MODELING HIV AIDS DYNAMICS WITH FUNDING ALONG THE NORTHERN CORRIDOR HIGHWAY IN KENYA

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NOVEMBER , 2023

DECLARATION

This thesis is my original work and has not been presented for a degree or other award in any other University.

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DEDICATION

I dedicate this work to my wife Caro and my son Anthony Musyoka, my mum Mary who always stood with and encouraged me throughout the period.

To my brothers and sisters for prayers, emotional and psychological support. My love for you and may God bless you all.

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ACRONYMS AND ABBREVIATIONS

ACU	-	AIDS Control Unit
AIDS	-	Acquired Immune Deficiency Syndrome
ART	-	Antiretroviral Therapy
CD4	-	Cell cluster Differentiation antigen 4 cell
DNA	-	Deoxyribonucleic acid
DOT	-	Directly Observed Treatment
ELISA	-	enzyme-linked immunosorbent assay
FSWs	-	Female Sex Workers
HIV	-	Human Immunodeficiency Virus
IFA	-	Immunoflourescent assay
Ig	-	Immunoglobulin
IgG	-	Immunoglobulin G
LIA	-	Lineimmuno assay
NAAT	-	Nucleic Acid Amplification Testing
NACC	-	National AIDS Control Council
NASCOP	-	National AIDS and STIs Control Program
nef	-	Negative regulator factor
PCR	-	Polymerase Chain Reaction
PrEP	-	PreExposure Prophylaxis
rev	-	Regulator of Virion
RNA	-	Ribonucleic acid
RIPA	-	RadioImmuno Precipitation Assays
STDS	-	Sexually Transmitted Diseases
tat	-	Trans-activator of transcription
UN	-	United Nations
UNAIDS	-	United Nations Joint HIV/AIDS Program
vip	-	Viral Infectivity factor
vpr	-	Viral protein r
vpu	-	Viral protein u
WHO	-	World Health Organization
NCH	-	Northern Corridor Highway

ABSTRACT

For the past three and a half decades, HIV/AIDS has been a worldwide health problem. Because of its severe repercussions, it necessitates a significant financial commitment to stem its spread and prevent death-related disorders. In 2019, 18.6 million dollars were spent globally to finance HIV response. UNAIDS estimated that the HIV response required $26.2 billion USD by 2020. This emerged as a result of increased infection and fatality rates between 2015 and 2020. East and Southern Africa are the most affected by HIV/AIDS in Africa. However, owing to financial commitments to combat the pandemic, Kenya and South Africa have seen enhanced prevention and treatment facilities, with the largest difficulty being reliance on donors for funding HIV response. Kenya has a record of more than 1.5 million cases of people living with HIV in 2019 with a prevalence of 4.8% among adults in the same year. This ranked Kenya as the seventh-largest HIV population in the world. A recent study among 3,805 truckers along the Northern corridor highway in Kenya found that 55.9% had commercial sex in the past 6 months and 46.6% had regular sex partners along their trucking route besides their wife or girlfriend at home. HIV is mostly spread sexually in Kenya, with a high rate of infection among critical demographics such as sex workers and truckers, among others. Our study was based on the Northern Corridor highway which runs from Mombasa to Busia-Malaba on the Kenya-Uganda border, passing through Nairobi, Mai Mahiu, and Salgaa. The major mechanism of HIV/AIDS transmission along Kenya's Northern Corridor route is transactional sex, which involves financial transfers. The sexual network of truckers are complex and can be a conduit for the widespread of HIV between truckers and FSWs along the corridor. This prompted the need to better understand the dynamics of transmission of HIV/AIDS between truckers and female sex workers. In this study, a model was formulated for HIV/AIDS dynamics along the Northern corridor highway in Kenya which included circumcision and funding of treatment as control measures. The reproduction number, disease-free equilibrium and endemic equilibrium points were determined and their stabilities were also determined using the next-generation matrix method. The disease-free equilibrium is stable when $R_{0u} < 1$, $R_{0c} < 1$ and $R_{0f} < 1$ while the endemic equilibrium point is stable when $R_{0u} > 1$, $R_{0c} > 1$ and $R_{0f} > 1$. It was found that circumcision can be used as an intervention to minimize the infection of HIV among truckers and female sex workers. In addition, a model was developed to investigate the impact of funding on HIV transmission between truckers and female sex workers. According to the model's findings, boosting circumcision and funding decreases the rate of transition from the Susceptible to the Infected classes. Additionally, boosting funding for any class of treatment raises the Treatment class and lowers the total number of AIDS-related deaths. This also indicates that the Kenyan government should increase internal funding for HIV/AIDS due to the dwindling donor funding since 2018. This is because funding is a key in increasing treatment which decreases the AIDS cases.

CHAPTER 1

INTRODUCTION AND BACKGROUND INFORMATION

1.1 Introduction

By mid-1980s, HIV had reached every continent on earth. A team of researchers and health specialists assembled under the aegis of the World Health Organization (WHO) to suggest a worldwide plan for the control and prevention of AIDS, which was later supported by the World Health Assembly and the United Nations Assembly. The formation of the Global Program on AIDS in 1987 and the Joint United Nations Program on HIV/AIDS (UNAIDS) in 1996 prompted the United Nations began to treat AIDS as a global health issue. At the UN General Assembly's Special Session on AIDS in 2001, 189 countries concurred that AIDS was a top-priority global health issue, signing a landmark Declaration of Commitment on HIV/AIDS that guaranteed practical responses, concerted action, and responsibility for breakthroughs against the disease. The Declaration outlined a detailed set of time-bound goals to help achieve the Millennium Development Goal of slowing down the spread of the epidemic and start reversing it by 2015. As at 2007, an estimated 33.2 million individuals had been diagnosed of HIV, with an additional 2.5 million new HIV infections. AIDS claimed the lives of nearly 2.1 million people in that year while the worst-affected region in the globe was Sub-Saharan Africa (UNAIDS, n.d.; WHO, 2010)

In low-income countries were Kenya is categorised, HIV/AIDS was a consequential factor impeding the improvement of the economy. Despite the fact that women lived longer than males globally, AIDS had reduced female life expectancy in Kenya, Malawi, Zambia, and Zimbabwe. Women's higher risk to HIV infection was linked to gender

inequity, poverty, and a lack of education among women (UNAIDS, 2020). According to a survey done in 1990 using samples of women who attended ante-natal clinics, the UNAIDS indicated that the HIV epidemic curve seemed to reach its maximum in the late 1990s, and overall infection rates fell in 2002 and 2003 to roughly 6.7 percent (*Kenya AIDS Response Progress Report*, 2016).

The epidemic was widespread, with the majority of infections resulting from unprotected heterosexual contact (UNAIDS, 2020). Bwayo (1994); Bwayo (1991); Bwayo *et al.,* (1991); Mbugua *et al.,* (1995); and Plummer *et al.,* (1991) discovered that Kenya's HIV/AIDS epidemiology revealed high incidence of HIV and other STIs. It was found among sex workers that the rate was between 34 – 75% and for truckers, the rates were between 26 – 27%. Baeten *et al.,* (2005) underlined the significant incidence of these vulnerable populations. Ferguson e*t al.,* (2006) found that there was a strong relationship between HIV prevalence and places along the NCH between Mombasa and the towns along the NCH up to Ugandan border towns. For several reasons, transport workers are not captured in the national policy, except for the general policy that covers the entire masses. Under the direction of the Kenya Government, National AIDS Control Council (NACC) and the Ministry of Health's National AIDS and STD Control Program (NASCOP, 2014), the STD project mapped the main truck stops on the NCH between Mombasa and Kampala, the busiest of the four East African transport corridors. The study focused on measuring the extent and the rate of transport-related transactional sex on the trans-African highway between Mombasa and Kampala. It was found that the cost and availability of FSWs were the most important basis that truckers considered when deciding where to pack overnight. A stopover was considered as a hotspot based on

several factors that determine how much transactional sex took place. The study included the number of truckers and the number of FSWs present at the location.

Human Immunodeficiency Virus (HIV) has been one of the most dangerous viral agents for the past three decades, and it has now reached pandemic proportions. The virus is spread through a variety of means, including blood transfusions, deep kissing, and the most prevalent route, unprotected heterosexual sexual interactions with infected individuals. The virus would also be spread to neonates through their mothers through nursing. In general, the HIV/AIDS (Acquired Immune Deficiency Syndrome) epidemic among most at risk groups, such as female sex workers (FSWs) and truckers, need extra attention in order to prevent and control its spread and deleterious effects. Despite the fact that the epidemic has been present in East Africa for nearly two decades, vulnerable populations such as truckers and sex workers have been largely ignored, with only localized programs designed to meet the needs of highway-based sex workers and their clients, which primarily included truck drivers and their assistants (Ghani and Aral, 2005; Hawken *et al.,* 2002; Voeten *et al.*, 2002). Transactional sex was the major HIV/AIDS transmission medium along Kenya's Northern Corridor Highway (NCH), These acts take place at specified points along the route, many of which were towns, where transactional sex was highly popular and the towns were dubbed "hotspots." To slow the spread of AIDS throughout the corridor, these hotspots demand a lot of monitoring.

Early surveillance revealed a substantial link between HIV prevalence and places along the NCH connecting Mombasa and Ugandan border towns. (Ferguson *et al.,* 2006). HIV/AIDS spread from its major concentration along major highway networks and into major cities, eventually reaching rural areas according to (NASCOP, 2014). The

HIV/AIDS pandemic has expanded off the major highway due to the mobility of the FSWs along the highway and then returning again to their rural regions. The link between truck driving and HIV/AIDS received a lot of attention from writers and journalists; Murphy's (1993) “The Ukimwi Road” and Conover (1993) “Trucking through the AIDS belt”, for insatnces, highlighted high-risk sexual behaviour among truckers in East Africa. According to Kenya H.I.V (2014), HIV financing in Kenya is donor-driven which has been reducing since 2018. Also, the national and county governments contribute 31% of the money spend on HIV and AIDS response. This indicates that the country must find innovative and sustainable financing options within the existing fiscal space as the national efforts to respond to HIV/AIDS. Table 1.1 compares the proposed allocation to actual allocation.

According to Ferguson and Morris (2007), parking overnight at a given place depends on several factors which included security, available parking, the price of meals and accommodation. Other considerations were accommodation quality and availability, as well as the relative cost and availability of FSWs. They also found that a stopover was considered a hotspot depending on the frequency of and rate at which transactional sex took place there. The key determining factors were the truckers and the number of FSWs present at the location. The truckers and the number of FSWs present at the place played a major role in determining the hotspot towns along the NCH. The stopovers were classified into 3 classes: (1) weighbridge and border crossing points (locations where vehicles are held up due to bureaucracy): Mariakani and Mlolongo are examples of weighbridge stations, while Malaba and Busia are examples of border crossing points; (2) pure truck shops (locations that cater solely to the needs of truckers and other travellers): Salgaa, Machakos Junction, Mtito Andei, Maungu, and Mai Mahiu are examples; (3)

stopovers (locations that serve a variety of roles such as border crossing stations, administrative centers, and market centers) and Mlolongo and Busia are in this category.

Table 1.1: Table of counties and HIV financing 2018/2019

COUNTY	HIV FINANCING	
	Proposed Allocation 2017/2018 (Kshs. in millions)	Actual allocation 2018/2019 (Kshs. in millions)
Mombasa	6.171	0.5
Taita Taveta	45.312	0.702
Machakos	66.372	2.5
Makueni	118.92	5.0
Nakuru	81.548	3.0
Busia	21.888	2.5
Kisumu	45.0	2.3
Nairobi	414.0	4.4
Uasin Gishu	50.03	0.075

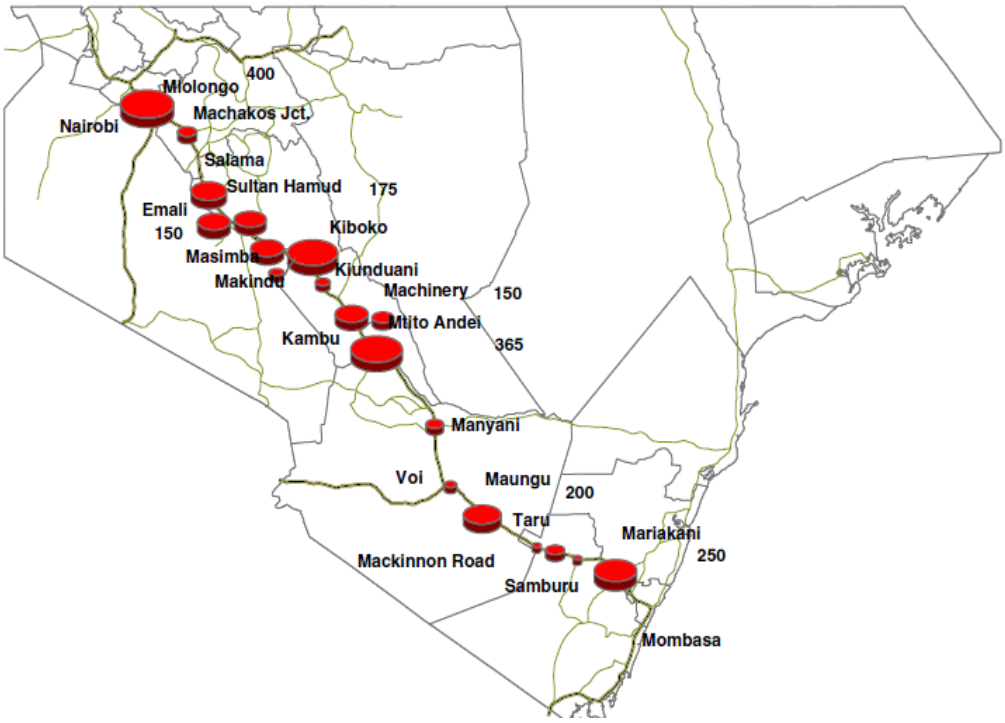

Figure 1.1: Estimates of sex worker populations—Mombasa–Nairobi

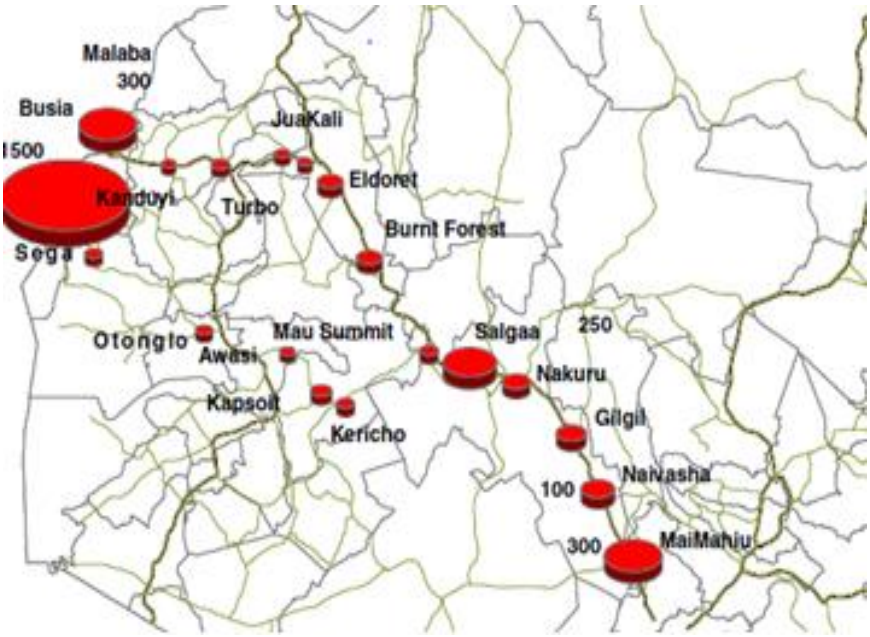

Figure 1.2: Estimates of sex worker populations—Nairobi–Uganda border

Ferguson and Morris (2007) surveyed 7700 FSWs's customers (30 percent truckers, 14.5 percent drivers of other vehicles and 7.4 percent police officers) and demonstrated the use of Geographical Information Systems (GIS) at every level.

