E_T I_{HC}^*}{I_{HC}} + \gamma_p S_P E_T + (\rho+\mu_p) S_P + \frac{\Lambda S_P^*}{S_P} \\ &+ (\omega+\mu_p) I_P + \frac{\gamma_p S_P E_T I_P^*}{I_P} + \frac{\omega I_P P_I^*}{P_I} + \gamma_c S_C E_T + (\sigma+\mu_c) S_C + \frac{\phi S_C^*}{S_C} + (\delta+\alpha_p) P_I \\ &+ (\eta+\mu_c) I_C + \frac{\gamma_c S_C E_T I_C^*}{I_C} + (\varepsilon+\alpha_b) B_I + \frac{\eta I_C B_I^*}{B_I} + \mu_e E_T + \frac{\nu I_{HT} E_T^*}{E_T}.\end{aligned} \tag{3.149}$$

It can be seen from equation (3.147) that if $\mathscr{J} < \mathscr{P}$ then $\frac{d\mathscr{L}}{dt} < 0$ and if $\Omega_1 = \Omega_1^*$ then $\frac{d\mathscr{L}}{dt} = 0$ where Ω_1 is given in equation (3.102) and Ω_1^* is the endemic equilibrium. Thus, the largest invariant set in Ω_1 is the endemic equilibrium. Hence from the LaSalle's invariant principle (LaSalle, 1976), we conclude that as $t \to \infty$, the solution of the model system (3.101) approaches the endemic equilibrium E_1^* when $R_0 > 1$. Therefore, the endemic equilibrium E_1^* is globally asymptotically stable in the invariant set Ω_1 if $\mathscr{J} < \mathscr{P}$. □

3.15 CTMC Model Formulation

In this section we formulate and analyze a continuous time markov chain (CTMC) stochastic model and apply multitype branching process to compute the probability of disease extinction or outbreak. The continuous-time Markov chain (CTMC) stochastic model is formulated based on deterministic model assumptions. The same notations and parameters as those in deterministic model (3.101) are used.

Let $\vec{Z} = [S_H, I_{HT}, I_{HC}, S_P, I_P, , P_I, S_C, I_C, B_I, E_T]^T$ be the associated random vector for all discrete random variables. The events and transition rates are summarized in Table 10. The values $1(-1)$ and 0 represent, respectively, an increase (decrease) by 1 and no change in state for the variable from time t to $(t+\Delta t)$.

The CTMC stochastic model is time-homogeneous and satisfies the Markov property which explains that the future state of the process at time $(t+\Delta t)$ depends on the current state at time t (Allen, 2010; Maliyoni *et al.*, 2017). From the Markov assumptions, the time between events

is exponentially distributed with parameter (Lahodny Jr *et al.*, 2015; Maliyoni *et al.*, 2017):

$$\begin{aligned}\psi(\vec{Z}) = \psi + \mu_h N_H + \Lambda_P + \mu_p N_P + \rho S_P + \omega I_P + (\delta + \alpha_p) P_I + \phi + \mu_c N_C + \nu I_{HT} \\ + \sigma S_C + \eta I_C + (\varepsilon + \alpha_b) B_I + \mu_e E_T + \beta(\alpha_p P_I + \alpha_b B_I) S_H + \gamma_p S_P E_T + \gamma_c S_C E_T\end{aligned} \tag{3.150}$$

Table 10: State transitions and rates for the CTMC model

Event	Transition $\Delta\vec{Z}(t)$	Transition rate, p
Recruitment of S_H	$(1,0,0,0,0,0,0,0,0,0)$	ψ
Human infection with infected pork	$(-1,1,0,0,0,0,0,0,0,0)$	$\beta_T \alpha_p P_I S_H$
Human infection with infected beef	$(-1,1,0,0,0,0,0,0,0,0)$	$\beta_T \alpha_b B_I S_H$
Human infection by *T. solium* eggs	$(-1,1,0,0,0,0,0,0,0,0)$	$\theta S_H E_T$
Natural death of S_H	$(-1,0,0,0,0,0,0,0,0,0)$	$\mu_h S_H$
Natural death of I_{HT}	$(0,-1,0,0,0,0,0,0,0,0)$	$\mu_h I_{TH}$
Natural death of I_{HC}	$(0,0,-1,0,0,0,0,0,0,0)$	$\mu_h I_{HC}$
Disease induced death of I_{HC}	$(0,0,-1,0,0,0,0,0,0,0)$	$\mu_d I_{HC}$
Recruitment of S_P	$(0,0,0,1,0,0,0,0,0,0)$	Λ_P
Slaughter of S_P	$(0,0,0,-1,0,0,0,0,0,0)$	ρS_P
Infection of S_P	$(0,0,0,-1,1,0,0,0,0,0)$	$\gamma_p S_P E_T$
Natural death of S_P	$(0,0,0,-1,0,0,0,0,0,0)$	$\mu_P S_P$
Slaughter of I_P	$(0,0,0,0,-1,1,0,0,0,0)$	ωI_P
Natural death of I_P	$(0,0,0,0,-1,0,0,0,0,0)$	$\mu_p I_P$
Throwing of infected pork	$(0,0,0,0,0,-1,0,0,0,0)$	δP_I
Consumption of infected pork	$(0,0,0,0,0,0,0,0,-1,0)$	$\alpha_p P_I$
Recruitment of S_C	$(0,0,0,0,0,0,1,0,0,0)$	ϕ
Slaughter of S_C	$(0,0,0,0,0,0,-1,0,0,0)$	σS_C
Infection of S_C	$(0,0,0,0,0,0,-1,1,0,0)$	$\gamma_c S_C E_T$
Natural death of S_C	$(0,0,0,0,0,0,-1,0,0,0)$	$\mu_c S_C$
Slaughter of I_C	$(0,0,0,0,0,0,0,-1,1,0)$	ηI_C
Natural death of I_C	$(0,0,0,0,0,0,0,-1,0,0)$	$\mu_c I_C$
Throwing of infected beef	$(0,0,0,0,0,0,0,0,-1,0)$	εB_I
Consumption of infected beef	$(0,0,0,0,0,0,0,0,-1,0)$	$\alpha_b B_I$
Shedding of taenia eggs	$(0,0,0,0,0,0,0,0,0,1)$	νI_{TH}
Death of taenia eggs	$(0,0,0,0,0,0,0,0,0,-1)$	$\mu_e E_T$

3.15.1 The Multitype Branching Process

The multitype branching process is used to study the dynamics of the CTMC model near the disease free equilibrium (DFE) (Allen & Lahodny Jr, 2012; Allen & van den Driessche, 2013; Maliyoni *et al.*, 2017). It is used to determine the probability of disease extinction or outbreak. If there are only few infectives at the beginning of disease outbreak, then the branching process either grows exponentially or hits zero (Allen, 2017). The multitype branching process is applied to infectious classes (infected humans, pigs,cattle, pork, beef) and taenia eggs in the en-

vironment which are the sources of infections. Susceptible humans, pigs and cattle are assumed to be at the DFE, that is $S_H^0 = \psi/\mu_h$, $S_P^0 = \Lambda/(\rho+\mu_p)$ and $S_C^0 = \phi/(\sigma+\mu_c)$ (Lahodny & Allen, 2013). We assume that infectious hosts of type i, I_i infects a susceptible individual of another type leading to infectious individuals of type j, I_j and that the number of infected individuals produced by an individual of type i is independent of the number of individuals produced by either type i or type j, $j \neq i$ (Allen & van den Driessche, 2013; Maliyoni *et al.*, 2017). Furthermore, we assume that the initial susceptible humans, pigs and cattle are sufficiently large, that is $S_H(0) \approx N_H(0) = S_H^0$, $S_P(0) \approx N_P(0) = S_P^0$ and $S_C(0) \approx N_C(0) = S_C^0$ where $N_H(0)$, $N_P(0)$ and $N_C(0)$ are the initial total human, pigs and cattle populations respectively. Since the multitype branching process is linear near the DFE, then the number of infections and deaths are independent. The offspring probability generating functions (pgfs) for infectious humans, pigs, cattle, taenia eggs, infectious pork and beef are defined. These offspring pgfs are then used to determine the probability of disease extinction or outbreak (Lahodny & Allen, 2013).

3.15.2 Stochastic Threshold for CTMC Epidemic Model

We apply the multitype branching process to the CTMC stochastic model by defining the probability generating functions for all infected classes. The model assumes that the initial susceptible humans and cattle are at disease free equilibrium and sufficiently large such that $S_H(0) = \Lambda_H/\mu_h = S_H^0$, $S_P(0) = \Lambda_P/(\rho+\mu_p) = S_P^0$ and $S_C(0) = \Lambda_C/(\sigma+\mu_b) = S_C^0$. If initially there were only one infected human with taeniasis $(I_{HT}(0) = 1)$ and there are no humans with cysticercosis, no infected pigs, pork, cattle, beef and *T. saginata* eggs in the environment, that is $(I_{HC}(0) = 0, I_P(0) = 0, P_I(0) = 0, I_C(0) = 0, B_I(0) = 0$ and $E_T(0) = 0$ respectively), we define the offspring probability generating function (pgf) by using equation (3.75). Thus, the offspring probability generating function for I_{TH} is given by:

$$f_1(u_1, u_2, .., u_7) = \frac{\nu u_1 u_7 + \mu_h}{\nu + \mu_h}. \tag{3.151}$$

The term $\nu/(\nu+\mu_h)$ represents the probability that the initial infectious human sheds a taenia egg in the environment and the initial host does not die resulting into one infectious human and one taenia egg in the environment while $\mu_h/(\nu+\mu_h)$ is the probability that an initial infected human is lost due to natural death leading to zero infectious humans and therefore no taenia eggs in the environment .

Since humans who are infected with cysticercosis do not play any role in the spread of the disease, therefore the offspring pgf for I_{HC} given that $I_{HT}(0) = 0, I_{HC}(0) = 1, I_P(0) = 0, P_I(0) = 0, I_C(0) = 0, B_I(0) = 0, E_T(0) = 0$ is:

$$f_2(u_1,u_2,...,u_7)=1. \tag{3.152}$$

The offspring pgf for I_P given that $I_{HT}(0)=0, I_{HC}(0)=0, I_P(0)=1, P_I(0)=0, I_C(0)=0, B_I(0)=0, E_T(0)=0$ is:

$$f_3(u_1,u_2,...,u_7)=\frac{\omega u_4+\mu_p}{\omega+\mu_p}. \tag{3.153}$$

The term $\omega/(\omega+\mu_p)$ is the probability that the initial infectious pig is slaughtered for consumption resulting to the presence of infected pork only whereas $\mu_p/(\omega+\mu_p)$ is the probability that the initial infectious pig may die before being slaughtered leading to zero infectious pigs and therefore no infectious pork.

The offspring pgf for P_I given that $I_{HT}(0)=0, I_{HC}(0)=0, I_P(0)=0, P_I(0)=1, I_C(0)=0, B_I(0)=0, E_T(0)=0$ is:

$$f_4(u_1,u_2,...,u_7)=\frac{\beta_T\alpha_p S_H^0 u_1 u_4+\delta}{\beta_T\alpha_p S_H^0+\delta}. \tag{3.154}$$

The term $\beta_T\alpha_p S_H^0/(\beta_T\alpha_p S_H^0+\delta)$ represents the probability that a proportion of initial infectious pork is lost due to consumption by susceptible humans resulting to one infectious human. The term $\delta/(\beta_T\alpha_p S_H^0+\delta)$ is the probability that the initial infectious pork is thrown before consumption following meat inspection leading to zero infectious pork and therefore no infectious humans.

The offspring pgf for I_C given that $I_{HT}(0)=0, I_{HC}(0)=0, I_P(0)=0, P_I(0)=0, I_C(0)=1, B_I(0)=0, E_T(0)=0$ is:

$$f_5(u_1,u_2,...,u_7)=\frac{\eta u_6+\mu_c}{\eta+\mu_c}. \tag{3.155}$$

The term $\eta/(\eta+\mu_b)$ is the probability that the initial infectious cattle is slaughtered for consumption resulting to the presence of infectious beef only whereas $\mu_c/(\eta+\mu_c)$ is the probability that the initial infectious cattle may die before being slaughtered leading to zero infectious cattle and thus no infectious beef.

The offspring pgf for B_I given that $I_{HT}(0)=0, I_{HC}(0)=0, I_P(0)=0, P_I(0)=1, I_C(0)=1, B_I(0)=1, E_T(0)=0$ is:

$$f_6(u_1,u_2,...,u_7)=\frac{\beta_T\alpha_b S_H^0 u_1 u_6+\varepsilon}{\beta_T\alpha_b S_H^0+\varepsilon}. \tag{3.156}$$

The term $\beta_T\alpha_b S_H^0/(\beta_T\alpha_b S_H^0+\varepsilon)$ represents the probability that a proportion of initial infectious beef is lost due to consumption by susceptible humans resulting to one human who is infected

with taeniasis. The term $\varepsilon/(\beta_T \alpha_b S_H^0 + \varepsilon)$ is the probability that the initial infectious beef is thrown before consumption following meat inspection leading to zero infectious beef.

The offspring pgf for E_T given that $I_{HT}(0) = 0, I_{HC}(0) = 0, I_P(0) = 0, P_I(0) = 0, I_C(0) = 0, B_I(0) = 0, E_T(0) = 1$ is:

$$f_7(u_1, u_2, ..., u_7) = \frac{\theta S_H^0 u_2 u_7 + \gamma_p S_P^0 u_3 u_7 + \gamma_b S_C^0 u_5 u_7 + \mu_e}{\theta S_H^0 + \gamma_p S_P^0 + \gamma_c S_C^0 + \mu_e}. \tag{3.157}$$

The term $\theta S_H^0/(\theta S_H^0 + \gamma_p S_P^0 + \gamma_c S_C^0 + \mu_e)$ represents the probability that the initial taenia egg infects a susceptible human resulting to one human with cysticercosis and a taenia egg. The term $\gamma_p S_P^0/(\theta S_H^0 + \gamma_p S_P^0 + \gamma_c S_C^0 + \mu_e)$ represents the probability that the initial taenia egg infects a susceptible pig leading to one infectious pig and a taenia egg. The term $\gamma_c S_C^0/(\theta S_H^0 + \gamma_p S_P^0 + \gamma_c S_C^0 + \mu_e)$ represents the probability that the initial taenia egg infects a susceptible cattle resulting to one infectious cattle and a taenia egg while $\mu_e/(\theta S_H^0 + \gamma_p S_P^0 + \gamma_c S_C^0 + \mu_e)$ is the probability that a taenia egg is lost through death leading to zero taenia eggs in the environment.

The expectation matrix $\mathbf{M}$ of the offspring probability generating function (pgf) is given by:

$$\mathbf{M} = \begin{pmatrix} \frac{\nu}{\nu+\mu_h} & 0 & 0 & \frac{\beta_T \alpha_p S_H^0}{\beta_T \alpha_p S_H^0 + \delta} & 0 & \frac{\beta_T \alpha_b S_H^0}{\beta_T \alpha_b S_H^0 + \varepsilon} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\theta S_H^0}{J} \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\gamma_p S_P^0}{J} \\ 0 & 0 & \frac{\omega}{\omega+\mu_p} & \frac{\beta_T \alpha_p S_H^0}{\beta_T \alpha_p S_H^0 + \delta} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\gamma_c S_C^0}{J} \\ 0 & 0 & 0 & 0 & \frac{\eta}{\eta+\mu_c} & \frac{\beta_T \alpha_b S_H^0}{\beta_T \alpha_b S_H^0 + \varepsilon} & 0 \\ \frac{\nu}{\nu+\mu_h} & 0 & 0 & 0 & 0 & 0 & \frac{J-\mu_e}{J} \end{pmatrix}, \tag{3.158}$$

where $J = \theta S_H^0 + \gamma_p S_P^0 + \gamma_c S_C^0 + \mu_e$.

The stochastic threshold for disease extinction or outbreak in humans, pigs and cattle for the CTMC stochastic model is the spectral radius of the expectation matrix, $\rho(\mathbf{M})$. The basic reproduction number $\mathscr{R}_0$ for deterministic model and the stochastic threshold $\rho(\mathbf{M})$ for CTMC stochastic model are closely related (Maliyoni *et al.*, 2017). The diseases die in humans, pigs

and cattle if $\rho(\mathbf{M}) \leq 1$ or $\mathscr{R}_0 \leq 1$. In deterministic models, the diseases persist in humans, pigs and cattle if $\mathscr{R}_0 > 1$. However in stochastic models, if $\rho(\mathbf{M}) > 1$ there is a possibility for disease outbreak or extinction depending on number of infectives that were initially available at the begining of disease outbreak (Allen & van den Driessche, 2013; Lahodny & Allen, 2013; Maliyoni, 2021). Thus, if $\rho(\mathbf{M}) > 1$, there exist a fixed point $(q_1, q_2, q_3, q_4, q_5, q_6, q_7) \in (0,1)^7$ of the offspring generating functions (3.151)-(3.157) that is used in writing the probability of disease extinction (Allen & Lahodny Jr, 2012; Maliyoni *et al.*, 2017). To get the fixed point, we set $f_i(q_1, q_2, q_3, q_4, q_5, q_6, q_7) = q_i$ for $i = 1, 2, .., 7$. That is:

$$\begin{aligned}
q_1 &= \frac{\nu q_1 q_7 + \mu_h}{\nu + \mu_h}, \\
q_2 &= 1, \\
q_3 &= \frac{\omega q_4 + \mu_p}{\omega + \mu_p}, \\
q_4 &= \frac{\beta_T \alpha_p S_H^0 q_1 q_4 + \delta}{\beta_T \alpha_p S_H^0 + \delta}, \\
q_5 &= \frac{\eta q_6 + \mu_c}{\eta + \mu_c}, \\
q_6 &= \frac{\beta_T \alpha_b S_H^0 q_1 q_6 + \varepsilon}{\beta_T \alpha_b S_H^0 + \varepsilon}, \\
q_7 &= \frac{\theta S_H^0 q_2 q_7 + \gamma_p S_P^0 q_3 q_7 + \gamma_c S_C^0 q_5 q_7 + \mu_e}{\theta S_H^0 + \gamma_p S_P^0 + \gamma_c S_C^0 + \mu_e}.
\end{aligned} \tag{3.159}$$

Due to non-linearity of probability generating functions (pgf) particularly the pgf in (3.157), q_i's are computed numerically in Chapter 4.

3.16 Dynamics of Cysticercosis and Taeniasis in Humans, Pigs and Cattle with Control Measures

Recently, the theory of optimal control and cost effectiveness analysis have gained popularity due to their power to analyze various mathematical models for determining effective control strategies. Currently, there is no study that has applied the optimal control theory and cost effectiveness analysis to assess the best strategy for controlling taeniasis and cysticercosis in humans, pigs and cattle. In this section, we formulate and analyze the optimal control model for taeniasis and cysticercosis and apply the cost-effectiveness analysis to determine the most effective strategy for disease control.

3.17 Model Formulation

A mathematical model for the transmission dynamics of taeniasis and cyticercosis with control measures is formulated basing on the basic model in Mwasunda *et al.* (2021). We divide the human population into S_H, I_{HT} and I_{HC} classes that represent susceptible humans, humans who are infected with taeniasis and humans who are infected with cysticercosis respectively. Pig and cattle populations are divided into susceptible, vaccinated, infected and recovered classes represented by S_P, V_P, I_P, R_P and S_C, V_C, I_C, R_C respectively. The compartments P_I and B_I denote pork and beef infected with larval cysts respectively, while E_T is the number of taenia eggs in the environment.

Susceptible humans are recruited through birth at a rate Λ_H and decrease at rates α_P and α_b due to consumption of raw or insufficiently cooked infected pork or beef respectively. Susceptible humans further decrease when they consume *T. solium* eggs from the contaminated water, fruits and vegtables or by putting contaminated fingers in their mouth (Del Brutto, 2013). The parameter β_T is the probability of susceptible humans to acquire taeniasis following consumption of infected pork and beef whereas $\lambda_{HT} = \beta_T(\alpha_p P_I + \alpha_b BI)$ and $\lambda_{HC} = \theta E_T$ are the forces of infections for humans to acquire taeniasis and cysticercosis due to consumption of raw or undercooked infected meat and teania eggs from the contaminated environment respectively. Humans who are infected with taeniasis and cysticercosis recover due to treatment at rates χ and χ_c respectively and move to the susceptible class. All humans are assumed to die naturally at a rate μ_h, except humans infected with cysticercosis who have an additional disease induced mortality rate μ_d. Taenia eggs in the environment grow at a rate ν due to open defecation by humans who are infected with taeniasis and diminish at a rate μ_e due to natural death.

Susceptible pigs and cattle are recruited at per capita rates Λ_P and Λ_c respectively due to birth. Parameters γ_p and γ_b are the rates at which cattle and pigs acquire cysticercosis from the contaminated environment respectively, and ρ_b is the vaccine efficacy for protecting vaccinated cattle and pigs against infection. The parameters λ_p and ω are the rates at which infected pigs are treated and slaughtered for consumption respectively while λ_b and η are the rates at which infected cattle are treated and slaughtered for consumption respectively. Both recovered pigs and cattle lose their immunity at rates π_p and π_b respectively and move to susceptible classes. Susceptible pigs and cattle are vaccinated at rates ϕ_s and ψ_s respectively and slaughtered for consumption at rates ρ_s and σ_s respectively. The parameters ϕ_v and ψ_v are the rates at which the vaccines wane in vaccinated pigs and cattle respectively. Moreover, vaccinated pigs and cattle are slaughtered for consumption at rates ρ_v and σ_v respectively while recovered pigs and cattle are slaughtered at the rates ρ_p and and σ_b respectively. All pigs and cattle suffer natural death at rates μ_p and μ_b respectively. The parameters δ and ε are the proportions of infected pork and

beef which are not consumed by susceptible humans.

In formulating the optimal control problem, we consider the free range farming system for both pigs and cattle, and we do not consider migration. We assume that humans can be infected by either taeniasis or cysticercosis; the number of taenia eggs consumed by humans, pigs and cattle has negligible effect on the total number of eggs in the environment and infected humans, pigs and cattle cannot recover naturally without treatment. We further assume that pigs and cattle do not suffer disease induced mortality, they become carriers for their life and the rates at which susceptible humans consume infected raw or undercooked pork or beef depend on the amount of infected pork or beef that is present. Humans, pigs and cattle contact rates with taenia eggs in the environment are also assumed to follow the principle of mass action. Furthermore, we assume that infected humans, pigs and cattle cannot recover from infections without treatment and that the vaccines which are provided to susceptible pigs and cattle are not 100% effective, hence they wane after sometime. The model parameters are summarized in Table 11. Figure 9 describes the interaction of humans, pigs and cattle when some interventions are implemented.

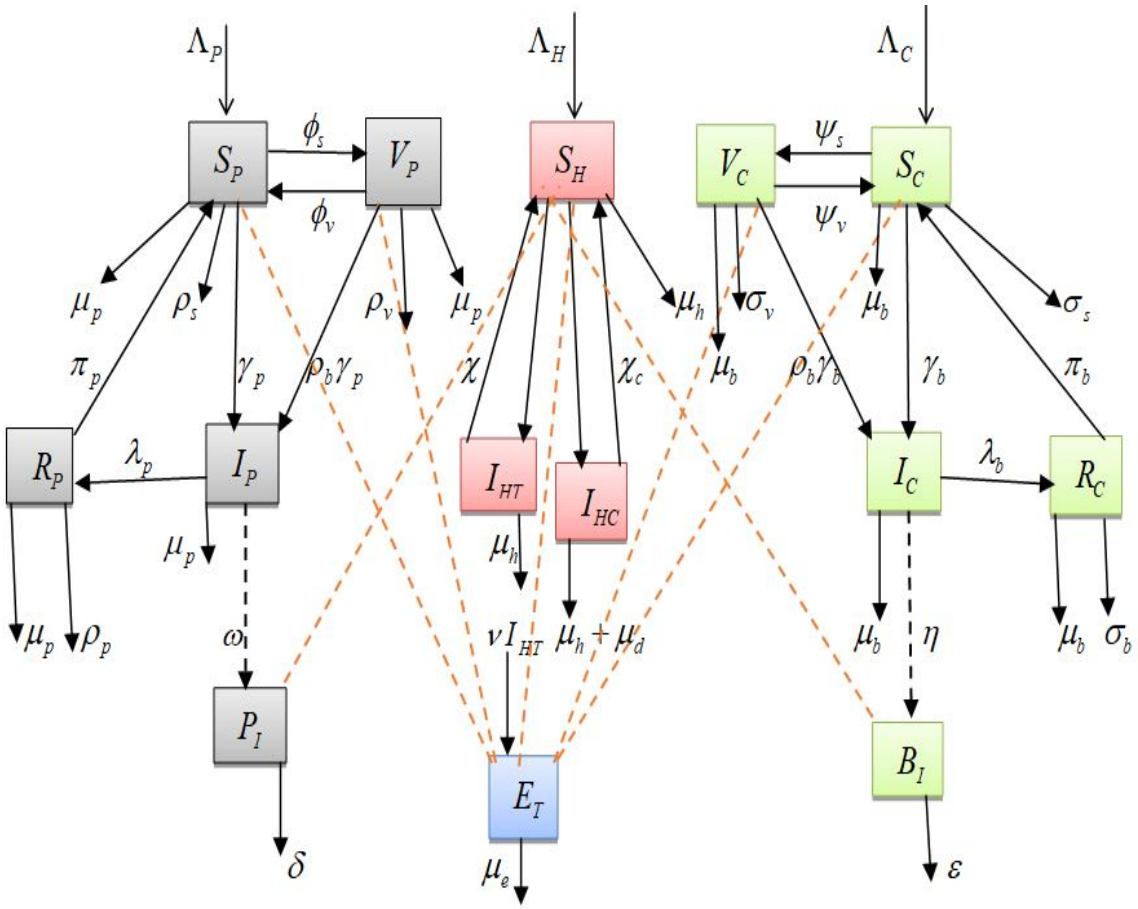


Figure 9: Model schematic diagram

Table 11: Description of model parameters

Parameter	Description
Λ_H	Per capita recruitment rate of human population
Λ_C	Per capita recruitment rate of cattle population
Λ_P	Per capita recruitment rate of pig population
μ_h	Per capita natural death rate of humans
α_p	Rate of eating raw/undercooked infected pork
λ_p	Recovery rate of infected pigs from cysticercosis
π_p	Immunity waning rate for recovered pigs
λ_b	Recovery rate of infected cattle from cysticercosis
π_b	Immunity waning rate for recovered cattle
χ	Recovery rate of infected humans from taeniasis
χ_c	Recovery rate of infected humans from cysticercosis
α_b	Rate of eating raw/undercooked infected beef
γ_p	*T. solium* eggs to pig transmission rate
γ_b	*T. saginata* eggs to susceptible cattle transmission rate
ρ_b	Vaccine efficacy to protect cattle and pigs against infection
ω	Slaughter rate of infected pigs
θ	*T. solium* eggs to human cysticercosis transmission rate
η	Slaughter rate of infected cattle
μ_b	Per capita natural death rate of cattle
μ_p	Per capita natural death rate of pigs
μ_d	Human cysticercosis disease induced death rate
ε	Proportion of unconsumed infected beef by humans
δ	Proportion of unconsumed infected pork by humans
β_T	Probability of humans getting taeniasis
ν	Defecation rate by humans with taeniasis
μ_e	Per capita death rate of taenia saginata eggs
σ_s	Harvesting rate of susceptible cattle
σ_v	Harvesting rate of vaccinated cattle
σ_b	Harvesting rate of recovered cattle
ρ_s	Harvesting rate of susceptible pigs
ρ_v	Harvesting rate of vaccinated pigs
ρ_p	Harvesting rate of recovered pigs
ϕ_s	Rate at which susceptible pigs are vaccinated
ϕ_v	Waning rate of vaccine in pigs
ψ_s	Rate at which susceptible cattle are vaccinated
ψ_v	Waning rate of vaccine in cattle

Following the description above, the mathematical model for the transmission dynamics and control of taeniasis and cysticercosis in human, pig and cattle populations is governed by the following system of differential equations:

$$
\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H + \chi I_{HT} + \chi_c I_{HC} - \beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \theta S_H E_T - \mu_h S_H,\\
\frac{dI_{HT}}{dt} &= \beta_T(\alpha_p P_I + \alpha_b B_I)S_H - (\chi + \mu_h)I_{HT},\\
\frac{dI_{HC}}{dt} &= \theta S_H E_T - (\mu_h + \mu_d + \chi_c)I_{HC},\\
\frac{dS_P}{dt} &= \Lambda_P + \pi_p R_P + \phi_v V_P - \gamma_p S_P E_T - (\rho_s + \mu_p + \phi_s)S_P,\\
\frac{dV_P}{dt} &= \phi_s S_P - \rho_b \gamma_p V_P E_T - (\rho_v + \mu_p + \phi_v)V_P,\\
\frac{dI_P}{dt} &= \gamma_p S_P E_T + \rho_b \gamma_p V_P E_T - (\omega + \lambda_p + \mu_p)I_P,\\
\frac{dR_P}{dt} &= \lambda_p I_P - (\rho_p + \pi_p + \mu_p)R_P,\\
\frac{dP_I}{dt} &= \omega I_P - (\delta + \alpha_P)P_I,\\
\frac{dS_C}{dt} &= \Lambda_C + \pi_b R_C + \psi_v V_C - \gamma_b S_C E_T - (\sigma_s + \mu_b + \psi_s)S_C,\\
\frac{dV_C}{dt} &= \psi_s S_C - \rho_b \gamma_b V_C E_T - (\sigma_v + \mu_b + \psi_v)V_C,\\
\frac{dI_C}{dt} &= \gamma_b S_C E_T + \rho_b \gamma_b V_C E_T - (\eta + \lambda_b + \mu_b)I_C,\\
\frac{dR_C}{dt} &= \lambda_b I_C - (\sigma_b + \pi_b + \mu_b)R_C,\\
\frac{dB_I}{dt} &= \eta I_C - (\varepsilon + \alpha_b)B_I,\\
\frac{dE_T}{dt} &= \nu I_{HT} - \mu_e E_T,
\end{aligned}
\tag{3.160}
$$

with initial conditions:

$$
\begin{aligned}
&S_H(0) > 0; I_{HT}(0) \geq 0; I_{HC}(0) \geq 0; S_P(0) > 0; V_P(0) > 0; I_P(0) \geq 0; R_P(0) \geq 0;\\
&P_I(0) \geq 0; S_C(0) > 0; V_C(0) > 0; I_C(0) \geq 0; R_C(0) \geq 0; B_I(0) \geq 0 \text{ and } E_T(0) \geq 0.
\end{aligned}
$$

3.18 Model Basic Properties

For the model system (3.160) to be epidemiologically meaningful, we need to show that all its state variables are non-negative and bounded.

3.18.1 Positivity of Model Solutions

Theorem 3.15

Let the initial conditions for the model system (3.101) be:

$$S_H(0)>0; I_{HT}(0)\geq 0; I_{HC}(0)\geq 0; S_P(0)>0; V_P(0)>0; I_P(0)\geq 0; R_P(0)\geq 0; P_I(0)\geq 0;$$
$$S_C(0)>0; V_C(0)>0; I_C(0)\geq 0; R_C(0)\geq 0; B_I(0)\geq 0; E_T(0)\geq 0.$$

Then the solutions $(S_H, I_{HT}, I_{HC}, S_P, V_P, I_P, R_P, P_I, S_C, V_C, I_C, R_C, B_I, E_T)$ of the model with positive initial conditions, will remain non-negative for all time $t>0$.

Proof.

$$\begin{aligned}\text{Let } t_1 = sup\{t>0 : S_H>0, I_{HT}>0, I_{HC}>0, S_P>0, V_P>0, I_P>0, R_P>0, P_I>0,\\ S_C>0, V_C>0, I_C>0, R_C>0, B_I>0, E_T>0 \in [0,t]\},\end{aligned}$$

so that $t_1>0$. Then, from the first equation for susceptible humans in the model system (3.160), we have:

$$\begin{aligned}\frac{dS_H}{dt} &= \Lambda_H + \chi I_{HT} + \chi_c I_{HC} - \beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \theta S_H E_T - \mu_h S_H,\\ \frac{dS_H}{dt} &\geq -(\beta_T(\alpha_p P_I + \alpha_b B_I) + \theta E_T + \mu_h) S_H,\\ \frac{dS_H}{S_H} &\geq -(\beta_T(\alpha_p P_I + \alpha_b B_I) + \theta E_T + \mu_h)\, dt,\\ S_H(t_1) &\geq S_H(0) e^{\int_0^{t_1} -(\beta_T(\alpha_p P_I(s) + \alpha_b B_I(s)) + \theta E_T(s) + \mu_h)\, ds} \geq 0.\end{aligned} \tag{3.161}$$

In similar manner, it can be shown that:

$$\begin{aligned}&I_{HT}(t)\geq 0; I_{HC}(t)\geq 0; S_P(t)\geq 0; V_P(t)\geq 0; I_P(t)\geq 0; R_P(t)\geq 0; P_I(t)\geq 0;\\ &S_C(t)\geq 0; V_C(t)\geq 0; I_C(t)\geq 0; R_C(t)\geq 0; B_I(t)\geq 0; E_T(t)\geq 0, \forall t\geq 0.\end{aligned} \tag{3.162}$$

Therefore, all solutions of the model system (3.160) will remain positive for all non-negative initial conditions.

3.18.2 Invariant Region

To find the biologically feasible region, we let $N_H = S_H + I_{HT} + I_{HC}, N_P = S_P + V_P + I_P + R_P$ and $N_C = S_C + V_C + I_C + R_C$ to be total populations for humans, pigs and cattle respectively.

Adding all the equations of the model system (3.160) for human population, we obtain:

$$\begin{aligned} &\frac{dN_H}{dt} = \Lambda_H - \mu_h N_H - \mu_d I_{HC}, \\ \Longrightarrow\ &\frac{dN_H}{dt} + \mu_h H \leq \Lambda_H. \end{aligned} \tag{3.163}$$

Integrating throughout and assuming that the initial total human population $N_H(0)$ cannot exceed Λ_H/μ_h, then it can be shown that the total human population $N_H \to \Lambda_H/\mu_h$ as $t \to \infty$. Also, using the same procedure the total pig population $N_P \to \Lambda_P/(\rho_s + \mu_b)$ and cattle population $N_C \to \Lambda_C/(\sigma_s + \mu_b)$ as $t \to \infty$.

Considering the last equation of the model system (3.160) for taenia eggs in the environment, we have:

$$\frac{dE_T}{dt} = \nu I_{HT} - \mu_e E_T. \tag{3.164}$$

Since the total human population $N_H \to \Lambda_H/\mu_h$, it can be concluded that $I_{HT} \leq \Lambda_H/\mu_h$. Thus we have:

$$\begin{aligned} &\frac{dE_T}{dt} \leq \nu\Lambda_H/\mu_h - \mu_e E_T, \\ \Longrightarrow\ &\frac{dE_T}{dt} + \mu_e E_T \leq \nu\Lambda_H/\mu_h. \end{aligned} \tag{3.165}$$

Applying the same procedure as for equation (3.163), it can be shown that the number of taenia eggs in the environment $E_T \to \nu\Lambda_H/\mu_h\mu_e$ as $t \to \infty$. Similarly, considering the equations for infected pork and beef in the mode system (3.160), it can be shown that $P_I \to \omega\Lambda_P/(\alpha_b + \delta)(\rho_s + \mu_b)$ and $B_I \to \eta\Lambda_C/(\varepsilon + \alpha_b)(\sigma_s + \mu_b)$ as $t \to \infty$. Therefore, this indicates that the biological feasible region:

$$\begin{aligned} \Omega_2 = \Big\{ &(S_H, I_{HT}, I_{HC}, S_P, V_P, I_P, R_P, P_I, S_C, V_C, I_C, R_C, B_I, E_T) \in \mathbb{R}_+^{14} : \\ &N_H \leq \Lambda_H/\mu_h; N_P \leq \Lambda_P/(\rho_s + \mu_b);\ N_C \leq \Lambda_C/(\sigma_s + \mu_b); E_T \leq \nu\Lambda_H/\mu_h\mu_e; \\ &P_I \leq \omega\Lambda_P/(\alpha_b + \delta)(\rho_s + \mu_b); B_I \leq \eta\Lambda_C/(\varepsilon + \alpha_b)(\sigma_s + \mu_b) \Big\} \end{aligned} \tag{3.166}$$

is positive invariant and hence the model system (3.160) is epidemiologically and mathematically well posed. Therefore, it is sufficient to consider the model system (3.160) for analysis.

3.19 Disease Free Equilibrium and Effective Reproduction Number

When there are no diseases in human, pig and cattle populations, then the disease free equilibrium E_2^0 is given by:

$$\begin{aligned} &E_2^0(S_H, I_{HT}, I_{HC}, S_P, V_P, I_P, R_P, P_I, S_C, V_C, I_C, R_C, B_I, E_T) \\ &= \left(\frac{\Lambda_H}{\mu_h}, 0, 0, \frac{d_0\Lambda_P}{M_0}, \frac{\phi_s\Lambda_P}{M_0}, 0, 0, 0, \frac{c_0\Lambda_C}{K_0}, \frac{\psi_s\Lambda_C}{K_0}, 0, 0, 0, 0\right), \end{aligned} \tag{3.167}$$

where

$$\begin{aligned} c_0 &= (\sigma_v + \psi_v + \mu_b)\ ,\ K_0 = c_0(\sigma_s + \mu_b) + \psi_s(\sigma_v + \mu_b), \\ d_0 &= (\rho_v + \phi_v + \mu_p)\ ,\ M_0 = d_0(\rho_s + \mu_p) + \phi_s(\rho_v + \mu_p). \end{aligned} \tag{3.168}$$

The effective reproduction number R_e is the expected number of new infections that may arise as a consequence of introducing one infected individual in a susceptible population when some interventions are implemented to control the spread of the disease Diekmann *et al.* (1990). The controls are effective if $R_e < 1$ and ineffective if $R_e > 1$. In this section, we adopt the next generation matrix method to compute R_e Van den Driessche & Watmough (2002). Let $\mathscr{F}_i$ and $\mathscr{V}_i$ be new infections and transfer terms in compartment i respectively, given by:

$$\mathscr{F}_i = \begin{pmatrix} \beta_T(\alpha_p P_I + \alpha_b B_I)S_H \\ \theta S_H E_T \\ \gamma_p S_P E_T + \rho_b\gamma_p V_P E_T \\ 0 \\ \gamma_b S_C E_T + \rho_b\gamma_c V_C E_T \\ 0 \\ 0 \end{pmatrix},\ \mathscr{V}_i = \begin{pmatrix} (\chi + \mu_h)I_{HT} \\ (\mu_h + \mu_d + \chi_c)I_{HC} \\ (\omega + \lambda_p + \mu_p)I_P \\ -\omega I_P + (\delta + \alpha_p)P_I \\ (\eta + \lambda_b + \mu_b)I_C \\ -\eta I_C + (\varepsilon + \alpha_b)B_I \\ -\nu I_{HT} + \mu_e E_T \end{pmatrix}. \tag{3.169}$$

The effective reproduction number R_e is given by:

$$R_e = \rho\left(FV^{-1}\right), \tag{3.170}$$

where F and V are Jacobian matrices evaluated at disease free equilibrium and they are given by:

$$F = \frac{\partial \mathscr{F}_i}{\partial x_j}\left(E_2^0\right),\ V = \frac{\partial \mathscr{V}_i}{\partial x_j}\left(E_2^0\right). \tag{3.171}$$

Using the definitions in (3.171), we have:

$$F = \begin{pmatrix} 0 & 0 & 0 & \frac{\beta_T \alpha_p \psi}{\mu_h} & 0 & \frac{\beta_T \alpha_b \psi}{\mu_h} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{\theta \psi}{\mu_h} \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{(d_0+\rho_b\phi_s)\gamma_p\Lambda_P}{M_0} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{(c_0+\rho_b\psi_s)\gamma_p\Lambda_C}{K_0} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{3.172}$$

and

$$V = \begin{pmatrix} (\chi+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & (\mu_h+\mu_d+\chi_c) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (\omega+\lambda_p+\mu_p) & 0 & 0 & 0 & 0 \\ 0 & 0 & -\omega & (\delta+\alpha_p) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & (\eta+\lambda_b+\mu_b) & 0 & 0 \\ 0 & 0 & 0 & 0 & -\eta & (\varepsilon+\alpha_b) & 0 \\ -\nu & 0 & 0 & 0 & 0 & 0 & \mu_e \end{pmatrix}. \tag{3.173}$$

Using the definition in (3.170), the effective reproduction number R_e is given by:

$$R_e = \sqrt{R_1 + R_2}, \tag{3.174}$$

where

$$\begin{aligned} R_1 &= \frac{\beta_T \nu \omega \alpha_p \gamma_p \Lambda_H \Lambda_P (d_o + \rho_b \phi_s)}{M_0 \mu_h \mu_e (\chi+\mu_h)(\delta+\alpha_p)(\omega+\lambda_p+\mu_p)} \\ R_2 &= \frac{\beta_T \nu \eta \alpha_b \gamma_b \Lambda_H \Lambda_C (c_o + \rho_b \psi_s)}{K_0 \mu_h \mu_e (\chi+\mu_h)(\varepsilon+\alpha_b)(\eta+\lambda_b+\mu_b)}. \end{aligned} \tag{3.175}$$

R_{e1} and R_{e2} are the partial reproduction numbers due to interaction of humans with pigs and with cattle respectively.