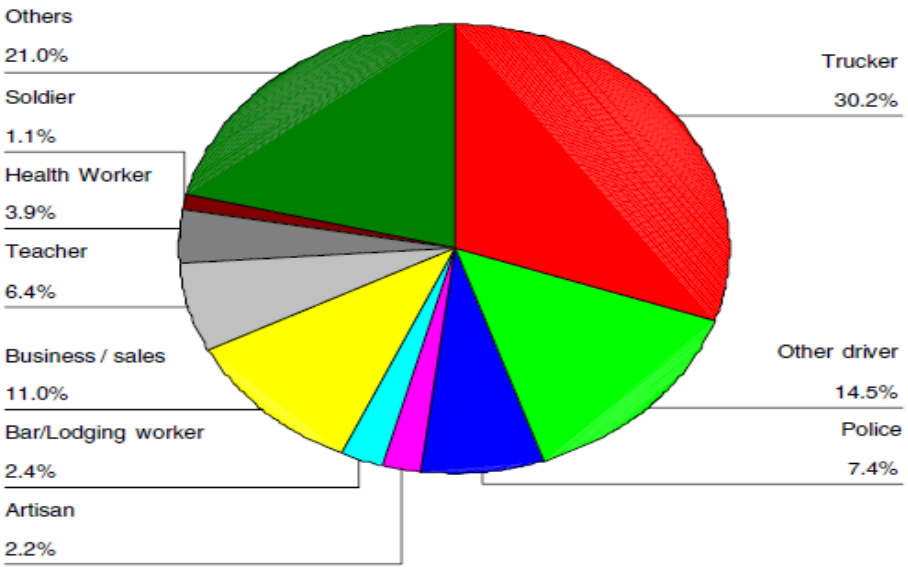

Figure 1.3: Occupational mix of highway-based sex worker clients

This study focused on developing a mathematical model of the transmission of HIV/AIDS among FSWs and truckers. The model included the presence of funding on treatment and circumcision, and a proper analysis of the model was carried out to determine their effect on the spread of the deadly virus between the targeted population.

1.2 HIV/AIDS

Calles *et al.,* (2010) considered HIV as a retrovirus from the family of lentiviruses and which attacks the body's natural defence system. HIV has a longer incubation period but eventually causes signs and symptoms of AIDS. The attack on the immune system weakens the immune system and makes it unable to fight off diseases. The HIV, with all the infections it causes, is called AIDS. The human body is able to fight infections due to the presence of the CD4+ cells of the white blood cells but HIV attacks the CD4+ cells to destroy them, and ultimately kill the individual. The destruction of more $CD4+$ cells means the body becomes vulnerable to other opportunistic infections. The final stage of HIV where the number of CD4+ cells have drastically reduced is called AIDS. People living with AIDS easily contract other infections and cancers that rarely occur in healthy people. It is important to reiterate that having HIV doesn't necessarily mean that the person has AIDS. Even in absence of treatment, it usually takes 10 – 12 years for HIV to progress to AIDS (clinical latency or incubation period). Early diagnosis of HIV and using the right medicines can elongate the incubation period, reduce damages to the immune sytem and return the immune system to a healthier state. There are two known HIV types; HIV type 1 (HIV-1) responsible for almost all AIDS cases worldwide and HIV-2 which cause AIDS-like illness. HIV-2 progresses more slowly than HIV-1.

HIV is made of a cylindrical centre surrounded with a sphere-shaped lipid bilayer envelope. In this lipid bilayer are two major viral glycoproteins which are gp120 and gp41. These proteins mediate recognition of CD4+ cells and chemokine receptors which enables the virus to attach and invade CD4+ cells (Calles et al. 2010). The inner sphere comprises of two-single stranded copies of genomic material (RNA) and also multiple proteins and enzymes necessary for HIV replication and maturation and include p17, p24, integrase, protease and reverse transcriptase. HIV is different from other retroviruses in the sense that it uses nine genes to code for the necessary proteins and enzymes. Three principal genes include gag, pol and env. The gag encodes the core proteins, the env gene encodes the HIV structural components called glycoproteins and the pol gene encodes the integrase, enzyme reverse transcriptase and protease. The other genes which include rev, nef, vif, vpu, vpr and tat are used for viral replication and enhancement of HIV's infectivity rate.

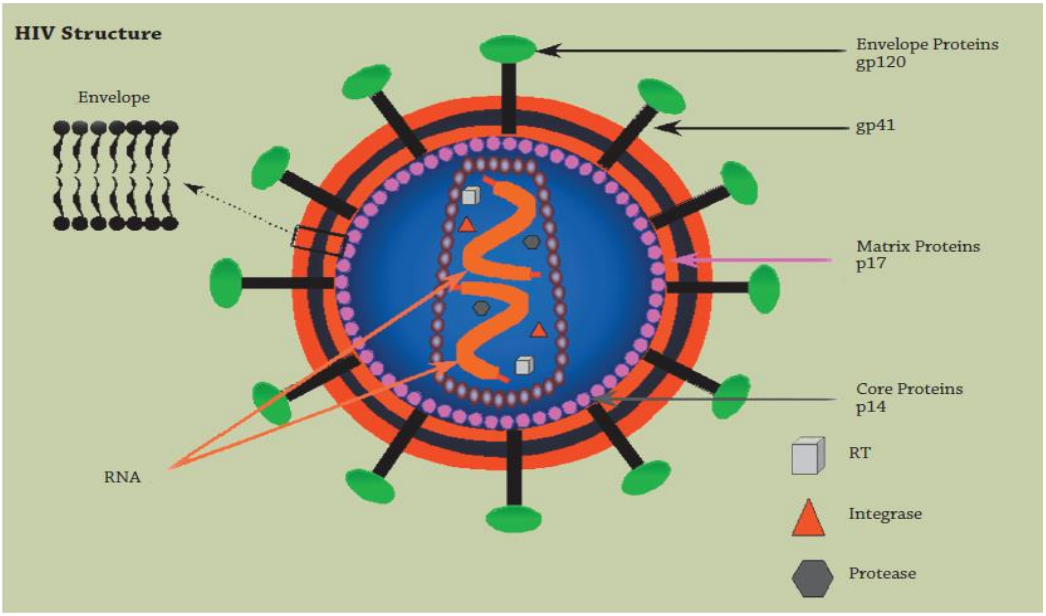

Figure 1.4: The Human Immunodeficiency Virus

The lifespan of any cell that has been infected by HIV is reduced because the virus uses the host cell to produce more virus. New host cells are used by HIV to continuously replicate, producing between 10 million to 10 billion virions daily. It only takes an approximate of one day after exposure for the HIV to attack the dendritic cells and capture them into the mucous membranes and the skin. The infected cells move to the lymph nodes and hence the peripheral blood and thereby increasing the rate of replication. The CD4+ cells remitted to fight the viral antigen also move to the lymph nodes. All these sequential actions exposes the CD4+ cells more to infection which is responsible for lymphadenopathy characteristics of the acute retroviral syndrome in people and not children. The monocytes infected by HIV act as a home of HIV since they enhance replication and protect the disease from being killed. The HIV-cycle revolves around 6 phases which are binding and entry, reverse transcription, integration, replication, budding and maturation.

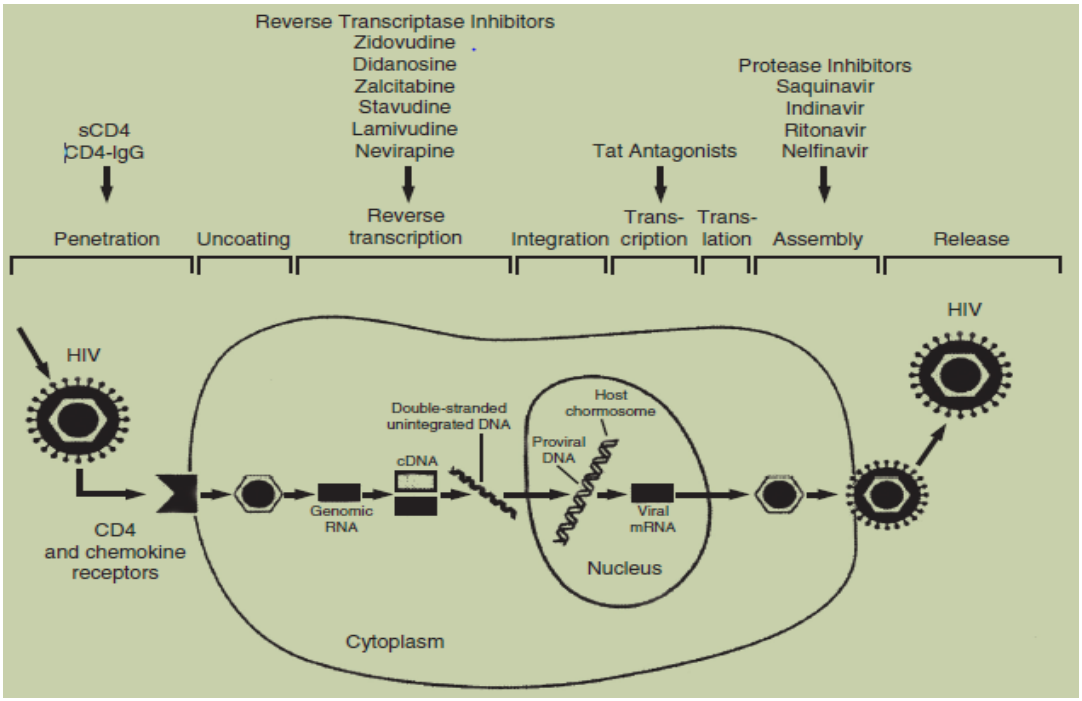

Figure 1.5: HIV life cycle

HIV infection is classified into five categories, namely; primary infection, Clinical Stages 1, 2, 3, and 4 (i.e. CS1, CS2, CS3 and CS4). The Primary Infection stage is the point in time when the virus gets inside the human body. The individual's blood transport a lot of the viruses per millilitre of plasma (viral-load), which can even exceed one million. A freshly infected individual starts experiencing an acute retroviral syndrome between 2-4 weeks after infection and whose signs and symptoms include: myalgia, headache, tendency to vomit, fever, weight loss, diarrhoea, nocturnal hyperhidrosis and rash. A significant rapid decrease in the CD4+ is experienced, although it doesn't often drop below 200 cells/μL. During this time, the virus combats CD4+ cells and this leaves the infected individual very vulnerable to opportunistic infections. Also, the ability of the thymus to produce T-lymphocytes is limited. The effectiveness of using ELISA HIV-testing at this stage depends on the time of seroconversion. The second stage is the Clinical Latency Stage 1.

At this point, a "viral set point" is formed and the body system, on realising the presence of these infections, signals the body immune system to activate the antibodies. The anitbodies are secreted to shield the immune system from been attacked. Due to a high viral load, the rate at which diseases progressions occur can be estimated. During this period, the patients does not necessarily show signs and symptoms but persistent lymphadenopathy is common. This period may last for about 8-10 years in adults. The HIV ELISA testing is reliable and CD4+ count is $\geq$500 cells/μL for people above age of 5 years. The third stage is the CS2. An infected person may appear healthy for some years before the mild signs and symptoms starts appearing. The following might develop in the infected person: lymphadenopathy, persistent hepatosplenomegaly, herpes zoster,

popular pruritic eruptions, molluscom contagiosum, and candidiasis. The viral load rises throughout this stage, whereas the CD4+ count drops to 350-499 cells/μL. The fourth stage is the CS3 where the immune system of the infected individuals becomes very weak and this can develop into a infections that are life-threatening like tuberculous lymphadenitis, persistent fever, development of cryptosporidiosis, candidiasis, bacterial pneumonia and also suffer from weight loss. The viral load continues increasing while the CD4+ cells count reduceds to less than 200-349 cells per μL in adults. The fifth and final stage is the CS4. With the virus taking over many cells and killing the CD4+ cells, individuals with advanced HIV and/or AIDS continue to acquire secondary infections such Pneumocystis jirovecii pneumonia, CMV infection, cryptococcal meningitis, Kaposi sarcoma, and a variety of other diseases that take advantage of the immune system's critical state. This is the highest stage of the diseases where the viral load is highest and CD4+ $<$ 200 cells/μL and may cause death.

HIV transmission happens when the mucous membrane is exposed to infected blood, breast milk and secretions around the genitals. The transmission takes place via sexual interactions with an infected individual, from mothers to babies at birth or by breastfeeding, also among drug users who share infected needles and transfusion of infected blood products. Among these methods of transmission of HIV, sexual transmission contributes the largest percentage worldwide (Ferris *et al.*, (2010). In a similar manner transactional sex which is very high along the NCH in Kenya has been the main mode of transmission of HIV along the highway. Ferris *et al.* (2010) highlighted that the chance of contracting HIV from sexual intercourse is determined by; (1) HIV status of the sexual partner. (2) Number of sexual partners. (3) Nature of the sexual

interaction. (4) Viral load in the body fluids of the infected partner. (5) Presence of other STIs.

Sharma & Marfatia (2008) enumerated some laboratory tests for diagnosing HIV infection as ELISA, Rapid Tests, Western Blot, p24 antigen, PCR, Saliva HIV tests, Oraquick Advance Rapid HIV Test, Urine tests, Nucleic Acid Amplification Testing.

1.3 Definition of Terms

HIV: It's a collection of retroviruses that target and destroy the immune system of the human body (CD4+ cells).

AIDS: The last stage of HIV infection, when the body's capacity to fight infections of any type is lost.

Truckers: The team of drivers and their aides entrusted by different companies with trucks to carry luggage to various locations along the NCH in Kenya and beyond.

FSWs: These are women who accept monetary payments or material items in return for sexual gratification. This includes those who intentionally characterize these activities as means of earning, even if sex work is not their profession (Cheryl, 2002).

CD4+ Cells: These are white blood cells in the human body that are in charge of immunity. T-helped lymphocytes, or T4 cells, are another name for them.

Transactional Sex: This is any sexual relationship based on exchange of sex for gifts or services. It is a superset of prostitution, in that the exchange of gifts for sex (usually non-marital) includes a broader set that does not necessarily involve a

predetermined payment of gift but where there is a definite motivation to benefit materially from the sexual exchange. The participants do not necessarily frame themselves in terms of prostitutes/ clients but often as girlfriends/ boyfriends or sugar babies/ sugar daddies (Hoefinger, 2010, 2013). Those offering sex may or may not feel affection for their partners.

Sexually Transmitted Infections (STIs): These are infections that can be transferred from person to person through sexual intercourse or sexual contact. The causative agents are often bacteria, viruses or parasites such as gonorrhoea, syphilis, chlamydia, chancroid and HIV/AIDS (Green, 2019).

1.4 Statement of the Problem

Transactional sex is the most common method of HIV/AIDS spread between truckers and FSWs along the NCH in Kenya according to Ferguson *et al.,* (2006). It is necessary to develop strategies for fully comprehending the dynamics of spread of HIV/AIDS between truckers and FSWs. As a result, mathematical models that depict this circumstance were developed in order to reduce the spread of HIV/AIDS among high-risk populations along the highway. The impact of the HIV/AIDS epidemic on the general population has been addressed in a majority of current models. The effect of circumcision and funding on the spread of the pandemic was not factored into in the models that looked at the HIV/AIDS epidemic along the transportation corridor. This research developed a mathematical model which incorporated circumcision and funding parameters in the prevention and treatment of the epidemic along the NCH in Kenya.

1.5 Justification

Ferguson & Morris (2007) found that for towns along the NCH, transactional sex between truckers and their regular and irregular female sex partners was very high. These Tonws were referred to as hotspots. Consequently the HIV prevalence was found to be high along the NCH and hence, this sparked the need to get ways to contain HIV/AIDS spread between truckers and their cohort along the corridor. This prompted our study on how circumcision and funding treatment would affect HIV/AIDS dynamics between truckers and FSWs along Kenya's NCH.

1.6 Objectives

1.6.1 General Objective

Modeling HIV AIDS dynamics with funding along the Northern Corridor Highway in Kenya.

1.6.2 Specific Objectives

i. Develop a deterministic mathematical model of HIV/AIDS dynamics along the Northern corridor highway incorporating circumcision and funding.

ii. Determine the endemic and disease-free equilibrium points of the model and their stability.

iii. Determine how circumcision affects HIV infection among the male population and between truckers and FSWs.

iv. Determine how funding affects HIV/AIDS dynamics between truckers and FSWs along the Northern corridor highway.