When there are no interventions ($\phi_s = \phi_v = \rho_v = \rho_b = \lambda_p = \lambda_b = \pi_p = \pi_b = \chi = \chi_c = \psi_s = \psi_v = \sigma_v = \sigma_b = 0$), then the effective reproduction number R_e reduces to the basic reproduction number R_0 given by:

$$R_0 = \sqrt{R_{HP} + R_{HC}}, \tag{3.176}$$

where

$$R_{HP} = \frac{\beta_T \nu \alpha_p \gamma_p \omega \Lambda_H \Lambda_P}{\mu_h^2 \mu_e (\omega + \mu_p)(\alpha_p + \delta)(\mu_p + \rho_s)},$$
$$R_{HC} = \frac{\beta_T \nu \alpha_b \gamma_b \eta \Lambda_H \Lambda_C}{\mu_h^2 \mu_e (\eta + \mu_b)(\alpha_b + \varepsilon)(\mu_b + \sigma_s)}. \tag{3.177}$$

3.20 The Optimal Control Problem for Taeniasis and Cysticercosis in Humans, Pigs and Cattle

In this section we administer time dependent control variables to determine if they can eradicate or contain taeniasis and cysticercosis in humans, pigs and cattle. The choice of control variables is based on the sensitivity analysis results for the basic reproduction number R_0 which are also presented in (Mwasunda *et al.*, 2021) that shows that interventions which focus on reducing the probability of human infection with taeniasis and defecation rate by humans with taeniasis will help in controlling the transmission of taeniasis and cysticercosis in humans, pigs and cattle. To optimally control taeniasis and cysticercosis in humans, pigs and cattle, we incorporate five time dependent control variables that are $u_1(t)$ which measures the effect of meat inspection in reducing the possibility of human infection with taeniasis, $u_2(t)$ and $u_3(t)$ which represent the control efforts due to vaccination of pigs and cattle respectively, $u_4(t)$ which measures treatment efforts of humans who are infected with taeniasis and $u_5(t)$ which measures the impact of improved hygiene and sanitation to reduce the rate of transfer of infections from contaminated environment to pigs, cattle and humans. Thus, incorporating these control variables in model system (3.101) we obtain:

$$
\begin{aligned}
\frac{dS_H}{dt} &= \Lambda_H + u_4 I_{HT} + \chi_c I_{HC} - \beta_T(1-u_1)(\alpha_p P_I + \alpha_b B_I)S_H \\
&\quad -\theta(1-u_5)S_H E_T - \mu_h S_H, \\
\frac{dI_{HT}}{dt} &= \beta_T(1-u_1)(\alpha_p P_I + \alpha_b B_I)S_H - (u_4+\mu_h)I_{HT}, \\
\frac{dI_{HC}}{dt} &= \theta(1-u_5)S_H E_T - (\mu_h + \mu_d + \chi_c)I_{HC}, \\
\frac{dS_P}{dt} &= \Lambda_P + \pi_p R_P + \phi_v V_P - \gamma_p(1-u_5)S_P E_T - (\rho_s + \mu_p + u_2)S_P, \\
\frac{dV_P}{dt} &= u_2 S_P - \rho_b\gamma_p(1-u_5)V_P E_T - (\rho_v + \mu_p + \phi_v)V_P, \\
\frac{dI_P}{dt} &= \gamma_p(1-u_5)S_P E_T + \rho_b\gamma_p(1-u_5)V_P E_T - (\omega + \lambda_p + \mu_p)I_P, \\
\frac{dR_P}{dt} &= \lambda_p I_P - (\rho_p + \pi_p + \mu_p)R_P, \\
\frac{dP_I}{dt} &= \omega I_P - (\delta + u_1 + \alpha_P)P_I, \\
\frac{dS_C}{dt} &= \Lambda_C + \pi_b R_C + \psi_v V_C - \gamma_b(1-u_5)S_C E_T - (\sigma_s + \mu_b + u_3)S_C, \\
\frac{dV_C}{dt} &= u_3 S_C - \rho_b\gamma_b(1-u_5)V_C E_T - (\sigma_v + \mu_b + \psi_v)V_C, \\
\frac{dI_C}{dt} &= \gamma_b(1-u_5)S_C E_T + \rho_b\gamma_b(1-u_5)V_C E_T - (\eta + \lambda_b + \mu_b)I_C, \\
\frac{dR_C}{dt} &= \lambda_b I_C - (\sigma_b + \pi_b + \mu_b)R_C, \\
\frac{dB_I}{dt} &= \eta I_C - (\varepsilon + u_1 + \alpha_b)B_I, \\
\frac{dE_T}{dt} &= \nu(1-u_5)I_{HT} - \mu_e E_T,
\end{aligned}
\tag{3.178}
$$

We are interested to minimize the number of infected humans, infected pigs and cattle, and the cost of administering the controls. The objective function that minimizes the number of infected humans, pigs, cattle and the cost for administering the controls is given as:

$$
J = \int_0^{T_f} \Big(C_1 I_{HT} + C_2 I_{HC} + C_3 I_P + C_4 I_C + C_5 u_2 S_P + C_6 u_3 S_C + \frac{1}{2}\sum_{j=1}^{j=5} A_j u_j^2 \Big) dt \tag{3.179}
$$

subject to system of differential equations (3.178), where C_1 and C_2 are the constants for minimizing humans who are infected with taeniasis and cysticercosis respectively, C_3 and C_4 are the weight constants for minimizing number of infected pigs and cattle that are slaughtered for consumption respectively whereas the terms $u_2 S_P$ and $u_3 S_C$ aim at minimizing the number of vaccines used for pigs and cattle with weight constants C_5 and C_6 respectively (Martcheva, 2015). The coefficients A_1, A_2, A_3, A_4, A_5 are relative cost weight for each individual control measure that are used to transform the integral into cost expended over a period of T_f years

which is the time period for applying the controls (Rong *et al.*, 2021). The control variables u_i^2 aim at minimizing the cost for implementing the controls. The square term of the controls reflects the non-linearity nature of the cost for implementing the controls, and the half-term minimizes the effect of applying the controls (Asamoah *et al.*, 2020). The initial values are set to be 2800, 2500, 550, 250, 140, 220, 100, 52, 340, 130, 250, 90, 83 and 100 for $S_H, I_{HT}, I_{HC}, S_P, V_P, I_P, R_P, P_I, S_C, V_C, I_C, R_C, B_I$ and E_T classes respectively.

Therefore, we seek to find the optimal controls $u_1^*, u_2^*, u_3^*, u_4^*$ and u_5^* such that:

$$J(u_1^*, u_2^*, u_3^*, u_4^*, u_5^*) = \min_U J(u_1, u_2, u_3, u_4, u_5), \tag{3.180}$$

where $U = \{u : u \text{ is measurable and } 0 \leq u_j(t) \leq 1 \text{ for } t \in [0, T_f]\}$ is the control set.

3.20.1 Characterization of the Optimal Control Problem

We apply Pontryagin's maximum principle (Biswas *et al.*, 2017; Pontryagin, 1962) which provides the necessary conditions that an optimal control problem must satisfy. This principle converts the system of differential equations (3.178) and equation (3.179) into minimization problem point-wise Hamiltonian ($\mathscr{H}$), with respect to control variables $(u_1, u_2, u_3, u_4, u_5)$.

If we defined a Lagrangian $\mathscr{L}$ for the control problem by:

$$\mathscr{L} = C_1 I_{HT} + C_2 I_{HC} + C_3 I_P + C_4 I_C + C_5 u_2 S_P + C_6 u_3 S_C + \frac{1}{2} \sum_{j=1}^{j=5} A_j u_j^2, \tag{3.181}$$

then the Hamiltonian function $\mathscr{H}$ for the control problem is given as:

$$\mathscr{H} = \mathscr{L} + \sum_{n=1}^{n=14} \lambda_n g_n, \tag{3.182}$$

where g_j are the right hand sides of the model system (3.178) and λ_n for $n = 1, 2, 3, ..., 14$ are the adjoint variables associated with state variables $S_H, I_{HT}, I_{HC}, S_P, V_P, I_P, R_P, P_I, S_C, V_C, I_C, R_C, B_I$ and E_T respectively.

Theorem 3.16

Given an optimal control set $(u_1^*, u_2^*, u_3^*, u_4^*, u_5^*)$ and solutions of the corresponding state system (3.178) that minimizes J over U, then there exists adjoint variables λ_n satisfying:

$$\begin{aligned}
&\frac{d\lambda_1}{dt} = -\frac{\partial \mathscr{H}}{\partial S_H}, \ \frac{d\lambda_2}{dt} = -\frac{\partial \mathscr{H}}{\partial I_{HT}}, \ \frac{d\lambda_3}{dt} = -\frac{\partial \mathscr{H}}{\partial I_{HC}}, \ \frac{d\lambda_4}{dt} = -\frac{\partial \mathscr{H}}{\partial S_P}, \ \frac{d\lambda_5}{dt} = -\frac{\partial \mathscr{H}}{\partial V_P}, \\
&\frac{d\lambda_6}{dt} = -\frac{\partial \mathscr{H}}{\partial I_P}, \ \frac{d\lambda_7}{dt} = -\frac{\partial \mathscr{H}}{\partial R_P}, \ \frac{d\lambda_8}{dt} = -\frac{\partial \mathscr{H}}{\partial P_I}, \ \frac{d\lambda_9}{dt} = -\frac{\partial \mathscr{H}}{\partial S_C}, \ \frac{d\lambda_{10}}{dt} = -\frac{\partial \mathscr{H}}{\partial V_C}, \\
&\frac{d\lambda_{11}}{dt} = -\frac{\partial \mathscr{H}}{\partial I_C}, \ \frac{d\lambda_{12}}{dt} = -\frac{\partial \mathscr{H}}{\partial R_C}, \ \frac{d\lambda_{13}}{dt} = -\frac{\partial \mathscr{H}}{\partial B_I}, \ \frac{d\lambda_{14}}{dt} = -\frac{\partial \mathscr{H}}{\partial E_T},
\end{aligned} \tag{3.183}$$

with transversality conditions:

$$\lambda_n(T_f) = 0. \tag{3.184}$$

Furthermore, the control variables $u_1^*, u_2^*, u_3^*, u_4^*$ and u_5^* are given as:

$$\begin{aligned}
u_1^* &= max\Big\{0, min\Big(1, \frac{(\lambda_2 - \lambda_1)\beta_T(\alpha_p P_I + \alpha_b B_I)S_H + \lambda_8 P_I + \lambda_{13} B_I}{A_1}\Big)\Big\}, \\
u_2^* &= max\Big\{0, min\Big(1, \frac{(\lambda_4 - \lambda_5 - C_5)S_P}{A_2}\Big)\Big\}, \\
u_3^* &= max\Big\{0, min\Big(1, \frac{(\lambda_9 - \lambda_{10} - C_6)S_C}{A_3}\Big)\Big\}, \\
u_4^* &= max\Big\{0, min\Big(1, \frac{(\lambda_2 - \lambda_1)I_{HT}}{A_4}\Big)\Big\}, \\
u_5^* &= max\Big\{0, min\Big(1, \frac{f(\lambda_n)}{A_5}\Big)\Big\},
\end{aligned} \tag{3.185}$$

where

$$\begin{aligned}
f(\lambda_n) =& (\lambda_3 - \lambda_1)\theta S_H E_T + (\lambda_6 - \lambda_4)\gamma_p S_P E_T + (\lambda_6 - \lambda_5)\rho_b \gamma_p V_P E_T + \\
& (\lambda_{11} - \lambda_9)\gamma_b S_C E_T + (\lambda_{11} - \lambda_{10})\rho_b \gamma_b V_C E_T + \lambda_{14} \nu I_{HT}.
\end{aligned}$$

Proof. Let $U = (u_1^*, u_2^*, u_3^*, u_4^*, u_5^*)$ be an optimal control set and $S_H^*, I_{HT}^*, I_{HC}^*, S_P^*, V_P^*, I_P^*, R_P^*$, $P_I^*, S_C^*, V_C^*, I_C^*, R_C^*, B_I^*, E_T^*$ be the corresponding state solutions. Using the Pontryagin's maximum principle (Pontryagin *et al.*, 1962; Pontryagin, 2018), there exist adjoint variables that satisfy:

$$\begin{aligned}
&\frac{d\lambda_1}{dt} = -\frac{\partial \mathscr{H}}{\partial S_H}, \ \frac{d\lambda_2}{dt} = -\frac{\partial \mathscr{H}}{\partial I_{HT}}, \ \frac{d\lambda_3}{dt} = -\frac{\partial \mathscr{H}}{\partial I_{HC}}, \ \frac{d\lambda_4}{dt} = -\frac{\partial \mathscr{H}}{\partial S_P}, \ \frac{d\lambda_5}{dt} = -\frac{\partial \mathscr{H}}{\partial V_P}, \\
&\frac{d\lambda_6}{dt} = -\frac{\partial \mathscr{H}}{\partial I_P}, \ \frac{d\lambda_7}{dt} = -\frac{\partial \mathscr{H}}{\partial R_P}, \ \frac{d\lambda_8}{dt} = -\frac{\partial \mathscr{H}}{\partial P_I}, \ \frac{d\lambda_9}{dt} = -\frac{\partial \mathscr{H}}{\partial S_C}, \ \frac{d\lambda_{10}}{dt} = -\frac{\partial \mathscr{H}}{\partial V_C}, \\
&\frac{d\lambda_{11}}{dt} = -\frac{\partial \mathscr{H}}{\partial I_C}, \ \frac{d\lambda_{12}}{dt} = -\frac{\partial \mathscr{H}}{\partial R_C}, \ \frac{d\lambda_{13}}{dt} = -\frac{\partial \mathscr{H}}{\partial B_I}, \ \frac{d\lambda_{14}}{dt} = -\frac{\partial \mathscr{H}}{\partial E_T},
\end{aligned} \tag{3.186}$$

with transversality conditions in (3.184).

Therefore, the adjoint system is given as:

$$
\begin{aligned}
\frac{d\lambda_1}{dt} &= \beta_T(1-u_1)(\lambda_1-\lambda_2)(\alpha_p P_I+\alpha_b B_I)+\theta(1-u_5)(\lambda_1-\lambda_3)E_T+\mu_h\lambda_1,\\
\frac{d\lambda_2}{dt} &= (u_4+\mu_h)\lambda_2-C_1-u_4\lambda_1-\nu(1-u_5)\lambda_{14},\\
\frac{d\lambda_3}{dt} &= (\mu_h+\mu_d+\chi_c)\lambda_3-C_2-\chi_c\lambda_1,\\
\frac{d\lambda_4}{dt} &= \gamma_p(1-u_5)(\lambda_4-\lambda_6)E_T+(\rho_s+\mu_p+u_2)\lambda_4-u_2\lambda_5-u_2C_5,\\
\frac{d\lambda_5}{dt} &= \rho_b\gamma_p(1-u_5)(\lambda_5-\lambda_6)E_T+(\rho_v+\mu_p+\phi_v)\lambda_5-\psi_v\lambda_4,\\
\frac{d\lambda_6}{dt} &= (\omega+\lambda_p+\mu_p)\lambda_6-\lambda_p\lambda_7-\omega\lambda_8-C_3,\\
\frac{d\lambda_7}{dt} &= (\rho_p+\pi_p+\mu_p)\lambda_7-\pi_p\lambda_4,\\
\frac{d\lambda_8}{dt} &= \beta_T(1-u_1)(\lambda_1-\lambda_2)\alpha_p S_H+(\delta+u_1+\alpha_P)\lambda_8,\\
\frac{d\lambda_9}{dt} &= \gamma_b(1-u_5)(\lambda_9-\lambda_{11})E_T+(\sigma_s+\mu_b+u_3)\lambda_9-u_3\lambda_{10}-u_3C_6,\\
\frac{d\lambda_{10}}{dt} &= \rho_b\gamma_b(1-u_5)(\lambda_{10}-\lambda_{11})E_T+(\sigma_v+\mu_b+\psi_v)\lambda_{10}-\psi_v\lambda_9,\\
\frac{d\lambda_{11}}{dt} &= (\eta+\lambda_b+\mu_b)\lambda_{11}-\lambda_b\lambda_{12}-\eta\lambda_{13}-C_4,\\
\frac{d\lambda_{12}}{dt} &= (\sigma_b+\pi_b+\mu_b)\lambda_{12}-\pi_b\lambda_9,\\
\frac{d\lambda_{13}}{dt} &= \beta_T(1-u_1)(\lambda_1-\lambda_2)\alpha_b S_H+(\varepsilon+u_1+\alpha_b)\lambda_{13},\\
\frac{d\lambda_{14}}{dt} &= \theta(1-u_5)(\lambda_1-\lambda_3)S_H+\gamma_p(1-u_5)(\lambda_4-\lambda_6)S_P+\rho_b\gamma_p(1-u_5)(\lambda_5-\lambda_6)V_P\\
&\quad+\gamma_b(1-u_5)(\lambda_9-\lambda_{11})S_C+\rho_b\gamma_b(1-u_5)(\lambda_{10}-\lambda_{11})V_C+\mu_e\lambda_{14}.
\end{aligned}
\tag{3.187}
$$

To obtain the optimality conditions, we differentiate the Hamiltonian function (3.182) with respect to the control variables and solve it when the derivative is zero, that is:

$$
\begin{aligned}
\frac{\partial \mathscr{H}}{\partial u_1} &= A_1 u_1 - (\lambda_2 - \lambda_1)\beta_T(\alpha_p P_I + \alpha_b B_I)S_H - \lambda_8 P_I - \lambda_{13} B_I = 0,\\
\frac{\partial \mathscr{H}}{\partial u_2} &= A_2 u_2 + C_5 S_P - (\lambda_4 - \lambda_5)S_P = 0,\\
\frac{\partial \mathscr{H}}{\partial u_3} &= A_3 u_3 + C_6 S_C - (\lambda_9 - \lambda_{10})S_C = 0,\\
\frac{\partial \mathscr{H}}{\partial u_4} &= A_4 u_4 - (\lambda_2 - \lambda_1)I_{HT} = 0,\\
\frac{\partial \mathscr{H}}{\partial u_5} &= A_5 u_5 - (\lambda_3 - \lambda_1)\theta S_H E_T + (\lambda_4 - \lambda_6)\gamma_p S_P E_T + (\lambda_5 - \lambda_6)\rho_b \gamma_p V_P E_T\\
&\quad + (\lambda_9 - \lambda_{11})\gamma_b S_C E_T + (\lambda_{10} - \lambda_{11})\rho_b \gamma_b V_C E_T - \lambda_{14}\nu I_{HT} = 0.
\end{aligned}
\tag{3.188}
$$

Since the characterization of the optimal control problem holds on the interior of the control set U, thus we have:

$$
\begin{aligned}
u_1^* &= max\Big\{0, min\Big(1, \frac{(\lambda_2 - \lambda_1)\beta_T(\alpha_p P_I + \alpha_b B_I)S_H + \lambda_8 P_I + \lambda_{13} B_I}{A_1}\Big)\Big\},\\
u_2^* &= max\Big\{0, min\Big(1, \frac{(\lambda_4 - \lambda_5 - C_5)S_P}{A_2}\Big)\Big\},\\
u_3^* &= max\Big\{0, min\Big(1, \frac{(\lambda_9 - \lambda_{10} - C_6)S_C}{A_3}\Big)\Big\},\\
u_4^* &= max\Big\{0, min\Big(1, \frac{(\lambda_2 - \lambda_1)I_{HT}}{A_4}\Big)\Big\},\\
u_5^* &= max\Big\{0, min\Big(1, \frac{f(\lambda_n)}{A_5}\Big)\Big\},
\end{aligned}
\tag{3.189}
$$

where

$$
\begin{aligned}
f(\lambda_n) =& (\lambda_3 - \lambda_1)\theta S_H E_T + (\lambda_6 - \lambda_4)\gamma_p S_P E_T + (\lambda_6 - \lambda_5)\rho_b \gamma_p V_P E_T +\\
& (\lambda_{11} - \lambda_9)\gamma_b S_C E_T + (\lambda_{11} - \lambda_{10})\rho_b \gamma_b V_C E_T + \lambda_{14}\nu I_{HT}
\end{aligned}
$$

and λ_n are solutions of the system of equations (3.187). □

CHAPTER FOUR

RESULTS AND DISCUSSION

4.1 Introduction

In this section we perform numerical simulations of mathematical models formulated in chapter 3 and discuss briefly the obtained results. These include sensitivity analysis of parameters and numerical simulation of both deterministic and CTMC stochastic models for *T. saginata* bovine cysticercosis and human taeniasis, dynamical model with its corresponding CTMC model for taeniasis and cysticercosis due to *T. solium* and *T. saginata* tapeworms in humans, pigs and cattle together with numerical simulations of the optimal control models to assess the most optimal and cost effective strategies for disease's control.

4.2 Taenia Saginata Deterministic Model

4.2.1 Simulation of the Basic Taenia Saginata Model

To understand well the transmission dynamics of *T. saginata* human taeniasis and bovine cysticercosis, we simulate the basic model (3.18) by using parameter values in Table 3. We quantify beef in terms of the number of infected cattle that are slaughtered for consumption. The Runge-Kutta order 4 numerical method is used to simulate the model in Matlab software.

The infected human population grows with time until when it reaches its equilibrium state due to consumption of raw or under-cooked infected beef as shown in Fig. 10(a). The increase in number of infected humans is direct proportional to the increase in number of Taenia eggs in the environment as shown in Fig. 10(c) as consequence of open human defecation. On the contrary, susceptible humans declines with time until when it stabilizes due to consumption of raw or inadequately cooked beef which is infected with tapeworms larval cysts.

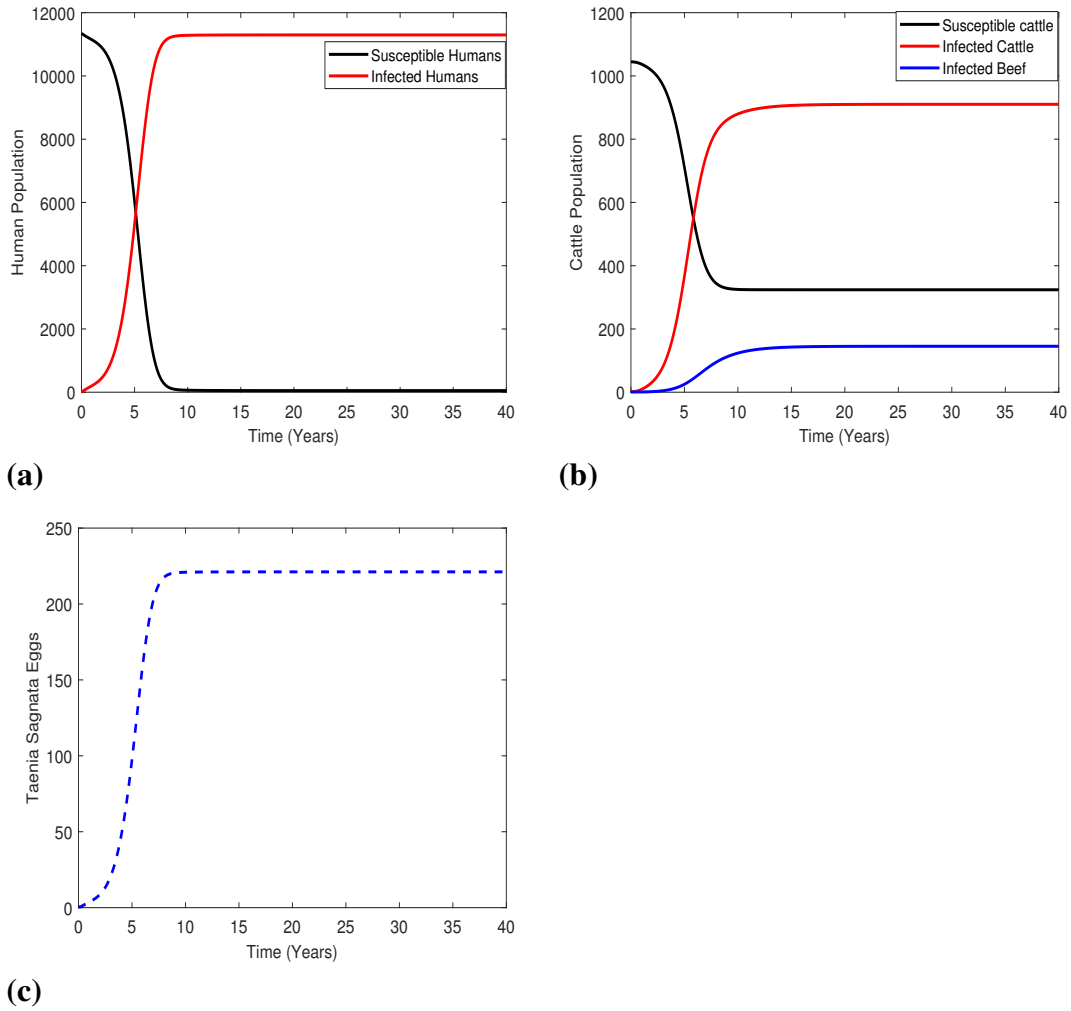


Figure 10: Dynamics of humans, cattle and T. sagnata eggs - basic model

From Fig. 10(b), it can be seen that the infected cattle increase initially to their maximum in the first six years until when they attain the steady state as a result of infection from the contaminated environment. Similarly, infected beef follows the same trend as the quantity of it depends on number of infected cattle that are slaughtered for consumption. On the other hand, susceptible cattle population declines with time until after the 10^{th} year when it stabilizes with about 320 cattle remaining uninfected.

4.2.2 Simulation of the Model with Controls

In this section, the model system (3.1) with control measures is simulated using parameter values in Table 3.

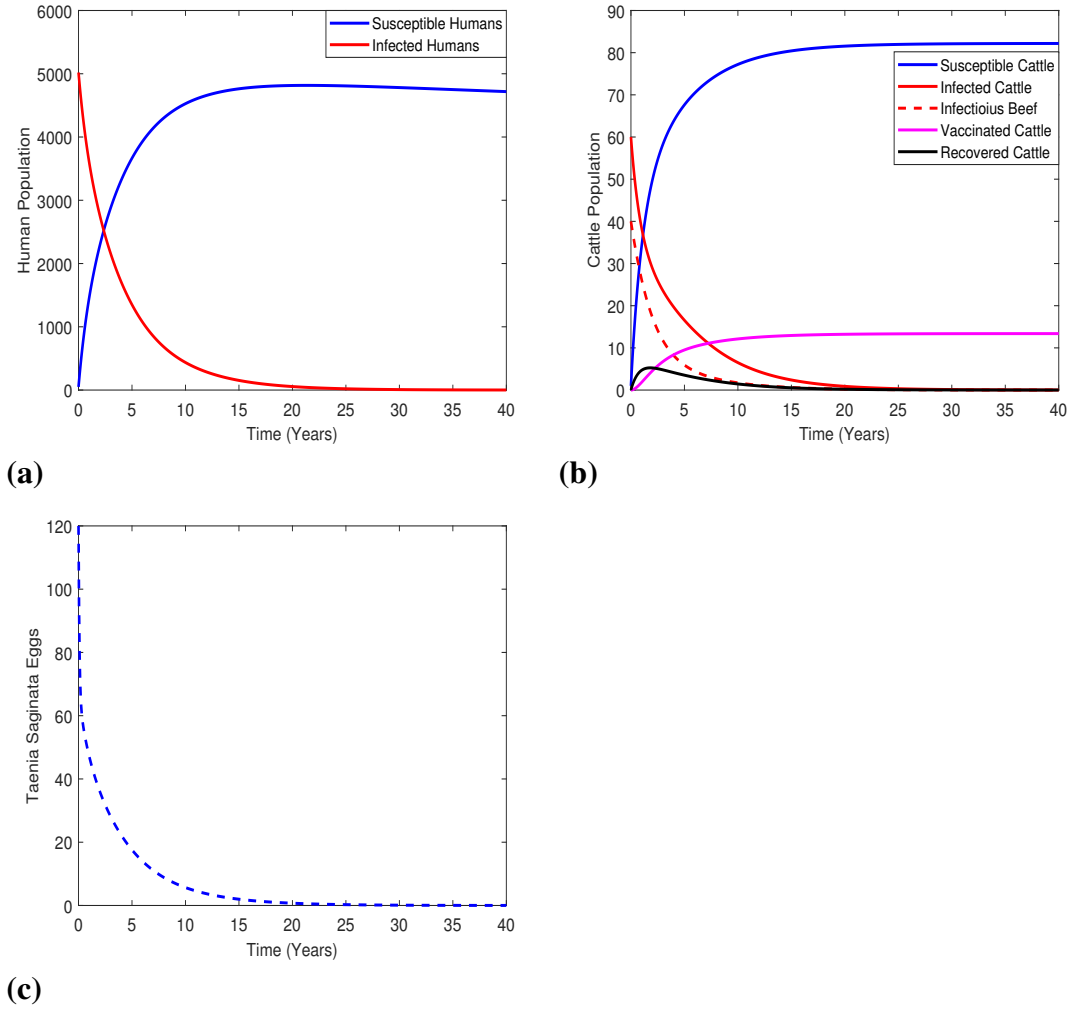


Figure 11: Dynamics of humans, cattle and T. saginata eggs - model with interventions

It can be observed from Fig. 11 that the trends for all sub-populations have significantly changed after implementing control strategies. In Fig. 11(b), infected cattle and infectious beef decline with time until when they approach zero after the 15^{th} year. Recovered cattle increase initial to the maximum in the first two years and then reduces with time until when they approach to zero in the 15^{th} year. On the other hand, vaccinated and susceptible cattle grow with time due to control measures and then stabilize after the 10^{th} year. In Fig. 11(a), infected humans have declined such that after the 20^{th} year there are no cases for infected humans whereas susceptible human population responds positively as a consequence of implementing control measures. The decline in cases of infected humans and infectious beef is in correspondence with decline in number of Taenia eggs in the environments as shown in Fig. 11(c) since infected humans

and infectious beef have significant contribution on the number of *Taenia sagnata* eggs in the environment.

4.2.3 Impact of Varying Control Options on Infected Humans, Infected Cattle and Taenia Saginata Eggs

In this section we simulate the model system (3.1) through varying control strategies that include cooking rate of infected beef, improvement in hygiene and sanitation (use of toilets), efforts in killing *Taenia saginata* eggs from contaminated environment, vaccination of susceptible cattle and treatment of both infected humans and cattle. The state variables I_{HT} and I_C represent humans who are infected with taeniasis and cattle infected with cystcercosis respectively. The controls were initially set at: $\kappa = 0.30, \tau = 0.20, c_h = 0.05, \psi_s = 0.08, \lambda = 0.15$ and $\chi = 0.07$ that represent cooking rate of infected beef, rate of using toilets, humans' effort to kill *T. saginata* eggs from contaminated environment, vaccination of susceptible cattle, recovery rates of infected humans and cattle due to treatment respectively. These control were increased concurrently in five different phases to observe the changes in number of infected humans and cattle, and the corresponding effective reproduction number as indicated in Table 12. Other parameter values were kept constant as shown in Table 3.

Table 12: Impact of varying controls on effective reproduction number R_{e1}

Control	1	2	3	4	5	6
κ	0.30	0.40	0.43	0.50	0.60	0.70
τ	0.20	0.40	0.45	0.60	0.70	0.80
c_h	0.05	0.10	0.15	0.20	0.30	0.40
ψ_s	0.08	0.12	0.18	0.22	0.32	0.42
λ	0.15	0.25	0.30	0.35	0.45	0.55
χ	0.07	0.13	0.20	0.23	0.33	0.43
R_{e1}	5.0498	3.2242	2.3864	1.7458	1.0821	0.6425

The results show that when intervention strategies are increased, the number of infected humans and cattle decline with time as shown in Fig. 12(a) and 12(b) respectively. Moreover, the results indicate that large increase of controls provides sufficient control of *T. saginata* bovine cysticercosis and human taeniasis within short time. These trends are inline with the decrease in number of *T. saginata* eggs in the environment as depicted in Fig. 12(c) and the corresponding decrease in effective reproduction number R_{e1}.

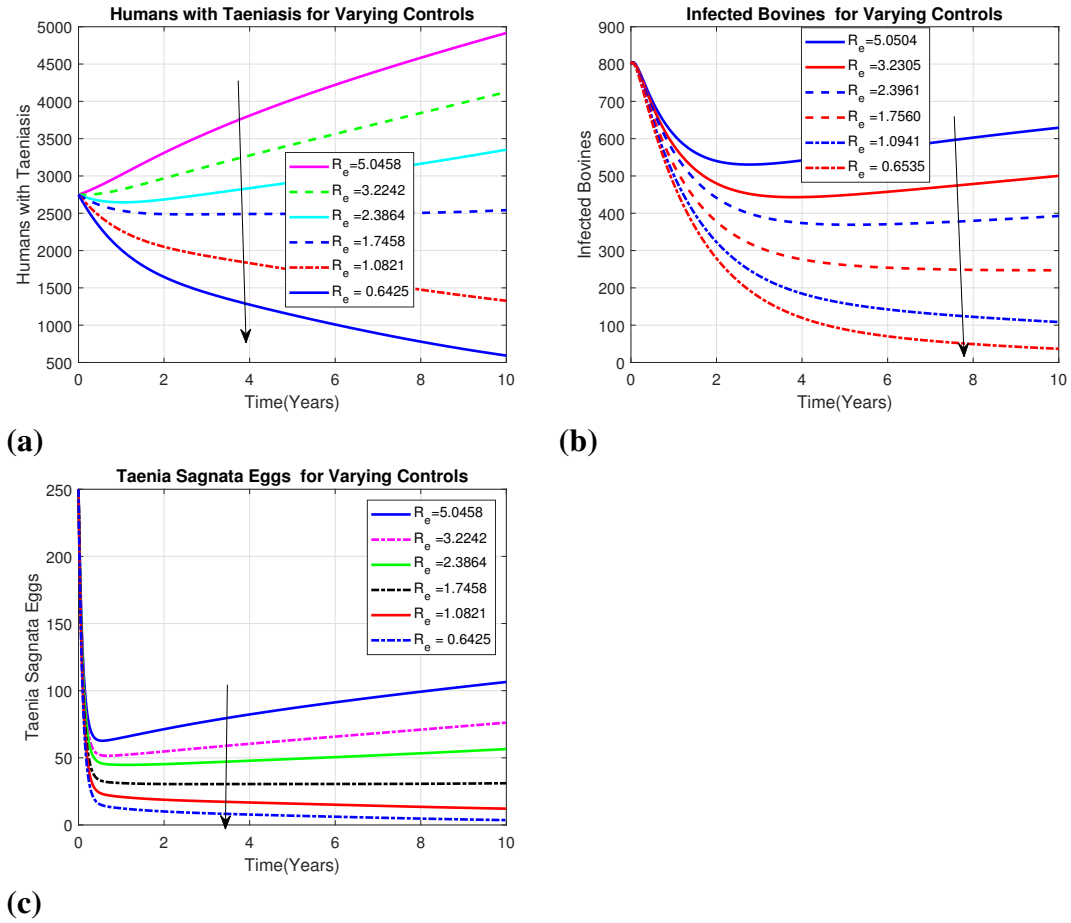


Figure 12: Impact of varying control options on infected humans, cattle and T. saginata eggs

4.3 Numerical Simulations for CTMC Taenia Saginata Model

In this section we simulate the deterministic model (3.18) with its corresponding cotinuous time Markov chain (CTMC) stochastic model to investigate the dynamics of taeniasis and cysticercosis in humans and cattle respectively using the parameter values in Table 3. Since the CTMC model assumes that susceptible populations are at the disease free equilibrium and sufficiently large, therefore the initial conditions for susceptible humans and cattle are $S_H(0) = 17\,518$ and $S_C(0) = 796$ respectively. The initial conditions for infected humans, cattle and *T. saginata* eggs in the environment are taken to be $I_{HT}(0) = 2$, $I_C(0) = 1, B_I(0) = \eta I_C(0)$ and $E_T(0) = 1$ respectively. The graphical representation of the CTMC solutions with their corresponding deterministic solutions are presented in Fig. 13-18.

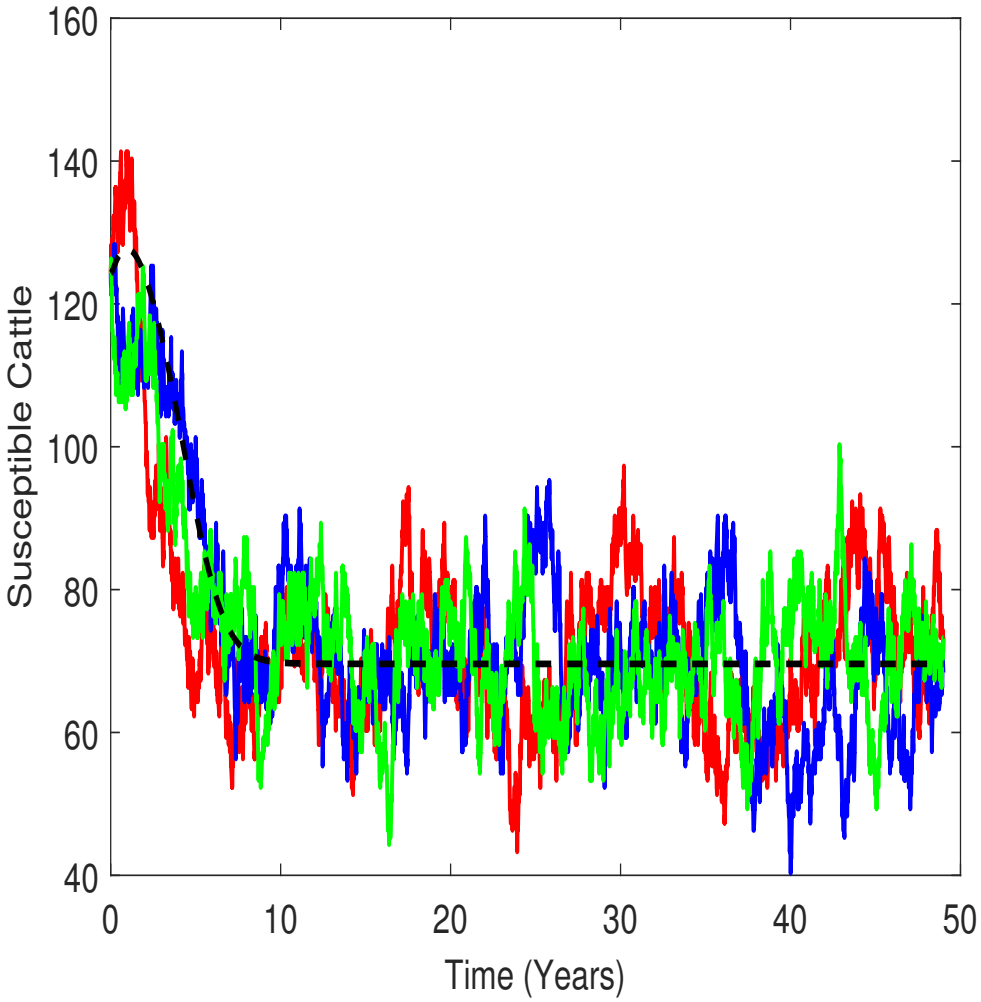


Figure 13: Comparison of ODE (dashed) and three sample paths of the CTMC (solid) for susceptible cattle

Figures 13-18 indicate the results of deterministic model for infected classes graphed with the corresponding sample paths for the continuous time Markov chain stochastic epidemic model. The disease dynamics of the CTMC stochastic model are different but not far from those of the deterministic model. It can be observed that CTMC solutions fluctuate within the solution of their corresponding deterministic equations. Figures 14-17 indicate that initially infected cattle, infected beef, *T. saginata* eggs in the environment and infected humans increase gradually with time and then grow rapidly before they are maintained at equilibrium. This situation shows that there is a disease outbreak in both human and cattle populations, and so *T. saginata* eggs persist in the environment.

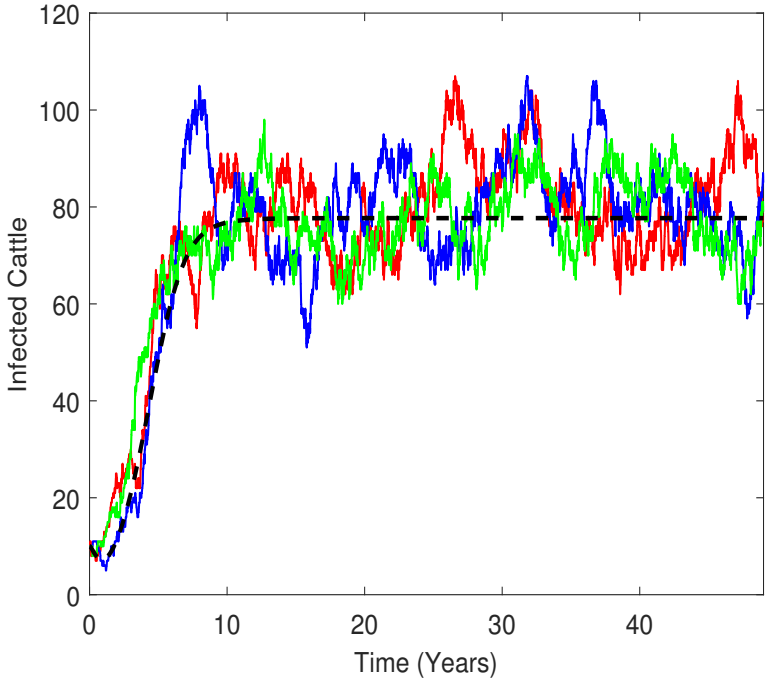


Figure 14: Comparison of ODE (dashed) and three sample paths of the CTMC (solid) for infected cattle

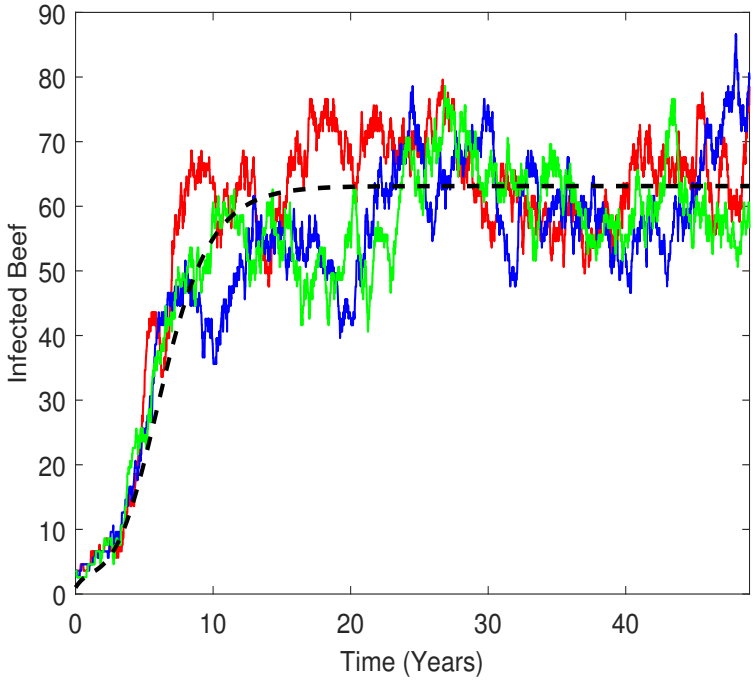


Figure 15: Comparison of ODE (dashed) and three sample paths of the CTMC (solid) for infected beef

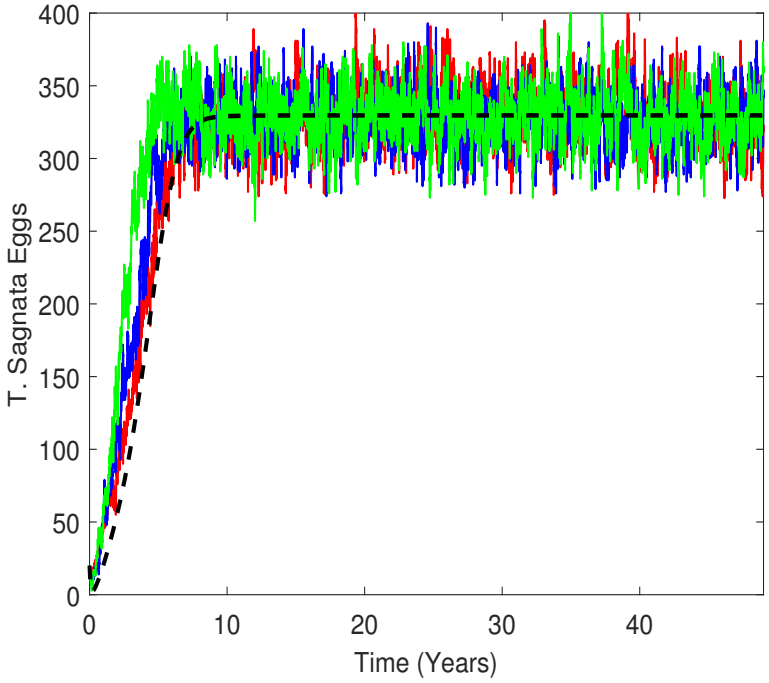


Figure 16: Comparison of ODE (dashed) and three sample paths of the CTMC (solid) for T. saginata eggs

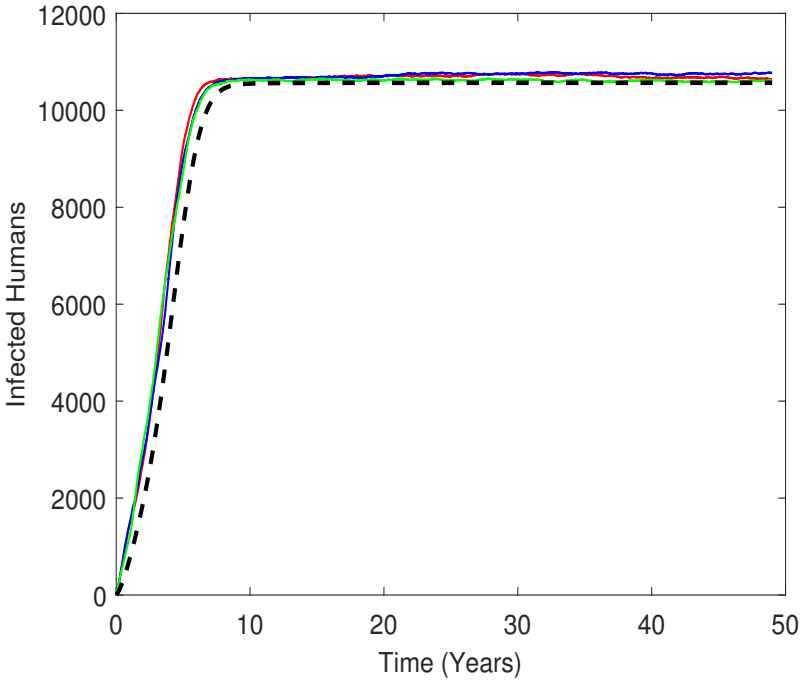


Figure 17: Comparison of ODE (dashed) and three sample paths of the CTMC (solid) for infected humans

Similarly, the solutions of the CTMC for susceptible cattle and humans are not far from the solutions of their corresponding deterministic equations. It can be seen form Fig. 13 & 18. that both susceptible cattle and humans decrease with time until when they are maintained at equilibrium, whereby a very small proportions of susceptible humans remain uninfected compared to susceptible cattle.

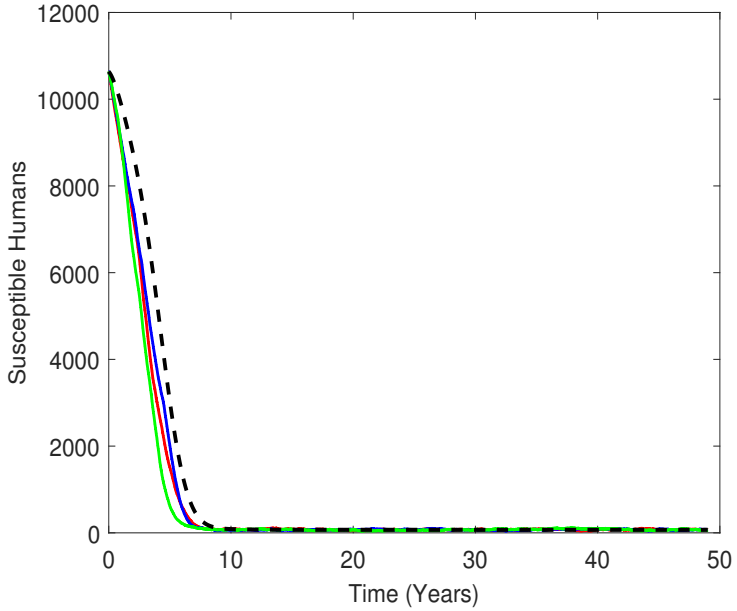


Figure 18: Comparison of ODE (dashed) and three sample paths of the CTMC (solid) for susceptible humans

4.4 Probability of Disease Extinction or Outbreak

The probability of disease extinction P_0, is calculated from the fixed point of the multitype branching process and is compared to the approximate probability of the disease extinction P_a, using 10 000 sample paths of the CTMC stochastic model. The fixed point in $(0,1)^4$ is given by $(q_1,q_2,q_3,q_4) = (0.3982, 0.8381, 0.0031, 0.8927)$. The approximation is carried out by computing the proportion of sample paths that hit zero before the occurrence of an outbreak. The approximate probabilities are calculated by using parameter values in Table 3 through varying initial size of infectious classes. The initial conditions are taken to be: $S_H(0) = \Lambda_H/\mu_h$, $S_C(0) = \Lambda_C/(\sigma+\mu_b)$, $I_{HT}(0) = y_1$, $I_C(0) = y_2$, $B_I(0) = y_3$ and $E_T(0) = y_4$. Figures 19-22 show that some sample paths of the CTMC model hit zero, implying that there is a possibility of bovine cysticercosis and human taeniasis to die even if the stochastic threshold is greater than one depending on the initial value of infectives.

Table 13: Probability of diseases' extinction

y_1	y_2	y_3	y_4	P_a	P_0
1	0	0	0	0.3980	0.3982
0	0	0	1	0.8924	0.8927
1	0	0	1	0.3552	0.3555
2	0	0	1	0.1413	0.1415
1	0	0	2	0.3169	0.3173
0	1	0	0	0.8378	0.8381
0	0	1	0	0.0029	0.0031
0	2	0	0	0.7021	0.7024
1	1	0	1	0.2974	0.2979
1	1	0	0	0.3335	0.3337
0	1	1	0	0.0025	0.0026
0	0	1	1	0.0026	0.0028
1	0	1	0	0.0011	0.0012
0	1	0	1	0.7463	0.7482
1	1	1	0	0.0008	0.0010
0	1	1	1	0.0021	0.0023
1	0	1	1	0.0009	0.0011
1	1	1	1	0.0010	0.0009

The results in Table 13 for cases when there is no infectious humans show that P_a and P_0 are in good agreement, meaning that P_a is a good estimate of P_0. Moreover, Table 13 shows that the disease dynamics depends on the initial number of infectious populations that were introduced in a fully susceptible population. For example, when the initial conditions are set to be $y_1 = 2, y_2 = 0, y_3 = 0$ and $y_4 = 1$, it can be seen in Fig. 4.10-4.13 that some sample paths of CTMC model go to zero in a finite time even though $\mathscr{R}_{01} = 23.1423 > 1$ and $\rho(\mathbf{M_1}) = 1.7256 > 1$ which means that the population following this sample path is quickly absorbed and finally settles at disease free equilibrium. The same situation can be observed for some other initial values of infected populations when they are varied without an infected cattle. This is not the case for the solution of deterministic equations which show that the disease is always endemic regardless the number of infected classes that were initially introduced in susceptible populations.

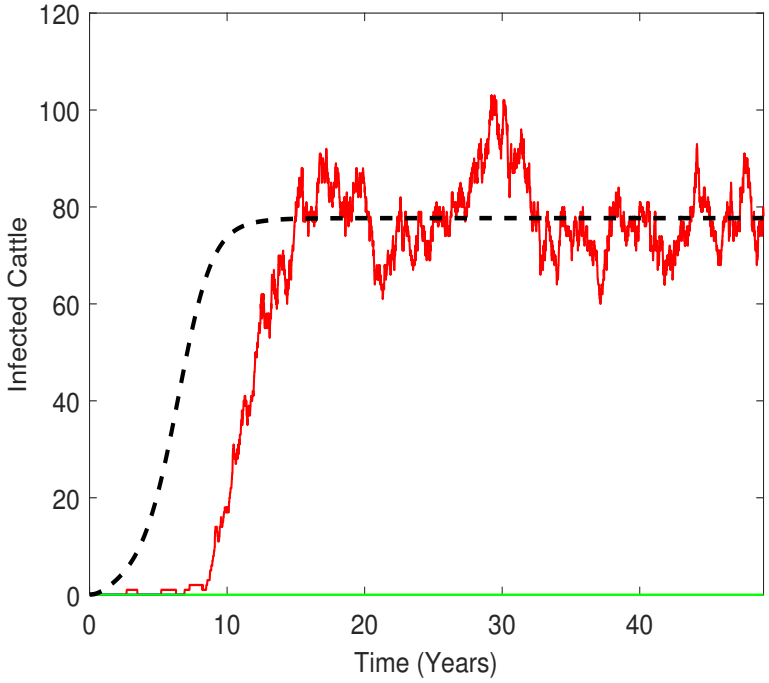


Figure 19: Solution of the ODE (dashed) with the corresponding three sample paths of the CTMC (solid) model for infected cattle

The probability of disease extinction is very high if the diseases emerge from a small number of *T. saginata* egg, followed by the case when there are only infected cattle at the beginning of the outbreak. The probability of disease is moderate if the diseases emerge from an infectious cattle and humans, or from either infectious humans and or from both infected humans and *T. saginata* eggs. Furthermore, it can be observed from Table 13 that, the disease will persist in populations if there is atleast an infected beef at the beginning of an outbreak. If the disease emerges from infected humans and or from both infected humans and *T. saginata* eggs, then the probability of disease extinction decline with high number of infective humans giving high probability of disease outbreak. This suggests that any intervention that focus on reducing the number of infected humans at the beginning of the epidemic, together with measures that focus on meat inspection and improved beef cooking reduce the transmission of taeniasis and cysticercosis in humans and cattle respectively.

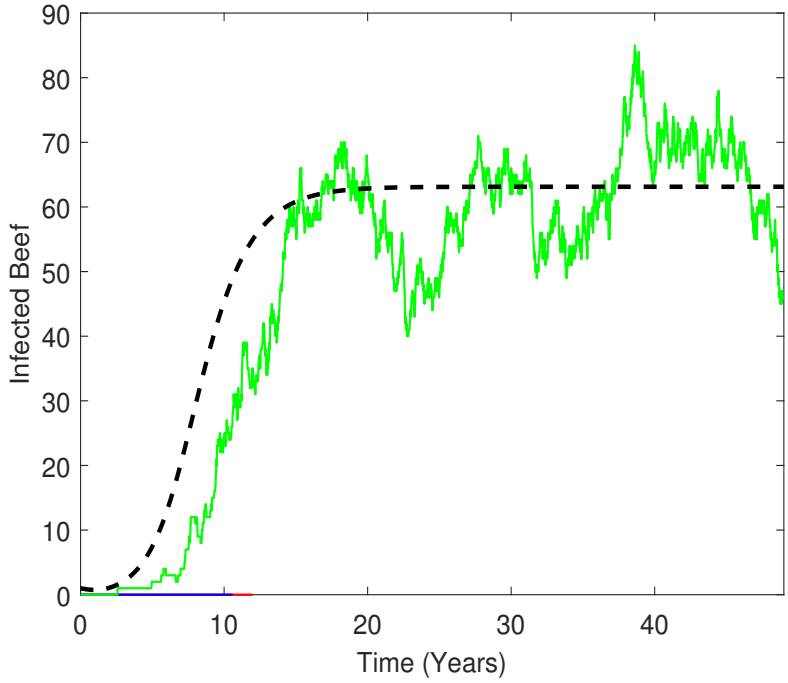


Figure 20: Solution of the ODE (dashed) with the corresponding three sample paths of the CTMC (solid) model for infected beef

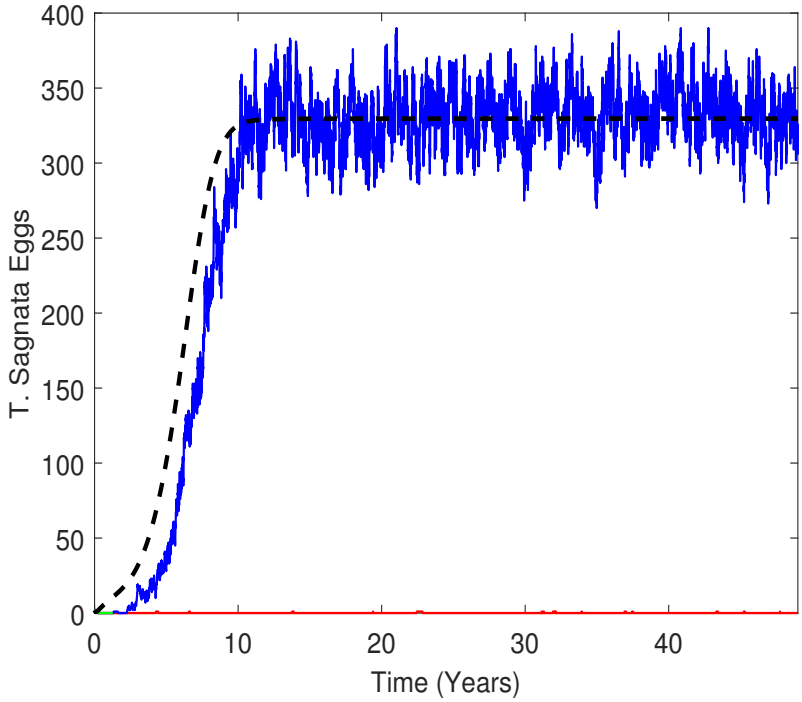


Figure 21: Solution of the ODE (dashed) with the corresponding three sample paths of the CTMC (solid) model for *T. saginata* eggs

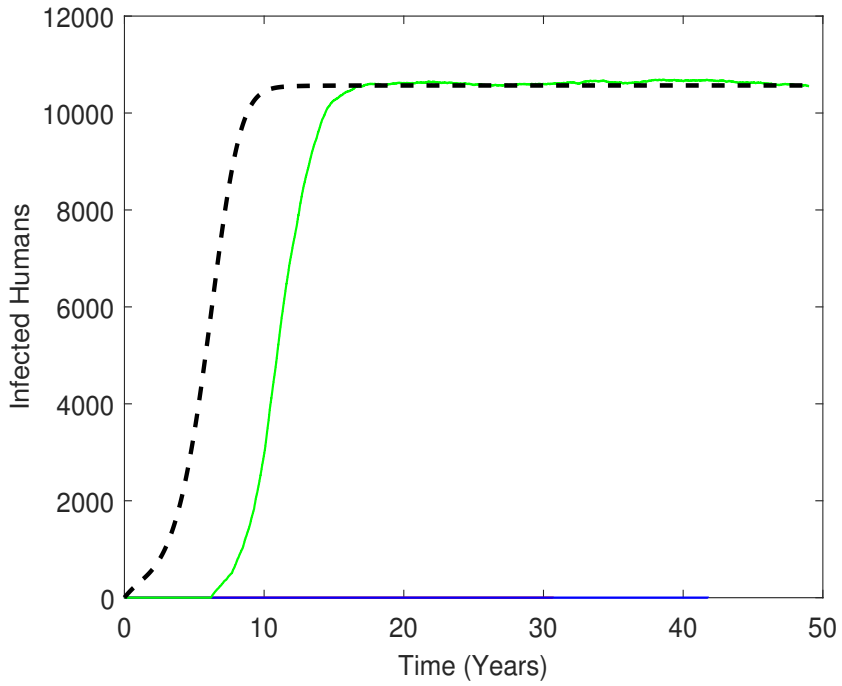


Figure 22: Solution of the ODE (dashed) with the corresponding three sample paths of the CTMC (solid) model for infected humans

4.5 Numerical Simulations of the Optimal Control Model for Bovine Cysticercosis and Human Taeniasis

In this section, numerical simulations of the optimal control model for the dynamics and control of taeniasis and cysticercosis in humans and cattle is carried out. To solve numerically the optimal control problem, we implement the forward-backward sweep method for the model system (3.90) and the adjoint system (3.98) in Matlab using parameters in Table 3. The method begins by solving the model system (3.90) forward in time using Runge Kutta method of the fourth order relying on the supplied initial values of the controls. Then, the backward fourth order Runge Kutta method uses the obtained values of the state variables and initial values of controls to solve the adjoint equations (3.98) with given final condition (3.97). The control variables $u_1(t), u_2(t), u_3(t)$ are then updated and used to solve the state and adjoint systems. The following control strategies are assessed when they are implemented either singularly or in combination to control the diseases in humans and cattle. Strategy 1: Combination of $u_1(t), u_2(t)$ and $u_3(t)$, Strategy 2: Combination of $u_2(t)$ and $u_3(t)$, Strategy 3: Combination of $u_1(t)$ and $u_2(t)$, Strategy 4: Combination of $u_1(t)$ and $u_3(t)$, Strategy 5: Implementation of $u_1(t)$ only, Strategy 6: Implementation of $u_3(t)$ only, Strategy 7: Implementation of $u_2(t)$ only.

4.5.1 When all Controls are Implemented

This strategy involves the combination of improved meat cooking, vaccination of cattle, and treatment of humans with taeniasis. The results in Fig. 23 shows a significant decrease of infected humans, infected cattle and *T. saginata eggs* in the environment when all time dependent controls are implemented. Humans with taeniasis reduces to zero in 2.5 years while infected cattle and taenia eggg reduce in 2.5 and 8 years respectively. The control profiles in Fig. 23(d) show that initially the control variables $u_2(t)$ is at its peak during and then declines gradually to zero in the first 4.5 years. The control profiles for $u_1(t)$ and $u_3(t)$ are fully utilized in the first 3 and 9.5 years respectively and eventually decline to zero in final year.

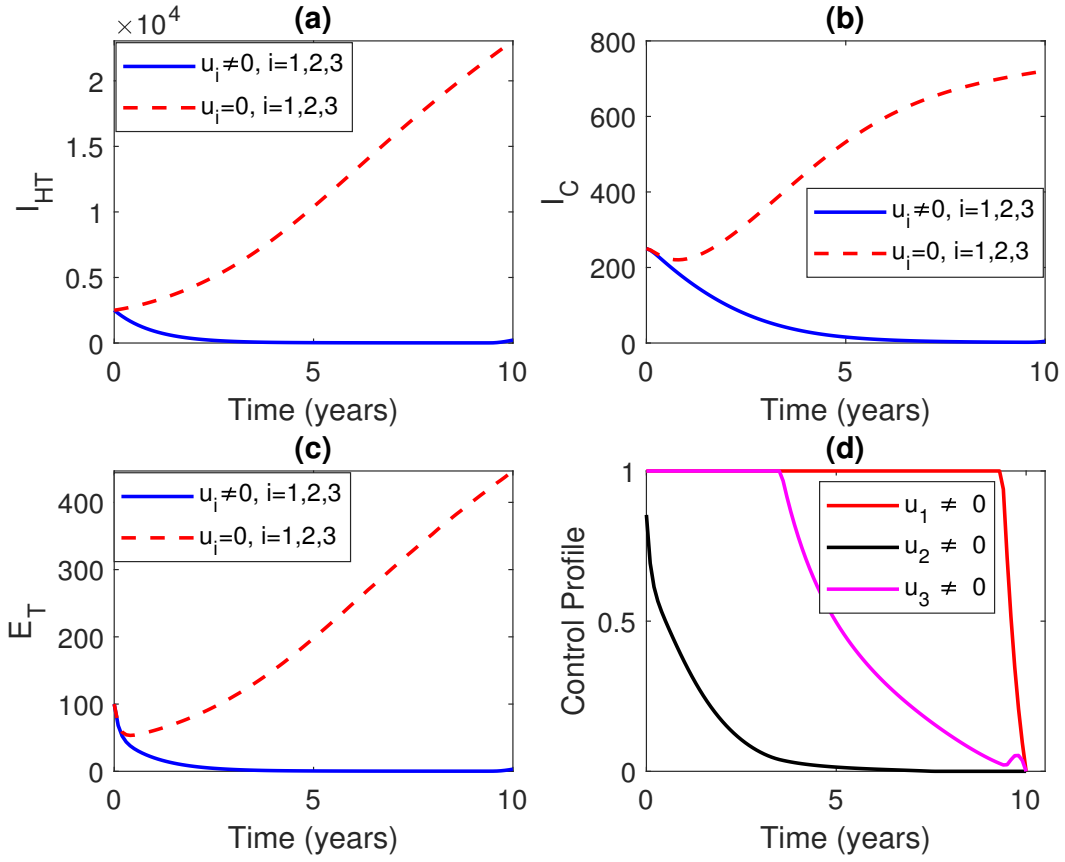


Figure 23: Impact of applying all controls on infected humans, cattle and taenia eggs

4.5.2 Vaccination of Cattle and Treatment of Humans with Taeniasis

In this strategy, the control variables $u_1(t)$ and $u_3(t)$ for cattle vaccination and treatment of humans who are infected with taeniasis respectively are used to optimize the objective function J while the control $u_1(t)$ on improving beef cooking rate is set to zero. The results in Fig. 24 show a reduction in number of cases for humans who are infected with taeniasis, infected cattle and taenia eggs in the environment. However, with this strategy it is not possible to control the

disease prevalence in humans and cattle. The control profiles in Fig. 24(d) show that the control variables $u_2(t)$ and $u_3(t)$ are fully utilized in their first 8.5 and 9.8 years and then decline to zero in the final time.

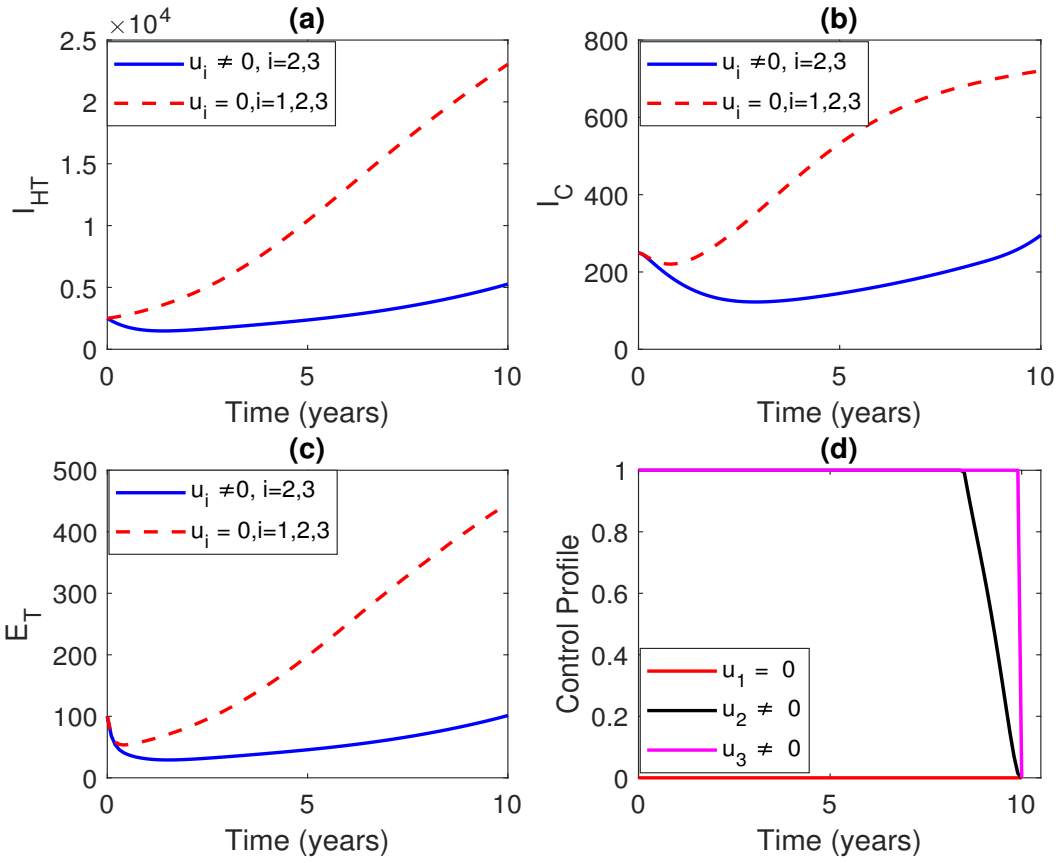


Figure 24: Impact of vaccination of cattle and treatment of humans with taeniasis on infected humans, cattle and taenia eggs

4.5.3 Improved Beef Cooking and Vaccination of Cattle

Here, we consider the combination of the control variables $u_1(t)$ for improved beef cooking and $(u_2(t))$ for vaccination of cattle. It can be observed in Fig. 25 that, although there is a decline in number of cases, however humans who are infected with taeniasis is maintained at 0.25×10^4 thoughout while infected cattle approaches zero after the first 1.5 years and a small increase in observed after the 9.5 year. on the other hand, taenia eggs declines in the first 0.5 years and stabilizes at 40. The control profiles in Fig. 25(d) show that the control variables $u_1(t)$ and $u_2(t)$ are at their peak in their first 8 and 9.9 years and finally decline to zero in the final time.

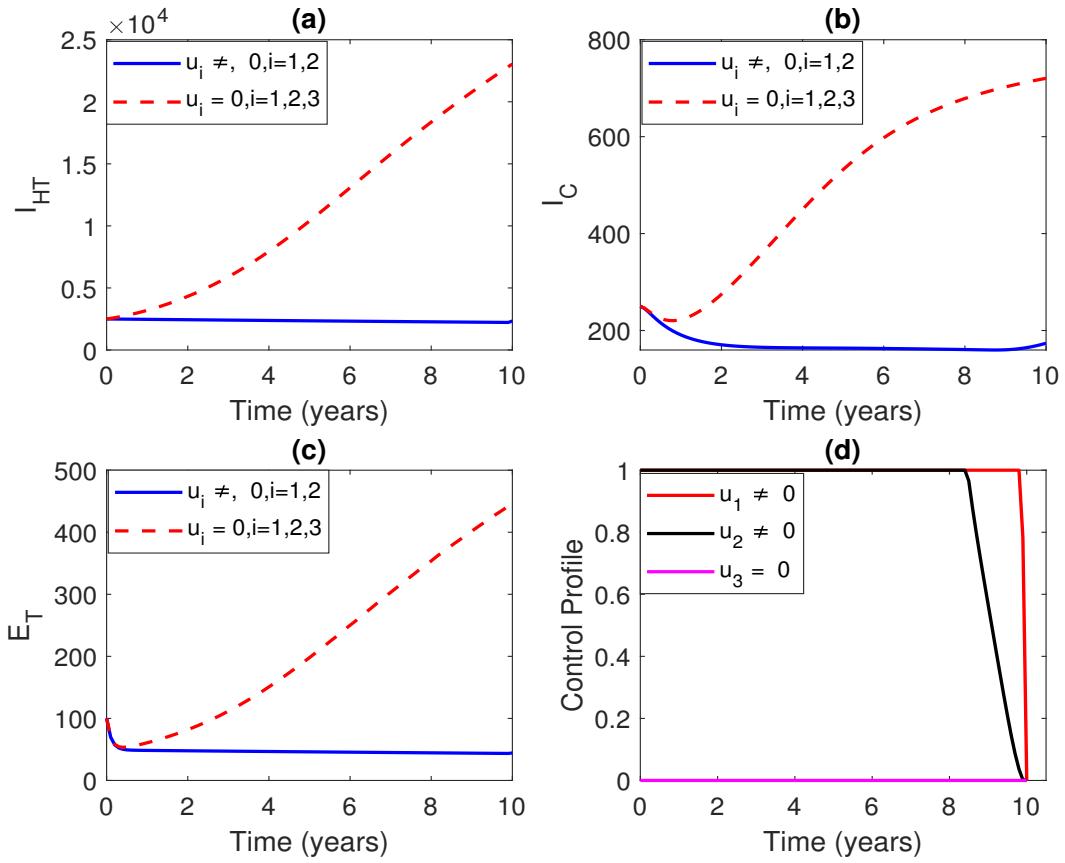


Figure 25: Impact of improved beef cooking and vaccination of cattle on infected humans, cattle and taenia eggs

4.5.4 Improved Meat Cooking and Treatment of Humans with Taeniasis

In this strategy, a combination of control variables $(u_1(t))$ for improved meat cooking and $(u_3(t))$ for treatment of humans who are infected with taeniasis are used to optimize the objective function J. The results for this strategy in Fig. 26 are the same as those for the first strategy. The control profile $u_1(t)$ is fully utilized for the first 9.5 years and quickly drops to zero at the final time while $u_3(t)$ is at its peak for the first 3 years and gradually declines to zero in the final time.

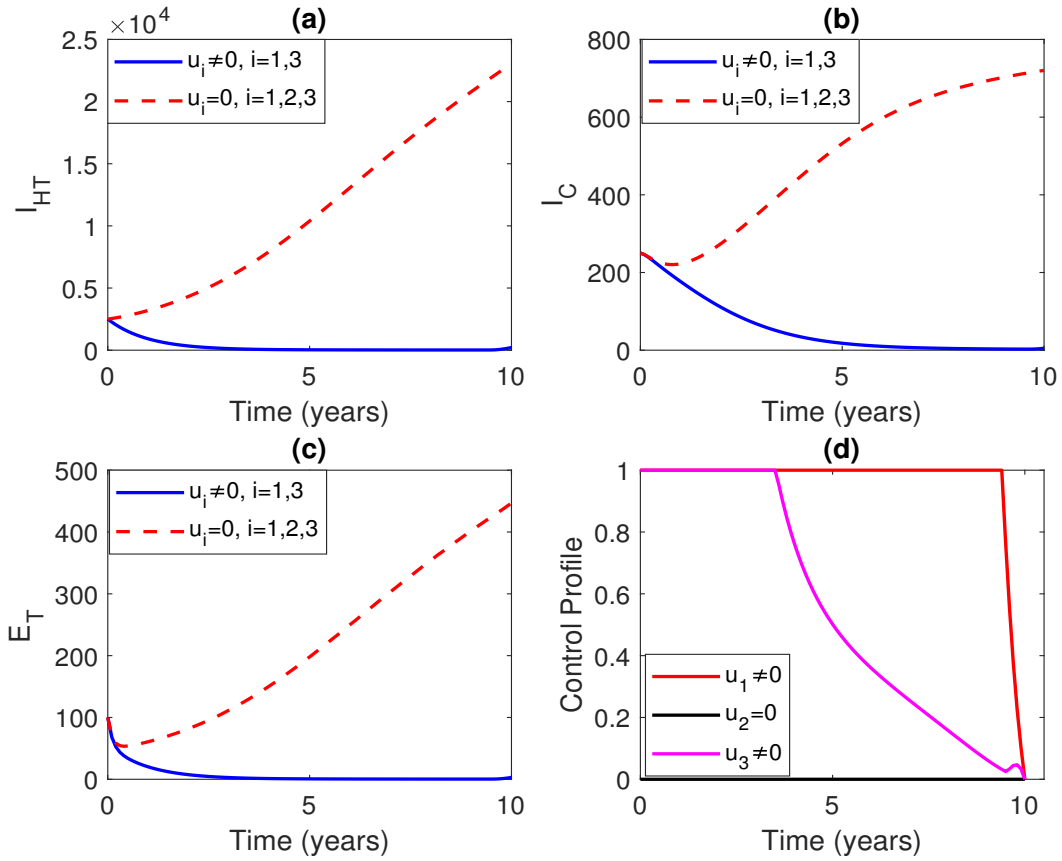


Figure 26: Impact of improved meat cooking and treatment of humans with taeniasis on infected humans, cattle and taenia eggs

4.5.5 Improved Meat Cooking Only

This strategy considers the implementation of improved meat cooking $(u_1(t))$ strategy only. The strategy yields similar results as the case for the second strategy with a slight difference in cases of infected cattle where there is a small decrease in number of cases in the first 0.5 years with a small increase up to the fifth year and thereafter it stabilizes. The control profile for the control variable $u_1(t)$ in Fig. 27(d) is fully utilized in the first 9.9 years and quickly decline to zero at the final time.

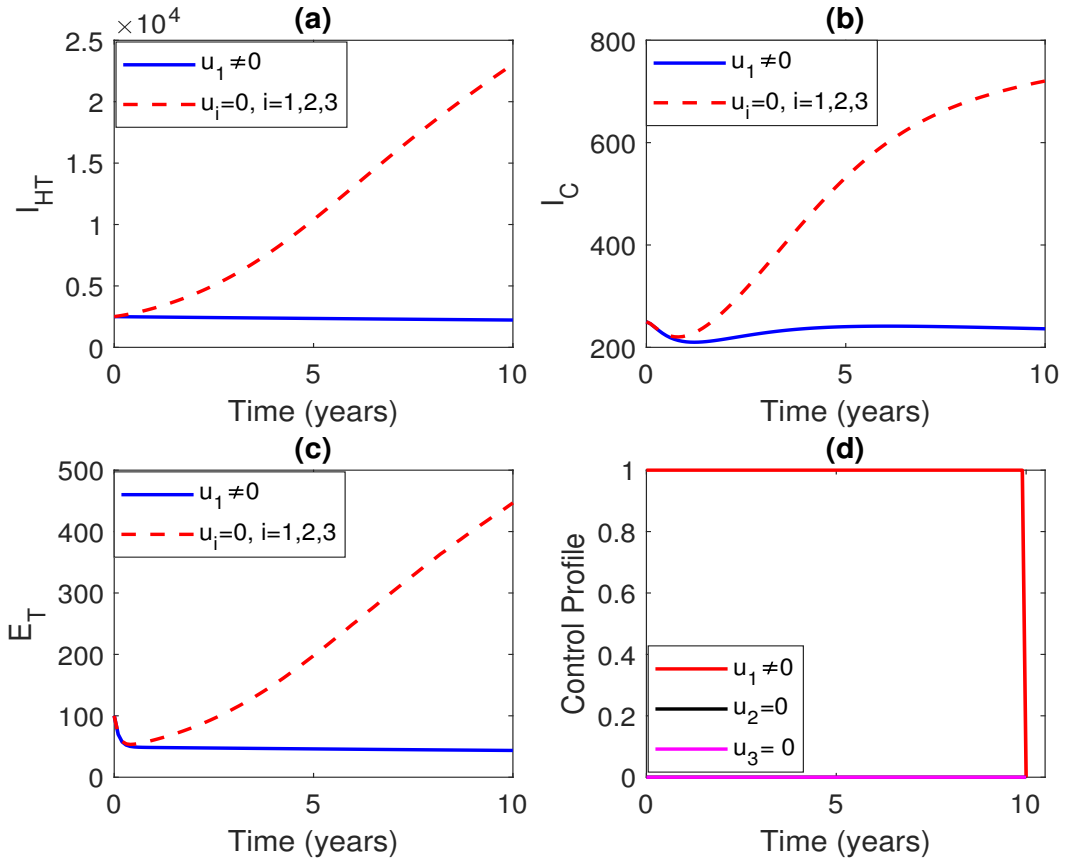


Figure 27: Impact of improved meat cooking on infected humans, cattle and taenia eggs

4.5.6 Treatment of Humans with Taeniasis Only

In this strategy, the control variable $u_3(t)$ is used to optimize the objective function J. It can be seen in Fig. 28 that although there is reduction in number of cases, however it is not possible to eliminate the disease in humans and cattle. The control profile in Fig. 28(d) indicated that the control variable $u_3(t)$ is at its peak for the first 9.9 years and then declines rapidly to zero in final time.

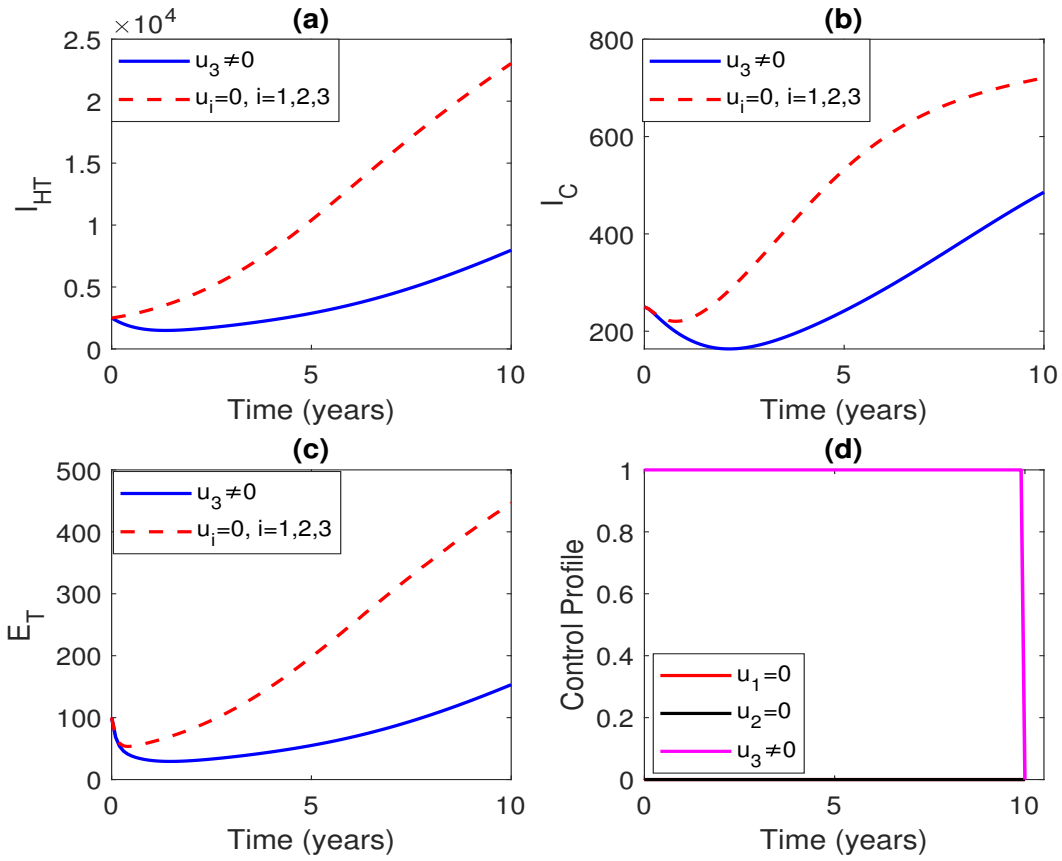


Figure 28: Impact of pigs and cattle vaccination on infected humans, cattle and taenia eggs

4.5.7 Cattle Vaccination Only

Here, we consider the implementation of cattle vaccination only for controlling the transmission of the diseases in humans and cattle. The results show that there is only a small decline in number of infected humans, cattle and *T. saginata* eggs in the environment. This implies that this is the least effective strategy for disease control. The control profiles in Fig. 29(d) for the control variable $u_2(t)$ is at its peak for the first 9 years and finally drops to zero in the final time.

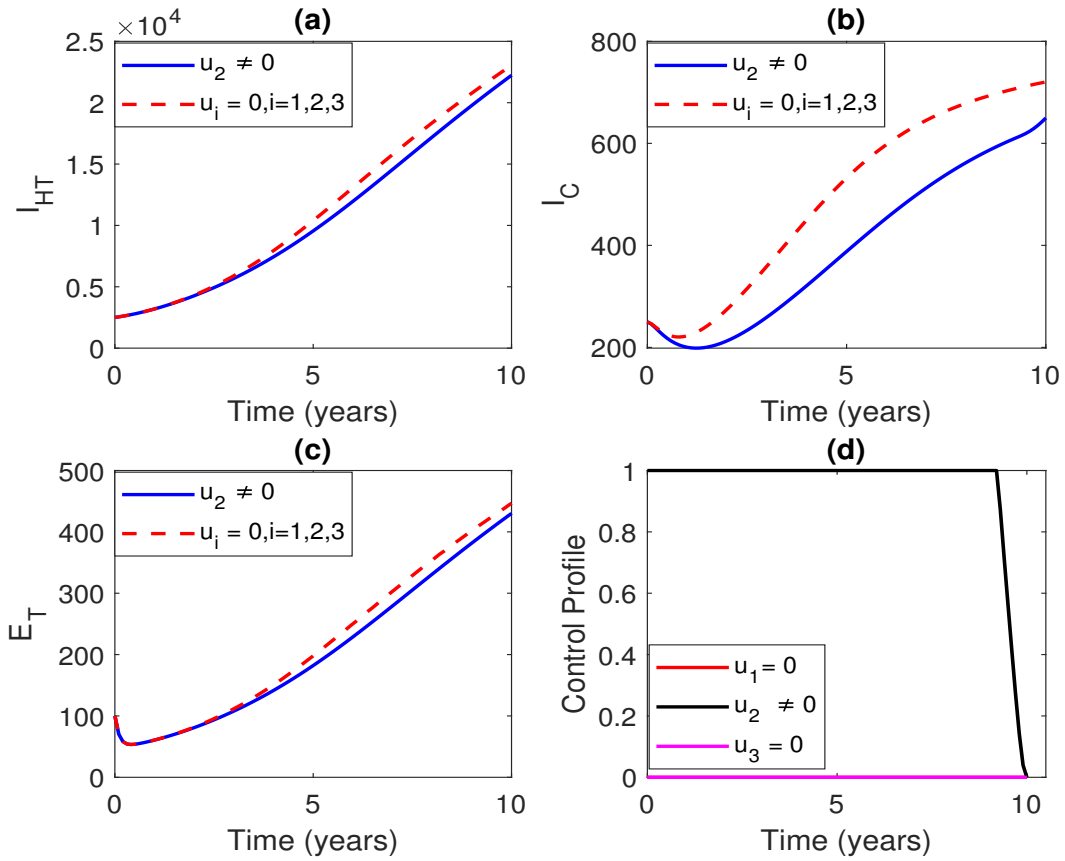


Figure 29: Impact of cattle vaccination on infected humans, cattle and taenia eggs

Generally, it can be observed from the graphs that the first and fourth strategies are more effective in controlling the transmission of taeniasis and cysticercosis in humans and cattle.

4.6 Numerical Simulations of the Basic Model for the Dynamics of Taeniasis and Cysticecosis in Humans, Pigs and Cattle

To understand well the dynamics of taeniasis and cysticercosis in humans, pigs and cattle, we simulate the model (3.101) using parameters from different literature and some are assumed as indicated in Table 7. To obtain initial conditions we consider a village with 5420 susceptible humans, 750 humans with taeniasis, 528 humans with Cysticercosis, 1050 susceptible pigs, 620 infected pigs, 1250 susceptible cattle, 850 infected cattle and 1000 taenia eggs in the environment. We quantify pork and beef in terms of the number of infected pigs and cattle that are slaughtered for consumption.

4.6.1 Model Simulation

The infected cattle and pigs increase initially to their maximum in the first three years and then decline until they settle to their steady states as illustrated in Fig. 30(a) & (b). A similar trend can be seen for infected pork and beef as they depend on number of infected pigs and cattle that are slaughtered for consumption. Also, infected humans with both taeniasis and cysticercosis increase with time following infections until when they attains the steady state after the fifth year as shown in Fig. 30(a). The increase in number of infected cattle and pigs is in correspondence with the increase in number of Taenia eggs in environment as shown in Fig. 30(d). Susceptible humans, pigs and cattle populations declines with time until when they approach their equilibrium states with only a small population of humans remaining uninfected.

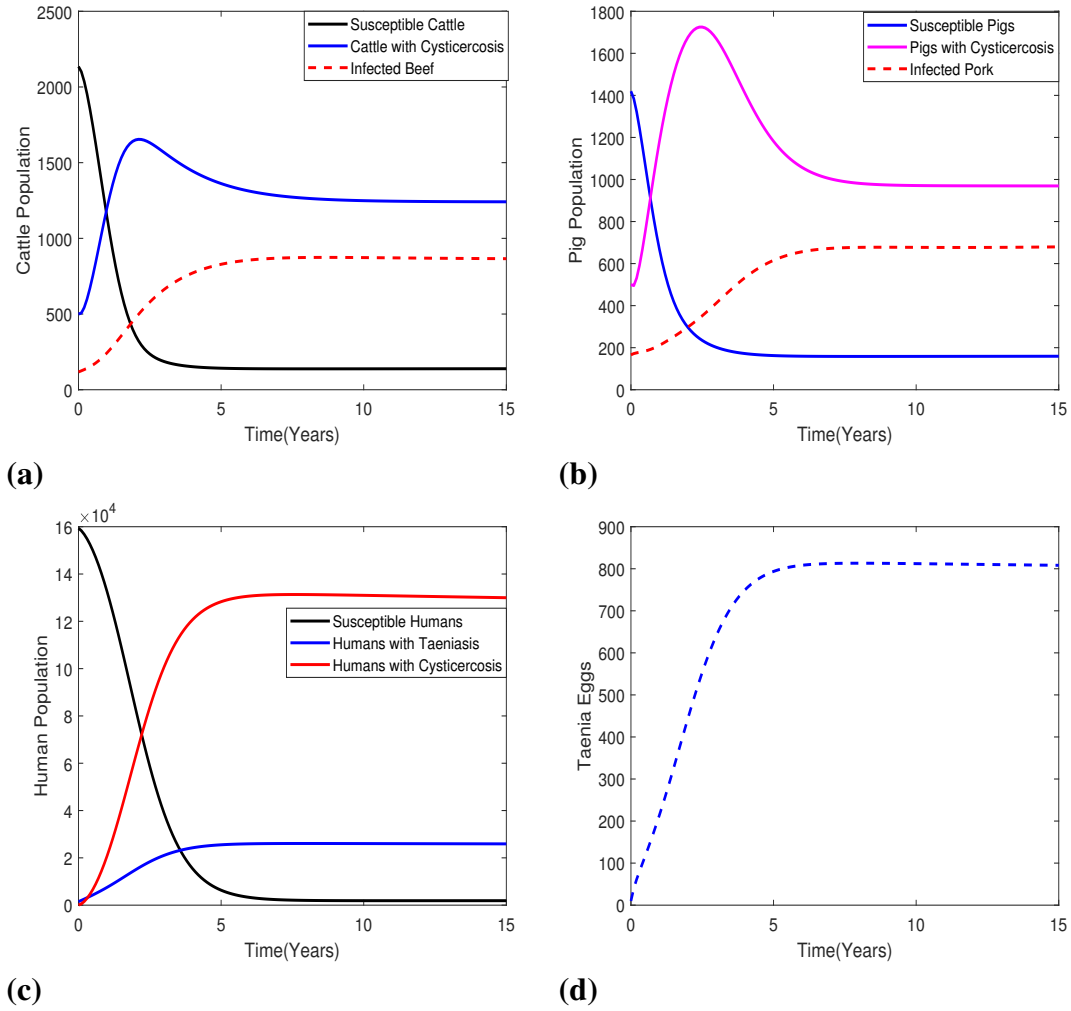


Figure 30: Dynamics of humans, pigs, cattle and taenia eggs

4.7 Effect of Varying the Most Sensitive Parameters

In this subsection we present numerical simulation by considering the most sensitive parameters in model system (3.101) to observe how they affect disease transmission in humans, pigs and cattle. In this case we consider the impact of varying the human recruitment rate (ψ), probability of humans to be infected with taeniasis (β_T) and the defection rate of humans with taeniasis (ν).