1.7 Significance of the Study

Transactional sex involving cash transfers is on the increase along the NCH due to the seductive dresses worn by the FSWs at night to seek clients. Also as a result of lack adequate awareneness on HIV/AIDS pre-exposure and preventive measures and long time taken by the truckers away from their wives, the truckers engage in unprotected heterosexual intercourse with the FSWs. This has contributed to an upsurge in HIV/AIDS spread which has invited interest from scientists to come up with methods of controlling its spread . This study developed a mathematical model showing the dynamics of the interactions between the truckers and the FSWs in the system. This research provides a deeper understanding of the response of the serial-killer HIV/AIDS epidemic to funding. Moreover, the analysis of the model equations provides an investigation into the effect of circumcision on HIV spread between truckers and FSWs along the Kenya's NCH. This research recommends some measures that would minimize the transmission of the epidemic among the population along the route, therefore limiting the spread of the pandemic outwards from the major highway

CHAPTER 2

LITERATURE REVIEW

This chapter explores the relevant literature on deterministic mathematical modeling of HIV AIDS to narrow down to the knowledge gap under study in our research.

Waziri *et al.* (2012) looked at the dynamics of HIV/AIDS with treatment and vertical transmission using a nonlinear deterministic mathematical model. The model was analysed qualitatively using the stability theory of differential equations. From the results of the model, the disease-free equilibrium was locally stable when $R_0 < 1$ and was unstable when $R_0 > 1$. The Disease Free Equilibrium was globally unstable due to the existence of the forward bifurcation at a threshold parameter equal to one. The use of ARVS treatment reduced greatly the rate of vertical transmission significantly. Endemic equilibrium existed when R_0 was greater than one. A mathematical model that considers the presence of complacency in a population where rate of HIV new infections is approaching zero was studied by Baryarama *et al.* (2006b). The analysis of the model indicated that complacency that arouse from the dependence of HIV transmission on the number of AIDS cases led to damped periodic oscillations in the number of infectives. The model also indicated the epidermic was stable at a higher number of AIDS infections. Also, Baryarama *et al.* (2006a) further considered the presence of gradual behavioural change in a population with HIV/AIDS infected individuals. The analysis of this model indicated a decrease in HIV prevalence in Uganda in the 1990s which was a result of the drastic decrease in the force of infection. It also showed that a decrease in behaviour parameters directly resulted to a drop in the number of secondary HIV infections. Waema and Olowofeso (2005) in their mathematical modelling for HIV

transmission using generating function approach in 2004, used the generating function technique to model HIV/AIDS transmission. The model was able to show highlight the response of the susceptible and the infected persons to different model parameters.

A mathematical model for HIV/AIDS transmission from mother to child by Narasimhamurthy *et. al.,* (2012) showed absence of vertical transmission into the community resulted to continuous decrese of the susceptible population. Analysis of the model indicated that when reproduction number $R_0 > 1$, the disease became endermic while when $R_0 < 1$, the disease died out. It was also concluded that rise in rate of vertical transmission lead to increased the infected population which resulted to increased pre-AIDS and AIDS population. Mukandavire *et. al.,* (2007) did a a sex-structured model for heterosexual transmission of HIV/AIDS with incubation period to investigate the effect of male circumcision as a preventive measure for HIV/AIDS. They also studied the effects of condom use as another preventive strategy for controlling HIV/AIDS. Their results indicated that in absence of a preventive vaccine for HIV/AIDS, male circumcision was a potential control strategy of HIV/AIDS to reduce the development of the HIV/AIDS epidemic and was more effective when implemented together with condom use. Mbare, (2021), modelled the function of long distance truck drivers on HIV transmission dynamics. From the analysis he found that truck drivers had the potential of increasing the HIV infections rates and transmissions within the susceptible population.

Espitia, *et. al.,* (2022), developed a deterministic mathematical model of HIV and AIDS in adult population considering sexual preferences like exclusive homosexual men contact, contact between homosexual men and women, contact between homosexual and heterosexual men, and heterosexual contact under antiretroviral therapy in Colombia.

Numerical simulations indicated that increase in the number of sexual partners in homosexual or heterosexual contact led to an increase in the number of HIV and AIDS infections. Chagoma, N. *et. al.,* (2023) used mathematical modeling to estimate the impact of COVID-19-related VMMC service disruptions on new HIV infections in Zimbabwe. Results showed that strategies to mitigate COVID-19 have had an impact on many public health outcomes, including for HIV. The reduction in the availability of VMC and other HIV prevention services contributed to additional new HIV infections depending on the length and frequency of disruptions. It was important for decision makers to consider strategic investments and policies to support COVID-19 pandemic control while maintaining and sustaining delivery of preventive health services such as VMC.

Teklu *et. al.,* (2021), developed a nonlinear compartmental mathematical model to assess the effect of treatment on HIV/AIDS dynamics and pneumonia coinfection in human population. Numerical simulations indicated that treatment against infection at every stage reduced the rate of infection and disease prevalence. Eaton, J. W *et. al.,* (2021) developed Naomi Bayesian small area estimation model to estimate quantities stratified by subnational administrative units, sex, and five-year age groups in sub- Saharan Africa. The model was estimated using empirical Bayes framework with probabilistic uncertainity in the output indicators.

Giddings, *et. al.,* (2023, studied infectious disease modeling of HIV prevention interventions using a systematic review and narrative synthesis of compartimental models. The results indicated that of 837 articles screened, 48 articles were included in the review and 32 unique mathematical models were identified. Majority of the studies

studied PrEP (83%), fewer modelled circumcision (54%) and only a few focussed on vaccination (10%) as means of HIV intervention measures.

Ibrahim *et. al.,* (2021), used a deterministic HIV transmission model to study dynamics of HIV transmission showing importance of counselling and treatment. Results from numerical simulation indicated that effective use of condom, counseling or the use of anti-retrovirus drug effectively reduced the HIV transmission. Also ART treatment rate reduced the basic reproduction number hence, leading to decease of HIV/AIDS. Ringa *et. al.,* (2022), developed a model COVID-19 and HIV and AIDS to assess the impact of COVID -19 on HIV dynamics in South Africa. Results indicated that HIV prevention measures significantly reduced the burden of co-infections with COVID-19, while effective treatment of COVID-19 could reduced co-infections with opportunistic infections such as HIV/AIDS. Furthermore, COVID-19 prevention strategy only averted about 10,500 new co-infection cases, with a similar number also averted by HIV-prevention control only.

Parker, *et. al.,* (2021), studied HIV infection in Eastern and Southern Africa (ESA) to advice on the challeges associated with HIV prevention within the region. The analysis indicated that elimination of HIV in ESA required continued investment, commitment to evidence-based programmes and persistence.

Omondi, *et. al.,* (2019) formulated mathematical compartmental models of HIV transmission within and between two age groups in Kenya. Analysis of the data fitted in the model showed that there was difference in the mean number of new HIV infections between males and females within the two age groups. Also analysis indicated that females had high HIV prevalence compared to their male counterparts. Furthermore the

analysis indicated that the HIV transmission rate was highest between young adults to adults and most HIV infections appeared in adult population. Omondi, *et. al.,* (2018), used a deterministic model to provide a quantification of HIV prevention, testing and treatment with ART as a fight against HIV infection. They used Lyapunov function to show the that model system was globally asymptotically stable when $R_0 < 1$. The results showed that there was need to promote prevention mechanism to reduce the occurrence of new infections. Also combination of several control mechanisms significantly reduced the spread of the disease, by maintaining the level of each control high. Omondi, *et. al.,* (2018), developed a mathematical model on HIV transmission dynamics incorporating sexual orientation of individuals. The analysis indicated a higher infectivity rate in the female population. Further the introduction of pre-exposure prophylaxis (PrEP) on the dynamics of the HIVproduced a positive effect by limiting the of spread of HIV.

Kimulu, A. M. (2023) studied HIV/AIDS dynamics among truckers and the local Community at Malaba and Busia Border Stops. The analysis found that increase in force of infections to males from females increased the male HIV infections due to prolonged time of sexual interactions as the truckers await clearance and also due to higher number of male truckers than females. Furthermore, the analysis showed that delays in clearance time increased both male and female HIV/AIDS infections leading to an increase in the number of AIDS cases in this border crossing points.

Based on the mentioned literature, mathematical modelling of HIV/AIDS dynamics has been studied in the general population. The review above clearly indicates that none of these models studied HIV/AIDS dynamics along the NCH in Kenya with the specific effects of either the truckers' circumcision or funding to influence the prevention and

treatment of HIV/AIDS. This research developed two deterministic mathematical models; the first model investigated the effect of circumcision as a control measure of HIV/AIDS transmission between truckers and female sex workers along NCH and the second investigated the significance of both funding and circumcision on the HIV/AIDS transmission dynamics between the same target group. This was also in accordance with Kenya Vision 2030, which aims to eliminate all new infections by 2030.

CHAPTER 3

RESEARCH DESIGN AND METHODOLOGY

3.1 Introduction

A mathematical model is developed for HIV spread between truckers and FSWs under the influence of circumcision along the NCH in Kenya from Mombasa to the Malaba-Busia border. In 2022, it was estimated that 39 million people were already infected with HIV, with new infections standing at 1.7 million, 770,000 AIDS-related deaths, and HIV prevalence of adults was 0.8% (Case *et al.,* 2019) globally. Most of the infected living human beings were in the low- and middle-income regions, with 68% estimated to be living in sub-Saharan Africa.

Case *et al.,* (2019) reported in the same study that 20.6 million out of the 37.9 million infected people were living in Eastern and Southern Africa in 2018, with recorded 800,000 new infections. Out of the 25.7 million people who were living with HIV in the entire African continent in 2018, 20.6 million (about 80%) were from Eastern and Southern Africa. About 1.1 million new infections were recorded in the African continent in 2018, and this contributed to about 67% of the new infections globally in that same year. Kenya claims the seventh-largest HIV epidemic in the world harbouring over 1.5 million infected individuals and 21,000 people die from AIDS-related sicknesses yearly (Galvani *et al.,* 2018). Maina *et al.,* (2014) explained that in 1996, 10.5 percent of Kenyans were infected, although the prevalence level declined to a half-standing at 5.9% by 2015 when HIV treatment and care increased. In 2016, 64 percent of HIV infected patients were on treatment and 51% of these were virally suppressed, (Iwuji & Newell, 2017).

In sub-Saharan Africa, truckers and FSWs who provide services to the truckers at crucial transport stopovers, were at greater risk of acquiring HIV compared to other cohorts working in different environment (Ojo *et al.,* 2011). In East Africa, HIV rates were still high along the border crossings and major roadways (E. Kelvin *et al.,* 2019). A 1991 survey of Kenyan truck drivers realised that 18% were HIV-positive; 61% truckers reported they have had sex with FSWs and only 32 percent of them ever used condoms (Bwayo, 1991; Bwayo *et al.,* 1991). According to a survey of 3,805 Kenyan truckers by Kelvin *et al.,* (2018), 55.9 percent had transactional sex in the preceding six months, and 46.6 percent had a regular companion across their route in addition to their wife or girlfriend at home. Truckers could be a channel for HIV transmission across partner categories (commercial, regular partners on their route, spouses or girlfriends at home) as well as domestic and international boundaries due to their complicated sexual interactions according to (International Labor Organization, 2005).

Despite the fact that the epidemic has been present in East Africa for more than two decades, vulnerable populations such as truckers and sex workers have been mostly disregarded (Ghani and Aral, 2005; Hawken *et al.,* 2002; Voeten *et al.,* 2002). To meet the demands of highway-based sex workers and their customers, localized initiatives have been established. The data from 2011 showed that 29.3% of FSWs are infected, (Nyblade *et al.,* 2017). In 2015, it was revealed that around one-third of the FSWs were infected (Musyoki *et al.,* 2015). According to Atika (2016), FSWs are said to be better at protecting themselves against HIV transmission than other HIV-affected populations, such as males who have sex with other men. This might be due to the fact that 76 percent of FSWs receive access to HIV services, which is greater than other significant

demographic groups. However, transactional sex, which involves monetary transfers, has its own set of sites along the route, some of which are towns.

3.2 Model 1: Mathematical Model for HIV Transmission Between Truckers and Fsws in Kenya with Circumcision as a Control Measure

3.2.1 Model formulation

A mathematical model is formulated to study HIV/AIDS transmission between truckers and FSWs in Kenya with the influence of circumcision. The population is divided into eight compartments, namely; (1) three susceptible classes S_u, S_c, S_f of uncircumcised susceptible male group, circumcised susceptible male group and susceptible female group respectively. (2) three infected classes I_u, I_c, I_f of uncircumcised Infected males, circumcised Infected males and Infected females. (3) treated group T and (4) the AIDS group A.

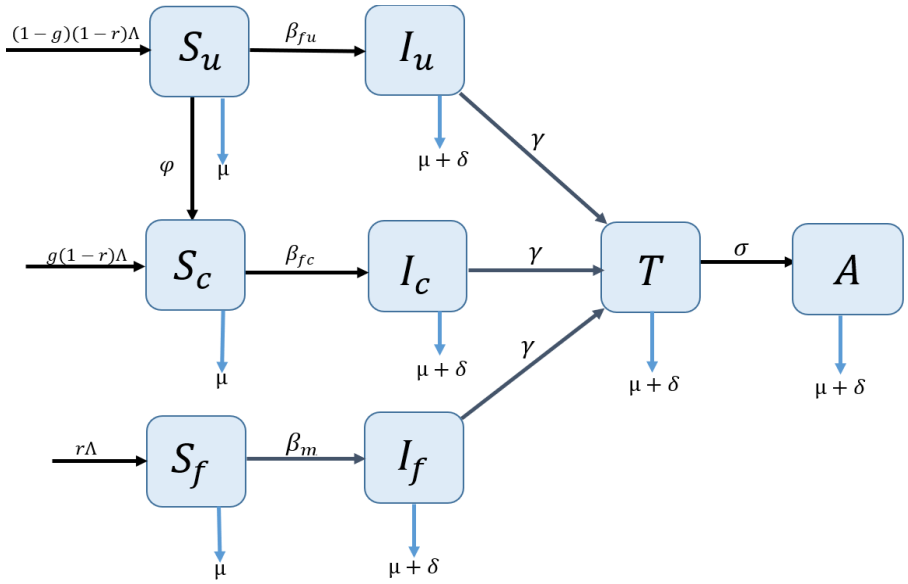

Figure 3.1: Flow chart of effect of circumcision on HIV transmission dynamics between truckers and FSWs

The model was based on the following assumptions:

i. All the infected individuals are subjected to treatment.

ii. The main clients of the female sex workers were truckers.

iii. Only the adult population is considered.

iv. Uncircumcised male drivers can be subjected to VMC

v. No births

Table 3.1: Definition of parameters

Parameter	Definition
g	circumcised proportion of males
r	the proportion of females recruited into female sex workers
Λ	recruitment rate into the susceptible population
γ	rate of accessing treatment of infected males or females
σ	rate of progression of treated individuals to AIDS
μ, δ	Natural mortality rate and disease-induced mortality rate respectively
φ	rate of circumcision
β_{fu}	the transmission rate of HIV from females to uncircumcised males
β_{fc}	the transmission rate of HIV from females to circumcised males
β_m	the transmission rate of HIV from males to females

From figure 3.1 the governing differential equations are;

$$\left.\begin{aligned}
\frac{dS_u}{dt} &= (1-g)(1-r)\Lambda - \beta_{fu}S_uI_u - (\varphi+\mu)S_u \\
\frac{dS_c}{dt} &= g(1-r)\Lambda + \varphi S_u - \beta_{fc}S_cI_c - \mu S_c \\
\frac{dS_f}{dt} &= r\Lambda - \beta_m S_f I_f - \mu S_f \\
\frac{dI_u}{dt} &= \beta_{fu}S_uI_u - (\mu+\gamma+\delta)I_u \\
\frac{dI_c}{dt} &= \beta_{fc}S_cI_c - (\mu+\gamma+\delta)I_c \\
\frac{dI_f}{dt} &= \beta_m S_f I_f - (\mu+\gamma+\delta)I_f \\
\frac{dT}{dt} &= (I_u + I_c + I_f)\gamma - (\mu+\delta+\sigma)T \\
\frac{dA}{dt} &= \sigma T - (\mu+\delta)A
\end{aligned}\right\} \qquad (3.1)$$

3.2.2 Analysis of the model

To investigate the feasible region, we sum all the human compartments to get the total population N,

$$N = S_u + S_c + S_f + I_u + I_c + I_f + T + A.$$

Obtaining the time derivatives of the total population along the solution path gives

$$\left.\begin{aligned}
\frac{dN}{dt} &= \frac{dS_u}{dt} + \frac{dS_c}{dt} + \frac{dS_f}{dt} + \frac{dI_u}{dt} + \frac{dI_c}{dt} + \frac{dI_f}{dt} + \frac{dT}{dt} + \frac{dA}{dt}, \\
\frac{dN}{dt} &= \Lambda - \mu N - \delta(I+T+A), \\
I &= I_u + I_c + I_f
\end{aligned}\right\} \qquad (3.2)$$

In absence of disease mortality rate then $\delta = 0$, and (3.2) reduces to

$$\frac{dN}{dt} \leq \Lambda - \mu N \qquad (3.3)$$

Soving the linear differential equation gives,

$$N \leq \frac{\Lambda}{\mu} + \frac{C}{e^{\mu t}}$$

where C is the constant of integration. As $t \to \infty$

$$\lim_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}$$

hence $N(t)$ is clearly bounded and

$$0 < N(t) \leq \frac{\Lambda}{\mu}$$

therefore, the feasible solution of the system of equations enter and remains in the region Ω for all future time, where

$$\Omega = \left\{S_u, S_c, S_f, I_u, I_c, I_f, T, A \in \mathbb{R}_+^8 \ : \ 0 < N(t) \leq \frac{\Lambda}{\mu}\right\} \tag{3.4}$$

This implies that the model is well-posed and therefore we proceed to study the model in in the region Ω. Since our model describes the human population, it is important to show that all the state variables $S_u, S_c, S_f, S_u, I_u, I_c, I_f, T$, and A are all positive for all time t.