4.7.1 Effect of Varying Human Recruitment Rate (ψ)

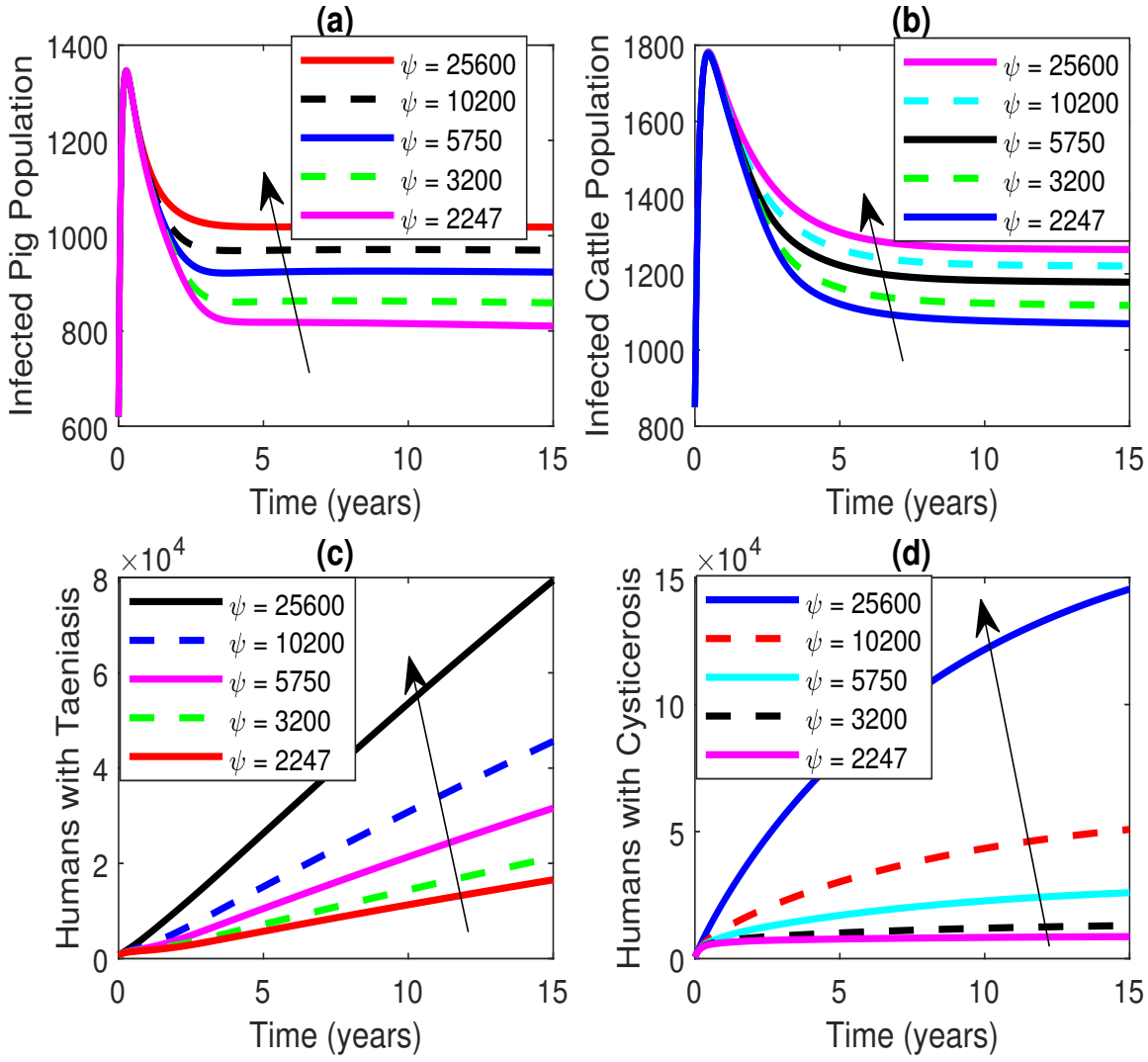


Figure 31: Impact of varying human recruitment rate on infected populations

The dynamics of taeniasis and cysticercosis shows that humans with taeniasis and cysticercosis, and infected cattle and pigs will increase in proportion to human recruitment rate as illustrated in Fig. 31.

4.7.2 Effect of Varying Defecation Rate (ν)

Infected pigs and cattle, and humans who are infected with cysticercosis increase with time as a result of increase in defecation rate as depicted by Fig. 32(a),(b),(d). A different trend can be observed for humans who are infected with taeniasis in Fig. 32(c) where there is a decrease in cases of humans who are infected when human defecation rate is increased. This is due to the fact that high defecation rate can result in expel of high number of taenia eggs and so the possibility of recovery.

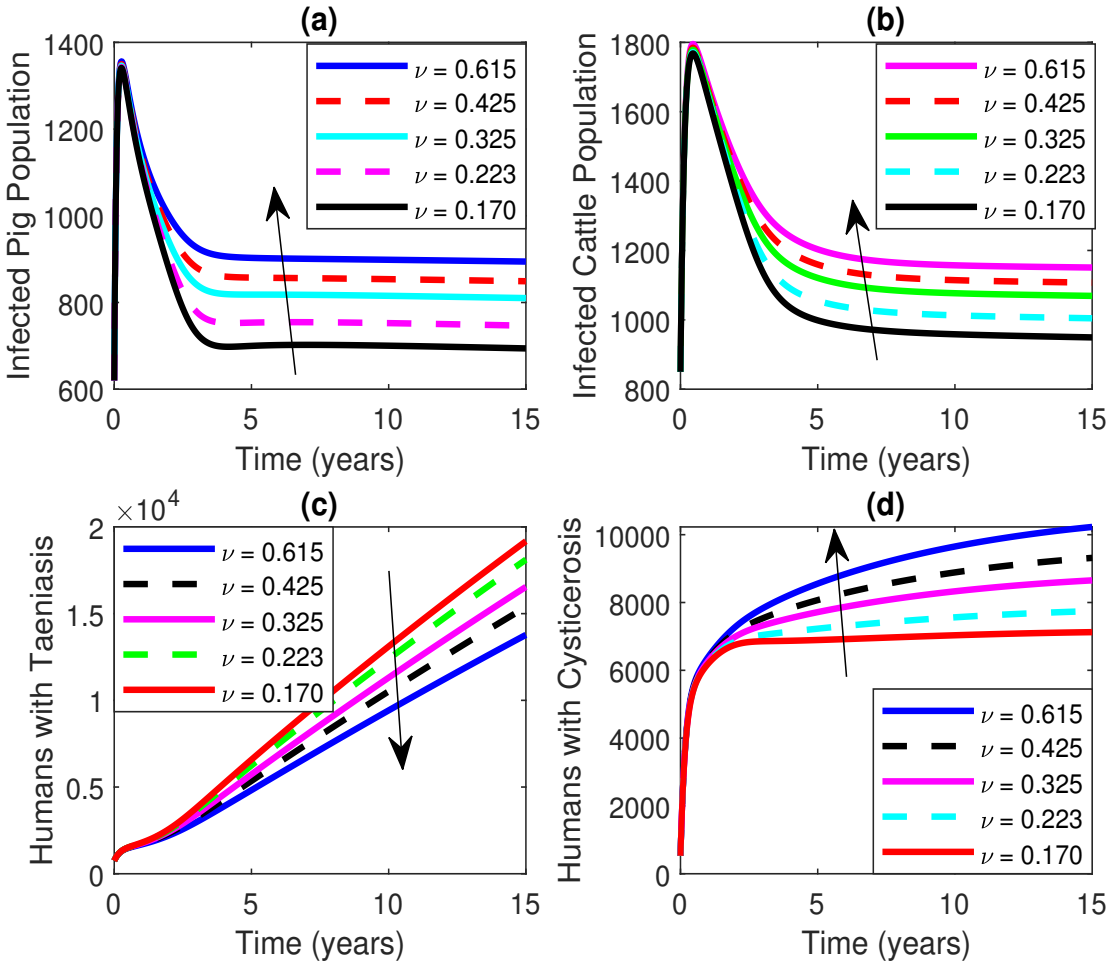


Figure 32: Impact of varying defecation rate on infected populations

4.7.3 Effect of Varying Probability of Taeniasis Infection (β_T)

In Fig. 33(a),(b),(c), the infected pigs, cattle and humans who are infected with taeniasis show an increase with time when the probability of taeniasis infection is increased. A different trend can be seen for humans who are infected with cysticercosis in Fig. 33 (d) that shows the decrease in disease prevalence with increase in probability of taeniasis infection. This is because humans

cysticercosis does not depend on probability of human infection with taeniasis but on the rate at which humans consume *T. solium* eggs from the contaminated environment.

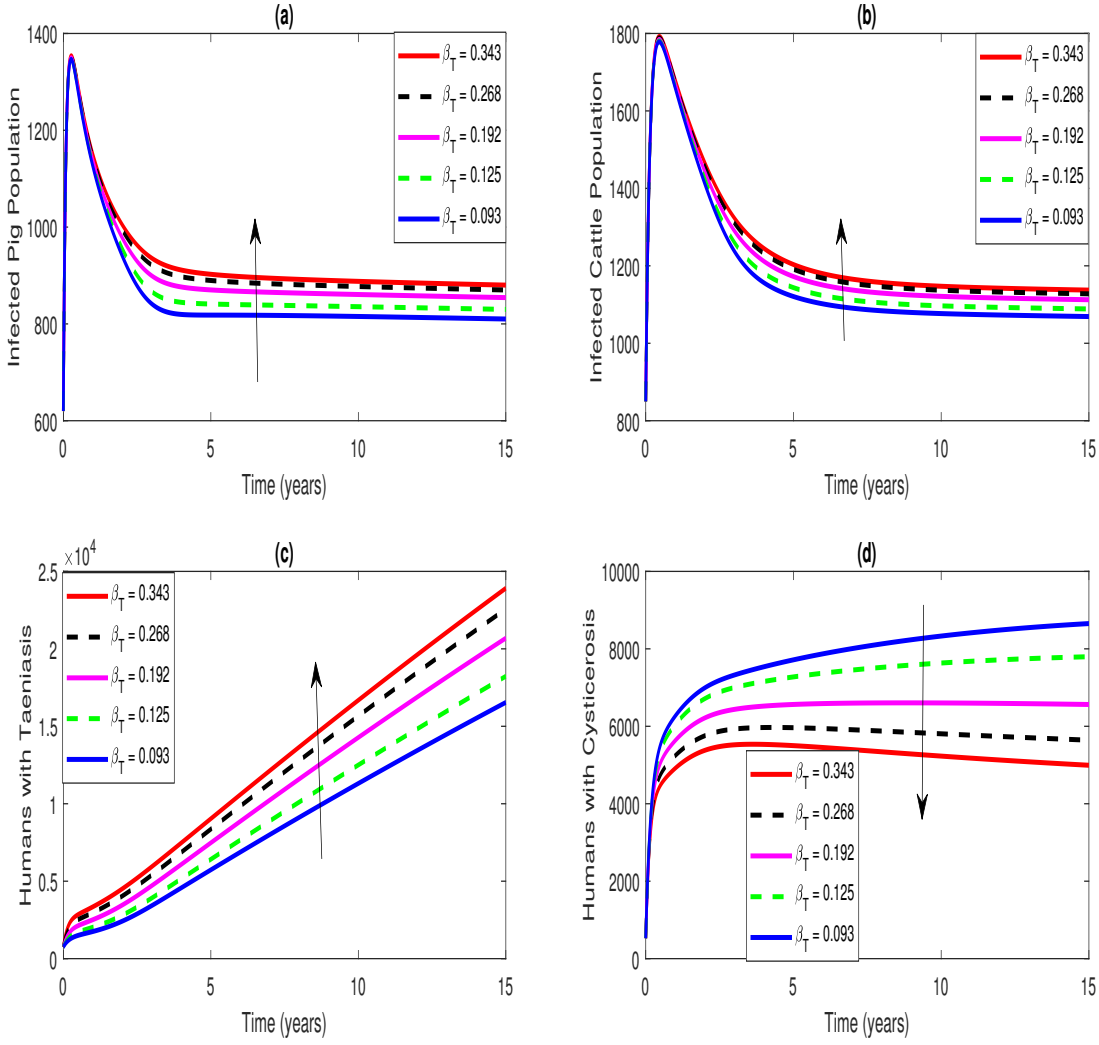


Figure 33: Impact of varying probability of infection on infected populations

4.8 CTCM Model Simulation

In this subsection, the deterministic model (3.101) is simulated with its corresponding continuous time Markov chain (CTMC) stochastic model to study the dynamics of taeniasis and cysticercosis in humans, pigs and cattle using the parameter values in Table 3.7 and initial con-

ditions:

$$I_{HT}(0)=1, I_{HC}(0)=0, S_H(0)=\psi/\mu_h-\Big(I_{HT}(0)+I_{HC}(0)\Big), I_P(0)=1, P_I(0)=1,$$
$$S_P(0)=\Lambda/(\rho+\mu_p)-I_P(0), I_C(0)=1, B_I(0)=1, S_C(0)=\phi/(\sigma+\mu_b)-I_C(0), E_T(0)=2. \tag{4.1}$$

Simulation results are presented in Fig. 34 and 35. The results show that continuous time Markov chain (CTMC) model solutions are relatively close to the corresponding deterministic model solutions. CTMC model results fluctuates within the solutions of deterministic model.

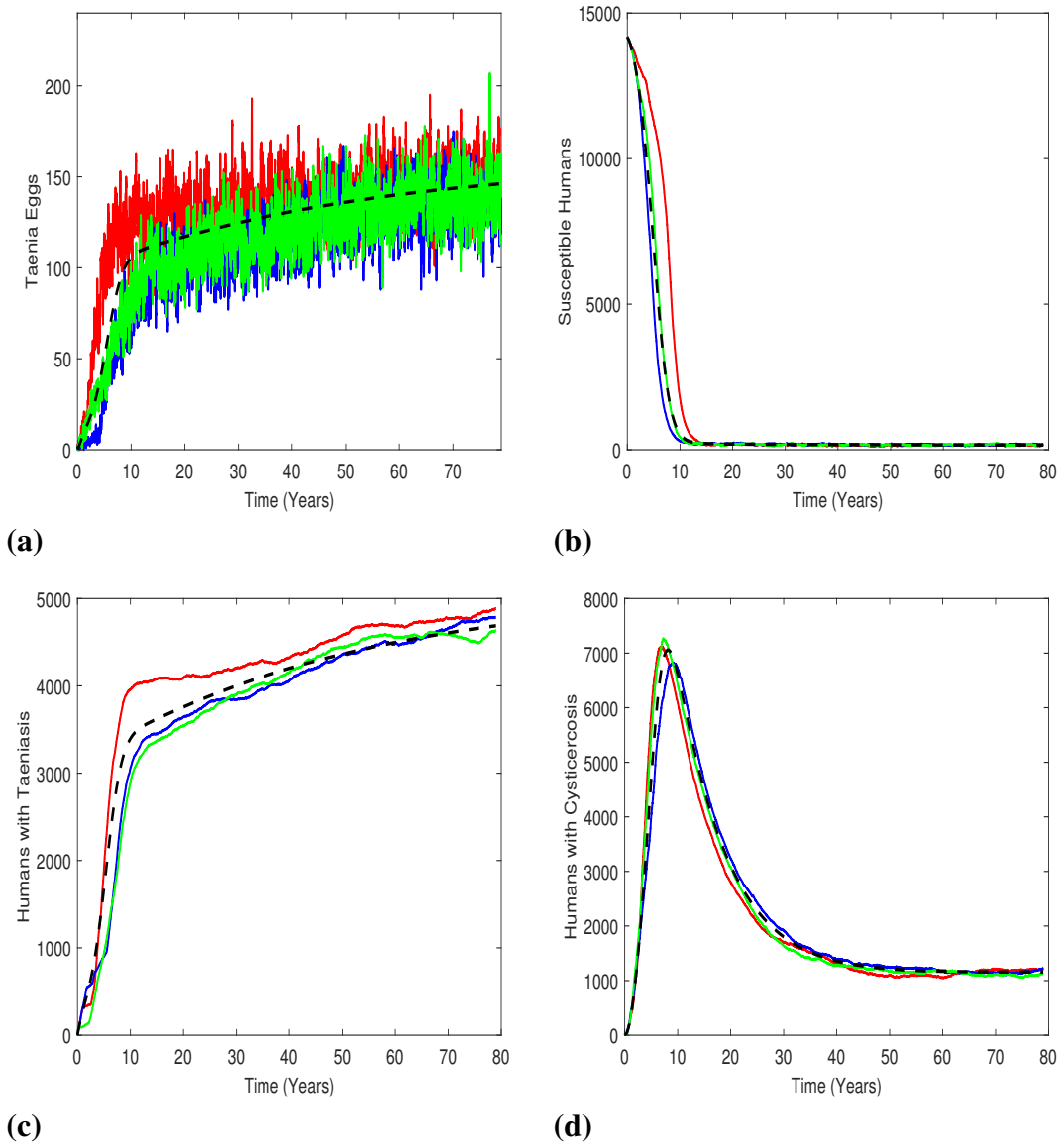


Figure 34: Comparison of deterministic solution (dashed) and three sample paths of the CTMC (solid) for humans and taenia eggs in the environment

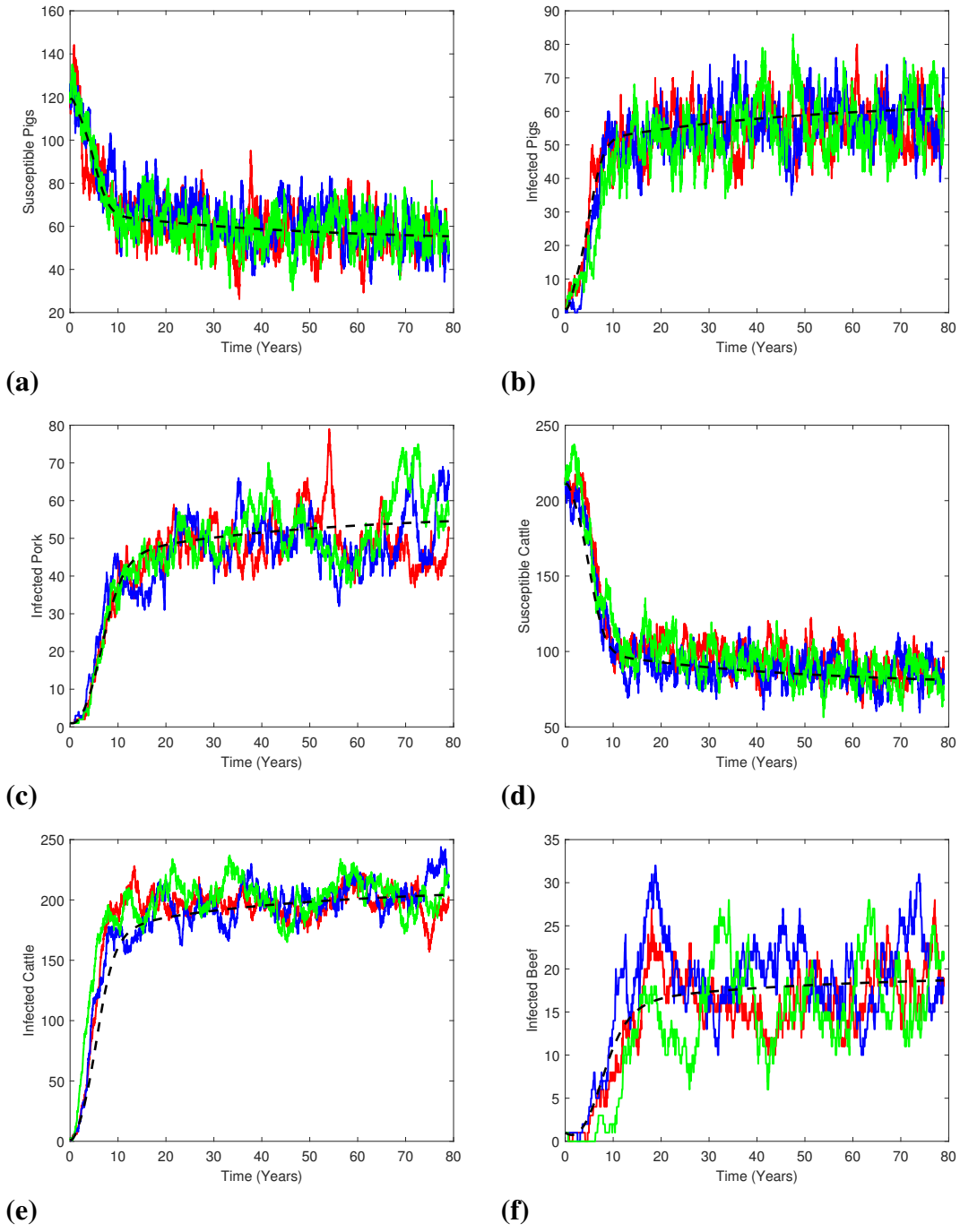


Figure 35: Comparison of deterministic solution (dashed) and three sample paths of the CTMC (solid) for pigs, cattle, infected pork and beef

4.9 Probability of Disease Outbreak or Extinction

The approximate probability of disease extinction P_a, is computed from the fixed point of the multitype branching process using 10 000 sample paths of the CTMC stochastic model. The approximation is carried out by computing the proportion of sample paths that hit zero before

the occurrence of disease outbreak. These probabilities are computed using parameter values in Table 7 through varying initial sizes for infectious classes.

Table 14: Probability of diseases' extinction

y_1	y_2	y_3	y_4	y_5	y_6	y_7	P_a
1	0	0	0	0	0	0	0.0008
0	1	0	0	0	0	0	1.0000
1	1	0	0	0	0	0	0.0013
0	0	0	0	0	0	1	0.5076
0	0	0	0	1	0	0	0.0241
0	0	0	0	0	1	0	0.0015
0	0	0	0	1	1	0	0.0000
0	0	1	0	0	0	0	0.0809
0	0	0	1	0	0	0	0.0170
0	0	1	1	0	0	0	0.0011
1	1	1	1	0	0	0	0.0000
1	1	0	0	1	1	0	0.0000
1	1	0	0	0	0	1	0.0003
0	0	0	0	1	1	1	0.0000
0	0	1	1	0	0	1	0.0008
0	0	1	1	1	1	0	0.0000
0	0	1	1	1	1	1	0.0000
1	1	1	1	1	1	1	0.0000

In computing the probabilities of diseases' extinction, many more combinations would have been included in Table 14. However using equation (3.79), the probabilities of diseases' extinction in those respective classes will be much lower compared to the cases when there are no infectives in some of the infectious classes. The graphs in Fig. 36 and 37 show that some sample paths of the CTMC model hit zero, implying that there is a possibility of taeniasis and cysticercosis to die in humans, pigs and cattle even if the stochastic threshold is greater than one depending on the initial value of infectives at the begining of disease outbreak.

The results in Table 14 show that the disease dynamics depend on the initial number of infectives that were introduced in susceptible populations. For example, when the initial conditions are set to be $I_{HT}(0) = y_1 = 1, I_{HC}(0) = y_2 = 0, I_P(0) = y_3 = 1, P_I(0) = y_4 = 0, I_C(0) = y_5 = 1, B_I(0) = y_6 = 0$ and $E_T(0) = y_7 = 1$, it can be seen in Fig. 36 and 37 that some sample paths of CTMC model hit zero in a finite time even though $R_0 = 19.4975 > 1$ and $\rho(\mathbf{M}) = 1.5877 > 1$ which means that the population following this sample path is quickly absorbed and finally settles at disease free equilibrium.

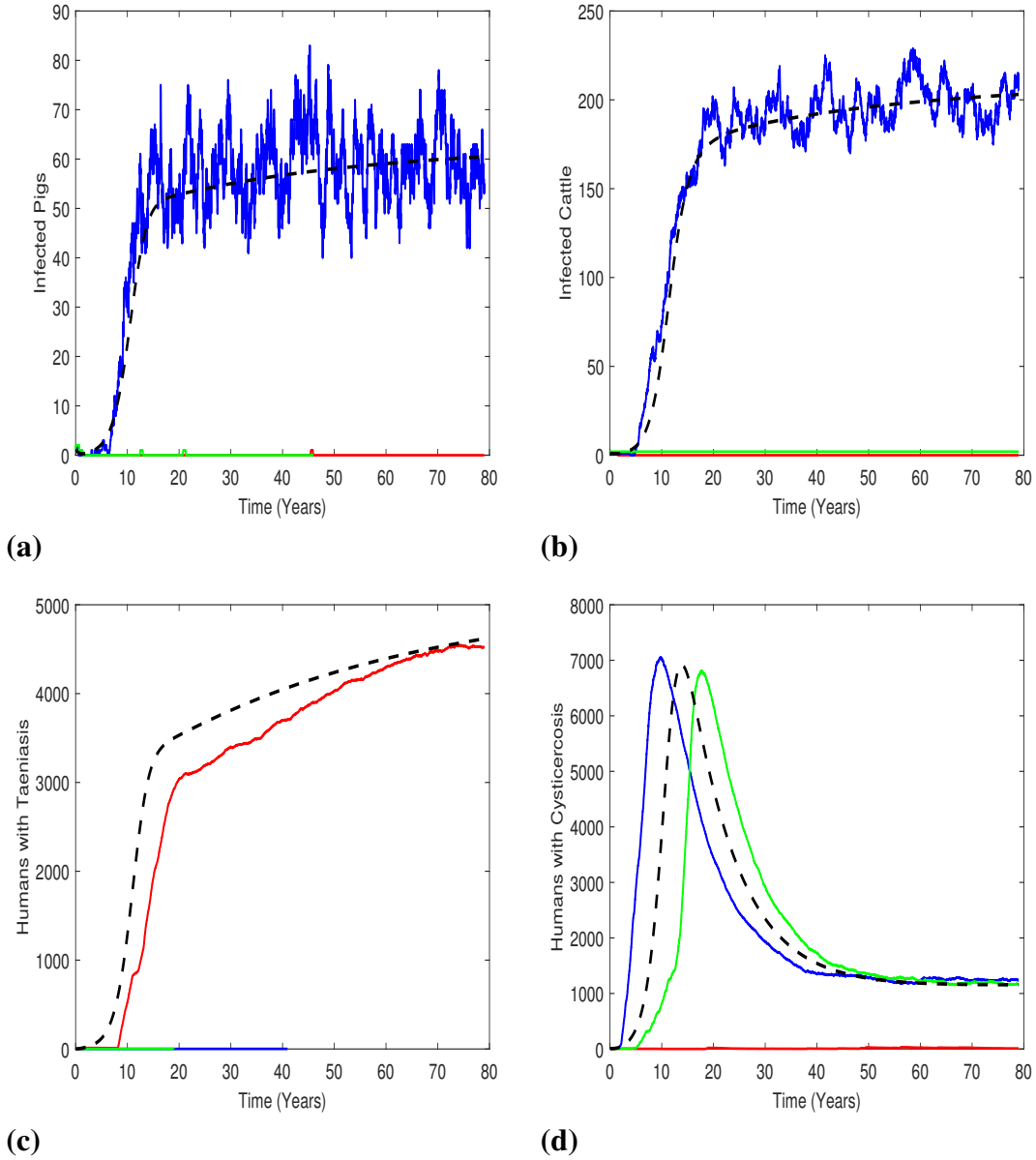


Figure 36: Comparison of deterministic solution (dashed) and three sample paths of the CTMC (solid) for infected humans, pigs and cattle

The results in Table 14 show that probability of disease extinction is very high if the diseases emerge from a small number of *T. saginata* egg and the disease dies automatically if it emerges from humans who are infected with cysticercosis since these are just dead end hosts. In all other case, there is major disease outbreak in humans, pigs and cattle. These results signify that any intervention that focus on reducing the number of infected humans with taeniasis, and infectious pork and beef at the beginning of the disease outbreak will play a great role in the control of taeniasis and cysticercosis in humans, pig and cattle.

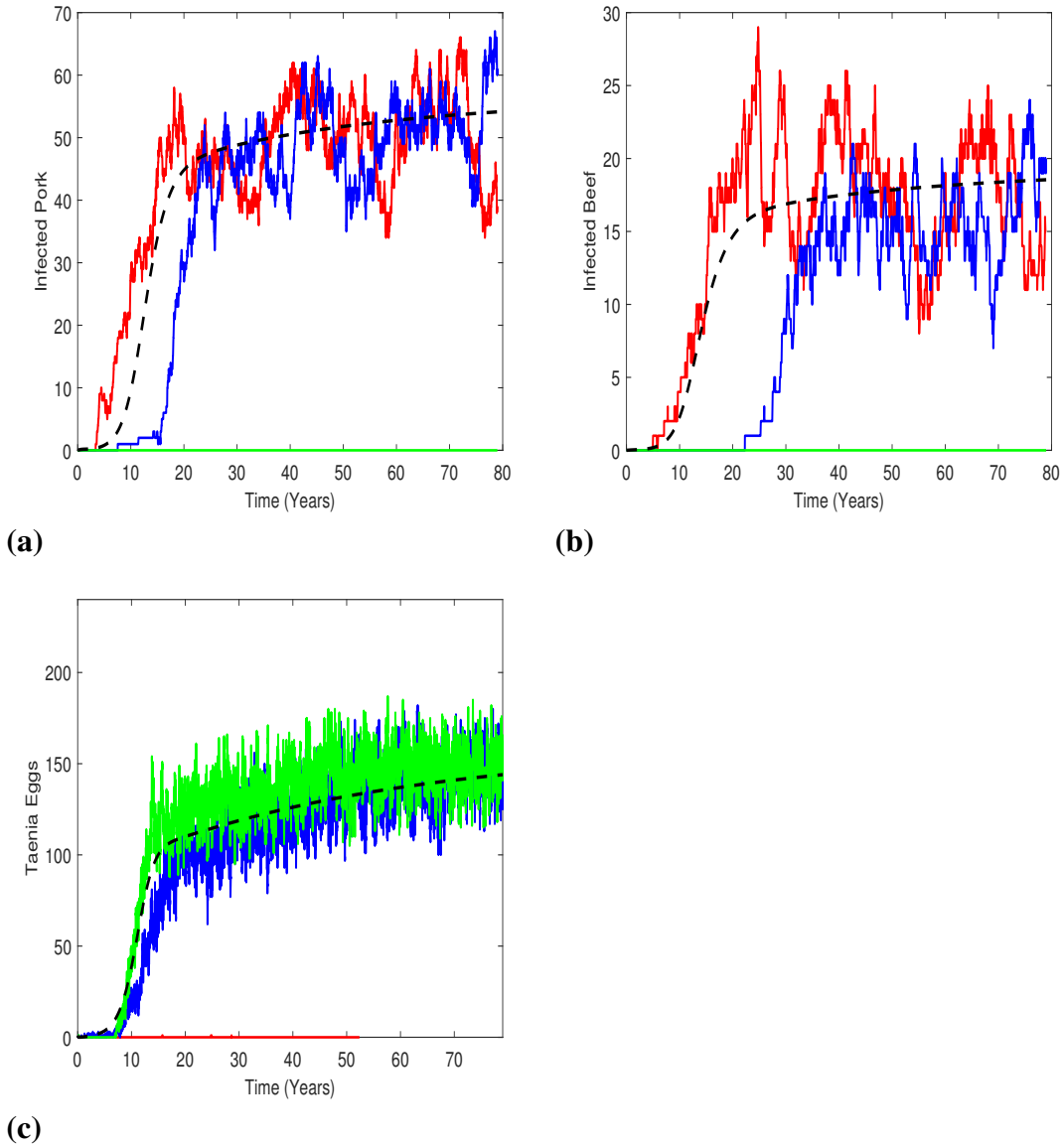


Figure 37: Comparison of deterministic solution (dashed) and three sample paths of the CTMC (solid) for infected pork, beef and taenia eggs in the environment

4.10 Comparison of the Basic Reproduction Number R_0 with the Effective Reproduction Number R_e

Figure 38 shows the comparison between effective reproduction number R_e in (3.174) and basic reproduction number R_0 in (3.176) when some parameters are varied using parameter values in Table 7. The aim is to assess whether implementation of control measures have significant impact in reducing or eliminating taeniasis and cysticercosis in humans, pigs and cattle.

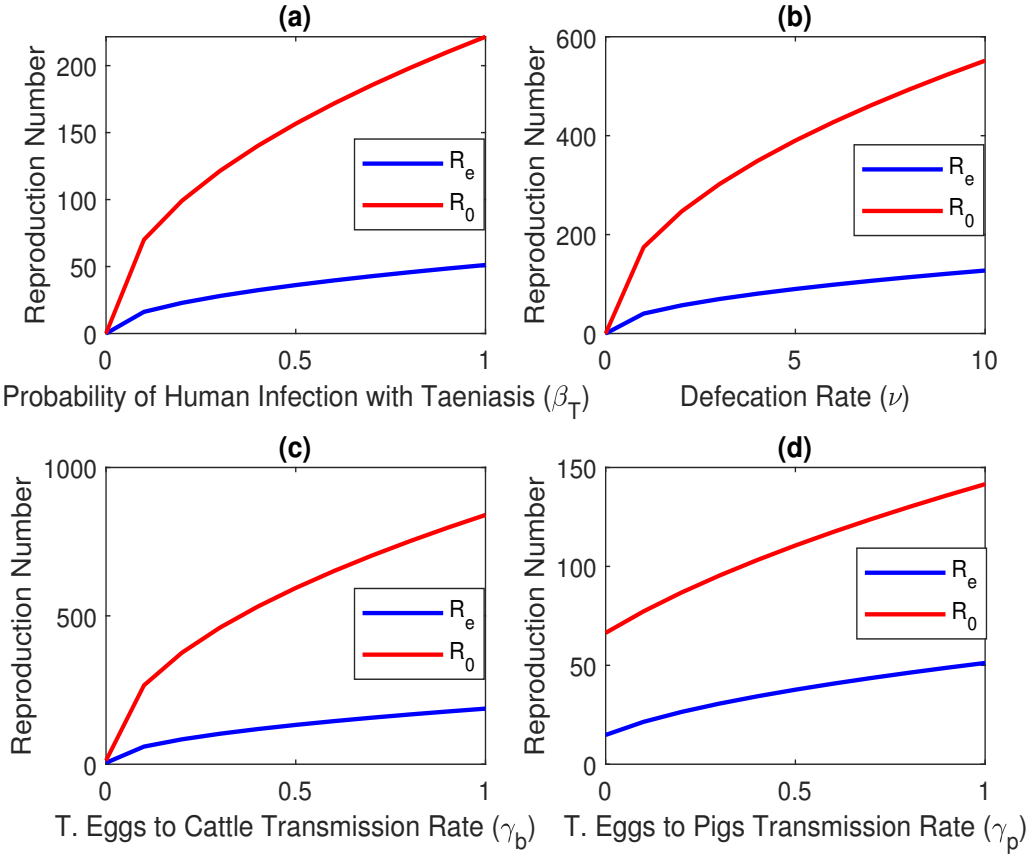


Figure 38: Variations in R_0 and R_e with respect to β_T, ν, γ_p and γ_b

From Fig. 38(a),(b),(c) and (d), it can be observed that, the effective reproduction number R_e is less than the basic reproduction number R_0, implying that there is a possibility of reducing the prevalence of taeniasis and cysticercosis in humans, pigs and cattle when some interventions are implemented.

4.11 Numerical Results for the Optimal Control and Cost-Effectiveness Analysis of Cysticercosis and Taeniasis in Humans, Pigs and Cattle

In this section, we present numerical simulations for the optimal control to assess the impact of various interventions on the control of taeniasis and cysticercosis. Cost-ffectiveness analysis is carried out thereafter to determine the most cost effective strategy for controlling the diseases. To obtain numerical solutions of the model, the forward-backward sweep method for the model system (3.178) and the adjoint system (3.187) are implemented in Matlab using parameter values in Table 15. The method begins by solving the model system (3.178) forward in time using Runge Kutta method of the fourth order relying on the supplied initial values of the controls. Then, the backward fourth order Runge Kutta method uses the obtained values

of the state variables and initial values of controls to solve the adjoint equations (3.187) with given final condition (3.184). The controls $u_1(t), u_2(t), u_3(t), u_4(t), u_5(t)$ are then updated and used to solve the state and adjoint systems. Some parameter values have been assumed since little has been done in this area and the diseases are common in rural areas where free range farming system for pigs and cattle is dominant, there is inadequate or no meat inspection and the treatment of these diseases is not readily available (Mkupasi *et al.*, 2011).

Table 15: Model parameter values (unit: yr^{-1})

Parameter	Value	Source	Parameter	Value	Parameter	Value
Λ_H	2247	Wu *et al.* (2013)	Λ_C	750	η	0.235
μ_p	0.996	Winskill *et al.* (2017)	Λ_P	1450	θ	0.00523
μ_d	0.0925	Wang *et al.* (2013)	ε	0.225	α_p	0.012
μ_h	0.0141	Wang *et al.* (2013)	δ	0.358	ρ_b	0.1968
ω	0.332	Kyvsgaard *et al.* (2007)	β_T	0.093	γ_b	0.00625
μ_e	10.42	Wang *et al.* (2013)	ν	0.150	λ_b	0.125
π_b	0.213	-	λ_p	0.125	α_b	0.023
μ_b	0.33	Wang *et al.* (2013)	σ_s	0.213	χ_c	0.192
γ_p	0.01	Kyvsgaard *et al.* (2007)	σ_v	0.183	χ	0.225
π_p	0.213	-	ρ_p	0.105	ϕ_v	0.413
ψ_v	0.115	-	ρ_v	0.075	σ_b	0.153
ψ_s	0.213	-	ρ_s	0.252	ϕ_s	0.317

Since the implementation of only one control may not be effective in disease control, we illustrate the impact of combining atleast two controls as follows: Strategy 1: Meat inspection ($u_1(t)$), pigs and cattle vaccination ($u_2(t)$ and $u_3(t)$ respectively), treatment of humans who are infected with taeniasis ($u_4(t)$) and improved hygiene and sanitation ($u_5(t)$), Strategy 2: Meat inspection ($u_1(t)$), pigs and cattle vaccination ($u_2(t)$ and $u_3(t)$ respectively) and treatment of humans who are infected with taeniasis ($u_4(t)$), Strategy 3: Meat inspection ($u_1(t)$), treatment of humans who are infected with taeniasis ($u_4(t)$) and improved hygiene and sanitation ($u_5(t)$), Strategy 4: Meat inspection ($u_1(t)$) with pigs and cattle vaccination ($u_2(t)$ and $u_3(t)$ respectively), Strategy 5: Pigs and cattle vaccination ($u_2(t)$ and $u_3(t)$ respectively), and treatment of humans who are infected with taeniasis ($u_4(t)$), Strategy 6: Meat inspection ($u_1(t)$) and improved hygiene and sanitation ($u_5(t)$), Strategy 7: Meat inspection ($u_1(t)$) and treatment of humans who are infected with taeniasis ($u_4(t)$), Strategy 8: Treatment of humans who are infected with taeniasis ($u_4(t)$) and improved hygiene and sanitation ($u_5(t)$), and Strategy 9: Pigs and cattle vaccination ($u_2(t)$ and $u_3(t)$ respectively).

4.11.1 When all Controls are Implemented

When all controls are implemented, all infected populations and taenia eggs in the environment decrease with time while humans with taeniasis decrease to zero in 3 years. Infected pigs, cattle and taenia eggs diminish to zero in 3, 5.5 and 1.5 years respectively. Only a small portion of humans who are infected with cysticercosis remains at the final time.

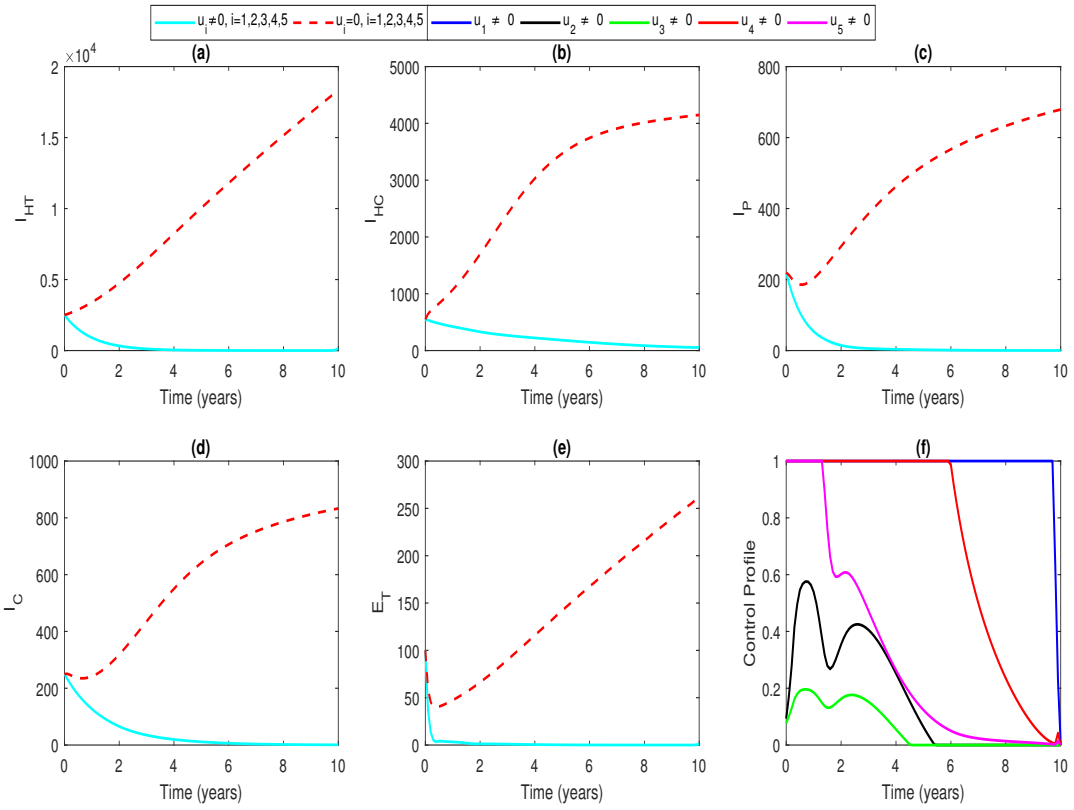


Figure 39: Impact of applying all controls on infected humans, cattle, pigs and taenia eggs

The control profiles for this strategy in Fig. 39(f) show that the control variables $u_1(t), u_4(t)$ and $u_5(t)$ are at their peak for the first 9.75, 6.5 and 1.5 years respectively and finally decline to zero. The control profiles for $u_2(t)$ and $u_3(t)$ variables have up and down movement and then decline to zero in the 5.5^{th} and 4.5^{th} years respectively.

4.11.2 Meat Inspection, Pigs and Cattle Vaccination, and Treatment of Humans Infected with Taeniasis

In this strategy, we consider a combination of meat inspection $(u_1(t))$, vaccination of pigs and cattle ($u_2(t)$ and $u_3(t)$ respectively) and treatment of humans with taeniasis $(u_4(t))$. It can be observed in Fig. 40 that, cysticercosis in pigs and cattle can be eradicated after 5 and 7 years

respectively while human taeniasis can be eradicated after 3 years. However, a small proportion of humans infected with cysticercosis remains at the final time. The control profiles in Fig. 40(f) show that the control variables $u_1(t)$, $u_2(t), u_3(t)$ and $u_4(t)$ are at their peak for the first 9.5, 3, 1.75 and 6 years respectively. Thereafter $u_1(t)$ and $u_4(t)$ decline to zero whereas $u_2(t)$ and $u_3(t)$ reduces to zero in the 5^{th} and 5.5^{th} years respectively.

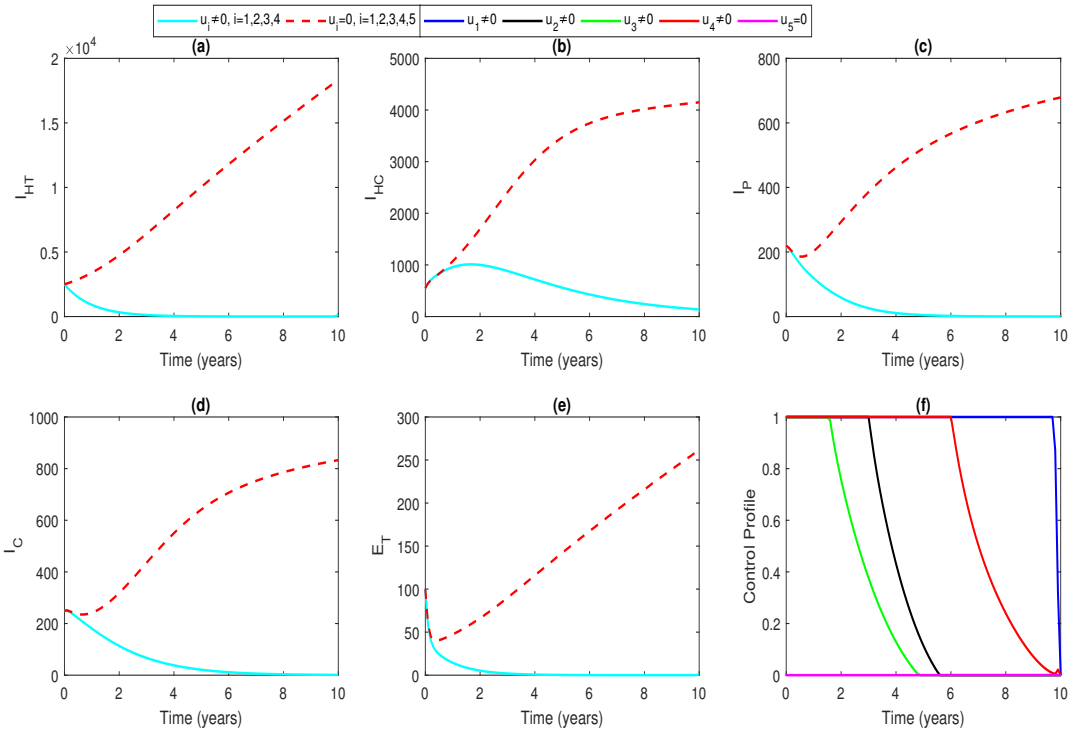


Figure 40: Impact of meat inspection, pigs and cattle vaccination, and treatment of humans with taeniasis on infected humans, cattle, pigs and taenia eggs

4.11.3 Meat Inspection, Improved Hygiene and Sanitation, and Treatment of Humans with Taeniasis

Here, a combination of meat inspection $(u_1(t))$, improved hygiene and sanitation $(u_5(t))$ and treatment of humans with taeniasis $(u_4(t))$ is considered . It can be seen in Fig. 41 that cysticercosis in pigs and cattle can be eliminated after 4 and 6 years respectively whereas human taeniasis and taenia eggs reduces to zero after 4 and 2 years respectively. Only a small proportion of humans remains infected with cysticercosis at the final time. The control profiles in Fig. 41 (f) show that the control variables $u_1(t)$ and $u_4(t)$ are at their peak for the first 9.5 and 6.5 years respectively and then decline to zero in the final time. On the other hand, the control profile for $u_5(t)$ variable has up and down movement in the first 1.75 years and then declines to zero in the final time.

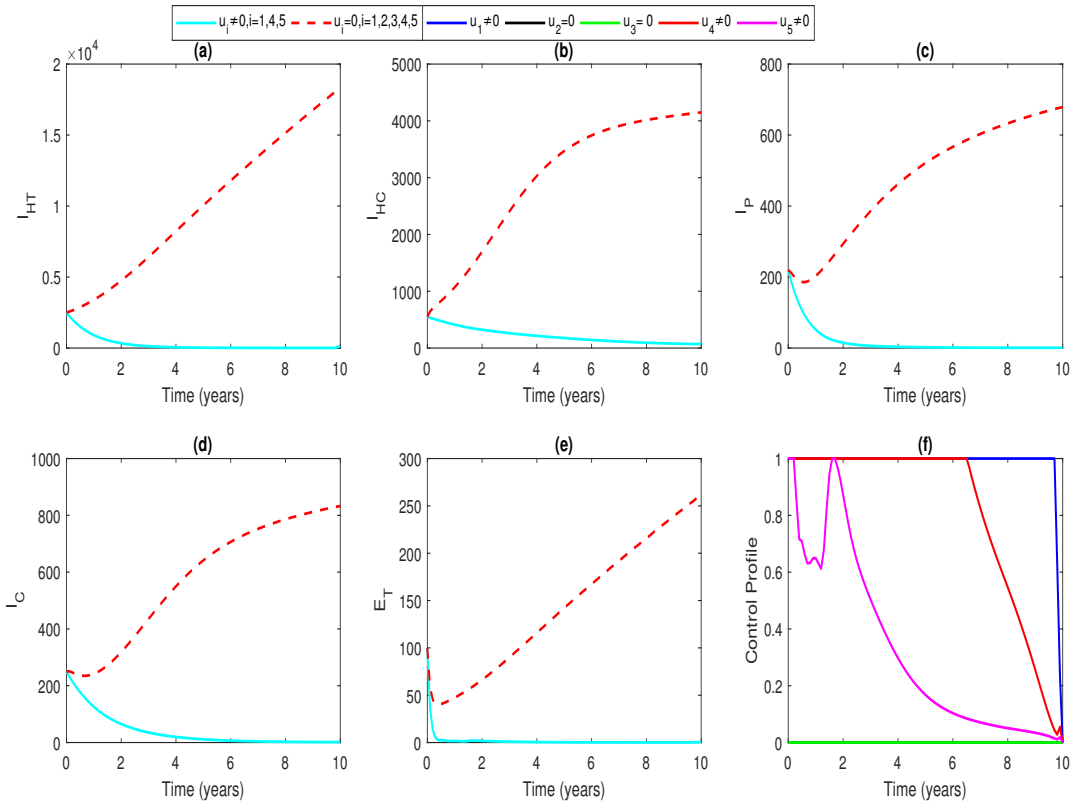


Figure 41: Impact of meat inspection, improved hygiene and sanitation, and treatment of Humans with taeniasis on infected humans, cattle, pigs and taenia eggs

4.11.4 Meat Inspection and Vaccination of Pigs and Cattle

In this strategy a combination of meat inspection $(u_1(t))$ and vaccination of pig and cattle populations ($u_2(t)$ and $u_3(t)$ respectively) is implemented. The results in Fig. 42 show that initially the infected pigs decrease with time in the first year and then attain the equilibrium. On the other hand, infected cattle approach to zero after the first 2 years while humans infected with taeniasis and taenia eggs decrease and remain intact. A different trend is observed for humans infected with cysticercosis where a small proportion declines for the first five years but flourish later. This is the case when hygiene and sanitation is not improved. The control profiles in Fig. 42(f) show that the control variable $u_1(t), u_2(t)$ and $u_3(t)$ are at their peak for the first 9.8, 9.75 and 9.25 years respectively and finally decline to zero.

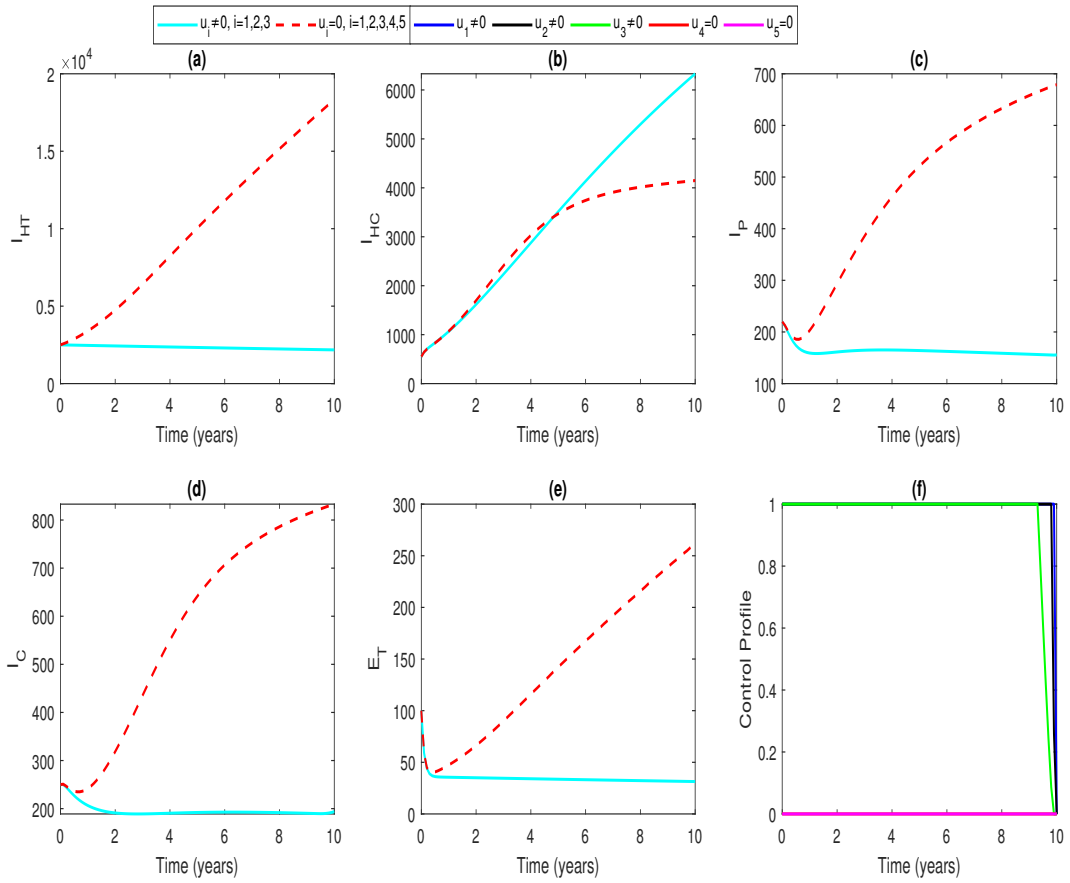


Figure 42: Impact of meat inspection and vaccination of pigs and cattle on infected humans, cattle, pigs and taenia eggs

4.11.5 Vaccination of Pigs and Cattle, and Treatment of Humans Infected with Taeniasis

In this strategy, the optimal control model with control variables that involve a combination of pigs and cattle vaccination ($u_2(t)$ and $u_3(t)$ respectively) and treatment of humans with taeniasis ($u_4(t)$) is simulated. The results in Fig. 43 indicate that cases of human taeniasis, taenia eggs, and porcine and bovine cysticercosis reduce after implementing this strategy, however there is no possibility to control the diseases. On the other hand, there is a decline of human cysticercosis cases in the first five years and thereafter exceed the case when no control is applied. The control profiles in Fig. 43 (f) show that the control variables $u_2(t)$, $u_3(t)$ and $u_4(t)$ are at their peak for the first 9.75, 9.5 and 9.8 years respectively and they eventually reduce to zero in the final time.

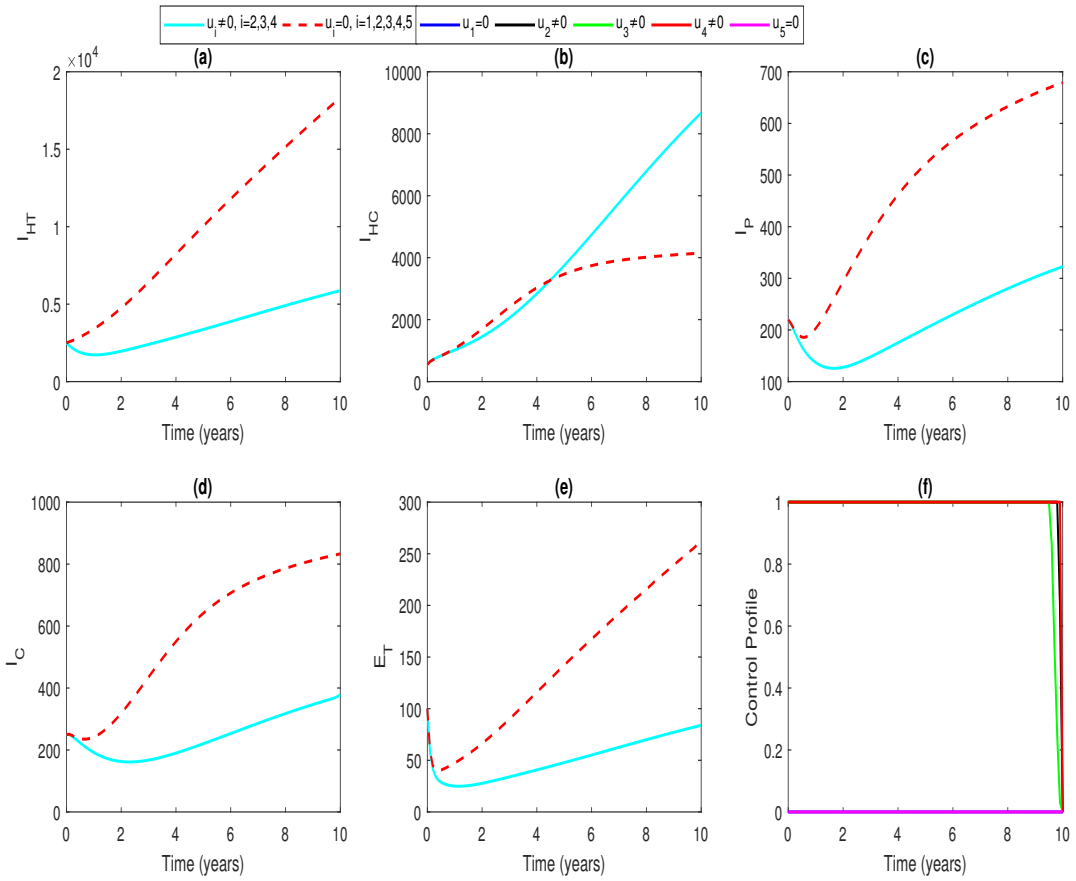


Figure 43: Impact of vaccination of pigs and cattle, treatment of humans with taeniasis on infected humans, cattle, pigs and taenia eggs

4.11.6 Meat Inspection, and Improved Hygiene and Sanitation

In this strategy a combination of meat inspection $(u_1(t))$ with improved hygiene and sanitation $(u_5(t))$ is implemented. It can be observed that infected pigs and cattle decline with time, reaching zero in 4 and 6 years respectively while taenia eggs diminish quickly in the first 0.5 years. On the other hand, there is reduction in number of humans with taeniasis and humans with cysticercosis, however human infections cannot be eliminated for the whole time of implementing the controls. The control profiles in Fig. 44 (f) show that the control variable $u_5(t)$ is at its peak for the first 1.75 years followed by up and down movements until the 9^{th} year when it drops down quickly to zero. On the other hand, the control profiles $u_1(t)$ is at its peak for the first 9.5 years and finally declines to zero.

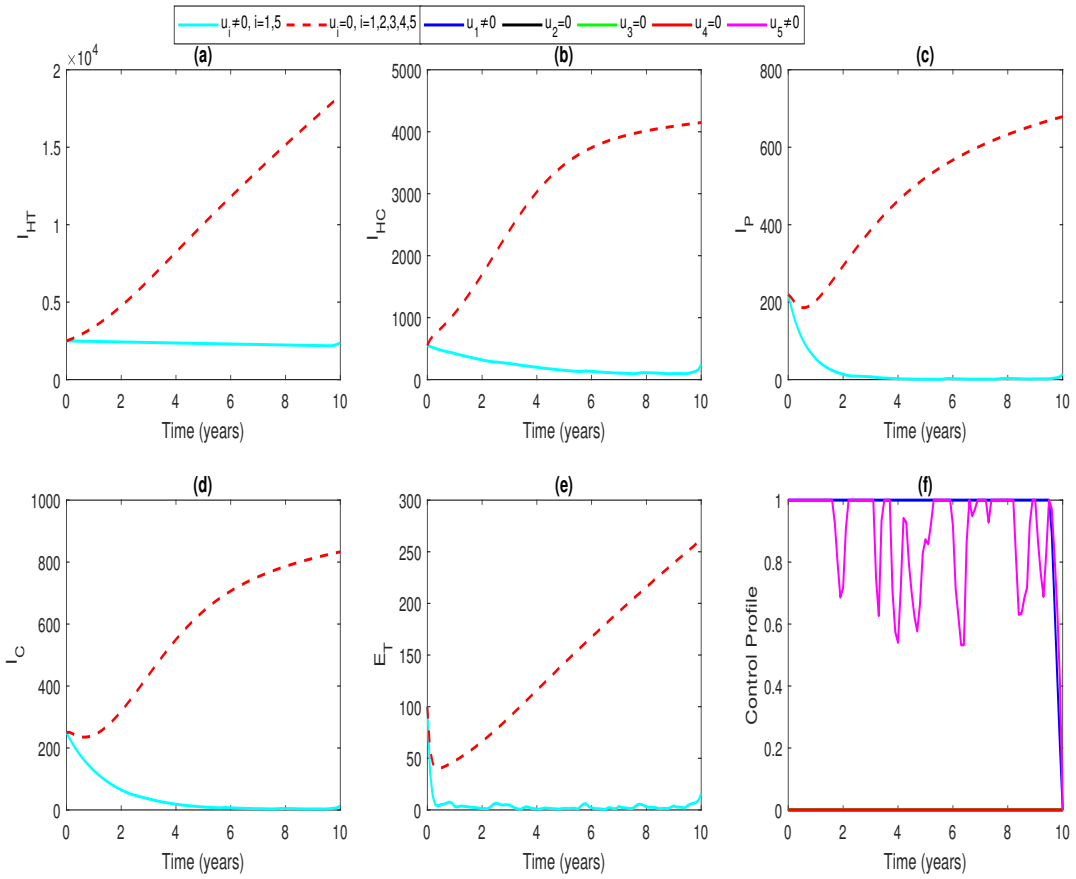


Figure 44: Impact of meat inspection and improved hygiene and sanitation on infected humans, cattle, pigs and taenia eggs

4.11.7 Meat Inspection and Treatment of Humans with Taeniasis

This strategy assesses the impact of implementing a combination of meat inspection $(u_1(t))$ with treatment of humans who are infected with taeniasis $(u_4(t))$. It can be seen in Fig. 45 that humans taeniasis, porcine cyticercosis, bovine cysticercosis, and taenia eggs in the environment can be elimnated within 4, 6, 8 and 4 years respectively. Also, humans who are infected with cysticercosis decline with time, however a proportion remains in the final time. The control profiles in Fig. 45 (f) indicated that the control variables $u_1(t)$ and $u_4(t)$ are at their peak for the first 9.5 and 6.25 years respectively and finally decline to zero.

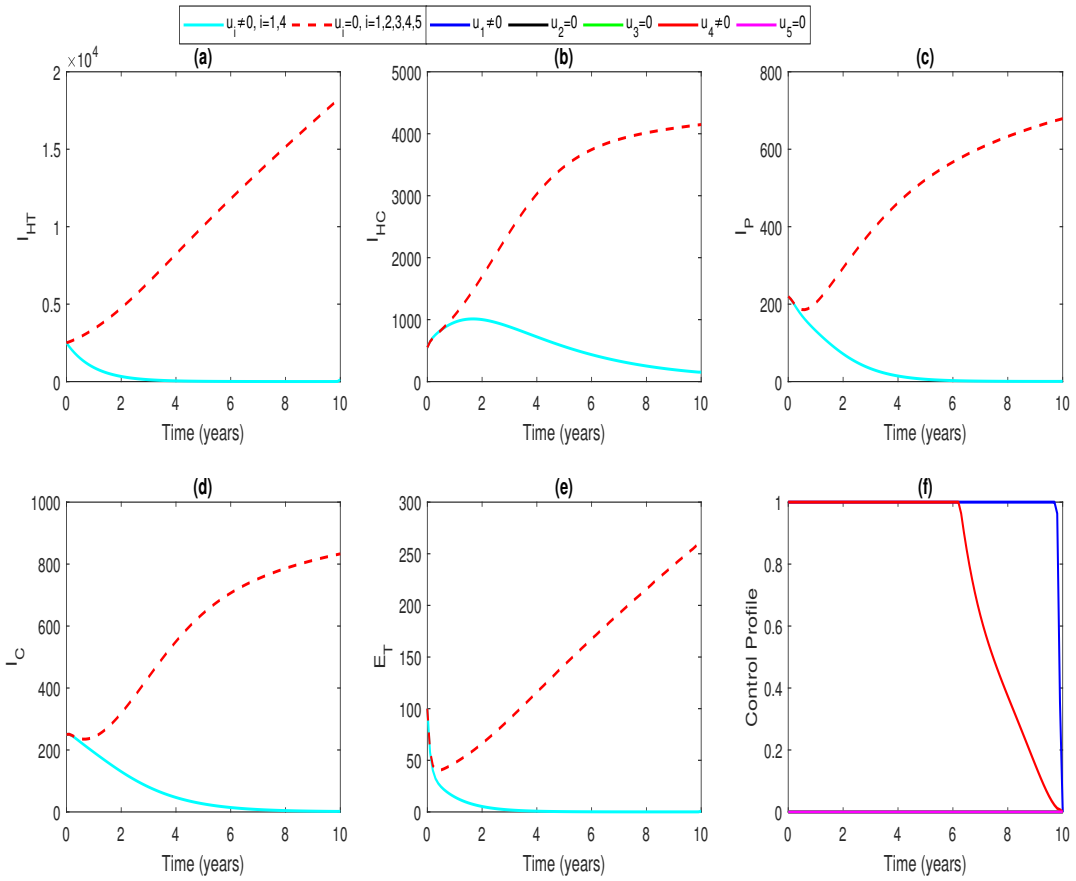


Figure 45: Impact of meat inspection and treatment of humans with taeniasis on infected humans, cattle, pigs and taenia eggs

4.11.8 Improved Hygiene and Sanitation & Treatment of Humans with Taeniasis

This strategy gives similar results as strategy 6 which combines meat inspection with improved hygiene and sanitation whereby porcine and bovine cysticercosis can be eliminated within 4 and 6 years respectively.This is due to the fact that when hygiene and sanitation is improved, humans with taeniasis and infected cattle and pigs are treated, the cases of taeniasis and cysticercosis in humans and the number of taenia eggs in the environment decrease significantly as portrayed in Fig. 46. The profiles in Fig. 46 (f) show that the control variable $u_5(t)$ peaks for the first year followed by up and down movements until the 9^{th} year after which it declines to zero while $u_4(t)$ peaks for the first 9.9 years and finally declines to zero.

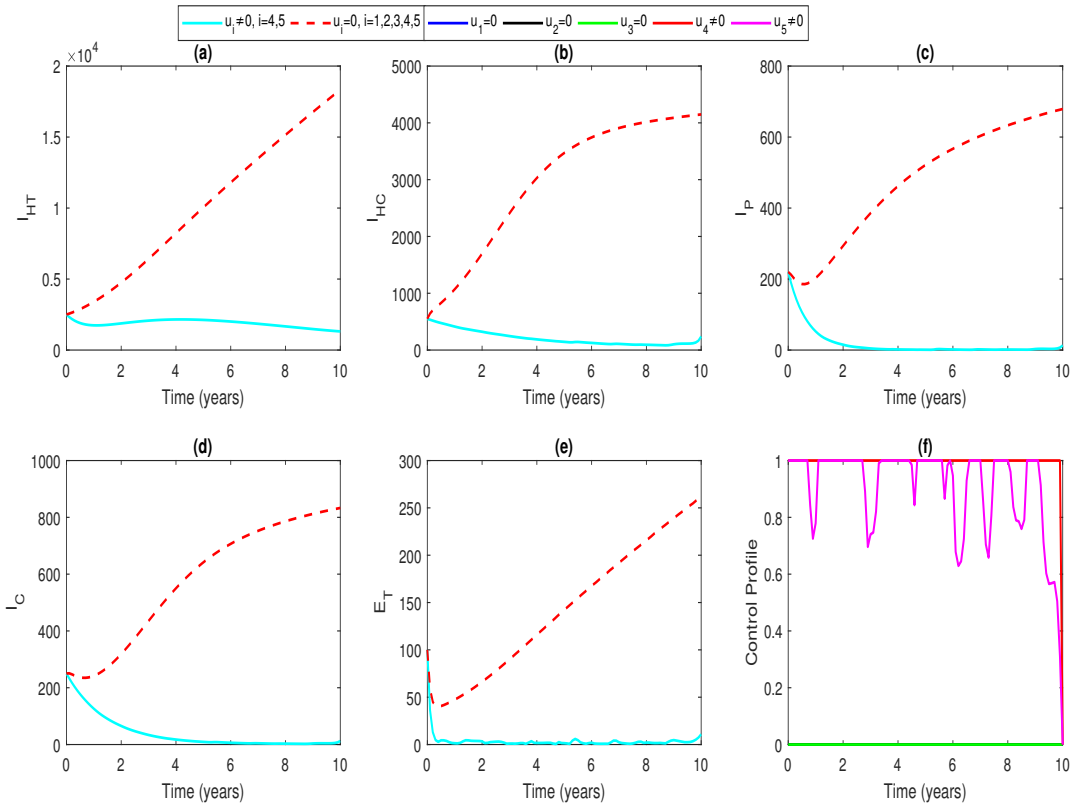


Figure 46: Impact of improved hygiene and sanitation with treatment of humans having taeniasis on infected humans, cattle, pigs and taenia eggs

4.11.9 Pigs and Cattle Vaccination

Here, we assess the impact of implementing pigs and cattle vaccination on disease control in humans, pigs and cattle. This strategy is ineffective since when it is implemented only a small change in humans with taeniasis, infected pigs and cattle, and taenia eggs in the environment is observed. For the case of humans with cysticercosis, there is no change in the first five years and thereafter the number of cases increases compared to the case when there is no control. The profiles in Fig. 47(f) show that the control variable $u_2(t)$ and $u_3(t)$ are always at their peak for the first 9.9 and 9.5 years respectively and quickly declines to zero.

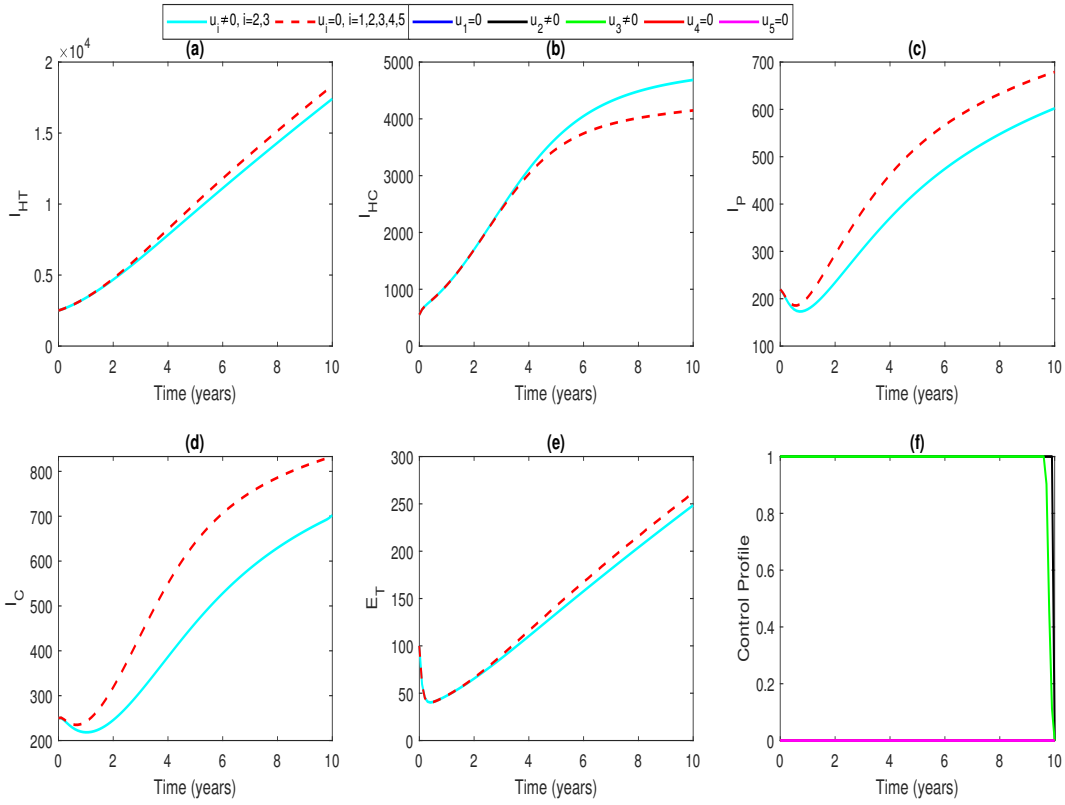


Figure 47: Impact of pigs and cattle vaccination on infected humans, cattle, pigs and taenia eggs

Generally, it can be observed that the first and third strategies that involve implementation of all control measures and when pigs cattle vaccination is excluded are effective in controlling the transmission of taeniasis and cysticercosis in humans, pigs and cattle.

4.12 Cost-Effectiveness Analysis

In this section, we carryout a cost-effectiveness analysis to justify the costs associated with various control strategies that include meat inspection, vaccination of pigs and cattle, treatment of humans who have taeniasis, and improved hygiene and sanitation. The analysis is based on incremental cost-effectiveness ratio (ICER) (Agusto, 2013; Agusto & ELmojtaba, 2017; Omame *et al.*, 2021) which is given by:

$$\text{ICER(X)} = \frac{\text{Difference in infection averted costs in strategies X and Y}}{\text{Difference in total number of infection averted in strategies X and Y}}$$

To implement the ICER, the optimal control model is simulated using various intervention strategies. Then, using the simulation results, the control strategies are ranked in increasing

order of effectiveness based on cases of infection averted. In computing the ICER for startegy 1, we consider the baseline when no intervention has been implemented to control the diseases. That is both total cost and total infection averted are zero. The ICER for various control strategies are given in Table 16

Table 16: ICER in increasing order of total infection averted

Strategy	Total Cost	Total Infection Averted	ICER
1	313 188.91	1 388 893.43	0.2255
3	312 002.18	1 388 898.50	-234.0690
2	442 817.08	1 390 133.11	105.9565
7	472 839.59	1 390 136.79	8 158.2908
8	515 079.26	1 390 326.56	222.5835
5	2 349 121.73	1 391 602.56	1 437.3374
6	569 467.05	1 392 116.77	-3 460.9492
4	1 874 540.84	1 393 457.10	973.6959
9	4 045 734.84	1 394 988.27	1 417.9967

By comparing two strategies with the highest ICER in Table 16, it can be observed that the ICER for strategy 7 is greater than the ICER for strategy 5. This shows that Strategy 7 is more expensive to implement compared to Strategy 5. Therefore, Strategy 7 can be excluded from the set of controls for implementation so as to preserve the available limited resources. Thus, Strategy 7 is removed and Strategy 5 is further compared with other Strategies. The procedure is repeated by computing the ICER and comparing the strategies until when two strategies remain for comparison purposes. Thus, the ICER for the remaining strategies are presented in Table 17.

Table 17: ICER in increasing order of total infection averted

Strategy	Total Cost	Total Infection Averted	ICER
1	313 188.91	1 388 893.43	0.2255
3	312 002.18	1 388 898.50	-234.0690
2	442 817.08	1 390 133.11	105.9565
8	515 079.26	1 390 326.56	373.5445
5	2 349 121.73	1 391 602.56	1 437.3374
6	569 467.05	1 392 116.77	-3 460.9492
4	1 874 540.84	1 393 457.10	973.6959
9	4 045 734.84	1 394 988.27	1 417.9967

It can be observed further from Table 17 that the ICER for Strategy 5 is greater than ICER for Strategy 9 which implies that Strategy 9 is less expensive to implement than Strategy 5. Thus, strategy 5 can be removed from the list of control options so that Strategy 9 can be compared with other strategies. The ICER for the remaining strategies are shown in Table 18.

Table 18: ICER in increasing order of total infection averted

Strategy	Total Cost	Total Infection Averted	ICER
1	313 188.91	1 388 893.43	0.2255
3	312 002.18	1 388 898.50	-234.0690
2	442 817.08	1 390 133.11	105.9565
8	515 079.26	1 390 326.56	373.5445
6	569 467.05	1 392 116.77	30.3807
4	1 874 540.84	1 393 457.10	973.6959
9	4 045 734.84	1 394 988.27	1 417.9967

Similarly, the ICER for Strategy 9 from Table 18 is greater than ICER for Strategy 4 which means that Strategy 4 is less costly than Strategy 9. Thus, we remove strategy 9 from the list of intervention strategies so that Strategy 4 can be compared with the remaining strategies. Thus, the ICER for the remaining strategies are shown in Table 19.

Table 19: ICER in increasing order of total infection averted

Strategy	Total Cost	Total Infection Averted	ICER
1	313 188.91	1 388 893.43	0.2255
3	312 002.18	1 388 898.50	-234.0690
2	442 817.08	1 390 133.11	105.9565
8	515 079.26	1 390 326.56	373.5445
6	569 467.05	1 392 116.77	30.3807
4	1 874 540.84	1 393 457.10	973.6959

Likewise, it can be seen from Table 19 that the ICER for Strategy 4 is greater than ICER for Strategy 8, implying that Strategy 4 is more expensive to implement than Strategy 8. Thus, Strategy 4 is excluded from the set of control strategies and Strategy 8 is compared with other strategies. Following the same procedure, the results are shown in Tables 20, 21, 22 and 23.

Table 20: ICER in increasing order of total infection averted

Strategy	Total Cost	Total Infection Averted	ICER
1	313 188.91	1 388 893.43	0.2255
3	312 002.18	1 388 898.50	-234.0690
2	442 817.08	1 390 133.11	105.9565
8	515 079.26	1 390 326.56	373.5445
6	569 467.05	1 392 116.77	30.3807

Table 21: ICER in increasing order of total infection averted

Strategy	Total Cost	Total Infection Averted	ICER
1	313 188.91	1 388 893.43	0.2255
3	312 002.18	1 388 898.50	-234.0690
2	442 817.08	1 390 133.11	105.9565
6	569 467.05	1 392 116.77	63.8466

Table 22: ICER in increasing order of total infection averted

Strategy	Total Cost	Total Infection Averted	ICER
1	313 188.91	1 388 893.43	0.2255
3	312 002.18	1 388 898.50	-234.0690
6	569 467.05	1 392 116.77	80.0010

Table 23: ICER in increasing order of total infection averted

Strategy	Total Cost	Total Infection Averted	ICER
1	313 188.91	1 388 893.43	0.2255
3	312 002.18	1 388 898.50	-234.0690

Table 23 indicates that Strategy 3 (with negative ICER) strongly dominates Strategy 1. This means that Strategy 3 is less costly and more effective to implement compared to Strategy 1. Thus, these results indicate that the strategy that involves a combination of meat inspection, improved hygiene and sanitation, and treatment of humans who are infected with taeniasis saves more money and gives the best outcomes. Therefore, this strategy is the most cost-effective strategy in combating taeniasis and cysticercosis transmission in humans, pigs and cattle.

CHAPTER FIVE

CONCLUSION AND RECOMMENDATIONS

5.1 Summary

This study aimed at developing and analyzing mathematical models for transmission dynamics of taeniasis and cysticercosis in humans, pigs and cattle, find important parameters for the diseases' transmission, determine the likelihood of diseases' extinction or outbreak, assess control strategies and establish the cost effective strategy. The general objective of the study was to develop and analyze mathematical models for transmission dynamics and control of taeniasis and cysticercosis in humans, pigs and cattle. To address the general objective of the study, we proposed four specific objectives which are to: formulate deterministic and continuous time Markov chain stochastic models for taeniasis and cysticercosis dynamics, analyze mathematical models and perform sensitivity analysis to determine sensitive parameters to the diseases, apply the multitype branching process to compute the probability of diseases' extinction or outbreak, apply optimal control theory to determine the diseases' optimal control strategy and apply cost effectiveness analysis method to determine the most cost effective strategy for diseases' control.

The deterministic models were developed with the aid of ordinary differential equations. To provide better understanding of the problem,we began by developing *Taenia saginata* model for bovine cysticercosis and human taeniasis followed by the full model for transmission dynamics of cysticercosis and taeniasis due to *Taenia solium* and *Taenia saginata* tapeworms in humans, pigs and cattle. The corresponding CTMC stochastic models were developed basing on assumptions of deterministic models. To make the models mathematically and biologically realistic, we stated several assumptions and tested for positivity and boundedness of model solutions. The next generation matrix approach and forward normalized sensitivity index were applied respectively to compute the basic or effective reproduction numbers and sensitivity indices to determine sensitive parameters for the transmission dynamics of taeniasis and cysticercosis in humans, pigs and cattle. Lyapunov functions, Metzler matrix method and cooperative systems were used to investigate the stability of disease free and endemic equilibria. The center manifold theory was used to study the occurrence of backward or forward bifurcation and the multitype branching process was applied to compute the probability of diseases' extinction or outbreak.

To develop optimal control policy for taeniasis and cysticercosis, different control strategies such as treatment of infected humans, pigs and cattle, vaccination for pigs and cattle, meat inspection, indoor keeping for cattle and pigs, improved hygiene and sanitation and improved meat cooking were proposed and Pontryagin maximum principle was applied to evaluate their impact in controlling the diseases and finally, the incremental cost-effectiveness ratio (ICER)

was applied to determine the most cost effective strategy. The models were simulated in Matlab software using either Runge Kutta 4th order or Euler methods.

5.2 Conclusion

From the study, it can be concluded that taeniasis and cysticercosis in humans, pigs and cattle is largely influenced by open defecation by humans with taeniasis, consumption of raw or undercooked infected beef and pork, and number of taenia eggs in the environment. Analysis of the CTMC stochastic model show that there is major outbreak of taeniasis and cysticercosis in humans, pigs and cattle when the diseases emerge from humans with taeniasis or infectious pork and beef. Thus to control taeniasis and cysticercosis, control measures should focus on treatment of humans with taeniasis, improving meat cooking, meat inspection and improving hygiene and sanitation.

When interventions were implemented, the *T. saginata* model exhibits forward bifurcation implying that reducing effective reproduction number R_{e1} below unit is a sufficient condition for diseases' eradication. The optimal control analysis results for *T. saginata* model indicate that the strategy which focuses on improving meat cooking and treatment of infected humans is the most optimal strategy for diseases' control. Similarly, the optimal control analysis results for *T. solium* and *T. saginata* model shows that the strategy which combines meat (pork and beef) inspection, pigs' and cattle vaccination, treatment of humans with taeniasis, improving hygiene and sanitation is the optimal strategy for diseases' control. However, the cost effectiveness analysis indicates that the strategy which excludes vaccination of pigs and cattle is the most cost-effective strategy for diseases' control.

5.3 Recommendations

Taenia solium and *Taenia saginata* parasitic infection will remain a major challenge to human and animal health, posing significant impacts to the livelihood of rural livestock farming communities if proper control measures are not put in place. Based on research findings, we recommend the following:

i. Model results have reveled that humans with taeniasis, infectious beef and pork, and taenia eggs in the environment play a grater role in the transmission dynamics of taeniasis and cysticercosis. Hence, to control these diseases there is a need to ensure that there is improved hygiene and sanitation, improving meat cooking and meat inspection as well as treatment of humans with taeniasis so as to minimize the rate a which these diseases spread.

ii. Using the optimal control and cost-effectiveness analysis, our results have shown that diseases' control measures which combines meat inspection, treatment of humans with taeniasis, and improving hygiene and sanitation is the most effective strategy for diseases' control. Thus, to effectively control the spread of taeniasis and cysticercosi at minimal costs, the suggested control measures should be emphasized and implemented in combination.

iii. Based on sensitivity analysis and simulation results, *T. solium* parasitic infection poses a great threat to human health. Thus to control its spread, we recommend that pigs' indoor keeping should be emphasized, and hygiene and sanitation need to be improved.

iv. Mathematical models should be utilized together with other non-mathematical approaches for designing more appropriate measures to combat the spread of taeniasis and cysticercosis.

v. Massive awareness programs through information campaigns, educational seminars and the use of mass media such as televisions, radio and magazines to enhance people's should be carried out to ensure that the community is aware how taeniasis and cysticercosis are transmitted.

vi. The government should strengthen collaboration with non-governmental and international organizations to establish measures and implement strategies for diseases' control.