Theorem 3.1: Given the initial conditions such that $S_u(0) > 0$, $S_c(0) > 0$, $S_f(0) > 0$, $I_u(0) > 0$, $I_c(0) > 0$, $I_f(0) > 0$, $A(0) > 0$, $T(0) > 0$, then $S_u(t)$, $S_c(t)$, $S_f(t)$, $S_u(t)$, $I_u(t), I_c(t), I_f(t), T(t), A(t)$ are all positive for time $t \geq 0$.

Proof: From the system (3.1) of governing equations of the model we obtain;

$$\frac{dS_u}{dt} = (1-g)(1-r)\Lambda - \left(\beta_{fu} I_u + \varphi + \mu\right) S_u \quad \Rightarrow \quad \frac{dS_u}{dt} \geq -(\varphi + \mu) S_u \tag{3.5}$$

Separating the variables and integrating we have,

$$\frac{dS_u}{S_u} \geq -(\varphi + \mu)dt \quad \Rightarrow \quad \ln S_u \geq -(\varphi + \mu)t + c \ \Rightarrow \ S_u(t) \geq e^{-(\varphi+\mu)t+c}$$

Substituting $t = 0$ and determining the value of C gives $S_u(0) \geq e^0 e^C$ and hence

$$S_u(0) = e^C \tag{3.6}$$

Thus, the solution becomes

$$S_u(t) \geq S_u(0)e^{-(\varphi+\mu)t} > 0$$

and $S_u(t)$ is positive for every t.

$$\frac{dS_c}{dt} = g(1-r)\Lambda + \varphi S_u - \beta_{fc} S_c I_c - \mu S_c, \quad \Rightarrow \quad \frac{dS_c}{dt} \geq -\mu S_c \tag{3.7}$$

Separating the variables and integrating we have,

$$\frac{dS_c}{S_c} \geq -\mu dt \qquad \Rightarrow \qquad \ln S_c \geq -\mu t + C,$$

and introducing exponential on both sides we obtain

$$S_c(t) \geq e^{-\mu t + C} \tag{3.8}$$

Substituting $t = 0$ and determining the value of C we get, $S_c(0) \geq e^0 e^C$ and hence

$$S_c(0) = e^C \tag{3.9}$$

Our solution becomes

$$S_c(t) \geq S_c(0)e^{-\mu t} > 0$$

Which shows that $S_c(t)$ is positive for every t. Solving the other differential equations involving S_f, I_u, I_c, I_f, T, A in a similar manner we obtain

$$\left.\begin{aligned}
&\frac{dS_f}{dt} \geq -\mu S_f,\ S_f(t) \geq\ S_f(0)e^{-\mu t} > 0 \\
&\frac{dI_u}{dt} \geq -(\mu+\gamma+\delta)I_u, I_u(t) \geq I_u(0)e^{-(\mu+\gamma+\delta)t} > 0 \\
&\frac{dI_c}{dt} \geq -(\mu+\gamma+\delta)I_c, I_c(t) \geq I_c(0)e^{-(\mu+\gamma+\delta)t} > 0 \\
&\frac{dI_f}{dt} \geq -(\mu+\gamma+\delta)I_f, I_f(t) \geq I_f(0)e^{-(\mu+\gamma+\delta)t} > 0 \\
&\frac{dT}{dt} \geq -(\mu+\delta+\sigma)T, T(t) \geq T(0)e^{-(\mu+\delta+\sigma)t} > 0 \\
&\frac{dA}{dt} \geq -(\mu+\delta)A, A(t) \geq A(0)e^{-(\mu+\delta)t} > 0
\end{aligned}\right\} \qquad (3.10)$$

Equation (3.10) indicates that $S_f,\ I_{u,}\ I_{c,}\ I_{f,}\ T,\ A$ are always positive for all time t. ■

3.2.3 Disese Free Equilibrium (DFE)

The DFE,s E^0 is obtained by setting $I_u = I_c = I_f = T = A = 0$ in equation (3.1) to get

$$S_u = \frac{(1-g)(1-r)\Lambda}{\varphi+\mu}, \qquad S_c = \frac{\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)}, \qquad S_f = \frac{r\Lambda}{\mu}$$

and we have

$$E^0 = \left(S_u^0,\ S_c^0,\ S_f^0,\ I_u^0,\ I_c^0,\ I_f^0,\ T^0, A^0\right)$$

$$= \left(\frac{(1-g)(1-r)\Lambda}{\varphi+\mu}, \frac{\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)}, \frac{r\Lambda}{\mu}, 0, 0, 0, 0, 0, \right) \qquad (3.11)$$

3.2.4 Reproduction number $(\boldsymbol{R_0})$

When an infected individual is introduced into a susceptible population, the reproduction number R_0 represents the number of secondary infections that can be caused. R_0 is calculated using the next-generation matrix. Define F_i and V_i as matrices that indicate the rate of new infections and the rate at which people are transferred out of compartments.

The spectral radius of the $F_i V_i^{-1}$ matrix is used to calculate the R_0. From system (3.1) then;

$$\mathcal{F} = \begin{pmatrix} \beta_{fu} S_u I_u \\ \beta_{fc} S_c I_c \\ \beta_m S_f I_f \\ 0 \\ 0 \end{pmatrix} \text{ and } \mathcal{V} = \begin{pmatrix} (\mu+\gamma+\delta) I_u \\ (\mu+\gamma+\delta) I_c \\ (\mu+\gamma+\delta) I_f \\ -\gamma I_u - \gamma I_c - \gamma I_f + (\mu+\delta+\sigma) T \\ -\sigma T + (\mu+\delta) A \end{pmatrix}$$

Let F and V be the Jacobian of $\mathcal{F}$ at DFE and the Jacobian of $\mathcal{V}$ at DFE so that,

$$F = \begin{pmatrix} \beta_{fu} S_u^0 & 0 & 0 & 0 & 0 \\ 0 & \beta_{fc} S_c^0 & 0 & 0 & 0 \\ 0 & 0 & \beta_m S_f^0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} \mu+\gamma+\delta & 0 & 0 & 0 & 0 \\ 0 & \mu+\gamma+\delta & 0 & 0 & 0 \\ 0 & 0 & \mu+\gamma+\delta & 0 & 0 \\ -\gamma & -\gamma & -\gamma & \mu+\delta+\sigma & 0 \\ 0 & 0 & 0 & -\sigma & \mu+\delta \end{pmatrix}$$

Hence for system (3.1), the next generation matrix (Bada *et al.*, 2020; Oke *et al.*, 2021) is

$$FV^{-1} = \begin{pmatrix} \frac{\beta_{fu} S_u^0}{\mu+\gamma+\delta} & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_{fc} S_c^0}{\mu+\gamma+\delta} & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_m S_f^0}{\mu+\gamma+\delta} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

The characteristic polynomial of $FV^{-1} - \lambda I$ at DFE is given by,

$$\begin{vmatrix} \frac{\beta_{fu}S_u^0}{\mu+\gamma+\delta}-\lambda & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_{fc}S_c^0}{\mu+\gamma+\delta}-\lambda & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_m S_f^0}{\mu+\gamma+\delta}-\lambda & 0 & 0 \\ 0 & 0 & 0 & -\lambda & 0 \\ 0 & 0 & 0 & 0 & -\lambda \end{vmatrix} = 0$$

Which reduces to the following equation;

$$\lambda^2\left(-\lambda+\frac{\beta_m r\Lambda}{\mu(\mu+\gamma+\delta)}\right)\left(-\lambda+\frac{\beta_{fc}\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)(\mu+\gamma+\delta)}\right)\left(-\lambda+\frac{\beta_{fu}(1-g)(1-r)\Lambda}{(\varphi+\mu)(\mu+\gamma+\delta)}\right) = 0$$

The eigenvalues are

$$\lambda_1=\lambda_2=0,\quad \lambda_3=\frac{\beta_m r\Lambda}{\mu(\mu+\gamma+\delta)}, \lambda_4=\frac{\beta_{fc}\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)(\mu+\gamma+\delta)},$$

$$\lambda_5=\frac{\beta_{fu}(1-g)(1-r)\Lambda}{(\varphi+\mu)(\mu+\gamma+\delta)}$$

λ_3, λ_4 and λ_5 gives the basic reproduction numbers for the system (3.1) as follows;

$$\left.\begin{aligned} R_{of} &= \frac{\beta_m r\Lambda}{\mu(\mu+\gamma+\delta)} \\ R_{oc} &= \frac{\beta_{fc}\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)(\mu+\gamma+\delta)} \\ R_{ou} &= \frac{\beta_{fu}(1-g)(1-r)\Lambda}{(\varphi+\mu)(\mu+\gamma+\delta)} \end{aligned}\right\} \quad (3.12)$$

Where R_{of} is the number of secondary infections that an infected female can cause in her entire infectious period when introduced in the susceptible population, R_{oc} is the number of secondary infections that an infected circumcised male can cause when introduced in

the susceptible population, R_{ou} is the number of secondary infections that an infected uncircumcised male can cause when introduced in the susceptible population. The reproduction number is therefore

$$R_o = \max(R_{of},\ R_{oc}, R_{ou}).$$

3.2.5 Local stability of the disease free equilibrium (DFE)

Theorem 3.2: Define $R_o = \max(R_{of},\ R_{oc}, R_{ou})$, then the disease-free equilibrium E^0 of the system (3.1) is locally asymptotically stable if $R_0 < 1$.

***Proof*:** The Jacobian matrix of the system is given by

$$J = \begin{pmatrix} -\beta_{fu}I_u - (\varphi+\mu) & 0 & 0 & -\beta_{fu}S_u & 0 & 0 & 0 & 0 \\ \varphi & -\beta_{fc}I_c - \mu & 0 & 0 & -\beta_{fc}S_c & 0 & 0 & 0 \\ 0 & 0 & -\beta_m I_f - \mu & 0 & 0 & -\beta_m S_f & 0 & 0 \\ \beta_{fu}I_u & 0 & 0 & \beta_{fu}S_u - k & 0 & 0 & 0 & 0 \\ 0 & \beta_{fc}I_c & 0 & 0 & \beta_{fc}S_c - k & 0 & 0 & 0 \\ 0 & 0 & \beta_m I_f & 0 & 0 & \beta_m S_f - k & 0 & 0 \\ 0 & 0 & 0 & \gamma & \gamma & \gamma & -k_1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \sigma & -k_2 \end{pmatrix}$$

Where $k = \mu + \gamma + \delta,\ \ k_1 = \mu + \delta + \sigma,\ \ k_2 = \mu + \delta.$

The first two eigenvalues are $\lambda_1 = -(\mu + \delta)$, and $\lambda_2 = -(\mu + \delta + \sigma)$, and the remaining six eigenvalues $\lambda_3, \lambda_4, \cdots, \lambda_8$ are obtained from the equations;

$$\lambda^2 - \big(\beta_m(S_f - I_f) - k - \mu\big)\lambda + \beta_m\big(kI_f - \mu S_f\big) + \mu k = 0,$$

$$\lambda^2 - \big(\beta_{fc}(S_c - \mathrm{I_c}) - k - \mu\big)\lambda + \beta_{fc}(kI_c - \mu S_c) + \mu k = 0,$$

$$\lambda^2 - \big(\beta_{fu}(S_u - I_u) - (\varphi+\mu) - k\big)\lambda + \beta_{fu}(kI_u - (\varphi+\mu)S_u) + k(\varphi+\mu) = 0.$$

It is clear that λ_1 and λ_2 are negative. The requirements for the other eigenvalues to be negative are given below;

For negative λ_3, λ_4, we require

$$\frac{\beta_m(S_f - I_f)}{k+\mu} < 1, \text{ and } \frac{\beta_m(\mu S_f - kI_f)}{\mu k} < 1. \tag{3.13}$$

For negative λ_5, λ_6, we require

$$\frac{\beta_{fc}(S_c - I_c)}{k+\mu} < 1, \text{ and } \frac{\beta_{fc}(\mu S_c - kI_c)}{\mu k} < 1. \tag{3.14}$$

For negative λ_7, λ_8, we require

$$\frac{\beta_{fu}(S_u - I_u)}{k+\mu+\varphi} < 1, \text{ and } \frac{\beta_{fu}((\mu+\varphi)S_u - kI_u)}{k(\mu+\varphi)} < 1 \tag{3.15}$$

Substituting DFE

$$E^0 = \left(\frac{(1-g)(1-r)\Lambda}{\varphi+\mu}, \frac{\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)}, \frac{r\Lambda}{\mu}, 0,0,0,0,0, \right)$$

The requirements for stability therefore are;

i. λ_3, λ_4, are negative if

$$\frac{\beta_m r\Lambda}{\mu(2\mu+\gamma+\delta)} < 1, \text{ and } \frac{\beta_m r\Lambda}{\mu(\mu+\gamma+\delta)} < 1$$

and thus

$$\frac{\beta_m r\Lambda}{\mu(2\mu+\gamma+\delta)} < \frac{\beta_m r\Lambda}{\mu(\mu+\gamma+\delta)} < 1 \Rightarrow R_{of} < 1. \tag{3.16}$$

ii. λ_5, λ_6 are negative if

$$\frac{\Lambda\beta_{fc}(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)(2\mu+\gamma+\delta)} < 1, \quad \frac{\Lambda\beta_{fc}(1-r)(g\mu+\varphi)}{\mu(\mu+\gamma+\delta)(\varphi+\mu)} < 1$$

and thus

$$\frac{\Lambda\beta_{fc}(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)(2\mu+\gamma+\delta)} < \frac{\Lambda\beta_{fc}(1-r)(g\mu+\varphi)}{\mu(\mu+\gamma+\delta)(\varphi+\mu)} < 1 \Rightarrow R_{oc} < 1 \qquad (3.17)$$

iii. λ_7, λ_8 are negative if

$$\frac{\beta_{fu}(1-g)(1-r)\Lambda}{(2\mu+\gamma+\delta+\varphi)(\varphi+\mu)} < 1, \quad \text{and} \quad \frac{\beta_{fu}(1-g)(1-r)\Lambda}{(\mu+\gamma+\delta)(\varphi+\mu)} < 1$$

and thus

$$\frac{\beta_{fu}(1-g)(1-r)\Lambda}{(2\mu+\gamma+\delta+\varphi)(\varphi+\mu)} < \frac{\beta_{fu}(1-g)(1-r)\Lambda}{(\mu+\gamma+\delta)(\varphi+\mu)} \Rightarrow R_{ou} < 1. \qquad (3.18)$$

Hence, E^0 is locally asymptotically stable if $R_0 < 1$. ■

3.2.6 Global stability of the disease free equilibrium (DFE)

Theorem of Castillo – Chavez and Song, B. (2004) was used to prove global stability of DFE,

Theorem 3.3. The disease free equilibrium point

$$E^0 = \left(\frac{(1-g)(1-r)\Lambda}{\varphi+\mu}, \frac{\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)}, \frac{r\Lambda}{\mu}, 0, 0, 0, 0, 0, \right)$$

is globally asymptotically stable if $R_0 < 1$ and locally asymptotically stable if assumptions $(H1)$ and $(H2)$ are satisfied;

H1: for $\frac{dX}{dt} = F(X, 0)$, E^0 is globally asymptotically stable.