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APPENDICES

MATLAB Codes

APPENDIX I

```
%MATLAB code for Bifurcation diagram
%By Joshua Mwasunda
%PhD Student (AMCS) - NM-AIST, Arusha, Tz
%24 May 2021
close all;
clear;
R0_value=0:0.00001:2;
Root_array=zeros(length(R0_value),2);
%=============================================================
%Parameter Values
%c=Lambda_H,h=Beta_T, g=alpha_b,n=theta,r=mu_h;b=Lambda_C;
%e=gamma_b;j3=rho_s; s=mu_b;l=eta;p=epsilon;m=nu;t=mu_e
% x1=chi; k=kappa; r1=psi_v;r2=pi;j3=sigma_s, r3=psi_s;
%r4=lambda;r0=rho_b; j3=sigma_r; j2=sigma_v;c1=tau; c2=c_h
%=============================================================
c=227;x1=0.425;h=0.093;g=0.023;k=0.350;r=0.0141;b=750;
r1=0.248;j2=0.183;q=0.083*12; r2=0.213; e=0.00625;
j1=0.213;r3=0.115; s=0.33;ro=0.1968;r4=0.125;l=0.235;
j3=0.153;m=0.325;p=0.325;t1=10.42;c1=0.525;c2=0.623;
%=============================================================
c0=(j2+r1+s+ro*r3);
k0=c0*(j1+s)+r3*(j2+s);
h0=(1-c1)*h*g*e*b*l*(r2+j3+s);
D=ro*(r2*(l+r4)+(l+r2+r4)*(j1+r3)+(j3*(l+r4+j1+r3)));
O=r2*(l+r4+r2)+(r1+j2)+j3*(l+r4+r1+j2);
h1=ro*(r2*l+s*(l+r2+r4+s)+(l+r4+s)*j3);
h2=(l+r4+s)*(r2+j3+s)*((j2+s)*(j1+r3+s)+(j1+s)*r1);
h3=s^3*(1-ro)+ro*((r2+r4)*(r2+j3)*j1+(l*r2+(l+r4)*j3)*r3)...
+(l*r2+(l+r4)*j3)*(j2+r1)+s^2*(l+r4+r2+j2+j3+ro*...
(l+r4+r2+j2+j3+ro+r3)+r1)
+s*(D+O);
%=============================================================
for i=1:length(R0_value); R0=R0_value(i);
%Coefficients of quadratic equation
A=(c2+t1)*r*e*(ro*h0+e*h1*(p+g)*(x1+r));
B=(c2+t1)*r*(h0*(c0+ro*r3)+e*(p+g)*(x1+r)*h3)-h0*(1-c1)*m*ro*r3*c;
C=h2*(p+g)*(x1+r)*(1+R0)*(1-R0);
p1=[A,B,C];
y=roots(p1); len=length(y);
```

```
for t=1:len
if (imag(y(t))~=0) || (real(y(t))<0); Root_array(i,t)=0;
else
Root_array(i,t)=y(t);
end
end
end
%=================================================================
f=1;
f=f+1;
R0_value_Cr=f;
for j=R0_value_Cr:length(R0_value)
Root_array(j,:)=sort(Root_array(j,:));
end
f1=R0_value_Cr;
while (Root_array(f1,1)~=0) f1=f1+1;
end
R0_value_Cr2=f1;
Zero_1st=R0_value(1,1:R0_value_Cr2-1);...
y_zero=zeros(1,length(Zero_1st));
Unstable=R0_value(1,R0_value_Cr:length(R0_value));
%=================================================================
plot(Unstable,Root_array(R0_value_Cr:length(R0_value),2),'b',...
'LineWidth',3)
xlabel('Reproduction number, R_0','FontSize',12)
ylabel('Force of Infection','FontSize',12)
hold off
%=================================================================
plot(R0_value,Root_array(:,1),'r--',R0_value,Root_array(:,2),'b',...
'LineWidth',3)
xlabel('R_e','FontSize',12)
ylabel('E_T','FontSize',12)
title('Occurence of Forward Bifurcation')


%=================================================================
%MATLAB code for Basic Model Taenia saginata
%By Joshua Mwasunda
%PhD Student (AMCS) - NM-AIST, Arusha, Tz
%24 May 2021
clc;
clear;
close all;
%Parameter Values
%c=Lambda_H, h=Beta_T, g=alpha_b, r=mu_h; b=Lambda_C;
%e=gamma_b; i=sigma_s; s=mu_b; l=eta; p=epsilon; m=nu; t=mu_e
```

```
%============================================================
b=750; c=2247; e=0.00625; g=0.023; h=0.093; i=0.213;
l=0.235; m=0.325; p=0.325; r=0.0141; s=0.33; t1=10.42;
%============================================================
%Variables
%S_H=V, I_{HT}=W, S_C=B, I_C=C, B_I=D, E_T=E
%============================================================

fV=@(t,V,W,B,C,D,E)   c-h*(g*D)*V-r*V;
fW=@(t,V,W,B,C,D,E)   h*(g*D)*V-r*W;
fB=@(t,V,W,B,C,D,E)   b-e*B*E-(i+s)*B;
fC=@(t,V,W,B,C,D,E)   e*B*E-(l+s)*C;
fD=@(t,V,W,B,C,D,E)   l*C-(p+g)*D;
fE=@(t,V,W,B,C,D,E)   m*W-t1*E;
%============================================================
t(1)=0;
V(1)=5420;
W(1)=750;
B(1)=1250;
C(1)=850;
D(1)=l*C(1);
E(1)=1000;
tfinal=8;h=0.01;
N=ceil(tfinal/h);
%============================================================
for i=1:N
t(i+1)=t(i)+h;
k1V=fV(t(i), V(i), W(i), B(i),  C(i), D(i), E(i));
k1W=fW(t(i), V(i), W(i), B(i),  C(i), D(i), E(i));
k1B=fB(t(i), V(i), W(i), B(i),  C(i), D(i), E(i));
k1C=fC(t(i), V(i), W(i), B(i),  C(i), D(i), E(i));
k1D=fD(t(i), V(i), W(i), B(i),  C(i), D(i), E(i));
k1E=fE(t(i), V(i), W(i), B(i),  C(i), D(i), E(i));

k2V=fV(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2W=fW(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2B=fB(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2C=fC(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2D=fD(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2E=fE(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);


k3V=fV(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3W=fW(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3B=fB(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3C=fC(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
```

```
k3D=fD(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3E=fE(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);


k4V=fV(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4W=fW(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4B=fB(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4C=fC(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4D=fD(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4E=fE(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);

V(i+1)=V(i)+(h/6)*(k1V+2*k2V+2*k3V+k4V);
W(i+1)=W(i)+(h/6)*(k1W+2*k2W+2*k3W+k4W);
B(i+1)=B(i)+(h/6)*(k1B+2*k2B+2*k3B+k4B);
C(i+1)=C(i)+(h/6)*(k1C+2*k2C+2*k3C+k4C);
D(i+1)=D(i)+(h/6)*(k1D+2*k2D+2*k3D+k4D);
E(i+1)=E(i)+(h/6)*(k1E+2*k2E+2*k3E+k4E);
end
%=================================================================
figure(1)
subplot(2,2,1)
plot(t,V,'k','linewidth',2)
hold on
plot(t,W,'b','linewidth',2)
grid on
legend('S_H','I_{HT}')
title('(a)')
xlabel('Time(Years)')
ylabel('Human Population')
%=================================================================
subplot(2,2,2)
plot(t,B,'m','linewidth',2)
hold on
plot(t,C,'b','linewidth',2)
hold on
plot(t,D, 'r-.','linewidth',2)
xlabel('Time(Years)')
ylabel('Bovine Population')
title('(b)')
grid on
legend('S_C','I_C','B_I')
%=================================================================
subplot(2,2,3)
plot(t,E,'k-.','linewidth',2)
xlabel('Time(Years)')
ylabel('Taenia Eggs')
```

```
title('(c)')
grid on


 %=============================================================
 %MATLAB code for Taenia saginata Model with Interventions
 %By Joshua Mwasunda
 %PhD Student (AMCS) - NM-AIST, Arusha, Tz
 %24 May 2021
 clc;
 clear;
 close all;
 %=============================================================
 %Parameter Values
 %c=Lambda_H,h=Beta_T, g=alpha_b,n=theta,r=mu_h;b=Lambda_C;
 %e=gamma_b;j3=rho_s; s=mu_b;l=eta;p=epsilon;m=nu;t=mu_e
 % x1=chi; k=kappa; r1=psi_v;r2=pi;j3=sigma_s, r3=psi_s;
 %r4=lambda;r0=rho_b; j3=sigma_r; j2=sigma_v;c1=tau; c2=c_h
 %=============================================================
 c=227;x1=0.425;h=0.093;g=0.023;k=0.350;r=0.0141;b=750;
 r1=0.248;j2=0.183;q=0.083*12; r2=0.213; e=0.00625;
 j1=0.213;r3=0.115; s=0.33;ro=0.1968;r4=0.125;l=0.235;
 j3=0.153;m=0.325;p=0.325;t1=10.42;c1=0.525;c2=0.623;
 %=============================================================
 %Variables
 %S_H=V, I_{HT}=W, S_C=B, V_C=G, I_C=C, R_C=H, B_I=D, E_T=E
 %=============================================================
 %Parameters to Vary for Figure 3.6
 %k=0.30; c1=0.20; c2=0.05; r3=0.08; r4=0.15; x1=0.07;
 %k=0.40; c1=0.40; c2=0.10; r3=0.12; r4=0.25; x1=0.13;
 %k=0.43; c1=0.45; c2=0.15; r3=0.18; r4=0.30; x1=0.20;
 %k=0.50; c1=0.60; c2=0.20; r3=0.22; r4=0.35; x1=0.23;
 %k=0.60; c1=0.70; c2=0.30; r3=0.32; r4=0.45; x1=0.33;
 %k=0.70; c1=0.80; c2=0.40; r3=0.42; r4=0.55; x1=0.43;
 % PLOT ONLY THE GRAPHS FOR INFECTIOUS CLASSESS (I_{HT},I_C,E_T)
 %=============================================================
 fV=@(t,V,W,B,G,C,H,D,E)   c+x1*W-h*g*(1-k)*D*V-r*V;
 fW=@(t,V,W,B,G,C,H,D,E)   h*g*(1-k)*D*V-(r+x1)*W;
 fB=@(t,V,W,B,G,C,H,D,E)   b+r1*G+r2*H-e*B*E-(j1+r3+s)*B;
 fG=@(t,V,W,B,G,C,H,D,E)   r3*B-ro*e*G*E-(j2+r1+q)*G;
 fC=@(t,V,W,B,G,C,H,D,E)   e*B*E+ro*e*G*E-(l+r4+s)*C;
 fH=@(t,V,W,B,G,C,H,D,E)   r4*W-(r2+j3+q)*H;
 fD=@(t,V,W,B,G,C,H,D,E)   l*C-(p+g)*D;
 fE=@(t,V,W,B,G,C,H,D,E)   m*(1-c1)*W-(t1+c2)*E;

 t(1)=0;
 V(1)=1500;
```

```
W(1)=2750;
B(1)=250;
G(1)= 1250;
C(1)=800;
H(1)= 25;
D(1)=l*C(1);
E(1)=250;
tfinal=10;
h=0.01;
N=ceil(tfinal/h);
%============================================================
for i=1:N
t(i+1)=t(i)+h;

k1V=fV(t(i), V(i), W(i), B(i), G(i), C(i), H(i), D(i), E(i));
k1W=fW(t(i), V(i), W(i), B(i), G(i), C(i), H(i), D(i), E(i));
k1B=fB(t(i), V(i), W(i), B(i), G(i), C(i), H(i), D(i), E(i));
k1G=fG(t(i), V(i), W(i), B(i), G(i), C(i), H(i), D(i), E(i));
k1C=fC(t(i), V(i), W(i), B(i), G(i), C(i), H(i), D(i), E(i));
k1H=fH(t(i), V(i), W(i), B(i), G(i), C(i), H(i), D(i), E(i));
k1D=fD(t(i), V(i), W(i), B(i), G(i), C(i), H(i), D(i), E(i));
k1E=fE(t(i), V(i), W(i), B(i), G(i), C(i), H(i), D(i), E(i));

k2V=fV(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2W=fW(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2B=fB(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2G=fG(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2C=fC(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2H=fH(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2D=fD(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2E=fE(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);


k3V=fV(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3W=fW(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3B=fB(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3G=fG(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3C=fC(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3H=fH(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3D=fD(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3E=fE(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);

k4V=fV(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4W=fW(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4B=fB(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4G=fG(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
```

```
k4C=fC(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4H=fH(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4D=fD(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4E=fE(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);

V(i+1)=V(i)+(h/6)*(k1V+2*k2V+2*k3V+k4V);
W(i+1)=W(i)+(h/6)*(k1W+2*k2W+2*k3W+k4W);
B(i+1)=B(i)+(h/6)*(k1B+2*k2B+2*k3B+k4B);
G(i+1)=G(i)+(h/6)*(k1G+2*k2G+2*k3G+k4G);
C(i+1)=C(i)+(h/6)*(k1C+2*k2C+2*k3C+k4C);
H(i+1)=H(i)+(h/6)*(k1H+2*k2H+2*k3H+k4H);
D(i+1)=D(i)+(h/6)*(k1D+2*k2D+2*k3D+k4D);
E(i+1)=E(i)+(h/6)*(k1E+2*k2E+2*k3E+k4E);

end
%=========================================
figure(1)
subplot(2,2,1)
plot(t,V,'m','linewidth',2)
hold on
plot(t,W,'b','linewidth',2)
xlabel('Time(Years)')
ylabel('Human Population')
title('(a)')
grid on
legend('S_H','I_{HT}')
%=========================================
subplot(2,2,2)
plot(t,B,'m','linewidth',2)
hold on
plot(t,G,'b','linewidth',2)
hold on
plot(t,C,'g','linewidth',2)
hold on
plot(t,H,'r','linewidth',2)
hold on
plot(t,D, 'k--','linewidth',2)
xlabel('Time(Years)')
ylabel('Bovine Population')
title('(b)')
grid on
legend('S_C','V_C','I_C','R_C','B_I')
%=========================================
subplot(2,2,3)
plot(t,E,'b-.','linewidth',2)
xlabel('Time(Years)')
```

```
ylabel('Taenia saginata Eggs')
title('(c)')
grid on
%=======================================
```

APPENDIX II

```
%Bovine Cysticecosis and Human Taeniasis Epidemic Model
%CTMC Matlab Code
%By Joshua A Mwasunda
%PhD Student - NM-AIST, Arusha, Tz
%25 May, 2021

clear all
close all
clc;
set(0,'DefaultAxesFontSize', 12)%Increases axes labels
set(gca,'fontsize',18);%Changes font size in a figure.
%-----------------------------------------------------------------
%Parameter values
time=49; %time
lamb_C=100;lamb_H=250;alpha=0.23;beta=0.093;mu_h=0.0141;gam=0.00625;
sig=0.235;mu_b=0.33;eta=0.0325;eps=0.125;nu=0.325;mu_e=10.42;
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++%
%State Variables for deterministic Model
%x=S_H; x1=I_{HT}; y=S_C; y1=I_C; y2=B_I; z=E_T;
%State Variables for CTMC Model
%X=S_H; X1=I_{HT}; Y=S_C; Y1=I_C; Y2=B_I; Z=E_T;
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Euler's method for solving ODE
S_hum= lamb_H/mu_h;
S_cat= lamb_C/(sig+mu_b);
%++++++++++++++++++++++++++++++++++%
dt=0.05; tim=time/dt; kk=0:dt:time;

%Initial conditions
x0=2; y0=1;s0=1; z0=1;
%-----------------------------------------------------------------

x1(1)=x0; y1(1)=y0; y2(1)=s0; z(1)=z0; x(1)=S_hum-x0; y(1)=S_cat-y0;

for jj=1:tim
%Using Euler's formula i.e. y(n+1)=yn + h*f(xn,yn) where h=dt
x(jj+1)  = x(jj)  + dt*(lamb_H-beta*alpha*x(jj)*y2(jj)-mu_h*x(jj));
x1(jj+1) = x1(jj) + dt*(beta*alpha*x(jj)*y2(jj)-mu_h*x1(jj));
y(jj+1) = y(jj) + dt*(lamb_C-gam*z(jj)*y(jj)-(sig+mu_b)*y(jj));
y1(jj+1) = y1(jj) + dt*(gam*z(jj)*y(jj)-(eta+mu_b)*y1(jj));
y2(jj+1) = y2(jj) + dt*(eta*y1(jj)-(eps+alpha)*y2(jj));
z(jj+1)= z(jj)+ dt*(nu*x1(jj)-mu_e*z(jj));
end
%=================================================================%
```

```
%Three sample paths for CTMC (Gillespie's algorithm)
for k=1:3
clear t X X1 Y Y1 Y2 Z
j=1;
X1(1)=x0; Y1(1)=y0; Y2(1)=s0; Z(1)=z0;
X(1)=S_hum-x0; Y(1)=S_cat-y0;
t(1)=0; %Starting at zero
%-------------------------------------------------------------------
while (X1(j)+Y1(j)+Y2(j)+Z(j))>0 && t(j)<time
% Stop if it hits zero or at end time

randm1=rand; %Uniform random number
randm2=rand; %Uniform random number
%-------------------------------------------------------------------
H(j)=X(j)+X1(j);
Tc(j)=Y(j)+Y1(j);
%-------------------------------------------------------------------
%Total sum of rates of all possible events at time (t+1)
Total=lamb_H+mu_h*H(j)+lamb_C+mu_b*Tc(j)+nu*X1(j)+sig*Y(j)+eta*Y1(j)+...
(eps+alpha)*Y2(j)+mu_e*Z(j)+beta*alpha*Y2(j)*X(j)+gam*Y(j)*Z(j);
%-------------------------------------------------------------------
%Defining all possible events at time (t+1)
ev1 = lamb_H/Total;
ev2 = ev1 + beta*alpha*Y2(j)*X(j)/Total;
ev3 = ev2 + mu_h*X(j)/Total;
ev4 = ev3 + mu_h*X1(j)/Total;
ev5 = ev4 + nu*X1(j)/Total;
ev6 = ev5 + lamb_C/Total;
ev7 = ev6 + sig*Y(j)/Total;
ev8 = ev7 + gam*Z(j)*Y(j)/Total;
ev9 = ev8 + mu_b*Y(j)/Total;
ev10= ev9 + eta*Y1(j)/Total;
ev11= ev10+ mu_b*Y1(j)/Total;
ev12= ev11 + (eps+alpha)*Y2(j)/Total;
ev13= ev12+ mu_e*Z(j)/Total;
%-------------------------------------------------------------
t(j+1)=t(j)-log(randm1)/Total; % Time to next event
%-------------------------------------------------------------
%Computing probabilities of each event occuring at time (t+1)
if randm2<ev1;     % Recruitment of Susceptible Humans
X(j+1)  = X(j)+1;
X1(j+1) = X1(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
```

```
elseif randm2>=ev1 & randm2<ev2  % Infection of Susceptible humans
X(j+1)  = X(j)-1;
X1(j+1) = X1(j)+1;
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j)-1;
Z(j+1) = Z(j);
elseif randm2>=ev2 & randm2<ev3  % Death of Susceptible humans
X(j+1)  = X(j)-1;
X1(j+1) = X1(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev3 & randm2<ev4   % Death of Infected Humans
X(j+1)  = X(j);
X1(j+1) = X1(j)-1;
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev4 & randm2<ev5 % Shedding Taenia Eggs
X(j+1)  = X(j);
X1(j+1) = X1(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j)+1;
elseif randm2>=ev5 & randm2<ev6 %Recruitment of Susceptible Cattle
X(j+1)  = X(j);
X1(j+1) = X1(j);
Y(j+1)  = Y(j)+1;
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev6 & randm2<ev7 %Slaughter of Susceptible Cattle
X(j+1)  = X(j);
X1(j+1) = X1(j);
Y(j+1)  = Y(j)-1;
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev7 & randm2<ev8  %Infection of Susceptible Cattle
X(j+1)  = X(j);
X1(j+1) = X1(j);
Y(j+1)  = Y(j)-1;
```

```
Y1(j+1) = Y1(j)+1;
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev8 & randm2<ev9  %Death of Susceptible Cattle
X(j+1)  = X(j);
X1(j+1) = X1(j);
Y(j+1)  = Y(j)-1;
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev9 & randm2<ev10 %Slaughter of Infected Cattle
X(j+1)  = X(j);
X1(j+1) = X1(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j)-1;
Y2(j+1) = Y2(j)+1;
Z(j+1) = Z(j);
elseif randm2>=ev10 & randm2<ev11 %Death of Infected Cattle
X(j+1)  = X(j);
X1(j+1) = X1(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j)-1;
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev11 & randm2<ev12 %Throwing Infected Beef
X(j+1)  = X(j);
X1(j+1) = X1(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j)-1;
Z(j+1) = Z(j);
else                        % Death of Taenia Eggs
X(j+1)  = X(j);
X1(j+1) = X1(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j)-1;
end
j=j+1;
end
%-------------------------------------------------------------
figure (1)
if k==1
plot(t,X1,'r-','linewidth',1.5)
hold on
```

```
end
if k==2
plot(t,X1,'b-','linewidth',1.5)
hold on
end
if k==3
plot(t,X1,'g-','linewidth',1.5)
hold on
end
plot(kk,x1,'k--','LineWidth',2)
xlabel('Time (Years)','Fontsize',12)
ylabel('Infected Humans','Fontsize',12)
%=================================================================%
%PLOT OTHER GRAPHS IN SIMILAR MANNER
%=================================================================%

%=================================================================
%Prob of Extinction for CTMC Epidemic Model using 10000 Sample Paths
%By Joshua A Mwasunda
%PhD Student (AMCS) - NM-AIST, Arusha, Tz
%24 May 2021

clear all
close all
clc;
set(0,'DefaultAxesFontSize', 18)%Increases axes labels
set(gca,'fontsize',18);%Changes font size in a figure.
%-----------------------------------------------------------------
%Parameter values of the model
time=49; %time
lambda_C=420;lambda_H=100;alpha=0.23;beta=0.093;
mu_h=0.0141;gam=0.00625;sig=0.235;mu_b=0.33;
eta=0.0325;eps=0.125;nu=0.325;mu_e=10.42;
%-----------------------------------------------------------------
S_hum= lambda_H/mu_h;
S_cat= lambda_C/(sig+mu_b);
%-----------------------------------------------------------------
%Reproduction Number
R0=sqrt((beta*nu*alpha*gam*eta*lambda_C*lambda_H)/...
(mu_h^2*mu_e*(eta+mu_b)*(eps+alpha)*(sig+mu_b)));
%-----------------------------------------------------------------
%Initial Conditions
%x0=0; y0=0;s0=1; z0=0;
x0=1; y0=0;s0=0; z0=0;
%x0=0; y0=1;s0=0; z0=0;
%x0=0; y0=0;s0=0; z0=1;
```

```
%x0=1; y0=1;s0=1; z0=1;
%x0=0; y0=2;s0=0; z0=0;
%x0=1; y0=1;s0=0; z0=0;
%x0=1; y0=1;s0=0; z0=1;
%x0=1; y0=0;s0=0; z0=1;
%x0=2; y0=0;s0=0; z0=1;
%x0=1; y0=0;s0=0; z0=2;
%============================
%Three sample paths for CTMC (Gillespie's algorithm)
z=0;
ext=10000; % number of sample paths to be used
for k=1:ext
k;
clear t X X1 Y Y1 Y2 Z
j=1;
X1=x0; Y1=y0;  Y2=s0; Z=z0;
%S=Sstar-(x0+y0+z0);
X=S_hum-x0; Y=S_cat-y0;
t=0; %Starting at zero
%-------------------------------------------------------------------
while (X1+Y1+Y2+Z)>0 && t<time  % Stop if it hits zero or at end time
randm1=rand; %Uniform random number
randm2=rand; %Uniform random number
%-------------------------------------------------------------------
%Defining all possible events at time (t+1)
ev1 = lambda_H;
ev2 = ev1 + beta*alpha*Y2(j)*X;
ev3 = ev2 + mu_h*X;
ev4 = ev3 + mu_h*X1;
ev5 = ev4 + nu*X1;
ev6 = ev5 + lambda_C;
ev7 = ev6 + sig*Y;
ev8 = ev7 + gam*Z*Y;
ev9 = ev8 + mu_b*Y;
ev10= ev9 + eta*Y1;
ev11= ev10+ mu_b*Y1;
ev12= ev11 + (eps+alpha)*Y2;
ev13= ev12+ mu_e*Z;
% ev13 and Total are the same thing, so just use ev13
%-----------------------------------------------------------
t=t-log(randm1)/ev13; % Time to next event
%-----------------------------------------------------------
randm2=randm2*ev13;  % rather than divide everything by Total,
%instead multiply random number by Total
% rather than working with Unif(0,1), we work with Unif(0,Total)
```

```
%Computing probabilities of each event occuring at time (t+1)
if randm2<ev1;
X = X+1;
X1= X1;
Y= Y;
Y1= Y1;
Y2= Y2;
Z= Z;
elseif randm2>=ev1 & randm2<ev2  % Infection of Susceptible humans
X = X-1;
X1 = X1+1;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev2 & randm2<ev3  % Death of Susceptible humans
X = X-1;
X1 = X1 ;
Y = Y ;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev3 & randm2<ev4  % Death of Infected Humans
X = X;
X1 = X1-1;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev4 & randm2<ev5 % Shedding of Taenia Eggs
X = X;
X1 = X1;
Y= Y;
Y1 = Y1;
Y2 = Y2;
Z = Z+1;
elseif randm2>=ev5 & randm2<ev6 %Recruitment of Susceptible Cattle
X = X;
X1 = X1;
Y = Y+1;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev6 & randm2<ev7 %Slaughter of Susceptible Cattle
X = X;
X1 = X1;
```

```
Y = Y-1;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev7 & randm2<ev8   %Infection of Susceptible Cattle
X = X;
X1  = X1;
Y = Y-1;
Y1  = Y1+1;
Y2 = Y2;
Z= Z;
elseif randm2>=ev8 & randm2<ev9  %Death of Susceptible Cattle
X  = X;
X1 = X1;
Y  = Y-1;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev9 & randm2<ev10   %Slaughter of Infected Cattle
X  = X;
X1 = X1;
Y = Y;
Y1 = Y1-1;
Y2 = Y2+1;
Z = Z;
elseif randm2>=ev10 & randm2<ev11 %Death of Infected Cattle
X = X;
X1 = X1;
Y = Y;
Y1 = Y1-1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev11 & randm2<ev12 %Throwing Infected Beef
X  = X;
X1  = X1;
Y  = Y;
Y1  = Y1;
Y2  = Y2-1;
Z = Z;
else                        % Death of Taenia Eggs
X = X;
X1 = X1;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z-1;
```

```
end

end
if (X1+Y1+Y2+Z)==0
z=z+1;
else
z=z;
end

end
z;  %number of sample paths that hit zero out of 1000 sample paths
probext=z/ext  %approximate probability of extinction
%=============================================================
```

APPENDIX III

```
%MATLAB Code: Basic Model for Dynamics of Taeniasis and Cysticecosis
% in Humans, Pigs and Cattle
%By Joshua Mwasunda
%PhD Student (AMCS) - NM-AIST, Arusha, Tz
%24 May 2021
clc;
clear;
close all;
%Parameter Values
%c=Lambda_H;a=Lambda_P; h=beta_T, g=alpha_b,n=theta,r=mu_h;d=gamma_p;
%e=gamma_b;i=rho_s;s=mu_b;l=eta;p=epsilon;m=nu;t1=mu_e;f=alpha_p;
%u=mu_d;q=mu_p;j=rho;k=omega;b=Lambda_C;
%-----------------------------------------------------------------
b=750;c=2247;e=0.00625;g=0.023;h=0.093;i=0.213;f=0.012;j=0.252;
l=0.235;m=0.325;n=0.00523;p=0.325;r=0.0141;s=0.33;t1=10.42;
k=0.083*4;o=0.358;q=0.083*12;u=0.0952;o=delta;a=1450;d=0.01;
%=================================================================
%Variables
%S_H=V,I_{HT}=W,I_{HC}=X,S_P=Y,I_P=Z,P_I=A,S_C=B,I_C=C,B_I=D,E_T=E
%=================================================================
fV=@(t,V,W,X,Y,Z,A,B,C,D,E)   c-h*(f*A+g*D)*V-n*V*E-r*V;
fW=@(t,V,W,X,Y,Z,A,B,C,D,E)   h*(f*A+g*D)*V-r*W;
fX=@(t,V,W,X,Y,Z,A,B,C,D,E)   n*V*E-(r+u)*X;
fY=@(t,V,W,X,Y,Z,A,B,C,D,E)   a-d*Y*E-(j+q)*Y;
fZ=@(t,V,W,X,Y,Z,A,B,C,D,E)   d*Y*E-(k+q)*Z;
fA=@(t,V,W,X,Y,Z,A,B,C,D,E)   k*Z-(o+f)*A;
fB=@(t,V,W,X,Y,Z,A,B,C,D,E)   b-e*B*E-(i+s)*B;
fC=@(t,V,W,X,Y,Z,A,B,C,D,E)   e*B*E-(l+s)*C;
fD=@(t,V,W,X,Y,Z,A,B,C,D,E)   l*C-(p+g)*D;
fE=@(t,V,W,X,Y,Z,A,B,C,D,E)   m*W-t1*E;

t(1)=0;
V(1)=5420;W(1)=750;
X(1)=528;Y(1)=1050;
Z(1)=620;A(1)=k*Z(1);
B(1)=1250;C(1)=850;
D(1)=l*C(1);E(1)=1000;
tfinal=15;h=0.1;
N=ceil(tfinal/h);
%=================================================================
for i=1:N
t(i+1)=t(i)+h;
k1V=fV(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1W=fW(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1X=fX(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
```

```
k1Y=fY(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1Z=fZ(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1A=fA(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1B=fB(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1C=fC(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1D=fD(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1E=fE(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));

k2V=fV(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2W=fW(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2X=fX(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2Y=fY(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2Z=fZ(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2A=fA(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2B=fB(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2C=fC(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2D=fD(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2E=fE(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);

k3V=fV( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3W=fW( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3X=fX( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3Y=fY( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3Z=fZ( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3A=fA( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3B=fB( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3C=fC( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3D=fD( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3E=fE( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);

k4V=fV( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4W=fW( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4X=fX( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4Y=fY( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4Z=fZ( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4A=fA( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4B=fB( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4C=fC( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4D=fD( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4E=fE( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);

V(i+1)=V(i)+(h/6)*(k1V+2*k2V+2*k3V+k4V);
W(i+1)=W(i)+(h/6)*(k1W+2*k2W+2*k3W+k4W);
X(i+1)=X(i)+(h/6)*(k1X+2*k2X+2*k3X+k4X);
Y(i+1)=Y(i)+(h/6)*(k1Y+2*k2Y+2*k3Y+k4Y);
Z(i+1)=Z(i)+(h/6)*(k1Z+2*k2Z+2*k3Z+k4Z);
```

```
A(i+1)=A(i)+(h/6)*(k1A+2*k2A+2*k3A+k4A);
B(i+1)=B(i)+(h/6)*(k1B+2*k2B+2*k3B+k4B);
C(i+1)=C(i)+(h/6)*(k1C+2*k2C+2*k3C+k4C);
D(i+1)=D(i)+(h/6)*(k1D+2*k2D+2*k3D+k4D);
E(i+1)=E(i)+(h/6)*(k1E+2*k2E+2*k3E+k4E);

end
%=================================================
subplot(2,2,2)
plot(t,Y,'m','linewidth',2)
hold on
plot(t,Z,'k','linewidth',2)
hold on
plot(t,A, 'r-','linewidth',2)
xlabel('Time(years)')
ylabel('Pig Population')
title('(b)')
grid on
legend('S_P','I_P','P_I')
%=================================================
subplot(2,2,1)
plot(t,B,'m','linewidth',2)
hold on
plot(t,C,'b','linewidth',2)
hold on
plot(t,D, 'k','linewidth',2)
xlabel('Time(years)')
ylabel('Cattle Population')
title('(a)')
grid on
legend('S_C','I_C','B_I')
%=================================================
subplot(2,2,4)
plot(t,E,'b--','linewidth',2)
xlabel('Time(years)')
ylabel('Taenia Eggs')
title('(d)')
grid on
%=================================================
subplot(2,2,3)
plot(t,V,'r','linewidth',2)
hold on
plot(t,W,'b','linewidth',2)
hold on
plot(t,X, 'k','linewidth',2)
ylabel('Human Population')
```

```
xlabel('Time(years)')
legend('S_H','I_{HT}','I_{HC}')
title('(c)')
grid on
%================================================

%=====================================================
% IMPACT OF VARYING HUMAN RECRUITMENT RATE (c=psi)
%=====================================================
b=750;c=25600;e=0.00625;g=0.023;h=0.093;i=0.213;f=0.012;j=0.252;
l=0.235;m=0.325;n=0.00523;p=0.325;r=0.0141;s=0.33;t1=10.42;
k=0.083*4;o=0.358;q=0.083*12;u=0.0952;o=delta;a=1450;d=0.01;
%=====================================================================
fV=@(t,V,W,X,Y,Z,A,B,C,D,E)   c-h*(f*A+g*D)*V-n*V*E-r*V;
fW=@(t,V,W,X,Y,Z,A,B,C,D,E)   h*(f*A+g*D)*V-r*W;
fX=@(t,V,W,X,Y,Z,A,B,C,D,E)   n*V*E-(r+u)*X;
fY=@(t,V,W,X,Y,Z,A,B,C,D,E)   a-d*Y*E-(j+q)*Y;
fZ=@(t,V,W,X,Y,Z,A,B,C,D,E)   d*Y*E-(k+q)*Z;
fA=@(t,V,W,X,Y,Z,A,B,C,D,E)   k*Z-(o+f)*A;
fB=@(t,V,W,X,Y,Z,A,B,C,D,E)   b-e*B*E-(i+s)*B;
fC=@(t,V,W,X,Y,Z,A,B,C,D,E)   e*B*E-(l+s)*C;
fD=@(t,V,W,X,Y,Z,A,B,C,D,E)   l*C-(p+g)*D;
fE=@(t,V,W,X,Y,Z,A,B,C,D,E)   m*W-t1*E;

t(1)=0;
V(1)=5420;W(1)=750;
X(1)=528;Y(1)=1050;
Z(1)=620;A(1)=k*Z(1);
B(1)=1250;C(1)=850;
D(1)=l*C(1);E(1)=1000;
tfinal=15;h=0.1;
N=ceil(tfinal/h);
%=====================================================================
for i=1:N
t(i+1)=t(i)+h;
k1V=fV(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1W=fW(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1X=fX(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1Y=fY(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1Z=fZ(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1A=fA(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1B=fB(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1C=fC(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1D=fD(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1E=fE(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
```

```
k2V=fV(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2W=fW(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2X=fX(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2Y=fY(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2Z=fZ(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2A=fA(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2B=fB(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2C=fC(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2D=fD(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2E=fE(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);

k3V=fV( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3W=fW( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3X=fX( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3Y=fY( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3Z=fZ( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3A=fA( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3B=fB( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3C=fC( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3D=fD( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3E=fE( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);

k4V=fV( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4W=fW( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4X=fX( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4Y=fY( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4Z=fZ( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4A=fA( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4B=fB( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4C=fC( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4D=fD( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4E=fE( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);

V(i+1)=V(i)+(h/6)*(k1V+2*k2V+2*k3V+k4V);
W(i+1)=W(i)+(h/6)*(k1W+2*k2W+2*k3W+k4W);
X(i+1)=X(i)+(h/6)*(k1X+2*k2X+2*k3X+k4X);
Y(i+1)=Y(i)+(h/6)*(k1Y+2*k2Y+2*k3Y+k4Y);
Z(i+1)=Z(i)+(h/6)*(k1Z+2*k2Z+2*k3Z+k4Z);
A(i+1)=A(i)+(h/6)*(k1A+2*k2A+2*k3A+k4A);
B(i+1)=B(i)+(h/6)*(k1B+2*k2B+2*k3B+k4B);
C(i+1)=C(i)+(h/6)*(k1C+2*k2C+2*k3C+k4C);
D(i+1)=D(i)+(h/6)*(k1D+2*k2D+2*k3D+k4D);
E(i+1)=E(i)+(h/6)*(k1E+2*k2E+2*k3E+k4E);

end
%=============================================
```

```
subplot(2,2,1)
plot(t,Z,'r','linewidth',2)
hold on
subplot(2,2,2)
plot(t,C,'m','linewidth',2)
hold on
subplot(2,2,3)
plot(t,W,'k','linewidth',2)
hold on
subplot(2,2,4)
plot(t,X,'b','linewidth',2)
hold on
b=750;c=10200;e=0.00625;g=0.023;h=0.093;i=0.213;f=0.012;j=0.252;
l=0.235;m=0.325;n=0.00523;p=0.325;r=0.0141;s=0.33;t1=10.42;
k=0.083*4;o=0.358;q=0.083*12;u=0.0952;o=delta;a=1450;d=0.01;
%================================================================
fV=@(t,V,W,X,Y,Z,A,B,C,D,E)   c-h*(f*A+g*D)*V-n*V*E-r*V;
fW=@(t,V,W,X,Y,Z,A,B,C,D,E)   h*(f*A+g*D)*V-r*W;
fX=@(t,V,W,X,Y,Z,A,B,C,D,E)   n*V*E-(r+u)*X;
fY=@(t,V,W,X,Y,Z,A,B,C,D,E)   a-d*Y*E-(j+q)*Y;
fZ=@(t,V,W,X,Y,Z,A,B,C,D,E)   d*Y*E-(k+q)*Z;
fA=@(t,V,W,X,Y,Z,A,B,C,D,E)   k*Z-(o+f)*A;
fB=@(t,V,W,X,Y,Z,A,B,C,D,E)   b-e*B*E-(i+s)*B;
fC=@(t,V,W,X,Y,Z,A,B,C,D,E)   e*B*E-(l+s)*C;
fD=@(t,V,W,X,Y,Z,A,B,C,D,E)   l*C-(p+g)*D;
fE=@(t,V,W,X,Y,Z,A,B,C,D,E)   m*W-t1*E;

t(1)=0;
V(1)=5420;W(1)=750;
X(1)=528;Y(1)=1050;
Z(1)=620;A(1)=k*Z(1);
B(1)=1250;C(1)=850;
D(1)=l*C(1);E(1)=1000;
tfinal=15;h=0.1;
N=ceil(tfinal/h);
%================================================================
for i=1:N
t(i+1)=t(i)+h;
k1V=fV(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1W=fW(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1X=fX(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1Y=fY(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1Z=fZ(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1A=fA(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1B=fB(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1C=fC(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
```

```
k1D=fD(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1E=fE(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));

k2V=fV(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2W=fW(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2X=fX(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2Y=fY(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2Z=fZ(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2A=fA(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2B=fB(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2C=fC(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2D=fD(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2E=fE(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);

k3V=fV( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3W=fW( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3X=fX( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3Y=fY( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3Z=fZ( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3A=fA( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3B=fB( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3C=fC( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3D=fD( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3E=fE( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);

k4V=fV( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4W=fW( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4X=fX( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4Y=fY( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4Z=fZ( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4A=fA( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4B=fB( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4C=fC( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4D=fD( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4E=fE( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);

V(i+1)=V(i)+(h/6)*(k1V+2*k2V+2*k3V+k4V);
W(i+1)=W(i)+(h/6)*(k1W+2*k2W+2*k3W+k4W);
X(i+1)=X(i)+(h/6)*(k1X+2*k2X+2*k3X+k4X);
Y(i+1)=Y(i)+(h/6)*(k1Y+2*k2Y+2*k3Y+k4Y);
Z(i+1)=Z(i)+(h/6)*(k1Z+2*k2Z+2*k3Z+k4Z);
A(i+1)=A(i)+(h/6)*(k1A+2*k2A+2*k3A+k4A);
B(i+1)=B(i)+(h/6)*(k1B+2*k2B+2*k3B+k4B);
C(i+1)=C(i)+(h/6)*(k1C+2*k2C+2*k3C+k4C);
D(i+1)=D(i)+(h/6)*(k1D+2*k2D+2*k3D+k4D);
E(i+1)=E(i)+(h/6)*(k1E+2*k2E+2*k3E+k4E);
```

```
end
%================================================
subplot(2,2,1)
plot(t,Z,'k--','linewidth',2)
hold on
subplot(2,2,2)
plot(t,C,'c--','linewidth',2)
hold on
subplot(2,2,3)
plot(t,W,'b--','linewidth',2)
hold on
subplot(2,2,4)
plot(t,X,'r--','linewidth',2)
hold on
b=750;c=5750;e=0.00625;g=0.023;h=0.093;i=0.213;f=0.012;j=0.252;
l=0.235;m=0.325;n=0.00523;p=0.325;r=0.0141;s=0.33;t1=10.42;
k=0.083*4;o=0.358;q=0.083*12;u=0.0952;o=delta;a=1450;d=0.01;
%================================================================
fV=@(t,V,W,X,Y,Z,A,B,C,D,E)   c-h*(f*A+g*D)*V-n*V*E-r*V;
fW=@(t,V,W,X,Y,Z,A,B,C,D,E)   h*(f*A+g*D)*V-r*W;
fX=@(t,V,W,X,Y,Z,A,B,C,D,E)   n*V*E-(r+u)*X;
fY=@(t,V,W,X,Y,Z,A,B,C,D,E)   a-d*Y*E-(j+q)*Y;
fZ=@(t,V,W,X,Y,Z,A,B,C,D,E)   d*Y*E-(k+q)*Z;
fA=@(t,V,W,X,Y,Z,A,B,C,D,E)   k*Z-(o+f)*A;
fB=@(t,V,W,X,Y,Z,A,B,C,D,E)   b-e*B*E-(i+s)*B;
fC=@(t,V,W,X,Y,Z,A,B,C,D,E)   e*B*E-(l+s)*C;
fD=@(t,V,W,X,Y,Z,A,B,C,D,E)   l*C-(p+g)*D;
fE=@(t,V,W,X,Y,Z,A,B,C,D,E)   m*W-t1*E;

t(1)=0;
V(1)=5420;W(1)=750;
X(1)=528;Y(1)=1050;
Z(1)=620;A(1)=k*Z(1);
B(1)=1250;C(1)=850;
D(1)=l*C(1);E(1)=1000;
tfinal=15;h=0.1;
N=ceil(tfinal/h);
%================================================================
for i=1:N
t(i+1)=t(i)+h;
k1V=fV(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1W=fW(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1X=fX(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1Y=fY(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1Z=fZ(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
```

```
k1A=fA(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1B=fB(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1C=fC(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1D=fD(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1E=fE(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));

k2V=fV(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2W=fW(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2X=fX(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2Y=fY(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2Z=fZ(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2A=fA(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2B=fB(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2C=fC(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2D=fD(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2E=fE(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);

k3V=fV( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3W=fW( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3X=fX( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3Y=fY( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3Z=fZ( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3A=fA( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3B=fB( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3C=fC( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3D=fD( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3E=fE( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);

k4V=fV( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4W=fW( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4X=fX( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4Y=fY( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4Z=fZ( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4A=fA( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4B=fB( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4C=fC( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4D=fD( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4E=fE( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);

V(i+1)=V(i)+(h/6)*(k1V+2*k2V+2*k3V+k4V);
W(i+1)=W(i)+(h/6)*(k1W+2*k2W+2*k3W+k4W);
X(i+1)=X(i)+(h/6)*(k1X+2*k2X+2*k3X+k4X);
Y(i+1)=Y(i)+(h/6)*(k1Y+2*k2Y+2*k3Y+k4Y);
Z(i+1)=Z(i)+(h/6)*(k1Z+2*k2Z+2*k3Z+k4Z);
A(i+1)=A(i)+(h/6)*(k1A+2*k2A+2*k3A+k4A);
B(i+1)=B(i)+(h/6)*(k1B+2*k2B+2*k3B+k4B);
```

```
C(i+1)=C(i)+(h/6)*(k1C+2*k2C+2*k3C+k4C);
D(i+1)=D(i)+(h/6)*(k1D+2*k2D+2*k3D+k4D);
E(i+1)=E(i)+(h/6)*(k1E+2*k2E+2*k3E+k4E);

end
%================================================
subplot(2,2,1)
plot(t,Z,'b','linewidth',2)
hold on
subplot(2,2,2)
plot(t,C,'k','linewidth',2)
hold on
subplot(2,2,3)
plot(t,W,'m','linewidth',2)
hold on
subplot(2,2,4)
plot(t,X,'c','linewidth',2)
hold on
b=750;c=3200;e=0.00625;g=0.023;h=0.093;i=0.213;f=0.012;j=0.252;
l=0.235;m=0.325;n=0.00523;p=0.325;r=0.0141;s=0.33;t1=10.42;
k=0.083*4;o=0.358;q=0.083*12;u=0.0952;o=delta;a=1450;d=0.01;
%============================================================
fV=@(t,V,W,X,Y,Z,A,B,C,D,E)   c-h*(f*A+g*D)*V-n*V*E-r*V;
fW=@(t,V,W,X,Y,Z,A,B,C,D,E)   h*(f*A+g*D)*V-r*W;
fX=@(t,V,W,X,Y,Z,A,B,C,D,E)   n*V*E-(r+u)*X;
fY=@(t,V,W,X,Y,Z,A,B,C,D,E)   a-d*Y*E-(j+q)*Y;
fZ=@(t,V,W,X,Y,Z,A,B,C,D,E)   d*Y*E-(k+q)*Z;
fA=@(t,V,W,X,Y,Z,A,B,C,D,E)   k*Z-(o+f)*A;
fB=@(t,V,W,X,Y,Z,A,B,C,D,E)   b-e*B*E-(i+s)*B;
fC=@(t,V,W,X,Y,Z,A,B,C,D,E)   e*B*E-(l+s)*C;
fD=@(t,V,W,X,Y,Z,A,B,C,D,E)   l*C-(p+g)*D;
fE=@(t,V,W,X,Y,Z,A,B,C,D,E)   m*W-t1*E;

t(1)=0;
V(1)=5420;W(1)=750;
X(1)=528;Y(1)=1050;
Z(1)=620;A(1)=k*Z(1);
B(1)=1250;C(1)=850;
D(1)=l*C(1);E(1)=1000;
tfinal=15;h=0.1;
N=ceil(tfinal/h);
%================================================================
for i=1:N
t(i+1)=t(i)+h;
k1V=fV(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1W=fW(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
```

```
k1X=fX(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1Y=fY(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1Z=fZ(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1A=fA(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1B=fB(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1C=fC(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1D=fD(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1E=fE(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));

k2V=fV(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2W=fW(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2X=fX(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2Y=fY(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2Z=fZ(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2A=fA(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2B=fB(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2C=fC(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2D=fD(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2E=fE(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);

k3V=fV( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3W=fW( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3X=fX( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3Y=fY( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3Z=fZ( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3A=fA( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3B=fB( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3C=fC( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3D=fD( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3E=fE( t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);

k4V=fV( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4W=fW( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4X=fX( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4Y=fY( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4Z=fZ( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4A=fA( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4B=fB( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4C=fC( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4D=fD( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4E=fE( t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);

V(i+1)=V(i)+(h/6)*(k1V+2*k2V+2*k3V+k4V);
W(i+1)=W(i)+(h/6)*(k1W+2*k2W+2*k3W+k4W);
X(i+1)=X(i)+(h/6)*(k1X+2*k2X+2*k3X+k4X);
Y(i+1)=Y(i)+(h/6)*(k1Y+2*k2Y+2*k3Y+k4Y);
```

```
Z(i+1)=Z(i)+(h/6)*(k1Z+2*k2Z+2*k3Z+k4Z);
A(i+1)=A(i)+(h/6)*(k1A+2*k2A+2*k3A+k4A);
B(i+1)=B(i)+(h/6)*(k1B+2*k2B+2*k3B+k4B);
C(i+1)=C(i)+(h/6)*(k1C+2*k2C+2*k3C+k4C);
D(i+1)=D(i)+(h/6)*(k1D+2*k2D+2*k3D+k4D);
E(i+1)=E(i)+(h/6)*(k1E+2*k2E+2*k3E+k4E);

end
%================================================
subplot(2,2,1)
plot(t,Z,'g--','linewidth',2)
hold on
subplot(2,2,2)
plot(t,C,'g--','linewidth',2)
hold on
subplot(2,2,3)
plot(t,W,'g--','linewidth',2)
hold on
subplot(2,2,4)
plot(t,X,'k--','linewidth',2)
xlabel('Time (years)')
ylabel('Infected Pig Population')
hold on
b=750;c=2247;e=0.00625;g=0.023;h=0.093;i=0.213;f=0.012;j=0.252;
l=0.235;m=0.325;n=0.00523;p=0.325;r=0.0141;s=0.33;t1=10.42;
k=0.083*4;o=0.358;q=0.083*12;u=0.0952;o=delta;a=1450;d=0.01;
%============================================================
fV=@(t,V,W,X,Y,Z,A,B,C,D,E)   c-h*(f*A+g*D)*V-n*V*E-r*V;
fW=@(t,V,W,X,Y,Z,A,B,C,D,E)   h*(f*A+g*D)*V-r*W;
fX=@(t,V,W,X,Y,Z,A,B,C,D,E)   n*V*E-(r+u)*X;
fY=@(t,V,W,X,Y,Z,A,B,C,D,E)   a-d*Y*E-(j+q)*Y;
fZ=@(t,V,W,X,Y,Z,A,B,C,D,E)   d*Y*E-(k+q)*Z;
fA=@(t,V,W,X,Y,Z,A,B,C,D,E)   k*Z-(o+f)*A;
fB=@(t,V,W,X,Y,Z,A,B,C,D,E)   b-e*B*E-(i+s)*B;
fC=@(t,V,W,X,Y,Z,A,B,C,D,E)   e*B*E-(l+s)*C;
fD=@(t,V,W,X,Y,Z,A,B,C,D,E)   l*C-(p+g)*D;
fE=@(t,V,W,X,Y,Z,A,B,C,D,E)   m*W-t1*E;

t(1)=0;
V(1)=5420;W(1)=750;
X(1)=528;Y(1)=1050;
Z(1)=620;A(1)=k*Z(1);
B(1)=1250;C(1)=850;
D(1)=l*C(1);E(1)=1000;
tfinal=15;h=0.1;
N=ceil(tfinal/h);
```

```
%=================================================================
for i=1:N
t(i+1)=t(i)+h;
k1V=fV(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1W=fW(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1X=fX(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1Y=fY(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1Z=fZ(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1A=fA(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1B=fB(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1C=fC(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1D=fD(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));
k1E=fE(t(i),V(i),W(i),X(i),Y(i),Z(i),A(i),B(i),C(i),D(i),E(i));

k2V=fV(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2W=fW(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2X=fX(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2Y=fY(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2Z=fZ(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2A=fA(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2B=fB(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2C=fC(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2D=fD(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);
k2E=fE(t(i)+h/2, V(i)+(h/2)*k1V, W(i)+(h/2)*k1W, ..., E(i)+(h/2)*k1E);

k3V=fV(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3W=fW(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3X=fX(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3Y=fY(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3Z=fZ(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3A=fA(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3B=fB(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3C=fC(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3D=fD(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);
k3E=fE(t(i)+h/2, V(i)+(h/2)*k2V, W(i)+(h/2)*k2W, ..., E(i)+(h/2)*k2E);

k4V=fV(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4W=fW(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4X=fX(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4Y=fY(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4Z=fZ(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4A=fA(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4B=fB(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4C=fC(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4D=fD(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
k4E=fE(t(i)+h, V(i)+(h)*k3V, W(i)+(h)*k3W, ..., E(i)+(h)*k3E);
```

```
V(i+1)=V(i)+(h/6)*(k1V+2*k2V+2*k3V+k4V);
W(i+1)=W(i)+(h/6)*(k1W+2*k2W+2*k3W+k4W);
X(i+1)=X(i)+(h/6)*(k1X+2*k2X+2*k3X+k4X);
Y(i+1)=Y(i)+(h/6)*(k1Y+2*k2Y+2*k3Y+k4Y);
Z(i+1)=Z(i)+(h/6)*(k1Z+2*k2Z+2*k3Z+k4Z);
A(i+1)=A(i)+(h/6)*(k1A+2*k2A+2*k3A+k4A);
B(i+1)=B(i)+(h/6)*(k1B+2*k2B+2*k3B+k4B);
C(i+1)=C(i)+(h/6)*(k1C+2*k2C+2*k3C+k4C);
D(i+1)=D(i)+(h/6)*(k1D+2*k2D+2*k3D+k4D);
E(i+1)=E(i)+(h/6)*(k1E+2*k2E+2*k3E+k4E);

end
%================================================
subplot(2,2,1)
plot(t,Z,'m','linewidth',2)
xlabel('Time (years)')
ylabel('Infected Pig Population')
title('(a)')
legend('\psi=25600','\psi=10200','\psi=5750','\psi=3200','\psi=2247')
hold on
subplot(2,2,2)
plot(t,C,'b','linewidth',2)
xlabel('Time (years)')
ylabel('Infected Cattle Population')
title('(b)')
legend('\psi=25600','\psi=10200','\psi=5750','\psi=3200','\psi=2247')
hold on
subplot(2,2,3)
plot(t,W,'r','linewidth',2)
xlabel('Time (years)')
ylabel('Humans with Taeniasis')
title('(c)')
legend('\psi=25600','\psi=10200','\psi=5750','\psi=3200','\psi=2247')
hold on
subplot(2,2,4)
plot(t,X,'m','linewidth',2)
xlabel('Time (years)')
ylabel('Humans with Cysticerosis')
legend('\psi=25600','\psi=10200','\psi=5750','\psi=3200','\psi=2247')
%================================================================
%================================================================
%THE SAME CODE CAN BE USED FOR VARYING OTHER MOST SENSITIVE PARAMETERS
%================================================
% IMPACT OF VARYING DEFECATION RATE (m=nu)
% Use values: m=0.615, 0.425, 0.325,0.223, 0.170
```

```
%=================================================
% IMPACT OF VARYING PROB. OF INFECTION(h=beta_T)
% Use values: h=0.343, 0.268, 0.192,0.125, 0.093
%=================================================
% IMPACT OF VARYING HUMAN DEATH RATE (r=mu_h)
% Use values: r=0.00141, 0.0980, 0.1350,0.2150, 0.2850
%=================================================
```

APPENDIX IV

```
%CTCM for Taeniasis and Cysticercosis in Humans, Pigs and Cattle
%By Joshua Mwasunda
%PhD Student (AMCS) - NM-AIST, Arusha, Tz
%04 June, 2021

clear all
close all
clc;
set(0,'DefaultAxesFontSize', 12)%Increases axes labels
set(gca,'fontsize',18);%Changes font size in a figure.
%-------------------------------------------------------------------------
%% Parameter values of the model
time=79; %time
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++%
lamb_C=120;lamb_P=150;lamb_H=200;

alpha=0.23;beta=0.093;mu_h=0.0141;gam=0.00625;
sig=0.235;mu_b=0.33;eta=0.0325;eps=0.125;nu=0.325;mu_e=10.42;
theta=0.00523;gam_p=0.01;delta=0.358;w=0.083*4;alpha_p=0.012;
rho=0.252;mu_d=0.0925;mu_p=0.083*12;
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++%
%% Euler's method for solving ODE
S_hum= lamb_H/mu_h;
S_pig= lamb_P/(rho+mu_p);
S_cat= lamb_C/(sig+mu_b);
%++++++++++++++++++++++++++++++++++%
dt=0.05; tim=time/dt; kk=0:dt:time;
%Initial conditions
a0=1; b0=0;c0=1; k0=1; h0=2;
m0=1;n0=1;
%-------------------------------------------------------------------------
x1(1)=a0; x2(1)=b0;
p1(1)=k0;
p2(1)=m0;
%p2(1)=w*k0;
%y2(1)=eta*c0;
y2(1)=n0;
y1(1)=c0;
z(1)=h0;
x(1)=S_hum-(a0+b0);
p(1)=S_pig-k0;
y(1)=S_cat-c0;
%-------------------------------------------------------------------------
for jj=1:tim
%Using Euler's formula i.e. y(n+1)=yn + h*f(xn,yn) where h=dt
```

```
%============================================================
% HUMAN POPULATION
x(jj+1)= x(jj)+ dt*(lamb_H-beta*(alpha_p*p2(jj)+alpha*y2(jj))*x(jj)-
theta*x(jj)*z(jj)-mu_h*x(jj));% Equation 1
x1(jj+1) = x1(jj) + dt*(beta*(alpha_p*p2(jj)+alpha*y2(jj))*x(jj)
-mu_h*x1(jj)); % Equation 2
x2(jj+1) = x2(jj) + dt*(theta*x(jj)*z(jj)-(mu_d+mu_h)*x2(jj)); % Eqn 3
%============================================================
%PIG POPULATION
p(jj+1)= p(jj)+dt*(lamb_P-gam_p*p(jj)*z(jj)-(rho+mu_p)*p(jj)); % Eqn 4
p1(jj+1)= p1(jj)+ dt*(gam_p*p(jj)*z(jj)-(w+mu_p)*p1(jj)); % Eqn 5
p2(jj+1)= p2(jj)+ dt*(w*p1(jj)-(delta+alpha_p)*p2(jj)); % Eqn 6
%============================================================
%CATTLE POPULATION
y(jj+1) = y(jj) + dt*(lamb_C-gam*y(jj)*z(jj)-(sig+mu_b)*y(jj)); % Eqn 7
y1(jj+1) = y1(jj) + dt*(gam*y(jj)*z(jj)-(eta+mu_b)*y1(jj)); % Eqon 8
y2(jj+1) = y2(jj) + dt*(eta*y1(jj)-(eps+alpha)*y2(jj)); % Eqn 9
%============================================================
%ENVIRONMENT
z(jj+1)= z(jj)+ dt*(nu*x1(jj)-mu_e*z(jj)); % Equation 10
%============================================================
end
%Three sample paths for CTMC (Gillespie's algorithm)
for l=1:3
clear t X X1 X2 P P1 P2 Y Y1 Y2 Z
j=1;
X1(1)=a0; X2(1)=b0; P1(1)=c0; P2(1)=m0;%P2(1)=w*c0;
Y1(1)=k0; Y2(1)=n0;%Y2(1)=eta*k0;
Z(1)=h0;
X(1)=S_hum-(a0+b0);Y(1)=S_cat-(c0);P=S_pig-(k0);
t(1)=0; %Starting at zero
%------------------------------------------------------------
while (X1(j)+P1(j)+P2(j)+Y1(j)+Y2(j)+Z(j))>0 && t(j)<time
% Stop if it hits zero or at end time
randm1=rand; %Uniform random number
randm2=rand; %Uniform random number
%------------------------------------------------------
H(j)=X(j)+X1(j)+X2(j);
Tp(j)=P(j)+P1(j)+P2(j);
Tc(j)=Y(j)+Y1(j)+Y2(j);
%-----------------------------------------------
%-----------------------------------------------
%Total sum of rates of all possible events at time (t+1)
Total=lamb_H+mu_h*H(j)+beta*(alpha_p*P2(j)+alpha*Y2(j))*X(j)+
theta*X(j)*Z(j)+mu_d*X2(j)+lamb_P+mu_p*Tp(j)+rho*P(j)+
w*P1(j)+(delta+alpha_p)*P2(j)+gam_p*P(j)*Z(j)+lamb_C+mu_b*Tc(j)+
```

```
sig*Y(j)+eta*Y1(j)+(eps+alpha)*Y2(j)+gam*Y(j)*Z(j)+nu*X1(j)+mu_e*Z(j);
%-------------------------------------------------------------
%Defining all possible events at time (t+1)
ev1 = lamb_H/Total;
ev2 = ev1 + beta*alpha_p*P2(j)*X(j)/Total;
ev3 = ev2 + beta*alpha*Y2(j)*X(j)/Total;
ev4 = ev3 + theta*Z(j)*X(j)/Total;
ev5 = ev4 + mu_h*X(j)/Total;
ev6 = ev5 + mu_h*X1(j)/Total;
ev7 = ev6 + mu_h*X2(j)/Total;
ev8 = ev7 + mu_d*X2(j)/Total;
%===========================
ev9 = ev8 + lamb_P/Total;
ev10 = ev9 + rho*P(j)/Total;
ev11 = ev10 + gam_p*Z(j)*P(j)/Total;
ev12 = ev11 + mu_p*P(j)/Total;
ev13= ev12 + w*P1(j)/Total;
ev14= ev13+ mu_p*P1(j)/Total;
ev15= ev14 + delta*P2(j)/Total;
ev16= ev15 + alpha_p*P2(j)/Total;
%===================================
ev17 = ev16 + lamb_C/Total;
ev18 = ev17 + sig*Y(j)/Total;
ev19 = ev18 + gam*Z(j)*Y(j)/Total;
ev20 = ev19 + mu_b*Y(j)/Total;
ev21= ev20 + eta*Y1(j)/Total;
ev22= ev21+ mu_b*Y1(j)/Total;
ev23= ev22 + eps*Y2(j)/Total;
ev24= ev23 + alpha*Y2(j)/Total;
%===================================
ev25 = ev24 + nu*X1(j)/Total;
ev26= ev25+ mu_e*Z(j)/Total;
%-------------------------------------------------
t(j+1)=t(j)-log(randm1)/Total; % Time to next event
%-------------------------------------------------
%Computing probabilities of each event occuring at time (t+1)
if randm2<ev1;             % Recruitment of SH
X(j+1)  = X(j)+1;
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
```

```
Z(j+1) = Z(j);
elseif randm2>=ev1 & randm2<ev2  % Inf. of SH Pork Consup
X(j+1)  = X(j)-1;
X1(j+1) = X1(j)+1;
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev2 & randm2<ev3  % Inf. of SH beef Consup
X(j+1)  = X(j)-1;
X1(j+1) = X1(j)+1;
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev3 & randm2<ev4     % Inf. of SH from Env.
X(j+1)  = X(j)-1;
X1(j+1) = X1(j);
X2(j+1) = X2(j)+1;
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev4 & randm2<ev5   % Death of SH
X(j+1)  = X(j)-1;
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev5 & randm2<ev6  % Death of IHT
```

```
X(j+1)  = X(j);
X1(j+1) = X1(j)-1;
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev6 & randm2<ev7     % Death of IHC
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j)-1;
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev7 & randm2<ev8  % Disease Ind. Death of IHC
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j)-1;
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev8 & randm2<ev9   %Recruitment of S_P
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j)+1;
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev9 & randm2<ev10    %Slaughter of S_P
X(j+1)  = X(j);
X1(j+1) = X1(j);
```

```
X2(j+1) = X2(j);
P(j+1)  = P(j)-1;
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev10 & randm2<ev11  %Infection of S_P
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j)-1;
P1(j+1) = P1(j)+1;
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev11 & randm2<ev12 %Death of S_P
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j)-1;
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev12 & randm2<ev13   %Slaughter of I_P
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j)-1;
P2(j+1) = P2(j)+1;
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev13 & randm2<ev14  %Death of I_P
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1)  = X2(j);
P(j+1) = P(j);
```

```
P1(j+1) = P1(j)-1;
P2(j+1) = P2(j);
Y(j+1) = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev14 & randm2<ev15 %Throwing Infected Pork
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j)-1;
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev15 & randm2<ev16 %Cons. Infected Pork
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j)-1;
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev16 & randm2<ev17  %Recruitment of S_C
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j)+1;
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev17 & randm2<ev18  %Slaughter of S_C
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
```

```
Y(j+1)  = Y(j)-1;
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev18 & randm2<ev19  %Infection of S_C
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j)-1;
Y1(j+1) = Y1(j)+1;
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev19 & randm2<ev20  %Death of S_C
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j)-1;
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev20 & randm2<ev21  %Slaughter of I_C
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j)-1;
Y2(j+1) = Y2(j)+1;
Z(j+1) = Z(j);
elseif randm2>=ev21 & randm2<ev22 %Death of  I_C
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j)-1;
```

```
Y2(j+1) = Y2(j);
Z(j+1) = Z(j);
elseif randm2>=ev22 & randm2<ev23  %Throwing Infected Beef
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j)-1;
Z(j+1) = Z(j);
elseif randm2>=ev23 & randm2<ev24  %Cons Infected Beef
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j)-1;
Z(j+1) = Z(j);
elseif randm2>=ev24 & randm2<ev25  % Shedding rate by I_{HT}
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j)+1;
else                        % Death of Taenia Eggs
X(j+1)  = X(j);
X1(j+1) = X1(j);
X2(j+1) = X2(j);
P(j+1)  = P(j);
P1(j+1) = P1(j);
P2(j+1) = P2(j);
Y(j+1)  = Y(j);
Y1(j+1) = Y1(j);
Y2(j+1) = Y2(j);
Z(j+1) = Z(j)-1;
```

```
end
j=j+1;
end
figure (1)
%subplot(2,3,6)
if l==1
plot(t,X,'r-','linewidth',1.5)
hold on
end
if l==2
plot(t,X,'b-','linewidth',1.5)
hold on
end
if l==3
plot(t,X,'g-','linewidth',1.5)
hold on
end
end
% %subplot(2,2,1)
plot(kk,x,'k--','LineWidth',2)
xlabel('Time (Years)','Fontsize',12)
ylabel('Susceptible Humans','Fontsize',12)
%axis([0,time,0,120])
%=================================================
%PLOT OTHER GRAPHS IN SIMILAR MANNER
%=================================================

%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%
%Approximation of Prob of Disease Extinction in Humans,
%Pigs and Cattle for CTMC Epidemic Model using 10000 Sample Paths
%By Joshua Mwasunda
%PhD Student (AMCS) - NM-AIST, Arusha, Tz
%08 July 2021

% *** DO WE NEED TO SET SEED of RANDOM NUMBER GENERATOR?
format short
clear all
close all
clc;
set(0,'DefaultAxesFontSize', 18)%Increases axes labels
set(gca,'fontsize',18);%Changes font size in a figure.
%-----------------------------------------------------------------------
%Parameter values of the model
%% Model Parameter values
time=79; %time
lamb_C=120;lamb_P=150;lamb_H=300;
```

```
alpha=0.23;beta=0.093;mu_h=0.0141;gam=0.00625;
sig=0.235;mu_b=0.33;eta=0.0325;eps=0.125;nu=0.325;mu_e=10.42;
theta=0.00523;gam_p=0.01;delta=0.358;w=0.083*4;alpha_p=0.012;
rho=0.252;mu_d=0.0925;mu_p=0.083*12;
%=================================================================
%% Euler's method for solving ODE
S_hum= lamb_H/mu_h;
S_pig= lamb_P/(rho+mu_p);
S_cat= lamb_C/(sig+mu_b);
%++++++++++++++++++++++++++++++++++%
dt=0.05; tim=time/dt; kk=0:dt:time;
%-----------------------------------------------------------------
%Initial Conditions
%a0=1; b0=0;c0=0; d0=0;k0=0;m0=0;h0=0;
a0=0; b0=1;c0=0; d0=0;k0=0;m0=0;h0=0;
%a0=1; b0=1;c0=0; d0=0;k0=0;m0=0;h0=0;
%a0=0; b0=0;c0=0; d0=0;k0=0;m0=0;h0=1;
%a0=0; b0=0;c0=0; d0=0;k0=1;m0=0;h0=0;
%a0=0; b0=0;c0=0; d0=0;k0=0;m0=1;h0=0;
%a0=0; b0=0;c0=0; d0=0;k0=1;m0=1;h0=0;
%a0=0; b0=0;c0=1; d0=0;k0=0;m0=0;h0=0;
%a0=0; b0=0;c0=0; d0=1;k0=0;m0=0;h0=0;
%a0=0; b0=0;c0=1; d0=1;k0=0;m0=0;h0=0;
%a0=1; b0=1;c0=1; d0=1;k0=0;m0=0;h0=0;
%a0=1; b0=1;c0=0; d0=0;k0=1;m0=1;h0=0;
%a0=1; b0=1;c0=0; d0=0;k0=0;m0=0;h0=1;
%a0=0; b0=0;c0=0; d0=0;k0=1;m0=1;h0=1;
%a0=0; b0=0;c0=1; d0=1;k0=0;m0=0;h0=1;
%a0=0; b0=0;c0=1; d0=1;k0=1;m0=1;h0=0;
%a0=0; b0=0;c0=1; d0=1;k0=1;m0=1;h0=1;
%a0=0; b0=0;c0=1; d0=1;k0=1;m0=1;h0=1;
%a0=1; b0=0;c0=1;d0=0; k0=1;m0=0; h0=1;
%=================================================================
%Three sample paths for CTMC (Gillespie's algorithm)
z=0;
ext=10000; % number of sample paths to be used

for k=1:ext
k;
clear t SH EH IH SC EC IC DC SB EB IB EV
j=1;
X1=a0; X2=b0; P1=c0; P2=d0; Y1=k0; Y2=m0; Z=h0;
X=S_hum-(a0+b0);Y=S_cat-k0;P=S_pig-c0;
t(1)=0; %Starting at zero
%-----------------------------------------------------------------
while (X1+P1+P2+Y1+Y2+Z)>0 && t<time
```

```
 % Stop if it hits zero or at end time
%X1+P1+P2+Y1+Y2+Z
randm1=rand; %Uniform random number
randm2=rand; %Uniform random number
%-------------------------------------------------------------
H=X+X1+X2;
Tp=P+P1+P2;
Tc=Y+Y1+Y2;
%-------------------------------------------------------------
%Total sum of rates of all possible events at time (t+1)
Total=lamb_H+mu_h*H+beta*(alpha_p*P2+alpha*Y2)*X+theta*X*Z+mu_d*X2+...
lamb_P+mu_p*Tp+rho*P+w*P1+(delta+alpha_p)*P2+gam_p*P*Z+...
lamb_C+mu_b*Tc+sig*Y+eta*Y1+(eps+alpha)*Y2+gam*Y*Z+...
+nu*X1+mu_e*Z;
%-------------------------------------------------------------
%Defining all possible events at time (t+1)
ev1 = lamb_H/Total;
ev2 = ev1 + beta*alpha_p*P2*X/Total;
ev3 = ev2 + beta*alpha*Y2*X/Total;
ev4 = ev3 + theta*Z*X/Total;
ev5 = ev4 + mu_h*X/Total;
ev6 = ev5 + mu_h*X1/Total;
ev7 = ev6 + mu_h*X2/Total;
ev8 = ev7 + mu_d*X2/Total;
%============================
ev9 = ev8 + lamb_P/Total;
ev10 = ev9 + rho*P/Total;
ev11 = ev10 + gam_p*Z*P/Total;
ev12 = ev11 + mu_p*P/Total;
ev13= ev12 + w*P1/Total;
ev14= ev13+ mu_p*P1/Total;
ev15= ev14 + delta*P2/Total;
ev16= ev15 + alpha_p*P2/Total;
%===================================
ev17 = ev16 + lamb_C/Total;
ev18 = ev17 + sig*Y/Total;
ev19 = ev18 + gam*Z*Y/Total;
ev20 = ev19 + mu_b*Y/Total;
ev21= ev20 + eta*Y1/Total;
ev22= ev21+ mu_b*Y1/Total;
ev23= ev22 + eps*Y2/Total;
ev24= ev23 + alpha*Y2/Total;
%===================================
ev25 = ev24 + nu*X1/Total;
ev26= ev25+ mu_e*Z/Total;
```

```
% ev31 and Total are the same thing, so just use ev31
%-------------------------------------------------------
t=t-log(randm1)/ev26; % Time to next event
%-------------------------------------------------------
randm2=randm2*ev26;
%Computing probabilities of each event occuring at time (t+1)
if randm2<ev1;            % Recruitment of SH
X = X+1;
X1 = X1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev1 & randm2<ev2  % Inf. of SH Pork Consup
X = X-1;
X1 = X1+1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev2 & randm2<ev3  % Inf. of SH beef Consup
X = X-1;
X1 = X1+1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev3 & randm2<ev4     % Inf. of SH from Env.
X = X-1;
X1 = X1;
X2 = X2+1;
P = P;
P1= P1;
P2 = P2;
```

```
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev4 & randm2<ev5   % Death of SH
X = X-1;
X1 = X1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev5 & randm2<ev6  % Death of IHT
X = X;
X1 = X1-1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev6 & randm2<ev7     % Death of IHC
X = X;
X1 = X1;
X2 = X2-1;
P = P;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev7 & randm2<ev8  % Disease Ind. Death of IHC
X = X;
X1 = X1;
X2 = X2-1;
P = P;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
```

```
Y2 = Y2;
Z = Z;
elseif randm2>=ev8 & randm2<ev9   %Recruitment of S_P
X = X;
X1 = X1;
X2 = X2;
P = P+1;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev9 & randm2<ev10    %Slaughter of  S_P
X = X;
X1 = X1;
X2 = X2;
P = P-1;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev10 & randm2<ev11  %Infection of  S_P
X = X;
X1 = X1;
X2 = X2;
P = P-1;
P1= P1+1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev11 & randm2<ev12 %Death of  S_P
X = X;
X1 = X1;
X2 = X2;
P = P-1;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
```

```
elseif randm2>=ev12 & randm2<ev13   %Slaughter of I_P
X = X;
X1 = X1;
X2 = X2;
P = P;
P1= P1-1;
P2 = P2+1;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev13 & randm2<ev14  %Death of I_P
X = X;
X1 = X1;
X2 = X2;
P = P;
P1= P1-1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev14 & randm2<ev15 %Throwing Infected Pork
X = X;
X1 = X1;
X2 = X2;
P = P;
P1= P1;
P2 = P2-1;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev15 & randm2<ev16 %Cons. Infected Pork
X = X;
X1 = X1;
X2 = X2;
P = P;
P1= P1;
P2 = P2-1;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev16 & randm2<ev17  %Recruitment of S_C
X = X;
```

```
X1 = X1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y+1;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev17 & randm2<ev18  %Slaughter of S_C
X = X;
X1 = X1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y-1;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev18 & randm2<ev19  %Infection of S_C
X = X;
X1 = X1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y-1;
Y1 = Y1+1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev19 & randm2<ev20  %Death of S_C
X = X;
X1 = X1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y-1;
Y1 = Y1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev20 & randm2<ev21  %Slaughter of I_C
X = X;
X1 = X1;
X2 = X2;
```

```
P = P;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1-1;
Y2 = Y2+1;
Z = Z;
elseif randm2>=ev21 & randm2<ev22 %Death of I_C
X = X;
X1 = X1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1-1;
Y2 = Y2;
Z = Z;
elseif randm2>=ev22 & randm2<ev23  %Throwing Infected Beef
X = X;
X1 = X1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2-1;
Z = Z;
elseif randm2>=ev23 & randm2<ev24  %Cons Infected Beef
X = X;
X1 = X1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2-1;
Z = Z;
elseif randm2>=ev24 & randm2<ev25  % Shedding rate by I_{HT}
X = X;
X1 = X1;
X2 = X2;
P = P;
P1= P1;
```

```
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z+1;
else                    % Death of Taenia Eggs
X = X;
X1 = X1;
X2 = X2;
P = P;
P1= P1;
P2 = P2;
Y = Y;
Y1 = Y1;
Y2 = Y2;
Z = Z-1;
end
end
if (X1+P1+P2+Y1+Y2+Z)==0
z=z+1;
else
z=z;
end

end
z;  %number of sample paths that hit zero out of 1000 sample paths
probext=z/ext  %approximate probability of extinction
%=================================================================
```