H2: $G(X,Z) = AZ - \tilde{G}(X,Z),\ \tilde{G}(X,Z) \geq 0$ for $(X,Z) \in \mathbb{R}_+^5$ where $A = D_Z\, G(X,0)$ is an M- matrix (all off-diagonal elements are nonnegative) and $\mathbb{R}_+^5$ is the region where the model makes biological sense.

Equation (3.1) is written as

$$\frac{dX}{dt} = F(X,Z), \quad \frac{dZ}{dt} = G(X,Z),$$

where $X = (S_u, S_c, S_f)$ represents the disease free classes and $Z = (I_u, I_c, I_f, T, A)$ represents the infected classes.

Proof

In our case then; $F(X,0) = F\big((X_1,0),(X_2,0),(X_3,0)\big) = \Lambda - \mu S,\ S = (S_u, S_c, S_f)$, $X = (S_u, S_c, S_f)$ and $Z = (I_u, I_c, I_f, T, A)$.

$$A = \begin{pmatrix} \beta_{fu}S_u - \kappa & 0 & 0 & 0 & 0 \\ 0 & \beta_{fc}S_c - \kappa & 0 & 0 & 0 \\ 0 & 0 & \beta_m S_f - \kappa & 0 & 0 \\ \gamma & \gamma & \gamma & -\kappa_1 & 0 \\ 0 & 0 & 0 & \sigma & -\kappa_2 \end{pmatrix}$$

$$= \begin{pmatrix} \frac{\beta_{fu}(1-g)(1-r)\Lambda}{\varphi+\mu} - \kappa & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_{fc}\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)} - \kappa & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_m r\Lambda}{\mu} - \kappa & 0 & 0 \\ \gamma & \gamma & \gamma & -\kappa_1 & 0 \\ 0 & 0 & 0 & \sigma & -\kappa_2 \end{pmatrix} \quad (3.19)$$

AZ

$$= \begin{pmatrix} \frac{\beta_{fu}(1-g)(1-r)\Lambda}{\varphi+\mu} - \kappa & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_{fc}\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)} - \kappa & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_m r\Lambda}{\mu} - \kappa & 0 & 0 \\ \gamma & \gamma & \gamma & -\kappa_1 & 0 \\ 0 & 0 & 0 & \sigma & -\kappa_2 \end{pmatrix} \begin{pmatrix} I_u \\ I_c \\ I_f \\ T \\ A \end{pmatrix}$$

$$= \begin{pmatrix} \frac{\beta_{fu}(1-g)(1-r)\Lambda I_u}{\varphi+\mu} - \kappa I_u \\ \frac{\beta_{fc}\Lambda(1-r)(g\mu+\varphi)I_c}{\mu(\varphi+\mu)} - \kappa I_c \\ \frac{\beta_m r\Lambda I_f}{\mu} - \kappa I_f \\ \gamma I_u + \gamma I_c + \gamma I_f - \kappa_1 T \\ \sigma T - \kappa_2 A \end{pmatrix} \tag{3.20}$$

$G(X,Z)$

$$= \begin{pmatrix} \beta_{fu} S_u I_u - \kappa I_u \\ \beta_{fc} S_c I_c - \kappa I_c \\ \beta_m S_f I_f - \kappa I_f \\ \gamma I_u + \gamma I_c + \gamma I_f - \kappa_1 T \\ \sigma T - \kappa_2 A \end{pmatrix} \tag{3.21}$$

$$\tilde{G}(X,Z) = AZ - G(X,Z) = \begin{pmatrix} \frac{\beta_{fu} I_u}{\beta_{fu} S_u I_u}\left(\frac{(1-g)(1-r)\Lambda}{\varphi+\mu} - 1\right) \\ \frac{\beta_{fc} I_c}{\beta_{fc} S_c I_c}\left(\frac{\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)} - 1\right) \\ \frac{\beta_m I_f}{\beta_m S_f I_f}\left(\frac{r\Lambda}{\mu} - 1\right) \\ 0 \\ 0 \end{pmatrix} \tag{3.22}$$

From the second condition $\tilde{G}(X,Z) \geq 0$, Since all parameters lies between 0 and 1 then we have $\frac{(1-g)(1-r)\Lambda}{\varphi+\mu} - 1 \leq 0$, $\frac{\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)} - 1 \geq 0$ and $\frac{r\Lambda}{\mu} - 1 \geq 0$. Therefore condition $(H2)$, $G(X,Z) = AZ - \tilde{G}(X,Z)$, $\tilde{G}(X,Z) \geq 0$ for $(X,Z) \in \mathbb{R}_+^{\,5}$ is not satisfied and $E^0 = \left(\frac{(1-g)(1-r)\Lambda}{\varphi+\mu}, \frac{\Lambda(1-r)(g\mu+\varphi)}{\mu(\varphi+\mu)}, \frac{r\Lambda}{\mu}, 0, 0, 0, 0, 0, \right)$ is globally asymptotically unstable point of $\frac{dX}{dt} = F(X,0)$ when $R_0 < 1$ ■

3.2.7 Endemic Equilibrium Point (EEP)

This refers to the state at which the disease persists within the population. Let $E^* = \left(S_u^*, S_c^*, S_f^*, I_u^*, I_c^*, I_f^*, T^*, A^*\right)$ be the endemic equilibrium point, obtained by setting the LHS of equation (3.1) to zero, so that

$$\left.\begin{aligned}
&(1-g)(1-r)\Lambda - \beta_{fu}S_u^*I_u^* - (\varphi+\mu)S_u^* = 0\\
&g(1-r)\Lambda + \varphi S_u^* - \beta_{fc}S_c^*I_c^* - \mu S_c^* = 0\\
&r\Lambda - \beta_m S_f^* I_f^* - \mu S_f^* = 0\\
&\beta_{fu}S_u^*I_u^* - (\mu+\gamma+\delta)I_u^* = 0\\
&\beta_{fc}S_c^*I_c^* - (\mu+\gamma+\delta)I_c^* = 0\\
&\beta_m S_f^* I_f^* - (\mu+\gamma+\delta)I_f^* = 0\\
&\left(I_u^* + I_c^* + I_f^*\right)\gamma - (\mu+\delta+\sigma)\,T^* = 0\\
&\sigma\,T^* - (\mu+\delta)A^* = 0
\end{aligned}\right\} \qquad (3.23)$$

From which we obtain the endemic equilibrium point;

$$S_u^* = \frac{\mu+\delta+\gamma}{\beta_{fu}}, S_c^* = \frac{\mu+\delta+\gamma}{\beta_{fc}}, S_f^* = \frac{\mu+\delta+\gamma}{\beta_m}, I_f^* = \frac{\beta_m r\Lambda - \mu(\mu+\delta+\gamma)}{\beta_m(\mu+\delta+\gamma)},$$

$$I_u^* = \frac{\beta_{fu}(1-g)(1-r)\Lambda - (\varphi+\mu)(\mu+\delta+\gamma)}{\beta_{fu}(\mu+\delta+\gamma)},$$

$$I_c^* = \frac{\beta_{fu}\beta_{fc}g(1-r)\Lambda + \varphi\beta_{fc}(\mu+\delta+\gamma) - \mu\beta_{fu}(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}(\mu+\delta+\gamma)},$$

$$T^* = \frac{\beta_{fu}\beta_{fc}\beta_m\gamma(1-g)(1-r)\Lambda - \beta_{fc}\beta_m\gamma(\mu+\varphi)(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}\beta_m(\mu+\delta+\gamma)(\mu+\delta+\sigma)}$$

$$+ \frac{\beta_{fu}\beta_{fc}\beta_m r\Lambda\gamma - \beta_{fu}\beta_{fc}\mu\gamma(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}\beta_m(\mu+\delta+\gamma)(\mu+\delta+\sigma)}$$

$$+ \frac{\beta_{fu}\beta_{fc}\beta_m\gamma g(1-r)\Lambda - \varphi\gamma\beta_{fc}\beta_m(\mu+\delta+\gamma) - \mu\gamma\beta_{fu}\beta_m(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}\beta_m(\mu+\delta+\gamma)(\mu+\delta+\sigma)},$$

$$A^* = \frac{\beta_{fu}\beta_{fc}\beta_m\gamma\sigma(1-g)(1-r)\Lambda - \beta_{fc}\beta_m\gamma\sigma(\mu+\varphi)(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}\beta_m(\mu+\delta+\gamma)(\mu+\delta+\sigma)(\mu+\delta)}$$

$$+\frac{\beta_{fu}\beta_{fc}\beta_m r\Lambda\gamma\sigma-\beta_{fu}\beta_{fc}\mu\gamma\sigma(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}\beta_m(\mu+\delta+\gamma)(\mu+\delta+\sigma)(\mu+\delta)}$$

$$+\frac{\beta_{fu}\beta_{fc}\beta_m\gamma g\sigma(1-r)\Lambda-\varphi\gamma\sigma\beta_{fc}\beta_m(\mu+\delta+\gamma)-\mu\gamma\sigma\beta_{fu}\beta_m(\mu+\delta+\gamma)}{\beta_{fu}\beta_{fc}\beta_m(\mu+\delta+\gamma)(\mu+\delta+\sigma)(\mu+\delta)}$$

3.2.8 Local stability of the endemic equilibrium point (EEP)

Theorem 3.4: Define $R_0=\{R_{of},R_{oc},R_{ou}\}$, then the endemic equilibrium point E^* is locally asymptotically stable if $R_0>1$ and locally asymptotically unstable if $R_0<1$

***Proof*:** The Jacobian matrix of the system is given by

$$J=\begin{pmatrix}
-\beta_{fu}I_u-(\varphi+\mu) & 0 & 0 & -\beta_{fu}S_u & 0 & 0 & 0 & 0\\
\varphi & -\beta_{fc}I_c-\mu & 0 & 0 & -\beta_{fc}S_c & 0 & 0 & 0\\
0 & 0 & -\beta_m I_f-\mu & 0 & 0 & -\beta_m S_f & 0 & 0\\
\beta_{fu}I_u & 0 & 0 & \beta_{fu}S_u-k & 0 & 0 & 0 & 0\\
0 & \beta_{fc}I_c & 0 & 0 & \beta_{fc}S_c-k & 0 & 0 & 0\\
0 & 0 & \beta_m I_f & 0 & 0 & \beta_m S_f-k & 0 & 0\\
0 & 0 & 0 & \gamma & \gamma & \gamma & -k_1 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & \sigma & -k_2
\end{pmatrix}$$

where $k=\mu+\gamma+\delta,\ k_1=\mu+\delta+\sigma,\ k_2=\mu+\delta$. The first two eigenvalues are

$$\lambda_1=-(\mu+\delta),\qquad \text{and}\qquad \lambda_2=-(\mu+\delta+\sigma),$$

and the remaining six eigenvalues $\lambda_3,\ \lambda_4,\cdots,\ \lambda_8$ are obtained from the equations;

$$\lambda^2-\left(\beta_m\left(S_f^*-I_f^*\right)-k-\mu\right)\lambda+\beta_m\left(kI_f^*-\mu S_f^*\right)+\mu k=0$$

$$\lambda^2-\left(\beta_{fc}(S_c^*-I_c^*)-k-\mu\right)\lambda+\beta_{fc}(kI_c^*-\mu S_c^*)+\mu k=0,$$

$$\lambda^2-\left(\beta_{fu}(S_u^*-I_u^*)-(\varphi+\mu)-k\right)\lambda+\beta_{fu}(kI_u^*-(\varphi+\mu)S_u^*)+k(\varphi+\mu)=0.$$

Substituting the EEP $E^*=\left(S_u^*,S_c^*,S_f^*,I_u^*,I_c^*,I_f^*,T^*,A^*\right)$, The requirements for stability become;

i. λ_3, λ_4, are negative if

$$\frac{\beta_m(S_f^* - I_f^*)}{2\mu + \gamma + \delta} < 1, \quad \text{and} \quad \frac{\beta_m(\mu S_f^* - (\mu + \gamma + \delta)I_f^*)}{\mu(\mu + \gamma + \delta)} < 1,$$

which, on substitution of S_f^* and I_f^*, become

$$\frac{\beta_m r\Lambda}{(\mu + \delta + \gamma)(2\mu + \gamma + \delta)} > 0, \quad \text{and} \quad \frac{\beta_m r\Lambda}{\mu(\mu + \gamma + \delta)} = R_{of} > 1. \tag{3.24}$$

The first condition is already satisfied since $\beta_m, r, \Lambda, \mu, \delta, \gamma > 0$ and hence, λ_3 and λ_4 are negative if $R_{of} > 1$.

ii. λ_5, λ_6 are negative if

$$\frac{\beta_{fc}(S_c^* - I_c^*)}{2\mu + \gamma + \delta} < 1, \quad \text{and} \quad \frac{\beta_{fc}(\mu S_c^* - (\mu + \gamma + \delta)I_c^*)}{\mu(\mu + \gamma + \delta)} < 1$$

which, on substitution of S_c^* and I_c^*, become

$$\frac{(r-1)g\Lambda\beta_{fu} - \varphi(\mu + \delta + \gamma)}{(\mu + \delta + \gamma)(2\mu + \delta + \gamma)\beta_{fu}} < 1,$$

$$\text{and} \quad \frac{\beta_{fc}\left((1-r)g\Lambda\beta_{fu} + \varphi(\mu + \gamma + \delta)\right)}{\mu\beta_{fu}(\mu + \gamma + \delta)} > 1. \tag{3.25}$$

The first condition is true since $r - 1 < 0$ implies that

$$(r-1)g\Lambda\beta_{fu} - \varphi(\mu + \delta + \gamma) < 0.$$

The second condition becomes

$$\frac{\beta_{fc}\left((1-r)g\Lambda\beta_{fu} + \varphi(\mu + \gamma + \delta)\right)}{\mu\beta_{fu}(\mu + \gamma + \delta)(\mu + \varphi)} > \frac{1}{(\mu + \varphi)}$$

$$\Rightarrow \frac{\beta_{fc}(1-r)(g\mu + \varphi)\Lambda}{\mu(\mu + \gamma + \delta)(\mu + \varphi)}\frac{g}{(g\mu + \varphi)} + \frac{\beta_{fc}\varphi}{\mu\beta_{fu}(\mu + \varphi)} > \frac{1}{(\mu + \varphi)}$$

$$\Rightarrow R_{oc} > \frac{(g\mu + \varphi)}{g(\mu + \varphi)}\left(1 - \frac{\beta_{fc}\varphi}{\mu\beta_{fu}}\right) > 1. \tag{3.26}$$

iii. λ_7, λ_8 are negative if

$$\frac{\beta_{fu}(S_u^* - I_u^*)}{2\mu + \gamma + \delta + \varphi} < 1, \text{ and } \frac{\beta_{fu}\left((\mu + \varphi)S_u^* - (\mu + \gamma + \delta)I_u^*\right)}{(\mu + \gamma + \delta)(\mu + \varphi)} < 1.$$

which, on substitution of S_u^* and I_u^*, become

$$\frac{\beta_{fu}(1-g)(1-r)\Lambda}{(2\mu + \gamma + \delta + \varphi)(\mu + \delta + \gamma)} > 0, \quad \text{and} \quad \frac{\beta_{fu}(1-g)(1-r)\Lambda}{(\mu + \gamma + \delta)(\varphi + \mu)} > 1. \qquad (3.27)$$

The first condition is already satisfied since $0 < g < 1$, $0 < r < 1$ and hence, λ_7 and λ_8 are negative if $R_{ou} > 1$. Hence, E^* is locally asymptotically stable if $R_0 > 1$. ■

3.2.9 Global stability of the endemic equilibrium point (EEP)

Theorem 3.5: Let $E^* = \left(S_u^*, S_c^*, S_f^*, I_u^*, I_c^*, I_f^*, T^*, A^*\right)$ be the endemic equilibrium of (3.1) and V be a positive definite function on some neighborhood of E^*. Then if $\frac{dV}{dt} \leq 0$ then E^* is globally asymptotically stable.

Proof

The global stability of the EEP is investigated using the Lypunov function. We use the logarithmic Lyapunov function as proposed by Korobeinikov and Wake (2000) for an SIS model and extend it to our work. Let the Lyapunov function be

$$V = b_1\left(S_u - S_u^* - S_u^* ln\frac{S_u}{S_u^*}\right) + b_2\left(S_c - S_c^* - S_c^* ln\frac{S_c}{S_c^*}\right) + b_3\left(S_f - S_f^* - S_f^* ln\frac{S_f}{S_f^*}\right) +$$

$$b_4\left(I_u - I_u^* - I_u^* ln\frac{I_u}{I_u^*}\right) + b_5\left(I_c - I_c^* - I_c^* ln\frac{I_c}{I_c^*}\right) + b_6\left(I_f - I_f^* - I_f^* ln\frac{I_f}{I_f^*}\right) \qquad (3.28)$$

The time derivative of V is given by,

$$\frac{dV}{dt} = b_1\left(1-\frac{S_u^*}{S_u}\right)\frac{dS_u}{dt} + b_2\left(1-\frac{S_c^*}{S_c}\right)\frac{dS_c}{dt} + b_3\left(1-\frac{S_f^*}{S_f}\right)\frac{dS_f}{dt} + b_4\left(1-\frac{I_u^*}{I_u}\right)\frac{dI_u}{dt} +$$

$$b_5\left(1-\frac{I_c^*}{I_c}\right)\frac{dI_c}{dt} + b_6\left(1-\right.$$

$$\left.\frac{I_f^*}{I_f}\right)\frac{dI_f}{dt} \qquad (3.29)$$

Substituting $\frac{dS_u}{dt}, \frac{dS_c}{dt}, \frac{dS_f}{dt}, \frac{dI_u}{dt}, \frac{dI_c}{dt}$ and $\frac{dI_f}{dt}$ we obtain;

$$\frac{dV}{dt} = b_1\left(1-\frac{S_u^*}{S_u}\right)\left((1-g)(1-r)\Lambda - \beta_{fu}S_uI_u - (\varphi+\mu)S_u\right)$$

$$+b_2\left(1-\frac{S_c^*}{S_c}\right)\left(g(1-r)\Lambda + \varphi S_u - \beta_{fc}S_cI_c - \mu S_c\right)$$

$$+ b_3\left(1-\frac{S_f^*}{S_f}\right)\left(r\Lambda - \beta_m S_f I_f - \mu S_f\right) + b_4\left(1-\frac{I_u^*}{I_u}\right)\left(\beta_{fu}S_uI_u - \kappa I_u\right)$$

$$+b_5\left(1-\frac{I_c^*}{I_c}\right)\left(\beta_{fc}S_cI_c - \kappa I_c\right) + b_6\left(1-\frac{I_f^*}{I_f}\right)\left(\beta_m S_f I_f - \kappa I_f\right) \qquad (3.30)$$

From system (3.1), then at equilibrium the following relations are true;

$$\left.\begin{array}{c}(1-g)(1-r)\Lambda = \beta_{fu}S_u^*I_u^* - (\varphi+\mu)S_u^* \\ g(1-r)\Lambda = -\varphi S_u^* + \beta_{fc}S_c^*I_c^* + \mu S_c^* \\ r\Lambda = \beta_m S_f^* I_f^* + \mu S_f^* \\ \kappa = \beta_{fu}S_u^* = \beta_{fc}S_c^* = \beta_m S_f^*\end{array}\right\} \qquad (3.31)$$

Substituting (3.31) into (3.30) and simplifying gives;

$$\frac{dV}{dt} = -\left[b_1(\varphi+\mu)\frac{(S_u - S_u^*)^2}{S_u} + b_2\mu\varphi\frac{(S_c - S_c^*)^2(S_u - S_u^*)}{S_c} + b_3\mu\frac{\left(S_f - S_f^*\right)^2}{S_f}\right]$$

$$+\left[b_1\left(\frac{S_u - S_u^*}{S_u}\right)\left(\beta_{fu}S_u^*I_u^* - \beta_{fu}S_uI_u\right)\right.$$

$$+ b_2\left(\frac{S_c - S_c^*}{S_c}\right)\left(\beta_{fc}S_c^*I_c^* - \beta_{fc}S_cI_c\right) + b_3\left(\frac{S_f - S_f^*}{S_f}\right)\left(\beta_mS_f^*I_f^* - \beta_mS_fI_f\right)$$

$$+ b_4\beta_{fu}(S_u - S_u^*)(I_u - I_u^*) + b_5\beta_{fc}(S_c - S_c^*)(I_c - I_c^*)$$

$$\left.+ b_6\beta_m\left(S_f - S_f^*\right)\left(I_f - I_f^*\right)\right] \qquad (3.32)$$

$E^* = \left(S_u^*, S_c^*, S_f^*, I_u^*, I_c^*, I_f^*, T^*, A^*\right)$ is globally asymptotically stable if $\frac{dV}{dt} = 0$ and $\frac{dV}{dt} < 0$, using equation (5) then $\frac{dV}{dt} = 0$ if and only if $S_u = S_u^*$, $S_c = S_c^*$, $S_f = S_f^*$, $I_u = I_u^*$, $I_c = I_c^*$ and $I_f = I_f^*$.

Also $\frac{dV}{dt} < 0$ if and only if

$$-\left[b_1(\varphi+\mu)\frac{(S_u - S_u^*)^2}{S_u} + b_2\mu\varphi\frac{(S_c - S_c^*)^2(S_u - S_u^*)}{S_u} + b_3\mu\frac{\left(S_f - S_f^*\right)^2}{S_f}\right]$$

$$+\left[b_1\frac{(S_u - S_u^*)^2}{S_u}\left(\beta_{fu}S_u^*I_u^* - \beta_{fu}S_uI_u\right)\right.$$

$$+ b_2\frac{(S_c - S_c^*)^2}{S_u}\left(\beta_{fc}S_c^*I_c^* - \beta_{fc}S_cI_c\right) + b_3\frac{\left(S_f - S_f^*\right)^2}{S_f}\left(\beta_mS_f^*I_f^* - \beta_mS_fI_f\right)$$

$$+ b_4\beta_{fu}(S_u - S_u^*)(I_u - I_u^*) + b_5\beta_{fc}(S_c - S_c^*)(I_c - I_c^*)$$

$$\left.+ b_6\beta_m\left(S_f - S_f^*\right)\left(I_f - I_f^*\right)\right] < 0$$

Which implies that;

$$\left[b_1\frac{(S_u-S_u^*)^2}{S_u}\left(\beta_{fu}S_u^*I_u^*-\beta_{fu}S_uI_u\right)+b_2\frac{(S_c-S_c^*)^2}{S_u}\left(\beta_{fc}S_c^*I_c^*-\beta_{fc}S_cI_c\right)+\right.$$

$$b_3\frac{\left(S_f-S_f^*\right)^2}{S_f}\left(\beta_mS_f^*I_f^*-\beta_mS_fI_f\right)+b_4\beta_{fu}(S_u-S_u^*)(I_u-I_u^*)+b_5\beta_{fc}(S_c-S_c^*)(I_c-I_c^*)+$$

$$\left.b_6\beta_m\left(S_f-S_f^*\right)\left(I_f-I_f^*\right)\right]<\left[b_1(\varphi+\mu)\frac{(S_u-S_u^*)^2}{S_u}+b_2\mu\varphi\frac{(S_c-S_c^*)^2(S_u-S_u^*)}{S_u}+b_3\mu\frac{\left(S_f-S_f^*\right)^2}{S_f}\right]$$

■

3.3 Model 2 Mathematical Model for HIV Transmission Between Truckers and Fsws in Kenya With Circumcision and Funding as Control Measures

Global HIV funding is still not adequate to respond to the rising infections in the general population and the vulnerable populations. Recently, resources for HIV responses in low- and middle-income countries have been decreasing since 2018. UNAIDS ambitious Fast-Track approach is committed to ending the global HIV epidemic public health threat by 2030. As a result, UNAIDS estimated an annual investment of 26.2 billion USD for HIV-response in 2020, which would decrease steadily to 23.9 billion USD by 2030 (UNAIDS, 2020). Increases in resources for HIV responses in low- and middle-income countries stopped in 2017, with funding decreasing by 7% between 2017 and 2019 according to Dutta *et al.,* (2015). By the end of 2019, there was 18.6 billion USD available for the HIV response in low- and middle-income countries which was approximately 71% of the 2020 target. UNAIDS estimated that annual investments needed to be increased to 29 billion USD by 2025 to get the AIDS response back on track in low- and middle-income countries where Kenya is part (UNAIDS, 2020). To deliver enhanced integrated health care for critical groups in Kenya, HIV financing remains a major barrier that must be tackled.

Donor funding accounted for nearly 75% of Kenya's national HIV response in 2015 according to *Kenya AIDS Response Progress Report* (2016). However, diminishing foreign donor money creates a difficulty for Kenya's HIV response because government expenditure nearly doubled between 2006 and 2012 (from USD 57.49 million to USD 153 million) by the Kenyan Ministry of Health/National AIDS Control Council (*Kenya AIDS Response Progress Report 2016*, 2016). Kenya has made significant progress in combating the HIV pandemic through pioneering HIV prevention, including the use of VMMC, self-testing and PrEP. Also, the Kenya HIV Prevention Revolution Road Map is aiming to reduce 1.1 million new infections and 761,000 deaths associated with AIDS by 2030 according to Kenya H.I.V (2014). This information indicates a very close relationship between the HIV pandemic and funding and hence, this study considers the effect of funding on HIV transmission dynamics between truckers and female sex workers. This study identified the trend to expect at the different funding levels in its simulation.

3.3.1 Model formulation

A mathematical model is formulated to study the combined effects of funding and circumcision on HIV/AIDS transmission dynamics between truckers and FSWs along the NCH in Kenya. The population is divided into eight compartments, namely; (1) three susceptible classes S_u, S_c, S_f of uncircumcised susceptible male group, circumcised susceptible male group and susceptible female group respectively. (2) three infected classes I_u, I_c, I_f of uncircumcised Infected males, circumcised Infected males and Infected females. (3) treated group T and (4) the AIDS group A.

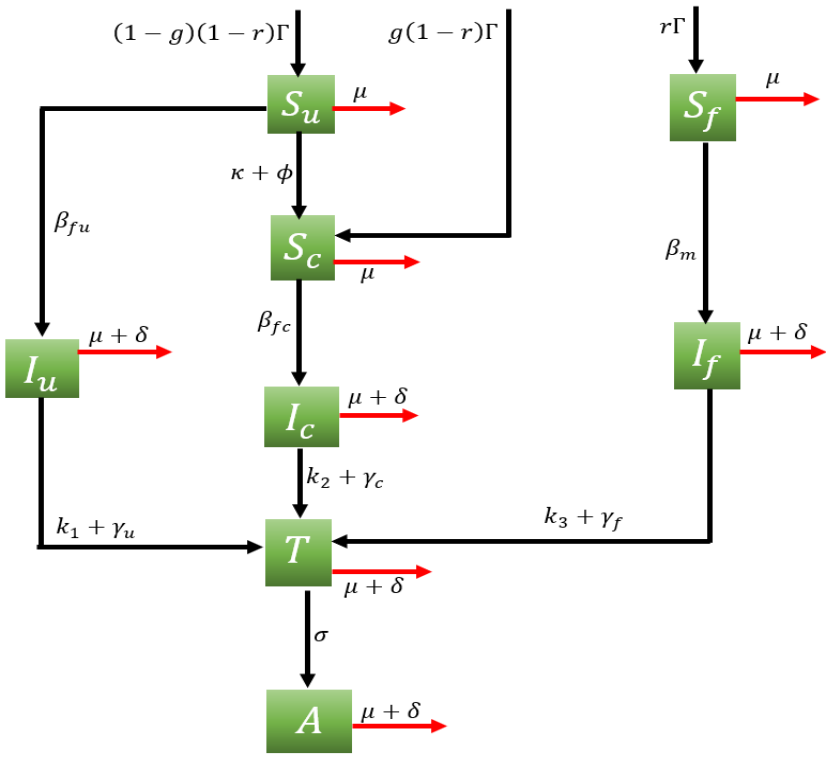

Figure 3.2: Flow diagram for the mathematical model of the effect of funding on HIV transmission dynamics between truckers and female sex workers

The model is based on the following assumptions;

(1) All infected population is subjected to treatment.

(2) Circumcision is done at a young age but adult male truckers can be subjected to VMC.

(3) The main clients of the female sex workers were truckers.

(4) Only the adult population is considered.

Table 3.3: Description of parameters

Parameter	Definition
Γ	recruitment rate into susceptible population
g	proportion of susceptible circumcised males
r	proportion of females recruited into FSWs
γ_u	Rate at which infected uncircumcised males access treatment
γ_c	Rate at which infected circumcised males access treatment
γ_f	Rate at which infected females access treatment
σ	Rate at which treated individuals progress to AIDS
μ	natural mortality rate
δ	disease-induced mortality rate
ϕ	Circumcision rate
β_{fu}	Transmission rate of HIV from females to uncircumcised males
β_{fc}	Transmission rate of HIV from females to circumcised males
β_m	Transmission rate of HIV from males to females
κ	Rate of funding circumcision
κ_1	Rate of funding infected uncircumcised males for treatment
κ_2	Rate of funding infected circumcised males for treatment
κ_3	Rate of funding infected females for treatment

The model is governed by the following ordinary differential equations;

$$\left.\begin{aligned}
\frac{dS_u}{dt} &= (1-g)(1-r)\Gamma - \beta_{fu}S_uI_u - (\kappa+\phi+\mu)S_u \\
\frac{dS_c}{dt} &= g(1-r)\Gamma + (\kappa+\phi)S_u - \beta_{fc}S_cI_c - \mu S_c \\
\frac{dS_f}{dt} &= r\Gamma - \beta_m S_f I_f - \mu S_f \\
\frac{dI_u}{dt} &= \beta_{fu}S_uI_u - [(\kappa_1+\gamma_u)+(\mu+\delta)]I_u \\
\frac{dI_c}{dt} &= \beta_{fc}S_cI_c - [(\kappa_2+\gamma_c)+(\mu+\delta)]I_c \\
\frac{dI_f}{dt} &= \beta_m S_f I_f - \left[\left(\kappa_3+\gamma_f\right)+(\mu+\delta)\right]I_f \\
\frac{dT}{dt} &= (\kappa_1+\gamma_u)I_u + (\kappa_2+\gamma_c)I_c + (\kappa_3+\gamma_f)I_f - ((\mu+\delta+\sigma)T \\
\frac{dA}{dt} &= \sigma T - (\mu+\delta)A
\end{aligned}\right\} \qquad (3.34)$$

Where all parameters lie between 0 and 1 and

$$0 < \kappa + \phi + \beta_{fu} < 1,$$

$$0 < \beta_{fc} + \mu < 1,$$

$$0 < \beta_m + \mu < 1,$$

$$0 < \kappa_1 + \gamma_u + \mu + \delta < 1,$$

$$0 < \kappa_2 + \gamma_c + \mu + \delta < 1,$$

$$0 < \kappa_3 + \gamma_f + \mu + \delta < 1,$$

$$0 < \sigma + \mu + \delta < 1.$$

3.3.2 Model Anaysis

In this section, we analyse the model which includes; invariant region, positivity of the model, reproduction number of HIV between the truckers and FSWs, the disease-free equilibrium (DFE), endemic equilibrium point (EEP) and local stability of DFE and EEP with funding parameters.