APPENDIX V

```
%FILE FOR RUNNING (File Name: Control_JAM)
%MATLAB code for the Optimal Control of T. saginata
%By Joshua Mwasunda
%PhD Student (AMCS) - NM-AIST, Arusha, Tz
%24 May 2021
clc;
clear;
close all;

format long
t0 = 0; tf=10; N=100;
time =linspace(t0,tf,N);
%INITIAL POPULATION VALUES
y0  = [2800,2500,340, 130, 250, 90, 83, 100];% initial conditions
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Constant=[2247 0.225 0.192 0.093 0.012 0.023 0.00523 0.0141 0.0925 ...
1450 0.213 0.01 0.252 0.083*12 0.317 0.1968 0.082 0.413 0.083*4 ...
0.125 0.105 0.358 750 0.213 0.00625 0.235 0.125 0.33 0.153 0.225...
0.150 10.42 0.213 0.183 0.115 0.248 2.8 70.2 0.05 2050 3000 1300];
%-------------------------------------------------------------------
%===================================================================
%Ah=Lambda_H; x1=chi; k=chi; bt=beta_T; ab=alpha_b; mh=mu_h;
%Ac=Lambda_C; rb=rho_b; gb=gamma_b; n1=eta; lb=lambda_b; mb=mu_b;
%nu=nu; delta1=delta;me=mu_e; w1=omega; e=epsilon; ss=sigma_s;
%sv=sigma_v; ps=psi_s; pv=psi_v; pb=pi; phs=phi_s; phv=phi_v;
%ap=alpha_p; o=theta; md=mu_d; Ap=Lambda_P; mp=mu_p; gp=gamma_p;
%kc=chi_c; pp=pi_p; rs =rho_s; sb=sigma_b; rv =rho_v; lp=lambda_p;
%rp =rho_p;
%===================================================================
Ah=Constant(1);k = Constant(2);kc= Constant(3);bt = Constant(4);
ap = Constant(5);ab = Constant(6);o = Constant(7);mh = Constant(8);
md =Constant(9);Ap= Constant(10); pp= Constant(11);gp = Constant(12);
rs = Constant(13);mp = Constant(14);phs=Constant(15);rb =Constant(16);
rv =Constant(17);phv =Constant(18);w1 =Constant(19);lp = Constant(20);
rp = Constant(21); delta1 =Constant(22);Ac  =Constant(23);
pb =Constant(24);gb = Constant(25);n1 = Constant(26);lb=Constant(27);
mb = Constant(28); sb=Constant(29);e =Constant(30);nu =Constant(31);
me =Constant(32); ss=Constant(33);sv =Constant(34);ps = Constant(35);
pv = Constant(36);C1= Constant(37);C2 = Constant(38);C3 =Constant(39);
A1=Constant(40);A2 =Constant(41);A3 =Constant(42);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
lf  = [0 0 0 0 0 0 0 0];
% TEST SECTION
init =y0;
init2 =lf;
```

```
h = (tf-t0)/N;
u = linspace(0,0,N+1);
u1=u'; u2=u'; u3=u';
U = [u1 u2 u3];
% IMPLIMENTATION OF THE ALGORITHM
%Test 1 stoping condition 1
delta = 0.001;
X=init;
i=0; %Initialize iteration counter
mm=size(X);
NumXX =10e10;
Xnew = rand(N+1,mm(2)).*(repmat(X,N+1,1));
DenXnew=norm(Xnew);
while  NumXX/DenXnew>delta
Xold =  Xnew;
oldu = U;
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%FORWARD RUNGE KUTTA  FOR STATES
[Tx, X]=rk4foward(@kims,t0, tf,N, init,U,Constant);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
% BACKWARD RUNGEKUTA FOR COSTATES
[Tp, P]=rk4back(@kims_costate,t0,tf,N,init2,U,X,Constant);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%UPDATE THE CONTROLS
f1 = X(1,:);g = X(2,:);r = X(3,:);s = X(4,:);
v= X(5,:);x = X(6,:);z = X(7,:);F = X(8,:);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
L1 = P(1,:); L2 = P(2,:); L3 = P(3,:); L4 = P(4,:); L5 = P(5,:);
L6 = P(6,:); L7 = P(7,:); L8 = P(8,:);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Implementation of all controls
% Case1:u1\neq 0, u2\neq 0, u3\neq 0, u4\neq 0,
%      u1 =max(0,min(1,(((L2-L1).*bt.*ab.*z.*f1)./A1)));
%      u2 =max(0,min(1,((r.*(L3-L4-C3))./A2)));
%      u3 =max(0,min(1,((g.*(L2-L1))./A3)));
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Cattle Vaccination and Treatment of Humans with Taeniasis
% Case2:u1\neq 0, u2\neq 0, u3\neq 0, u4\neq 0,
u1 =zeros(1,N+1);
u2 =max(0,min(1,((r.*(L3-L4-C3))./A2)));
u3 =max(0,min(1,((g.*(L2-L1))./A3)));
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Improved Beef Cooking and Cattle  Vaccination
% Case3:u1\neq 0, u2\neq 0, u3\neq 0, u4= 0,
%      u1 =max(0,min(1,(((L2-L1).*bt.*ab.*z.*f1)./A1)));
%      u2 =max(0,min(1,((r.*(L3-L4-C3))./A2)));
```

```
%      u3 =zeros(1,N+1);
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Improved Meat Cooking and Treatment of Humans with Taeniasis
% Case4:u1\neq 0, u2\neq 0, u3\neq 0, u4\neq 0,
%      u1 =max(0,min(1,(((L2-L1).*bt.*ab.*z.*f1)./A1)));
%      u2 =zeros(1,N+1);
%      u3 =max(0,min(1,((g.*(L2-L1))./A3)));
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Improved Meat Cooking
%      u1 =max(0,min(1,(((L2-L1).*bt.*ab.*z.*f1)./A1)));
%      u2 =zeros(1,N+1);
%      u3 =zeros(1,N+1);
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
% Treatment of Humans with Taeniasis
% Case6:u1\neq 0, u2= 0, u3= 0, u4= 0,
%      u1 =zeros(1,N+1);
%      u2 =zeros(1,N+1);
%      u3 =max(0,min(1,((g.*(L2-L1))./A3)));
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
% Cattle Vaccination Only
% Case7:u1= 0, u2= 0, u3= 0, u4\neq 0,
%      u1 =zeros(1,N+1);
%      u2 =max(0,min(1,((r.*(L3-L4-C3))./A2)));
%      u3 =zeros(1,N+1);
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Uu=[u1' u2' u3'];
U = 0.5*Uu + 0.5*oldu; % Convex combination of the controls
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Xnew =  X';
NumXX =abs(norm(Xnew-Xold));
DenXnew =norm(Xnew);
i=i+1; %Update iteration counter
end
% PLOTING
X=X';
Tx =Tx';
XX=X(:,1); YY=X(:,2); VV=X(:,3); ZZ=X(:,4); LL=X(:,5);
AA=X(:,6); BB=X(:,7); CC=X(:,8);
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Up = [0 0 0];
[T,Y] = ode45(@kims,time,y0,[],Up, Constant);
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Objective Function
J =sum((C1.*YY(1:end)+C2.*LL(1:end)+C3.*Uu(:,2).*VV(1:end))+...
((A1/2)*Uu(:,1).*Uu(:,1)+(A2/2)*Uu(:,2).*Uu(:,2)+...
(A3/2)*Uu(:,3).*Uu(:,3)));
```

```
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
S=[Tx,X];
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
figure(1)
subplot(2,2,1)
plot(Tx,X(:,2),'-b',T, Y(:,2),'--r','LineWidth',1.5);
ylabel('I_{HT}');
xlabel('Time (years)');
title('(a)')
legend('u_i\neq0, i=1,2,3','u_i=0, i=1,2,3')
%legend('u_i \neq,  0,i=2,3','u_i = 0,i=1,2,3')
%legend('u_i \neq,  0,i=1,2','u_i = 0,i=1,2,3')
%legend('u_i\neq0, i=1,3', 'u_i=0, i=1,2,3')
%legend('u_1\neq0','u_i=0, i=1,2,3')
%legend('u_3\neq0','u_i=0, i=1,2,3')
%legend('u_2 \neq','u_i = 0,i=1,2,3')
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
subplot(2,2,2)
plot(Tx,X(:,5),'-b',T, Y(:,5),'--r','LineWidth',1.5);
ylabel('I_{C}');
xlabel('Time (years)');
title('(b)')
legend('u_i\neq0, i=1,2,3','u_i=0, i=1,2,3')
%legend('u_i \neq,  0,i=2,3','u_i = 0,i=1,2,3')
%legend('u_i \neq,  0,i=1,2','u_i = 0,i=1,2,3')
%legend('u_i\neq0, i=1,3', 'u_i=0, i=1,2,3')
%legend('u_1\neq0','u_i=0, i=1,2,3')
%legend('u_3\neq0','u_i=0, i=1,2,3')
%legend('u_2 \neq','u_i = 0,i=1,2,3')
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
subplot(2,2,3)
plot(Tx,X(:,8),'-b',T, Y(:,8),'--r','LineWidth',1.5);
ylabel('E_{T}');
xlabel('Time (years)');
title('(e)')
legend('u_i\neq0, i=1,2,3','u_i=0, i=1,2,3')
%legend('u_i \neq,  0,i=2,3','u_i = 0,i=1,2,3')
%legend('u_i \neq,  0,i=1,2','u_i = 0,i=1,2,3')
%legend('u_i\neq0, i=1,3', 'u_i=0, i=1,2,3')
%legend('u_1\neq0','u_i=0, i=1,2,3')
%legend('u_3\neq0','u_i=0, i=1,2,3')
%legend('u_2 \neq','u_i = 0,i=1,2,3')
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
subplot(2,2,4)
plot(Tx,Uu(:,1),'-r',Tx,Uu(:,2),'-k',Tx,Uu(:,3),'m',...
Tx,2.0,'linewidth',1.5);
```

```
ylim([0 1])
xlim([0 10.5])
ylabel('Control Profile');
xlabel('Time (years)');
title('(f)')
legend('u_1 \neq  0','u_2 \neq  0','u_3 \neq  0')
%legend('u_1 =  0','u_2 \neq  0','u_3 \neq  0')
%legend('u_1 \neq  0','u_2 \neq  0','u_3 =  0')
%legend('u_1\neq0','u_2=0','u_3\neq0')
%legend('u_1\neq0','u_2=0','u_3= 0')
%legend('u_1=0','u_2=0','u_3\neq0')
%legend('u_1= 0','u_2  \neq 0','u_3 = 0')
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
% collect all the incidence terms in the ODE
X=XX'; % solution of the optimal control
U =[0 0 0]; % when no control
[Tx,Y] = ode45(@kims,time,y0,[],U, Constant);
Y=(Y);
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
% Infection Averted
Inew=sum(Y(:,2))-sum(X(:,2))+sum(Y(:,5))-sum(X(:,5))+...
sum(Y(:,8))-sum(X(:,8));
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Output=[J Inew]
%============================================================


%============================================================
% Defining function that solves the state system with 8 diff.eqns
%File Name: kims
%============================================================
function ydot = kims(t,yy,U,Constant)
f1=yy(1); g=yy(2); r=yy(3);s=yy(4);
v=yy(5);x=yy(6); z=yy(7);F=yy(8);
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Ah=Constant(1);k = Constant(2);kc= Constant(3);bt = Constant(4);
ap = Constant(5); ab = Constant(6);o = Constant(7);mh = Constant(8);
md =Constant(9);Ap= Constant(10); pp= Constant(11);gp = Constant(12);
rs = Constant(13);mp = Constant(14);phs=Constant(15);rb =Constant(16);
rv =Constant(17);phv =Constant(18);w1 =Constant(19);lp = Constant(20);
rp = Constant(21); delta1 =Constant(22);Ac  =Constant(23);
pb =Constant(24);gb = Constant(25); n1 = Constant(26);lb =Constant(27);
mb = Constant(28); sb=Constant(29);e =Constant(30); nu =Constant(31);
me =Constant(32); ss=Constant(33);sv =Constant(34);ps = Constant(35);
pv = Constant(36);C1= Constant(37); C2 = Constant(38);C3 =Constant(39);
A1=Constant(40);A2 =Constant(41);A3 =Constant(42);
```

```
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
u1 = U(1); u2=U(2); u3=U(3);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
ydot1=Ah+u3.*g-bt.*(1-u1).*ab.*z.*f1-mh.*f1;
ydot2=bt.*(1-u1).*ab.*z.*f1-(u3+mh).*g;
ydot3=Ac+pb.*x+pv.*s-gb.*r.*F-(ss+mb+u2).*r;
ydot4=u2.*r-rb.*gb.*s.*F-(sv+mb+pv).*s;
ydot5=gb.*r.*F+rb.*gb.*s.*F-(n1+lb+mb).*v;
ydot6=lb.*v-(sb+pb+mp).*x;
ydot7=n1.*v-(e+ab).*z;
ydot8=nu.*g-me.*F;
ydot  = [ydot1; ydot2; ydot3; ydot4; ydot5; ydot6; ydot7; ydot8];
%===============================================================

%===============================================================
%Defining function which solves co-state (adjoint) system with 8 Eqns
%File Name: kims_costate
%===============================================================
function ydot = kims_costate(t,y,U,X,Constant);
L1=y(1); L2=y(2); L3=y(3);L4=y(4);L5=y(5);L6=y(6);
L7=y(7);L8=y(8);
%===============================================================
Ah=Constant(1);k = Constant(2);kc= Constant(3);bt = Constant(4);
ap = Constant(5);ab = Constant(6);o = Constant(7);mh = Constant(8);
md =Constant(9);Ap= Constant(10);pp= Constant(11);gp = Constant(12);
rs = Constant(13);mp = Constant(14);phs=Constant(15); rb =Constant(16);
rv =Constant(17);phv =Constant(18);w1 =Constant(19);lp = Constant(20);
rp = Constant(21); delta1 =Constant(22);Ac  =Constant(23);
pb =Constant(24);gb = Constant(25);n1 = Constant(26);lb =Constant(27);
mb = Constant(28); sb=Constant(29);e =Constant(30); nu =Constant(31);
me =Constant(32); ss=Constant(33);sv =Constant(34);ps = Constant(35);
pv = Constant(36);C1= Constant(37); C2 = Constant(38);C3 =Constant(39);
A1=Constant(40);A2 =Constant(41);A3 =Constant(42);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
u1 = U(1); u2=U(2); u3=U(3);
% Variables
f1 = X(1,:);g = X(2,:);r = X(3,:);s = X(4,:);
v= X(5,:);x = X(6,:);z = X(7,:);F = X(8,:);

ydot1=bt.*(1-u1).*(L1-L2).*ab.*z+mh.*L1;% first ODE
ydot2=(u3+mh).*L2-C1-u3.*L1-nu.*L8; % second ODE
ydot3=gb.*(L3-L5).*F+(ss+mb+u3).*L3-u2.*L4-u2.*C3;
ydot4=gb.*rb.*(L4-L5).*F+(sv+mb+pv).*L4-pv.*L3;
ydot5=(n1+lb+mb).*L5-lb.*L6-n1.*L7-C2;
ydot6=(sb+pb+mb).*L6-pb.*L3;
ydot7=bt.*(1-u1).*(L1-L2).*ab.*f1+(e+ab).*L7;
```

```
ydot8=gb.*(L3-L5).*r+rb.*gb.*(L4-L5).*s+me.*L8;
ydot  = [ydot1; ydot2; ydot3; ydot4; ydot5; ydot6; ydot7; ydot8];
%=================================================================

%=================================================================
%Forward Runge-Kutta for States System
%File Name:rk4foward
%=================================================================
function [t, w]=rk4foward(f,t0, tf, N, init,U,Constant)
h      = (tf-t0)/N;         %the step size
t(1)   = t0;
w(:,1) = init;             %initial conditions
for i = 1:N
uu = U(i,:);
k1 = h*f(t(i),w(:,i),uu,Constant);
k2 = h*f(t(i)+h./2, w(:,i)+0.5*k1,uu,Constant);
k3 = h*f(t(i)+h./2, w(:,i)+0.5*k2,uu,Constant);
k4 = h*f(t(i)+h, w(:,i)+k3,uu,Constant);
w(:,i+1) = w(:,i) + (k1 + 2*k2 + 2*k3 + k4)/6;
t(i+1)   = t0 + i*h;
end
%=================================================================

%=================================================================
%Backward Runge-Kutta for States System
%File Name:rk4back
%=================================================================
function [t, w]=rk4back(f,t0, tf, N, init2,U,YY,Constant)
h = -(tf-t0)/N;     %the step size
t(N) = tf;
w(:,N) = init2;     %initial conditions
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
for i = N:-1:1
uu = U(i,:);
yy = YY(:,i);
k1 = h*f(t(i),w(:,i),uu,yy,Constant);
k2 = h*f(t(i)+h/2, w(:,i)+0.5*k1,uu,yy,Constant);
k3 = h*f(t(i)+h/2, w(:,i)+0.5*k2,uu,yy,Constant );
k4 = h*f(t(i)+h, w(:,i)+k3,uu,yy,Constant);
if i>1
w(:,i-1) = w(:,i) + (k1 + 2*k2 + 2*k3 + k4)/6;
t(i-1) = tf + i*h;
else
ww = w(:,i) + (k1 + 2*k2 + 2*k3 + k4)/6;
tt = tf + i*h;
end
```

```
end
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
w = [ww w];
t = [tt t];
%=============================================================

%FILE FOR RUNNING (File Name: Control_JAM)
%MATLAB code for the Optimal Control of T. saginata and T. Solium
%By Joshua Mwasunda
%PhD Student (AMCS) - NM-AIST, Arusha, Tz
%24 June 2021
clc
clear all
close all
format long
t0 = 0; tf=10; N=100;
time =linspace(t0,tf,N);
%INITIAL POPULATION VALUES
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
y0  = [2800,2500,550,250,140,220,100,52,340,130,250,90,83,100];
% initial condition for STATE SYSTEM
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Constant=[2247 0.225 0.192 0.093 0.012 0.023 0.00523 0.0141 0.0925 ...
1450 0.213 0.01 0.252 0.083*12 0.317 0.1968 0.082 0.413 ...
0.083*4 0.125 0.105 0.358 750 0.213 0.00625 0.235 0.125 0.33 ...
0.153 0.225 0.150 10.42 0.213 0.183 0.115 0.248 0.8 1.2 25 40.5 ...
0.0075 0.05 500 50 450 100 3000];
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Ah=Constant(1);k = Constant(2);kc= Constant(3);bt = Constant(4);
ap = Constant(5);ab = Constant(6);o = Constant(7);mh = Constant(8);
md =Constant(9);Ap=Constant(10);pp= Constant(11);gp=Constant(12);
rs = Constant(13);mp = Constant(14);phs=Constant(15);rb=Constant(16);
rv=Constant(17);phv=Constant(18);w1=Constant(19);lp = Constant(20);
rp=Constant(21);delta1=Constant(22);Ac=Constant(23);pb=Constant(24);
gb = Constant(25);n1 = Constant(26);lb =Constant(27);mb=Constant(28);
sb=Constant(29);e =Constant(30);nu =Constant(31);me =Constant(32);
ss=Constant(33);sv =Constant(34);ps = Constant(35);pv = Constant(36);
C1= Constant(37); C2 = Constant(38);C3 =Constant(39);C4 =Constant(40);
C5 =Constant(41);C6 =Constant(42);A1=Constant(43);A2 =Constant(44);
A3 =Constant(45);A4 =Constant(46);A5 =Constant(47);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
lf  = [0 0 0 0 0 0 0 0 0 0 0 0 0 0];
%% TEST SECTION
init =y0;
init2 =lf;
h = (tf-t0)/N;
```

```
u = linspace(0,0,N+1);
u1=u'; u2=u'; u3=u'; u4=u'; u5=u';
U = [u1 u2 u3 u4 u5];
%% IMPLIMENTATION OF THE ALGORITHM
%Test 1 stoping condition 1
delta = 0.001;
X=init;
i=0; %Initialize iteration counter
mm=size(X);
NumXX =10e10;
Xnew = rand(N+1,mm(2)).*(repmat(X,N+1,1));
DenXnew=norm(Xnew);
while  NumXX/DenXnew>delta
Xold =  Xnew;
oldu = U;
%++++++++++++++++++++++++++++++++++++++++++++++++++
%FORWARD RUNGE KUTTA  FOR STATES
[Tx, X]=rk4foward(@kims,t0, tf,N, init,U,Constant);
%++++++++++++++++++++++++++++++++++++++++++++++++++
% BACKWARD RUNGEKUTA FOR COSTATES
[Tp, P]=rk4back(@kims_costate,t0,tf,N,init2,U,X,Constant);
%++++++++++++++++++++++++++++++++++++++++++++++++++
%UPDATE THE CONTROLS
f1 = X(1,:);g = X(2,:);h = X(3,:);j = X(4,:);l = X(5,:);
m = X(6,:);p = X(7,:);q = X(8,:);r = X(9,:);s = X(10,:);
v= X(11,:);x = X(12,:);z = X(13,:);F = X(14,:);
%++++++++++++++++++++++++++++++++++++++++++++++++++
L1 = P(1,:); L2 = P(2,:); L3 = P(3,:); L4 = P(4,:); L5 = P(5,:);
L6 = P(6,:); L7 = P(7,:); L8 = P(8,:); L9 = P(9,:); L10 = P(10,:);
L11 = P(11,:); L12 = P(12,:); L13 = P(13,:); L14 = P(14,:);

%++++++++++++++++++++++++++++++++++++++++++++++++++
%Implementation of all controls
% Case1:u1\neq 0, u2\neq 0, u3\neq 0, u4\neq 0,
u1 =max(0,min(1,(((L2-L1).*bt.*(ap.*q+ab.*z).*f1+L8.*q+L13.*z)./A1)));
u2 =max(0,min(1,((j.*(L4-L5-C5))./A2)));
u3 =max(0,min(1,((r.*(L9-L10-C6))./A3)));
u4 =max(0,min(1,((g.*(L2-L1))./A4)));
u5 =max(0,min(1,(((L3-L1).*o.*f1.*F+(L6-L4).*gp.*j.*F+...
(L6-L5).*rb.*gp.*l.*F+(L11-L9).*gb.*r.*F+(L11-L10).*rb.*gb.*s.*F+...
nu.*g.*L14)./A5)));
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Combination of Improved Hygiene and Sanitation, Vaccination of Pigs
%and Cattle, Treatment of Humans with Taeniasis
% Case2:u1\neq 0, u2\neq 0, u3\neq 0, u4\neq 0,
%  u1 =zeros(1,N+1);
```

```
%  u2 =max(0,min(1,((j.*(L4-L5-C5))./A2)));
%  u3 =max(0,min(1,((r.*(L9-L10-C6))./A3)));
%  u4 =max(0,min(1,((g.*(L2-L1))./A4)));
%  u5=max(0,min(1,(((L3-L1).*o.*f1.*F+(L6-L4).*gp.*j.*F+...
%(L6-L5).*rb.*gp.*l.*F+(L11-L9).*gb.*r.*F+(L11-L10).*rb.*gb.*s.*F+...
%nu.*g.*L14)./A5)));
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Combination of Meat Inspection, Improved Hygiene and Sanitation,
%and Vaccination of Pigs and Cattle
% Case3:u1\neq 0, u2\neq 0, u3\neq 0, u4= 0,
% u1 =max(0,min(1,(((L2-L1).*bt.*(ap.*q+ab.*z).*f1+L8.*q+L13.*z)./A1)));
% u2 =max(0,min(1,((j.*(L4-L5-C5))./A2)));
% u3 =max(0,min(1,((r.*(L9-L10-C6))./A3)));
% u4 =zeros(1,N+1);
%  u5=max(0,min(1,(((L3-L1).*o.*f1.*F+(L6-L4).*gp.*j.*F+...
%(L6-L5).*rb.*gp.*l.*F+(L11-L9).*gb.*r.*F+(L11-L10).*rb.*gb.*s.*F+...
%nu.*g.*L14)./A5)));
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Meat Inspection, Vaccination of Pigs and Cattle, Treatment of
%Humans with Taeniasis
% Case4:u1\neq 0, u2\neq 0, u3\neq 0, u4\neq 0,
% u1 =max(0,min(1,(((L2-L1).*bt.*(ap.*q+ab.*z).*f1+L8.*q+L13.*z)./A1)));
% u2 =max(0,min(1,((j.*(L4-L5-C5))./A2)));
% u3 =max(0,min(1,((r.*(L9-L10-C6))./A3)));
% u4 =max(0,min(1,((g.*(L2-L1))./A4)));
% u5 =zeros(1,N+1);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Combination of Meat Inspection, Improved Hygiene and Sanitation,
%and Treatment of Humans with Taeniasis
% Case5:u1\neq 0, u2= 0, u3= 0, u4 \neq 0,
% u1 =max(0,min(1,(((L2-L1).*bt.*(ap.*q+ab.*z).*f1+L8.*q+L13.*z)./A1)));
% u2 =zeros(1,N+1);
% u3 =zeros(1,N+1);
% u4 =max(0,min(1,((g.*(L2-L1))./A4)));
% u5 =max(0,min(1,(((L3-L1).*o.*f1.*F+(L6-L4).*gp.*j.*F+...
%(L6-L5).*rb.*gp.*l.*F+(L11-L9).*gb.*r.*F+(L11-L10).*rb.*gb.*s.*F+...
%nu.*g.*L14)./A5)));
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Combination of Meat Inspection and Vaccination of Pigs and Cattle
% Case6:u1\neq 0, u2= 0, u3= 0, u4= 0,
% u1 =max(0,min(1,(((L2-L1).*bt.*(ap.*q+ab.*z).*f1+L8.*q+L13.*z)./A1)));
% u2 =max(0,min(1,((j.*(L4-L5-C5))./A2)));
% u3 =max(0,min(1,((r.*(L9-L10-C6))./A3)));
% u4 =zeros(1,N+1);
% u5 =zeros(1,N+1);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
```

```
%Combination of Improved Hygiene and Sanitation \& Pigs and
% Cattle Vaccination
% Case7:u1= 0, u2= 0, u3= 0, u4\neq 0,
% u1 =zeros(1,N+1);
% u2 =max(0,min(1,((j.*(L4-L5-C5))./A2)));
% u3 =max(0,min(1,((r.*(L9-L10-C6))./A3)));
% u4 =zeros(1,N+1);
% u5 =max(0,min(1,(((L3-L1).*o.*f1.*F+(L6-L4).*gp.*j.*F+...
%(L6-L5).*rb.*gp.*l.*F+(L11-L9).*gb.*r.*F+(L11-L10).*rb.*gb.*s.*F+...
%nu.*g.*L14)./A5)));
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
% Vaccination of Pigs and Cattle, Treatment of Humans with Taeniasis
% Case8:u1\neq 0, u2\neq 0, u3\neq 0, u4\neq 0,
%      u1 =zeros(1,N+1);
%      u2 =max(0,min(1,((j.*(L4-L5-C5))./A2)));
%      u3 =max(0,min(1,((r.*(L9-L10-C6))./A3)));
%      u4 =max(0,min(1,((g.*(L2-L1))./A4)));
%      u5 =zeros(1,N+1);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Combination of Meat Inspection, Improved Hygiene and Sanitation
% Case9:u1= 0, u2\neq 0, u3\neq 0, u4\neq 0,
%u1 =max(0,min(1,(((L2-L1).*bt.*(ap.*q+ab.*z).*f1+L8.*q+L13.*z)./A1)));
% u2 =zeros(1,N+1);
% u3 =zeros(1,N+1);
%u4 =zeros(1,N+1);
% u5 =max(0,min(1,(((L3-L1).*o.*f1.*F+(L6-L4).*gp.*j.*F+...
%(L6-L5).*rb.*gp.*l.*F+(L11-L9).*gb.*r.*F+(L11-L10).*rb.*gb.*s.*F+...
%nu.*g.*L14)./A5)));
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Combination of Meat Inspection and Treatment of Humans with Taeniasis
% Case10:u1= 0, u2\neq 0, u3\neq 0, u4= 0,
% u1 =max(0,min(1,(((L2-L1).*bt.*(ap.*q+ab.*z).*f1+L8.*q+L13.*z)./A1)));
% u2 =zeros(1,N+1);
% u3 =zeros(1,N+1);
% u4 =max(0,min(1,((g.*(L2-L1))./A4)));
% u5 =zeros(1,N+1);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Combination of Improved Hygiene and Sanitation \& Treatment of
%Humans with Taeniasis
% Case11:u1= 0, u2= 0, u3= 0, u4\neq 0,
% u1 =zeros(1,N+1);
% u2 =zeros(1,N+1);
% u3 =zeros(1,N+1);
%u4 =max(0,min(1,((g.*(L2-L1))./A4)));
% u5 =max(0,min(1,(((L3-L1).*o.*f1.*F+(L6-L4).*gp.*j.*F+...
%(L6-L5).*rb.*gp.*l.*F+(L11-L9).*gb.*r.*F+(L11-L10).*rb.*gb.*s.*F+...
```

```
%nu.*g.*L14)./A5)));
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Implementation of Pigs and Cattle Vaccination only
% Case12:u1\neq 0, u2\neq 0, u3\neq 0, u4\neq 0,
%      u1 =zeros(1,N+1);
%      u2 =max(0,min(1,((j.*(L4-L5-C5))./A2)));
%      u3 =max(0,min(1,((r.*(L9-L10-C6))./A3)));
%      u4 =zeros(1,N+1);
%      u5 =zeros(1,N+1);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Uu=[u1' u2' u3' u4' u5'];
U = 0.5*Uu + 0.5*oldu; % Convex combination of the controls
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Xnew =  X';
NumXX =abs(norm(Xnew-Xold));
DenXnew =norm(Xnew);
i=i+1; %Update iteration counter
end
%% PLOTING
X=X';
Tx =Tx';
XX=X(:,1); YY=X(:,2); VV=X(:,3); ZZ=X(:,4); LL=X(:,5);
AA=X(:,6); BB=X(:,7); CC=X(:,8); DD=X(:,9); EE=X(:,10);
FF=X(:,11); GG=X(:,12); HH=X(:,13); JJ=X(:,14);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Up = [0 0 0 0 0];
[T,Y] = ode45(@kims,time,y0,[],Up, Constant);
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
J =sum((C1.*YY(1:end)+C2.*VV(1:end)+C3.*AA(1:end)+C4.*FF(1:end)+...
C5.*Uu(:,2).*ZZ(1:end)+C6.*Uu(:,3).*DD(1:end))+...
((A1/2)*Uu(:,1).*Uu(:,1)+(A2/2)*Uu(:,2).*Uu(:,2)+...
(A3/2)*Uu(:,3).*Uu(:,3)+(A4/2)*Uu(:,4).*Uu(:,4)+...
(A5/2)*Uu(:,5).*Uu(:,5))); %Change to the suitable objective function
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
S=[Tx,X];
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
figure(1)
subplot(2,3,1)
plot(Tx,X(:,2),'-c',T, Y(:,2),'--r','LineWidth',1.5);
%ylim([0 700])
ylabel('I_{HT}');
xlabel('Time (years)');
title('(a)')
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
subplot(2,3,2)
plot(Tx,X(:,3),'-c',T, Y(:,3),'--r','LineWidth',1.5);
```

```
%%ylim([0 700])
ylabel('I_{HC}');
xlabel('Time (years)');
title('(b)')
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
subplot(2,3,3)
plot(Tx,X(:,6),'-c',T, Y(:,6),'--r','LineWidth',1.5);
%ylim([250 800])
ylabel('I_{P}');
xlabel('Time (years)');
title('(c)')
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
subplot(2,3,4)
plot(Tx,X(:,11),'-c',T, Y(:,11),'--r','LineWidth',1.5);
%ylim([250 800])
ylabel('I_{C}');
xlabel('Time (years)');
title('(d)')
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
subplot(2,3,5)
plot(Tx,X(:,14),'-c',T, Y(:,14),'--r','LineWidth',1.5);
%ylim([250 800])
ylabel('E_{T}');
xlabel('Time (years)');
title('(e)')
%legend('u_i\neq0, i=1,2,3,4,5','u_i=0, i=1,2,3,4,5')
%legend('u_i \neq,  0,i=2,3,4,5 ','u_i = 0,i=1,2,3,4,5 ')
%legend('u_i \neq,  0,i=1,2,3,4 ','u_i = 0,i=1,2,3,4,5 ')
%legend('u_i\neq0, i=1,2,3,4', 'u_i=0, i=1,2,3,4,5')
%legend('u_i\neq0, i=1,4,5','u_i=0, i=1,2,3,4,5')
%legend('u_i\neq0, i=1,2,3','u_i=0, i=1,2,3,4,5')
%legend('u_i \neq  0,i=2,3,5 ','u_i = 0,i=1,2,3,4,5 ')
%legend('u_i\neq0, i=2,3,4','u_i=0, i=1,2,3,4,5')
%legend('u_i\neq0, i=1,5','u_i=0, i=1,2,3,4,5')
%legend('u_i\neq0, i=1,4','u_i=0, i=1,2,3,4,5')
%legend('u_i\neq0, i=4,5','u_i=0, i=1,2,3,4,5')
legend('u_i\neq0, i=2,3','u_i=0, i=1,2,3,4,5 ')
%legend('u_1 \neq  0','u_i = 0,i=1,2,3,4,5 ')
%legend('u_4 \neq  0','u_i = 0,i=1,2,3,4,5 ')
%legend('u_5 \neq  0','u_i = 0,i=1,2,3,4,5 ')
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
subplot(2,3,6)
plot(Tx,Uu(:,1),'-b',Tx,Uu(:,2),'-k',Tx,Uu(:,3),'-g',...
Tx,Uu(:,4),'-r',Tx,Uu(:,5),'-m',Tx,2.0,'linewidth',1.5);
ylim([0 1])
xlim([0 10.5])
```

```
ylabel('Control Profile');
xlabel('Time (years)');
title('(f)')
%legend('u_1\neq 0','u_2 \neq0','u_3\neq0','u_4\neq0','u_5\neq 0')
%legend('u_1 =  0','u_2\neq0','u_3\neq 0','u_4\neq 0','u_5\neq 0')
%legend('u_1 \neq  0','u_2 \neq  0','u_3\neq 0','u_4\neq0','u_5= 0')
%legend('u_1\neq0','u_2\neq0','u_3\neq0','u_4\neq0','u_5=0')
%legend('u_1\neq0','u_2=0','u_3= 0','u_4\neq0','u_5\neq0')
%legend('u_1\neq0','u_2\neq0','u_3\neq0','u_4=0','u_5=0')
%legend('u_1 = 0','u_2\neq0','u_3\neq0','u_4 = 0','u_5\neq 0')
%legend('u_1=0','u_2\neq0','u_3\neq0','u_4\neq0','u_5=0')
%legend('u_1\neq0','u_2=0','u_3=0','u_4=0','u_5\neq0')
%legend('u_1\neq0','u_2=0','u_3=0','u_4\neq0','u_5=0')
%legend('u_1=0','u_2=0','u_3=0','u_4\neq0','u_5\neq0')
legend('u_1=0','u_2\neq0','u_3\neq0','u_4=0','u_5=0')
%legend('u_1 \neq 0','u_2 = 0','u_3 = 0','u_4 = 0','u_5 = 0')
%legend('u_1 = 0','u_2 = 0','u_3= 0','u_4 \neq  0','u_5 = 0')
%legend('u_1 = 0','u_2 = 0','u_3= 0','u_4 =  0','u_5 \neq 0')
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
% collect all the incidence terms in the ODE
X=XX'; % solution of the optimal control
U =[0 0 0 0 0]; % when no control
[Tx,Y] = ode45(@kims,time,y0,[],U, Constant);
Y=(Y);
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%Inew=sum(Y(:,2))-sum(X(:,2))+sum(Y(:,3))-sum(X(:,3)) %averted
Inew=sum(Y(:,2))-sum(X(:,2))+sum(Y(:,3))-sum(X(:,3))+...
sum(Y(:,6))-sum(X(:,6))+sum(Y(:,11))-sum(X(:,11));% averted
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Output=[J Inew]S
%============================================================

%============================================================
% Defining function that solves the state system with 14 diff.eqns
%File Name: kims
%============================================================
function ydot = kims(t,yy,U,Constant)
%This function solves MU infection on human type three equations
f1=yy(1); g=yy(2); h1=yy(3); j=yy(4);l=yy(5);
m=yy(6); p=yy(7);q=yy(8); r=yy(9);s=yy(10);
v=yy(11);x=yy(12); z=yy(13);F=yy(14);

%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Ah=Constant(1);k = Constant(2);kc= Constant(3);bt = Constant(4);
ap = Constant(5);ab = Constant(6);o = Constant(7);mh = Constant(8);
md =Constant(9);Ap=Constant(10);pp= Constant(11);gp=Constant(12);
```

```
rs = Constant(13);mp = Constant(14);phs=Constant(15);rb=Constant(16);
rv=Constant(17);phv=Constant(18);w1=Constant(19);lp = Constant(20);
rp=Constant(21);delta1=Constant(22);Ac=Constant(23);pb=Constant(24);
gb = Constant(25);n1 = Constant(26);lb =Constant(27);mb=Constant(28);
sb=Constant(29);e =Constant(30);nu =Constant(31);me =Constant(32);
ss=Constant(33);sv =Constant(34);ps = Constant(35);pv = Constant(36);
C1= Constant(37); C2 = Constant(38);C3 =Constant(39);C4 =Constant(40);
C5 =Constant(41);C6 =Constant(42);A1=Constant(43);A2 =Constant(44);
A3 =Constant(45);A4 =Constant(46);A5 =Constant(47);
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++

u1 = U(1); u2=U(2); u3=U(3); u4 =U(4); u5 =U(5);

ydot1=Ah+u4.*g+kc.*h1-bt.*(1-u1).*(ap.*q+ab.*z).*f1-...
o.*(1-u5).*f1.*F-mh.*f1;
ydot2=bt.*(1-u1).*(ap.*q+ab.*z).*f1-(u4+mh).*g;
ydot3=o.*(1-u5).*f1.*F-(mh+md+kc).*h1;
ydot4=Ap+pp.*p+phv.*l-gp.*(1-u5).*j.*F-(rs+mp+u2).*j;
ydot5=u2.*j-rb.*gp.*(1-u5).*l.*F-(rv+mp+phv).*l;
ydot6=gp.*(1-u5).*j.*F+rb.*gp.*(1-u5).*l.*F-(w1+lp+mp).*m;
ydot7=lp.*m-(rp+pp+mp).*p;
ydot8=w1.*m-(delta1+ap).*q;
ydot9=Ac+pb.*x+pv.*s-gb.*r.*(1-u5).*F-(ss+mb+u3).*r;
ydot10=u3.*r-rb.*gb.*(1-u5).*s.*F-(sv+mb+pv).*s;
ydot11=gb.*(1-u5).*r.*F+rb.*gb.*(1-u5).*s.*F-(n1+lb+mb).*v;
ydot12=lb.*v-(sb+pb+mp).*x;
ydot13=n1.*v-(e+ab).*z;
ydot14=nu.*(1-u5).*g-me.*F;
ydot= [ydot1;ydot2;ydot3;ydot4;ydot5;ydot6;ydot7;ydot8;ydot9;...
ydot10;ydot11;ydot12;ydot13;ydot14];

%====================================================================
%Defining function which solves co-state (adjoint) system with 14 Eqns
%File Name: kims_costate
%====================================================================
function ydot = kims_costate(t,y,U,X,Constant);
%This function solves a MU infection on human type three equations
L1=y(1); L2=y(2); L3=y(3);L4=y(4);L5=y(5);L6=y(6);
L7=y(7);L8=y(8);L9=y(9);L10=y(10);L11=y(11);
L12=y(12);L13=y(13);L14=y(14);

%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
Ah=Constant(1);k = Constant(2);kc= Constant(3);bt = Constant(4);
ap = Constant(5);ab = Constant(6);o = Constant(7);mh = Constant(8);
md =Constant(9);Ap=Constant(10);pp= Constant(11);gp=Constant(12);
rs = Constant(13);mp = Constant(14);phs=Constant(15);rb=Constant(16);
```

```
rv=Constant(17);phv=Constant(18);w1=Constant(19);lp = Constant(20);
rp=Constant(21);delta1=Constant(22);Ac=Constant(23);pb=Constant(24);
gb = Constant(25);n1 = Constant(26);lb =Constant(27);mb=Constant(28);
sb=Constant(29);e =Constant(30);nu =Constant(31);me =Constant(32);
ss=Constant(33);sv =Constant(34);ps = Constant(35);pv = Constant(36);
C1= Constant(37); C2 = Constant(38);C3 =Constant(39);C4 =Constant(40);
C5 =Constant(41);C6 =Constant(42);A1=Constant(43);A2 =Constant(44);
A3 =Constant(45);A4 =Constant(46);A5 =Constant(47);
%++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
%list your suitable parameters
u1 = U(1); u2=U(2); u3=U(3); u4=U(4); u5=U(5);
% variables x=S_c,y=I_c,v=S_s,z=I_s, S_h, I_h, R_h, B
f1 = X(1,:);g = X(2,:);h1 = X(3,:);j = X(4,:);l = X(5,:);
m = X(6,:);p = X(7,:);q = X(8,:);r = X(9,:);s = X(10,:);
v= X(11,:);x = X(12,:);z = X(13,:);F = X(14,:);

ydot1=bt.*(1-u1).*(L1-L2).*(ap.*q+ab.*z)+...
o.*(1-u5).*(L1-L3).*F+mh.*L1;% first ODE
ydot2=(u4+mh).*L2-C1-u4.*L1-nu.*(1-u5).*L14; % second ODE
ydot3=(mh+md+kc).*L3-C2-kc.*L1;
%ydot4=gp.*(L4-L6).*F+(rs+mp+u2).*L4-u2.*L5;
ydot4=gp.*(1-u5).*(L4-L6).*F+(rs+mp+u2).*L4-u2.*L5-u2.*C5;
ydot5=gp.*rb.*(1-u5).*(L5-L6).*F+(rv+mp+phv).*L5-phv.*L4;
ydot6=(w1+lp+mp).*L6-lp.*L7-w1.*L8-C3;
ydot7=(rp+pp+mp).*L7-pp.*L4;
ydot8=bt.*(1-u1).*(L1-L2).*ap.*f1+(delta1+u1+ap).*L8;
%ydot9=gb.*(L9-L11).*F+(ss+mb+u3).*L9-u3.*L10;
ydot9=gb.*(1-u5).*(L9-L11).*F+(ss+mb+u3).*L9-u3.*L10-u3.*C6;
ydot10=gb.*rb.*(1-u5).*(L10-L11).*F+(sv+mb+pv).*L10-pv.*L9;
ydot11=(n1+lb+mb).*L11-lb.*L12-n1.*L13-C4;
ydot12=(sb+pb+mb).*L12-pb.*L9;
ydot13=bt.*(1-u1).*(L1-L2).*ab.*f1+(e+u1+ab).*L13;
ydot14=o.*(1-u5).*(L1-L3).*f1+gp.*(1-u5).*(L4-L6).*j+...
rb.*gp.*(1-u5).*(L5-L6).*l+gb.*(1-u5).*(L9-L11).*r+...
rb.*gb.*(1-u5).*(L10-L11).*s+me.*L14;
ydot= [ydot1;ydot2;ydot3;ydot4;ydot5;ydot6;ydot7;ydot8;ydot9;...
ydot10;ydot11;ydot12;ydot13;ydot14];
%%*******************************************************

%================================================================
%Forward Runge-Kutta for States System
%File Name:rk4foward
%================================================================
function [t, w]=rk4foward(f,t0, tf, N, init,U,Constant)
h      = (tf-t0)/N;        %the step size
t(1)   = t0;
```

```
w(:,1) = init;            %initial conditions
for i = 1:N
uu = U(i,:);
k1 = h*f(t(i),w(:,i),uu,Constant);
k2 = h*f(t(i)+h./2, w(:,i)+0.5*k1,uu,Constant);
k3 = h*f(t(i)+h./2, w(:,i)+0.5*k2,uu,Constant);
k4 = h*f(t(i)+h, w(:,i)+k3,uu,Constant);
w(:,i+1) = w(:,i) + (k1 + 2*k2 + 2*k3 + k4)/6;
t(i+1)   = t0 + i*h;
end
%===================================================================

%===================================================================
%Backward Runge-Kutta for States System
%File Name:rk4back
%===================================================================
function [t, w]=rk4back(f,t0, tf, N, init2,U,YY,Constant)
h = -(tf-t0)/N;     %the step size
t(N) = tf;
w(:,N) = init2;     %initial conditions
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
for i = N:-1:1
uu = U(i,:);
yy = YY(:,i);
k1 = h*f(t(i),w(:,i),uu,yy,Constant);
k2 = h*f(t(i)+h/2, w(:,i)+0.5*k1,uu,yy,Constant);
k3 = h*f(t(i)+h/2, w(:,i)+0.5*k2,uu,yy,Constant );
k4 = h*f(t(i)+h, w(:,i)+k3,uu,yy,Constant);
if i>1
w(:,i-1) = w(:,i) + (k1 + 2*k2 + 2*k3 + k4)/6;
t(i-1) = tf + i*h;
else
ww = w(:,i) + (k1 + 2*k2 + 2*k3 + k4)/6;
tt = tf + i*h;
end
end
%+++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++++
w = [ww w];
t = [tt t];
%===================================================================
```

Outbreak/Extinction of Bovine Cysticercosis and Human Taeniasis & Optimal Control

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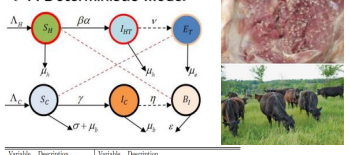
Introduction

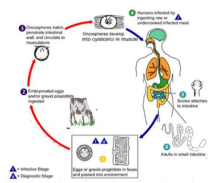
Bovine cysticercosis and human taeniasis are neglected food-borne diseases that pose challenges to food safety, peoples' health and the economy of rural farmers. Based on the deterministic model, a continuous time Markov chain stochastic model is formulated and analyzed to study the possibility of cysticercosis and taeniasis outbreak or extinction in cattle and humans respectively when initially there are few infectives. The analytical and numerical results for probability of diseases' extinction are in good agreement when initially there is no infectious human. Some variations in probabilities of disease's extinction are due to infectious beef being considered a discrete number in the CTMC stochastic model. The probability of disease's extinction is high when the diseases emerge from a small number of T. saginata eggs from the environment or from infected cattle. However, this probability becomes low when there is at least an infectious beef at the beginning of the diseases' outbreak.

Deterministic and Stochastic Modelling

❖ A Deterministic Model



Variable	Description	Variable	Description
S_H	Susceptible humans	I_{HT}	Humans with taeniasis
S_C	Susceptible cattle	I_C	Infected cattle
B_I	Infected beef	E_T	*Taenia saginata* eggs in the environment



MODEL EQUATIONS

$$\frac{dS_H}{dt} = \Lambda_H - \beta\alpha B_I S_H - \mu_h S_H,$$
$$\frac{dI_{HT}}{dt} = \beta\alpha B_I S_H - \mu_h I_{HT},$$
$$\frac{dS_C}{dt} = \Lambda_C - \gamma S_C E_T - (\sigma + \mu_b) S_C,$$
$$\frac{dI_C}{dt} = \gamma S_C E_T - (\eta + \mu_b) I_C,$$
$$\frac{dB_I}{dt} = \eta I_C - (\epsilon + \alpha) B_I,$$
$$\frac{dE_T}{dt} = \nu I_{HT} - \mu_e E_T,$$

$S_H(0) > 0$; $I_{HT}(0) \geq 0$; $S_C(0) > 0$;
$I_C(0) \geq 0$; $B_I(0) \geq 0$ and $E_T(0) \geq 0$.

Description of model parameters and their values (unit: yr^{-1}).

Parameter	Description	Value
Λ_H	Per capita recruitment rate of human population	100
μ_h	Per capita natural death rate of humans	0.0141
α	Rate of eating raw or undercooked infectious beef	0.23
Λ_C	Per capita recruitment rate of cattle	420
γ	*T. saginata* eggs to cattle transmission rate	0.00625
η	Slaughter rate of infected cattle	0.0325
μ_b	Per capita natural death rate of cattle	0.33
σ	The rate at which susceptible cattle are slaughtered	0.235
ϵ	Proportion of unconsumed infectious beef	0.225
β	Probability of humans to contract taeniasis	0.093
ν	Defecation rate by humans with taeniasis	0.325
μ_e	Per capita death rate of *T. saginata* eggs	10.42

❖ The Basic Reproduction Number

$$R_0 = \sqrt{\frac{\beta\nu\alpha\gamma\eta\Lambda_H\Lambda_C}{\mu_h^2\mu_e(\eta+\mu_b)(\alpha+\epsilon)(\sigma+\mu_b)}}.$$

❖ Sensitivity Analysis Results

Parameter	Sensitivity Index	Parameter	Sensitivity Index
Λ_H	+ 0.5000	Λ_C	+ 0.5000
β_T	+ 0.5000	μ_e	- 0.5000
μ_h	- 1.0000	η	+ 0.2920
ν	+ 0.5000	μ_b	- 0.5959
α_h	+ 0.4536	ϵ	- 0.4536
γ_h	+ 0.5000	σ_s	- 0.1961

❖ CTMC Model Formulation

The CTMC model transitions of states and their rates.

Event	Transition $\Delta Z(t)$	Transition rate, p
Recruitment of S_H	(1,0,0,0,0,0)	Λ_H
Infection of S_H	(−1, 1, 0, 0, 0, 0)	$\beta\alpha B_I S_H$
Death of S_H	(−1, 0, 0, 0, 0, 0)	$\mu_h S_H$
Death of I_{HT}	(0, −1, 0, 0, 0, 0)	$\mu_h I_{TH}$
Recruitment of S_C	(0,0,1,0,0,0)	Λ_C
Slaughter of S_C	(0, 0, −1, 0, 0, 0)	σS_C
Infection of S_C	(0, 0, −1, 1, 0, 0)	$\gamma S_C E_T$
Death of S_C	(0, 0, −1, 0, 0, 0)	$\mu_b S_C$
Slaughter of I_C	(0, 0, 0, −1, 1, 0)	ηI_C
Death of I_C	(0, 0, 0, −1, 0, 0)	$\mu_b I_C$
Throwing of infectious beef B_I	(0, 0, 0, 0, −1, 0)	ϵB_I
Consumption of infectious beef B_I	(0, 0, 0, 0, −1, 0)	αB_I
Shedding	(0,0,0,0,0,1)	νI_{TH}
Death of E_T	(0, 0, 0, 0, 0, −1)	$\mu_e E_T$

Analytical Vs Numerical Probabilities of Diseases' Extinction (Po, Pa)

$$q_1 = \frac{\mu_h}{\nu(1-q_4)+\mu_h},$$
$$q_2 = \frac{\epsilon\alpha\beta\eta\nu\Lambda_H}{(\epsilon+\alpha)(\alpha\beta\nu\Lambda_H+g_0)}\left(\frac{1}{R_0^2}\right)+\frac{\alpha\beta\nu\Lambda_H\mu_b+g_0}{\alpha\beta\nu\Lambda_H(\eta+\mu_b)+g_0},$$
$$q_3 = \frac{\epsilon\mu_h}{\beta\alpha\Lambda_H(1-q_1)+\epsilon\mu_h},$$
$$q_4 = \frac{\mu_e(\sigma+\mu_b)}{\gamma\Lambda_C(1-q_2)+\mu_e(\sigma+\mu_b)},$$

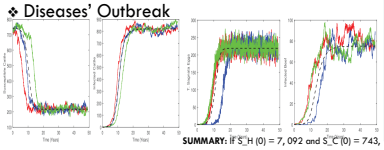
where $g_0 = \epsilon\mu_h(\eta+\mu_b)(\nu+\mu_h)$.

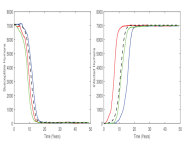
Probability of Disease Extinction .

y_1	y_2	y_3	y_4	P_o	P_a
1	0	0	0	0.3861	0.5306
0	0	0	1	0.9621	0.9616
1	0	0	1	0.3708	0.5102
2	0	0	1	0.1472	0.2707
1	0	0	2	0.3601	0.4906
0	1	0	0	0.9146	0.9105
0	0	1	0	0.0030	0.0018
0	2	0	0	0.8267	0.8290
1	1	0	1	0.3418	0.4646
1	1	0	0	0.3475	0.4831
1	1	1	1	0.0009	0.0008

Numerical Simulations

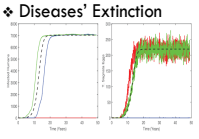
❖ Diseases' Outbreak

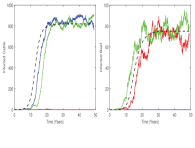




SUMMARY: If S_H (0) = 7, 092 and S_C (0) = 743, I_HT (0) = 2, I_C (0) = 1, B_I(0) = η, I_C (0) , E_T (0) = 1, there is disease's outbreak. If S_H (0) = 7, 092, S_C (0) = 743, I_HT (0) = 2, I_C = 0 B_I(0) = 0 and E_T (0) = 1, there is either diseases' outbreak or extinction (R0 = 19.8430 > 1 & stochastic threshold p(M) = 1.1461 > 1). Thus, the probability of disease's extinction is high if the diseases emerge from a small number of T. saginata eggs or from infected cattle. This probability becomes low when there is at least an infectious beef at the beginning of the diseases' outbreak. . Thus, interventions that focus on treatment of infected humans , meat inspection and improved beef cooking at the beginning of the epidemic, can help to control the diseases.

❖ Diseases' Extinction





The Optimal Control Problem

Basing on sensitivity analysis results we formulate and anlyze the optimal control problem for bovine cysticercosis and human taeniasis. In this case we focus on the time dependent control variable u_1(t) which for improved meat, u_2(t) for cattle vaccination and u_3(t) for treatment of humans with taeniasis. The results indicate that a strategy which focuses on improving meat cooking and treatment of humans with taeniasis is the optimal strategy for diseases' control.

The Optimal Control Model

$$\frac{dS_H}{dt} = \Lambda_H + w_5(t)I_{HT} - \beta_T\alpha_h(1-u_1(t))B_I S_H - \mu_h S_H,$$
$$\frac{dI_{HT}}{dt} = \beta_T\alpha_h(1-u_1(t))B_I S_H - (\mu_h + w_5(t))I_{HT},$$
$$\frac{dS_C}{dt} = \Lambda_C + \psi_c V_C + \pi R_C - \gamma_b S_C E_T - (\sigma_s + u_2(t) + \mu_b)S_C,$$
$$\frac{dV_C}{dt} = u_2(t)S_C - \rho_{\beta}\gamma_b V_C E_T - (\sigma_s + \psi_c + \mu_b)V_C,$$
$$\frac{dI_C}{dt} = \gamma_b S_C E_T + \rho_{\beta}\gamma_b V_C E_T - (\eta + \lambda + \mu_b)I_C,$$
$$\frac{dR_C}{dt} = \lambda I_C - (\pi + \sigma_r + \mu_b)R_C,$$
$$\frac{dB_I}{dt} = \eta I_C - (\varepsilon + \alpha_b)B_I,$$
$$\frac{dE_T}{dt} = \nu I_{HT} - \mu_e E_T.$$

We aim at minimizing the number of infected humans, cattl e and the cost associated with implementation of these int erventions. The objective function is:

$$J = \int_0^{T_f} (C_1 I_{HT} + C_2 I_C + C_3 u_2 S_C + \frac{1}{2}\sum_{i=1}^{i=3} A_i u_i^2)\,dt$$

we seek to find the optimal controls u_1^*, u_2^* and u_3^* such that:

$$J(u_1^*, u_2^*, u_3^*) = \min_U J(u_1, u_2, u_3),$$

where $U = \{u : u$ is measurable and $0 \leq u(t) \leq 1$ for $t \in [0, T_f]\}$ is the control set.

The Lagrangian $\mathcal{L}$ and Hamiltonian functions $\mathcal{H}$ for the control problem as:

$$\mathcal{L} = C_1 I_{HT} + C_2 I_C + C_3 u_2 S_C + \frac{1}{2}\sum_{i=1}^{i=3} A_i u_i^2, \quad \mathcal{H} = \mathcal{L} + \lambda_1\frac{dS_H}{dt} + \lambda_2\frac{dI_{HT}}{dt} + \lambda_3\frac{dS_C}{dt} + \lambda_4\frac{dV_C}{dt} + \lambda_5\frac{dI_C}{dt} + \lambda_6\frac{dR_C}{dt} + \lambda_7\frac{dB_I}{dt} + \lambda_8\frac{dE_T}{dt}$$

The adjoint system is given as:

$$\frac{d\lambda_1}{dt} = \beta_T(1-u_1)(\lambda_1 - \lambda_2)\alpha_h B_I + \mu_h\lambda_1,$$
$$\frac{d\lambda_2}{dt} = (w_5 + \mu_h)\lambda_2 - C_1 - w_5\lambda_1 - \nu\lambda_8,$$
$$\frac{d\lambda_3}{dt} = \gamma_b(\lambda_3 - \lambda_5)E_T + (\sigma_s + \mu_b + u_2)\lambda_3 - u_2\lambda_4 - u_2 C_3,$$
$$\frac{d\lambda_4}{dt} = \rho_{\beta}\gamma_b(\lambda_4 - \lambda_5)E_T + (\sigma_s + \mu_b + \psi_c)\lambda_4 - \psi_c\lambda_3,$$
$$\frac{d\lambda_5}{dt} = (\eta + \lambda_5 + \mu_b)\lambda_5 - \lambda_6\lambda_6 - \eta\lambda_7 - C_2,$$
$$\frac{d\lambda_6}{dt} = (\sigma_r + \pi_b + \mu_b)\lambda_6 - \pi_b\lambda_3,$$
$$\frac{d\lambda_7}{dt} = \beta_T(1-u_1)(\lambda_1 - \lambda_2)\alpha_h S_H + (\varepsilon + \alpha_b)\lambda_7,$$
$$\frac{d\lambda_8}{dt} = \gamma_b(\lambda_3 - \lambda_5)S_C + \rho_{\beta}\gamma_b(\lambda_4 - \lambda_5)V_C + \mu_e\lambda_8.$$

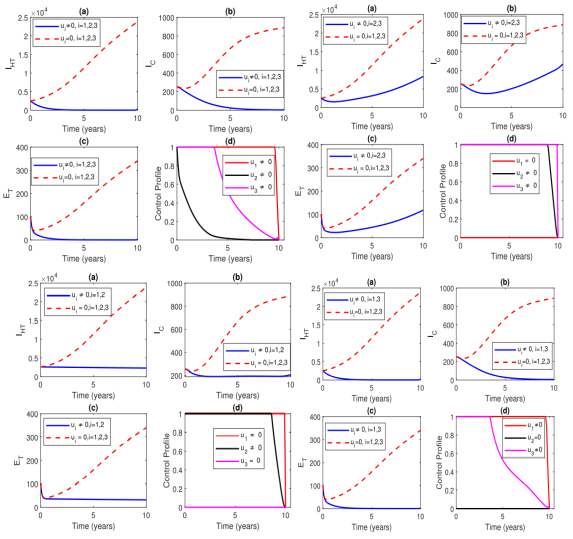
To obtain the control variables, we differentiate the Hamilto nian function and solve it when derivative is zero. That is:

$$\frac{\partial\mathcal{H}}{\partial u_1} = A_1 u_1 - (\lambda_2 - \lambda_1)\beta_T\alpha_h B_I S_H = 0,$$
$$\frac{\partial\mathcal{H}}{\partial u_2} = A_2 u_2 - (\lambda_3 - \lambda_4 - C_3)S_C = 0,$$
$$\frac{\partial\mathcal{H}}{\partial u_3} = A_3 u_3 - (\lambda_2 - \lambda_1)I_{HT} = 0.$$

From which we obtain:

$$u_1^* = \max\{0, \min\{1, \frac{(\lambda_2 - \lambda_1)\beta_T\alpha_h B_I S_H}{A_1}\}\},$$
$$u_2^* = \max\{0, \min\{1, \frac{(\lambda_3 - \lambda_4 - C_3)S_C}{A_2}\}\},$$
$$u_3^* = \max\{0, \min\{1, \frac{(\lambda_2 - \lambda_1)I_{HT}}{A_3}\}\}.$$

Numerical Results



It can be observed that a strategy which focus on improving meat cooking and treatm ent of humans with taeniasis is the most optimal strategy for diseases' control.

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