The invariant region of the model is where the solutions of the model are feasible. The total population N is defined as

$$N = S_u + S_c + S_f + I_u + I_c + I_f + T + A.$$

The time derivatives of our total population along the solution path are given by

$$\left.\begin{aligned} \frac{dN}{dt} &= \frac{dS_u}{dt} + \frac{dS_c}{dt} + \frac{dS_f}{dt} + \frac{dI_u}{dt} + \frac{dI_c}{dt} + \frac{dI_f}{dt} + \frac{dT}{dt} + \frac{dA}{dt}, \\ \frac{dN}{dt} &= \Gamma - \mu N - \delta(I + T + A), \\ I &= I_u + I_c + I_f \end{aligned}\right\} \qquad (3.35)$$

In absence of disease mortality rate then $\delta = 0$, and (3.35) reduces to

$$\frac{dN}{dt} \leq \Gamma - \mu N \qquad (3.36)$$

Integrating the differential equation with respect to time we get

$$N \leq \frac{\Gamma}{\mu} + \frac{C}{e^{\mu t}}$$

where C is the constant of integration. As t tends to infinity the limit of $N(t)$ becomes

$$\lim_{t \to \infty} N(t) \leq \frac{\Gamma}{\mu}$$

hence $N(t)$ is clearly bounded and

$$0 < N(t) \leq \frac{\Gamma}{\mu}$$

therefore, we conclude that the feasible solutions of the set of the system of equations enter and remains in the region Ω for all future time and

$$\Psi = \left\{S_u, S_c, S_f, I_u, I_c, I_f, T, A \in \mathbb{R}_+^8 : 0 < N(t) \leq \frac{\Gamma}{\mu}\right\} \qquad (3.37)$$

This implies that the model is well-posed and epidemiologically making sense, therefore we study the dynamics of the model in Ψ.

Theorem 3.6: If all parameters of the system (3.34) are nonnegative and satisfy the initial conditions $0 < S_u(0), S_c(0), S_f(0), I_u(0), I_c(0), I_f(0), T(0), A(0) \in \Psi$, then the solution set $\{S_u(t), S_c(t), S_f(t), I_u(t), I_c(t), I_f(t), T(t), A(t) > 0\}$ of the system (4.1) is also nonnegative for all $t \geq 0$

Proof

Consider the first equation of the system (3.34)

$$\frac{dS_u}{dt} = (1-g)(1-r)\Gamma - \beta_{fu} S_u I_u - (\kappa + \phi + \mu) S_u \tag{3.38}$$

Let

$$\omega = (1-g)(1-r), \quad \eta = \beta_{fu} I_u, \quad \zeta = \kappa + \phi + \mu, \tag{3.39}$$

then equation (4.5) becomes

$$\frac{dS_u}{dt} = \omega\Gamma - \eta S_u - \zeta S_u \tag{3.40}$$

Expression (4.7) can be written as

$$\frac{d}{dt}\left(S_u e^{\left\{\int_0^t \zeta(u)du + \mu t\right\}}\right) \leq (\omega\Gamma) e^{\left\{\int_0^t \zeta(u)du + \mu t\right\}} \tag{3.41}$$

On integrating both sides of (3.41) with respect to t we get

$$S_u(t) e^{\left\{\int_0^t \zeta(u)du + \mu t\right\}} - S_u(0) \leq (\omega\Gamma) e^{\{\int_0^\tau \zeta(\tau)d\tau + \mu x\}dx} \tag{3.42}$$

If we multiply expression (3.42) by $\exp\left(-\int_0^{t^*} \zeta(u)du - \mu_v t\right)$ we obtain

$$S_u(\hat{t}) \leq S_u(0)e^{\left\{-\int_0^t \zeta(u)du-\mu t\right\}} + \omega\Gamma e^{\left\{-\int_0^t \zeta(u)du-\mu t\right\}} e^{\left\{\int_0^{\hat{\tau}} \zeta(\tau)d\tau+\mu x\right\}dx}$$

$$> 0 \qquad (3.43)$$

The right-hand side of expression (3.43) is positive, then the solution $S_u(t)$ will remain positive for all $t > 0$. Similarly, using the above argument it can be shown that $S_c, S_f, I_u, I_c, I_f, T, A$ are nonnegative for all $t > 0$. ■

3.3.3 Equilibrium Points

The Disease-Free Equilibrium (DFE) Point is obtained by setting

$$I_u = I_c = I_f = T = A = 0$$

in the system (4.1) to get

$$S_u = \frac{(1-g)(1-r)\Gamma}{\kappa+\phi+\mu}, \qquad S_c = \frac{(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)}, \qquad S_f = \frac{r\Gamma}{\mu}$$

Therefore, the DFE is given by

$$E^0 = \left(S_u^0, S_c^0, S_f^0, I_u^0, I_c^0, I_f^0, T^0, A^0\right)$$

$$= \left(\frac{(1-g)(1-r)\Gamma}{\kappa+\phi+\mu}, \frac{(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)}, \frac{r\Gamma}{\mu}, 0,0,0,0,0\right) \qquad (3.44)$$

Endemic equilibrium points EEP is the points at which the disease persists within the population. For this system, let the endemic equilibrium points EEP, $E^* = \left(S_u^*, S_c^*, S_f^*, I_u^*, I_c^*, I_f^*, T^*, A^*\right)$, then the EEP is given by;

$$S_u^* = \frac{\kappa_1+\gamma_u+\kappa_5}{\beta_{fu}}, \quad S_c^* = \frac{\kappa_2+\gamma_c+\kappa_5}{\beta_{fc}}, \quad S_f^* = \frac{\kappa_3+\gamma_f+\kappa_5}{\beta_m},$$

$$I_u^* = \frac{\beta_{fu}(1-g)(1-r)\Gamma - (\kappa+\phi+\mu)(\kappa_1+\gamma_u+\kappa_5)}{\beta_{fu}(\kappa_1+\gamma_u+\kappa_5)},$$

$$I_f^* = \frac{\beta_m r\Gamma - \mu(\kappa_3+\gamma_f+\kappa_5)}{\beta_m(\kappa_3+\gamma_f+\kappa_5)},$$

$$I_c^* = \frac{\beta_{fc}(1-r)\Gamma(g\mu+\kappa+\phi) - \mu(\kappa_2+\gamma_c+\kappa_5)(\kappa+\phi+\mu)}{\beta_{fc}(\kappa_2+\gamma_c+\kappa_5)(\kappa+\phi+\mu)},$$

$$T^* = \frac{(\kappa_1+\gamma_u)\beta_{fu}(1-g)(1-r)\Gamma - (\kappa_1+\gamma_u)(\kappa_1+\gamma_u+\kappa_5)(\kappa+\phi+\mu)}{\kappa_4\beta_{fu}(\kappa_1+\gamma_u+\kappa_5)}$$

$$+\frac{(\kappa_2+\gamma_c)\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma - \mu(\kappa_2+\gamma_c)(\kappa_2+\gamma_c+\kappa_5)(\kappa+\phi+\mu)}{\kappa_4\beta_{fc}(\kappa_2+\gamma_c+\kappa_5)(\kappa+\phi+\mu)}$$

$$+\frac{(\kappa_3+\gamma_f)\beta_m r\Gamma - \mu(\kappa_3+\gamma_f)(\kappa_3+\gamma_f+\kappa_5)}{\kappa_4\beta_m(\kappa_3+\gamma_f+\kappa_5)},$$

$$A^* = \frac{\sigma}{\kappa_4\kappa_5}\left[\frac{(\kappa_1+\gamma_u)\beta_{fu}(1-g)(1-r)\Gamma - (\kappa_1+\gamma_u)(\kappa_1+\gamma_u+\kappa_5)(\kappa+\phi+\mu)}{\beta_{fu}(\kappa_1+\gamma_u+\kappa_5)}\right.$$

$$+\frac{(\kappa_2+\gamma_c)\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma - \mu(\kappa_2+\gamma_c)(\kappa_2+\gamma_c+\kappa_5)(\kappa+\phi+\mu)}{\kappa_4\beta_{fc}(\kappa_2+\gamma_c+\kappa_5)(\kappa+\phi+\mu)}$$

$$\left.+\frac{(\kappa_3+\gamma_f)\beta_m r\Gamma - \mu(\kappa_3+\gamma_f)(\kappa_3+\gamma_f+\kappa_5)}{\beta_m(\kappa_3+\gamma_f+\kappa_5)}\right] \qquad (3.45)$$

3.3.4 The Reproduction Number

The reproduction number R_0 is the number of secondary infections that an infectious individual can cause when introduced into a susceptible population. The next-generation matrix is used to obtain R_0. Define F_i and V_i as matrices that represent the rate at which new infections occurs and the rate of transfer of individuals out of compartments. The R_0 was obtained from the spectral radius of the $F_0 V_0^{-1}$ matrix. From system (3.34) then;

$$\mathcal{F} = \begin{pmatrix} \beta_{fu} S_u I_u \\ \beta_{fc} S_c I_c \\ \beta_m S_f I_f \\ 0 \\ 0 \end{pmatrix} \text{ and}$$

$$\mathcal{V} = \begin{pmatrix} (\kappa_1 + \gamma_u + \mu + \delta) I_u \\ (\kappa_2 + \gamma_c + \mu + \delta) I_c \\ (\kappa_3 + \gamma_f + \mu + \delta) I_f \\ -(\kappa_1 + \gamma_u) I_u - (\kappa_2 + \gamma_c) I_c - (\kappa_3 + \gamma_f) I_f + (\mu + \delta + \sigma) T \\ -\sigma T + (\mu + \delta) A \end{pmatrix} \quad (3.46)$$

Let F and V be the Jacobian of $\mathcal{F}$ at DFE and the Jacobian of $\mathcal{V}$ at DFE so that,

$$F = \begin{pmatrix} \beta_{fu} S_u^0 & 0 & 0 & 0 & 0 \\ 0 & \beta_{fc} S_c^0 & 0 & 0 & 0 \\ 0 & 0 & \beta_m S_f^0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} \kappa_1 + \gamma_u + \mu + \delta & 0 & 0 & 0 & 0 \\ 0 & \kappa_2 + \gamma_c + \mu + \delta & 0 & 0 & 0 \\ 0 & 0 & \kappa_3 + \gamma_f + \mu + \delta & 0 & 0 \\ -\kappa_1 - \gamma_u & -\kappa_2 - \gamma_c & -\kappa_3 - \gamma_f & \mu + \delta + \sigma & 0 \\ 0 & 0 & 0 & -\sigma & \mu + \delta \end{pmatrix}$$

Hence for system (3.34) the next generation matrix according to (Oke *et al.*, 2021; Oke & Bada, 2019) is;

$$FV^{-1} = \begin{pmatrix} \frac{\beta_{fu} S_u^0}{\kappa_1 + \gamma_u + \mu + \delta} & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_{fc} S_c^0}{\kappa_2 + \gamma_c + \mu + \delta} & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_m S_f^0}{\kappa_3 + \gamma_f + \mu + \delta} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

The characteristic polynomial of $FV^{-1} - \lambda I$ at DFE is given by

$$\lambda^2\left(-\lambda+\frac{\beta_m r\Gamma}{\mu\left(\kappa_3+\gamma_f+\mu+\delta\right)}\right)\left(-\lambda+\frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)(\kappa_2+\gamma_c+\mu+\delta)}\right)\left(-\lambda\right.$$

$$\left.+\frac{\beta_{fu}(1-g)(1-r)\Gamma}{(\kappa+\phi+\mu)(\kappa_1+\gamma_u+\mu+\delta)}\right)=0 \qquad (3.47)$$

The eigenvalues are

$$\lambda_1=\lambda_2=0,\ \ \lambda_3=\frac{\beta_m r\Gamma}{\mu\left(\kappa_3+\gamma_f+\mu+\delta\right)},$$

$$\lambda_4=\frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)(\kappa_2+\gamma_c+\mu+\delta)},\lambda_5=\frac{\beta_{fu}(1-g)(1-r)\Gamma}{(\kappa+\phi+\mu)(\kappa_1+\gamma_u+\mu+\delta)},$$

λ_3,λ_4 and λ_5 gives the basic reproduction numbers for the system (3.34) as;

$$R_{of}=\frac{\beta_m r\Gamma}{\mu\left(\kappa_3+\gamma_f+\mu+\delta\right)},$$

$$R_{oc}=\frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)(\kappa_2+\gamma_c+\mu+\delta)},$$

$$\text{and}\quad R_{ou}=\frac{\beta_{fu}(1-g)(1-r)\Gamma}{(\kappa+\phi+\mu)(\kappa_1+\gamma_u+\mu+\delta)}. \qquad (3.48)$$

3.3.5 Stability Analysis of DFE and EEP

Theorem 3.7: Let $R_0=max\left(R_{of},R_{oc},R_{ou}\right)$, then the disease-free equilibrium E^0 of the system (3.34) is locally asymptotically stable if $R_0<1$ while the endemic equilibrium point E^* is locally asymptotically stable if $R_0>1$.

***Proof*:**

The Jacobian matrix of the system is

$$J=\begin{pmatrix} -\beta_{fu}I_u-(\kappa+\phi+\mu) & 0 & 0 & -\beta_{fu}S_u & 0 & 0 & 0 & 0 \\ \kappa+\phi & -\beta_{fc}I_c-\mu & 0 & 0 & -\beta_{fc}S_c & 0 & 0 & 0 \\ 0 & 0 & -\beta_m I_f-\mu & 0 & 0 & -\beta_m S_f & 0 & 0 \\ \beta_{fu}I_u & 0 & 0 & \beta_{fu}S_u-(k_1+\gamma_u+\kappa_5) & 0 & 0 & 0 & 0 \\ 0 & \beta_{fc}I_c & 0 & 0 & \beta_{fc}S_c-(k_2+\gamma_c+\kappa_5) & 0 & 0 & 0 \\ 0 & 0 & \beta_m I_f & 0 & 0 & \beta_m S_f-(k_3+\gamma_f+\kappa_5) & 0 & 0 \\ 0 & 0 & 0 & k_1+\gamma_u & k_2+\gamma_c & k_3+\gamma_f & -k_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \sigma & -k_5 \end{pmatrix}$$

where $k_4=\mu+\delta+\sigma,\ k_5=\mu+\delta$. The first two eigenvalues are

$\lambda_1=-(\mu+\delta)$, and $\lambda_2=-(\mu+\delta+\sigma)$. λ_3 and λ_4 are obtained from;

$$\lambda^2-\Big(\big(\beta_{fu}S_u-(k_1+\gamma_u+\kappa_5)\big)-\big(\beta_{fu}I_u-(\kappa+\phi+\mu)\big)\Big)\lambda + \beta_{fu}\big(I_u(k_1+\gamma_u+\kappa_5)-S_u(\kappa+\phi+\mu)\big)+(\kappa+\phi+\mu)(k_1+\gamma_u+\kappa_5) = 0,$$

and therefore λ_3 and λ_4 are negative if

$$\frac{\beta_{fu}(S_u-I_u)}{\mu+k_1+\gamma_u+\kappa_5}<1,\ \text{and}\ \frac{\beta_{fu}\big(S_u(\kappa+\phi+\mu)-I_u(k_1+\gamma_u+\kappa_5)\big)}{(\kappa+\phi+\mu)(k_1+\gamma_u+\kappa_5)}<1. \qquad (3.49)$$

Similarly, λ_5 and λ_6 are obtained from

$$\lambda^2-\Big(\big(\beta_{fc}S_c-(k_2+\gamma_c+\kappa_5)\big)-\big(\beta_{fc}I_c+\mu\big)\Big)\lambda+\beta_{fc}(I_c(k_2+\gamma_c+\kappa_5)-\mu S_c) + \mu(k_2+\gamma_c+\kappa_5)=0,$$

and therefore λ_5 and λ_6 are negative if

$$\frac{\beta_{fc}(S_c-I_c)}{\mu+k_2+\gamma_c+\kappa_5}<1,\ \text{and}\ \frac{\beta_{fc}\big(\mu S_c-I_c(k_c+\gamma_c+\kappa_5)\big)}{\mu(k_2+\gamma_c+\kappa_5)}<1. \qquad (3.50)$$

In a similar manner λ_7 and λ_8 are obtained from

$$\lambda^2 - \left(\beta_m(S_f - I_f) - (\mu + k_3 + \gamma_f + \kappa_5)\right)\lambda + \beta_m\left(I_f(k_3 + \gamma_f + \kappa_5) - \mu S_f\right) + \mu(k_3 + \gamma_f + \kappa_5) = 0$$

and λ_7 and λ_8 are negative if

$$\frac{\beta_m(S_f - I_f)}{\mu + k_3 + \gamma_f + \kappa_5} < 1, \text{ and } \frac{\beta_m\left(\mu S_f - I_f(k_3 + \gamma_f + \kappa_5)\right)}{\mu(k_3 + \gamma_f + \kappa_5)} < 1. \quad (3.51)$$

Case 1: At the disease-free-equilibrium ***DFE***

$$E^0 = \left(\frac{(1-g)(1-r)\Gamma}{\kappa + \phi + \mu}, \frac{(1-r)(g\mu + \kappa + \phi)\Gamma}{\mu(\kappa + \phi + \mu)}, \frac{r\Gamma}{\mu}, 0,0,0,0,0\right)$$

The requirements for stability therefore are;

i. λ_3, λ_4, are negative if

$$\frac{\beta_{fu}(1-g)(1-r)\Gamma}{(2\mu + k_1 + \gamma_u + \delta)(\kappa + \phi + \mu)} < 1, \text{ and } \frac{\beta_{fu}(1-g)(1-r)\Gamma}{(\kappa + \phi + \mu)(k_1 + \gamma_u + \kappa_5)} < 1$$

and thus

$$\frac{\beta_{fu}(1-g)(1-r)\Gamma}{(2\mu + k_1 + \gamma_u + \delta)(\kappa + \phi + \mu)} < \frac{\beta_{fu}(1-g)(1-r)\Gamma}{(\kappa + \phi + \mu)(k_1 + \gamma_u + \kappa_5)} < 1 \Rightarrow R_{ou} < 1. \quad (3.52)$$

ii. λ_5, λ_6 are negative if

$$\frac{\beta_{fc}(1-r)(g\mu + \kappa + \phi)\Gamma}{\mu(\kappa + \phi + \mu)(2\mu + k_2 + \gamma_c + \delta)} < 1,$$

$$\frac{\beta_{fc}(1-r)(g\mu + \kappa + \phi)\Gamma}{\mu(\kappa + \phi + \mu)(k_2 + \gamma_c + \mu + \delta)} < 1$$

and thus

$$\frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)(2\mu+k_2+\gamma_c+\delta)} < \frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)(k_2+\gamma_c+\mu+\delta)} < 1$$

$$\Rightarrow R_{oc} < 1 \tag{3.53}$$

iii. λ_3, λ_4, are negative if

$$\frac{\beta_m r\Gamma}{\mu(2\mu+k_3+\gamma_f+\delta)} < 1, \text{ and } \frac{\beta_m r\Gamma}{\mu(k_3+\gamma_f+\mu+\delta)} < 1$$

and thus

$$\frac{\beta_m r\Gamma}{\mu(2\mu+k_3+\gamma_f+\delta)} < \frac{\beta_m r\Gamma}{\mu(k_3+\gamma_f+\mu+\delta)} < 1$$

$$\Rightarrow R_{of} < 1. \tag{3.54}$$

Hence, E^0 is locally asymptotically stable if $R_0 < 1$. ■

Theorem of Castillo – Chavez and Song, B. (2004) was used to prove global stability of DFE,

Theorem 3.8. The disease free equilibrium point

$$E^0 = \left(S_u^0, S_c^0, S_f^0, I_u^0, I_c^0, I_f^0, T^0, A^0\right)$$

$$= \left(\frac{(1-g)(1-r)\Gamma}{\kappa+\phi+\mu}, \frac{(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)}, \frac{r\Gamma}{\mu}, 0,0,0,0,0\right)$$

is globally asymptotically stable, if $R_0 < 1$ is locally asymptotically stable and assumption $(H1)$ and $(H2)$ are satisfied.

Proof. The equation (3.1) is written as $\frac{dX}{dt} = F(X,Z), \frac{dZ}{dt} = G(X,Z)$, where $X = (S_u, S_c, S_f)$ represents the disease free classes and $Z = \left(I_u, I_c, I_f, T, A\right)$ represents the

infected classes. $G(X,0)$ represents the disease free equilibrium point, E^0 of the model. The conditions $(H1)$ and $(H2)$ are;

H1: for $\frac{dX}{dt} = F(X,0)$, E^0 is globally asymptotically stable

H2: $G(X,Z) = AZ - \tilde{G}(X,Z)$, $\tilde{G}(X,Z) \geq 0$ for $(X,Z) \in {\mathbb{R}_+}^5$ where $A = D_Z\, G(X,0)$ is an M- matrix (all off-diagonal elements are nonnegative) and ${\mathbb{R}_+}^5$ is the region where the model makes biological sense.

In our case then;

$$F(X,0) = F\big((X_1,0),(X_2,0),(X_3,0)\,\big) = \Gamma - \mu S,$$

$$S = \big(S_u, S_c, S_f\big)$$

$$X = \big(S_u, S_c, S_f\big)$$

and

$$Z = \big(I_u, I_c, I_f, T, A\big).$$

Also $k_4 = \mu + \delta + \sigma$, $k_5 = \mu + \delta$

$$A = \begin{pmatrix} \beta_{fu}S_u - \big(\kappa_1 + \gamma_u + \kappa_5\big) & 0 & 0 & 0 & 0 \\ 0 & \beta_{fc}S_c - \big(\kappa_2 + \gamma_u + \kappa_5\big) & 0 & 0 & 0 \\ 0 & 0 & \beta_m S_f - \big(\kappa_3 + \gamma_u + \kappa_5\big) & 0 & 0 \\ \kappa_1 + \gamma_u & \kappa_2 + \gamma_u & \kappa_3 + \gamma_u & -\kappa_4 & 0 \\ 0 & 0 & 0 & \sigma & -\kappa_5 \end{pmatrix}$$

$$AZ = \begin{pmatrix} \beta_{fu}S_u^0 I_u - (\kappa_1 + \gamma_u + \kappa_5)I_u \\ \beta_{fc}S_c^0 I_c - (\kappa_2 + \gamma_u + \kappa_5)I_c \\ \beta_m S_f^0 I_f - (\kappa_3 + \gamma_u + \kappa_5)I_f \\ (\kappa_1 + \gamma_u)I_u + (\kappa_2 + \gamma_u)I_c + (\kappa_3 + \gamma_u)I_f - \kappa_4 T \\ \sigma T - \kappa_5 A \end{pmatrix} \qquad (3.55)$$

$$G(X,Z) = \begin{pmatrix} \beta_{fu}S_u I_u - (\kappa_1 + \gamma_u + \kappa_5)I_u \\ \beta_{fc}S_c I_c - (\kappa_2 + \gamma_u + \kappa_5)I_c \\ \beta_m S_f I_f - (\kappa_3 + \gamma_u + \kappa_5)I_f \\ (\kappa_1 + \gamma_u)I_u + (\kappa_2 + \gamma_u)I_c + (\kappa_3 + \gamma_u)I_f - \kappa_4 T \\ \sigma T - \kappa_5 A \end{pmatrix}$$

$$\tilde{G}(X,Z) = AZ - G(X,Z) = \begin{pmatrix} \frac{\beta_{fu} I_u}{\beta_{fu} S_u I_u}(S_u^0 - 1) \\ \frac{\beta_{fc} I_c}{\beta_{fc} S_c I_c}(S_c^0 - 1) \\ \frac{\beta_m I_f}{\beta_m S_f I_f}(S_f^0 - 1) \\ 0 \\ 0 \end{pmatrix} \tag{3.56}$$

Where $S_u^0 = \frac{(1-g)(1-r)\Gamma}{\kappa+\phi+\mu}, S_c^0 = \frac{(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)}$ and $S_f^0 = \frac{r\Gamma}{\mu}$

From the second condition $\tilde{G}(X,Z) \geq 0$, hence

$$\frac{(1-g)(1-r)\Gamma}{\kappa+\phi+\mu} - 1 \geq 0, \frac{(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)} - 1 \geq 0, \text{and } \frac{r\Gamma}{\mu} - 1 \geq 0.$$

Since all parameters lies between 0 and 1 then we have;

$$\frac{(1-g)(1-r)\Gamma}{\kappa+\phi+\mu} < 1, \frac{(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)} < 1 \text{ and } \frac{r\Gamma}{\mu} < 1$$

and therefore $\tilde{G}(X,Z) \leq 0$ which shows that

$$E^0 = \left(\frac{(1-g)(1-r)\Gamma}{\kappa+\phi+\mu}, \frac{(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\kappa+\phi+\mu)}, \frac{r\Gamma}{\mu}, 0,0,0,0,0\right)$$

is globally asymptotically unstable point of $\frac{dX}{dt} = F(X,0)$ when $R_0 < 1$ ■

Case 2: At the endemic equilibrium point ***EEP***,

$$E^* = \left(S_u^*, S_c^*, S_f^*, I_u^*, I_c^*, I_f^*, T^*, A^*\right)$$

The first two eigenvalues are $\lambda_1 = -(\mu+\delta)$, and $\lambda_2 = -(\mu+\delta+\sigma)$ and the other eigenvalues $\lambda_3 \ldots \lambda_8$ are negative under the following conditions:

- λ_3 and λ_4 are negative if

$$\frac{\beta_{fu}(S_{u2}^{*} - I_{u2}^{*})}{\mu + k_1 + \gamma_u + \kappa_5} < 1, \text{ and } \frac{\beta_{fu}\left(S_{u2}^{*}(\kappa + \phi + \mu) - I_{u2}^{*}(k_1 + \gamma_u + \kappa_5)\right)}{(\kappa + \phi + \mu)(k_1 + \gamma_u + \kappa_5)} < 1$$

which, on substitution of S_u^* and I_u^*, become

$$\frac{\beta_{fu}(1-g)(1-r)\Gamma}{(2\mu + \gamma + \delta + \kappa + \phi)(\mu + \delta + \gamma)} > 0, \text{ and } \frac{\beta_{fu}(1-g)(1-r)\Gamma}{(\mu + \gamma + \delta)(\kappa + \phi + \mu)} > 1.$$

The first condition is satisfied since $0 < g < 1$, $0 < r < 1$ and hence, λ_3 and λ_4 are negative if

$$R_{ou} > 1. \tag{3.57}$$

- λ_5 and λ_6 are negative if

$$\frac{\beta_{fc}(S_c^{*} - I_c^{*})}{\mu + k_2 + \gamma_c + \kappa_5} < 1, \text{ and } \frac{\beta_{fc}\left(\mu S_c^{*} - I_c^{*}(k_c + \gamma_c + \kappa_5)\right)}{\mu(k_2 + \gamma_c + \kappa_5)} < 1.$$

which, on substitution of S_c^* and I_c^*, become

$$\frac{(r-1)g\beta_{fu}\Gamma - (\kappa + \phi)(\mu + \delta + \gamma)}{(\mu + \delta + \gamma)(2\mu + \delta + \gamma)\beta_{fu}} < 1,$$

$$\text{and } \frac{\beta_{fc}\left((1-r)g\beta_{fu}\Gamma + (\kappa + \phi)(\mu + \gamma + \delta)\right)}{\mu\beta_{fu}(\mu + \gamma + \delta)} > 1.$$

The first condition is true since $r - 1 < 0$ implies that

$$(r-1)g\beta_{fu}\Gamma - \phi(\mu + \delta + \gamma) < 0.$$

The second condition becomes

$$\frac{\beta_{fc}\left((1-r)g\beta_{fu}\Gamma + (\kappa + \phi)(\mu + \gamma + \delta)\right)}{\mu\beta_{fu}(\mu + \gamma + \delta)(\mu + \kappa + \phi)} > \frac{1}{(\mu + \kappa + \phi)}$$

$$\frac{\beta_{fc}(1-r)(g\mu+\kappa+\phi)\Gamma}{\mu(\mu+\gamma+\delta)(\mu+\kappa+\phi)}\frac{g}{(g\mu+\kappa+\phi)}+\frac{\beta_{fc}(\kappa+\phi)}{\mu\beta_{fu}(\mu+\kappa+\phi)}>\frac{1}{(\mu+\kappa+\phi)}$$

$$R_{oc}>\frac{(g\mu+\kappa+\phi)}{g(\mu+\kappa+\phi)}\left(1-\frac{\beta_{fc}(\kappa+\phi)}{\mu\beta_{fu}}\right)>1. \tag{3.58}$$

- λ_7 and λ_8 are negative if

$$\frac{\beta_m(S_f^*-I_f^*)}{\mu+k_3+\gamma_f+\kappa_5}<1, \text{ and } \frac{\beta_m\left(\mu S_f^*-I_f^*(k_3+\gamma_f+\kappa_5)\right)}{\mu(k_3+\gamma_f+\kappa_5)}<1.$$

which, on substitution of S_f^* and I_f^*, become

$$\frac{\beta_m r\Gamma}{(\mu+\delta+\gamma)(2\mu+\gamma+\delta)}>0, \text{ and } \frac{\beta_m r\Gamma}{\mu(\mu+\gamma+\delta)}=R_{of}>1. \tag{3.59}$$

The first condition is already satisfied since $\beta_m, r, \Gamma, \mu, \delta, \gamma > 0$ and hence, λ_7 and λ_8 are negative if $R_{of} > 1$.

Hence, E^* is locally asymptotically stable if $R_0 > 1$. ■

Theorem 3.9: If $R_{ou} > 1$, $R_{oc} > 1$ and $R_{of} > 1$ and V be a positive definite function on some neighborhood of E^*. Then if $\frac{dV}{dt} \leq 0$ then $E^* = (S_u^*, S_c^*, S_f^*, I_u^*, I_c^*, I_f^*, T^*, A^*)$ is globally asymptotically stable.

Proof

To determine global stability of the endemic equilibrium point E^* we use the Lypunov's method and let the Lypunov function be;

$$V(S_u^*, S_c^*, S_f^*, I_u^*, I_c^*, I_f^*, T^*, A^*)$$

$$= \left(S_u - S_u^* - S_u^* \log\frac{S_u^*}{S_u}\right) + \left(S_c - S_c^* - S_c^* \log\frac{S_c^*}{S_c}\right)$$

$$+ \left(S_f - S_f^* - S_f^* \log\frac{S_f^*}{S_f}\right) + \left(I_u - I_u^* - I_u^* \log\frac{I_u^*}{I_u}\right) + \left(I_c - I_c^* - I_c^* \log\frac{I_c^*}{I_c}\right)$$

$$+ \left(I_f - I_f^* - I_f^* \log\frac{I_f^*}{I_f}\right) \tag{3.60}$$

The time derivative of V along the solution of system (3.34) gives;

$$\frac{dV}{dt} = \left(\frac{S_u - S_u^*}{S_u}\right)\frac{dS_u}{dt} + \left(\frac{S_c - S_c^*}{S_c}\right)\frac{dS_c}{dt} + \left(\frac{S_f - S_f^*}{S_f}\right)\frac{dS_f}{dt} + \left(\frac{I_u - I_u^*}{I_u}\right)\frac{dI_u}{dt} + \left(\frac{I_c - I_c^*}{I_c}\right)\frac{dI_c}{dt}$$

$$+ \left(\frac{I_f - I_f^*}{I_f}\right)\frac{dI_f}{dt} \tag{3.61}$$

Substituting $\frac{dS_u}{dt}, \frac{dS_c}{dt}, \frac{dS_f}{dt}, \frac{dI_u}{dt}, \frac{dI_c}{dt}$ and $\frac{dI_f}{dt}$ we obtain;

$$\frac{dV}{dt} = \left(\frac{S_u - S_u^*}{S_u}\right)\left((1-g)(1-r)\Gamma - \beta_{fu} S_u I_u - (\kappa + \phi + \mu)S_u\right)$$

$$+ \left(\frac{S_c - S_c^*}{S_c}\right)\left(g(1-r)\Gamma + (\kappa + \phi)S_u - \beta_{fc} S_c I_c - \mu S_c\right)$$

$$+ \left(\frac{S_f - S_f^*}{S_f}\right)\left(r\Gamma - \beta_m S_f I_f - \mu S_f\right)$$

$$+ \left(\frac{I_u - I_u^*}{I_u}\right)\left(\beta_{fu} S_u I_u - (\kappa_1 + \gamma_u + \kappa_5)I_u\right)$$

$$+ \left(\frac{I_c - I_c^*}{I_c}\right)\left(\beta_{fc} S_c I_c - (\kappa_2 + \gamma_c + \kappa_5)I_c\right)$$

$$+ \left(\frac{I_f - I_f^*}{I_f}\right)\left(\beta_m S_f I_f - (\kappa_3 + \gamma_f + \kappa_5)I_f\right) \tag{3.62}$$

Where $k_5 = \mu + \delta$. At equilibrium system (3.34) satisfies;

$$\left.\begin{aligned}(1-g)(1-r)\Gamma &= \beta_{fu}S_u^*I_u^* + (\kappa+\phi+\mu)S_u^*\\ g(1-r)\Gamma &= -(\kappa+\phi)S_u + \beta_{fc}S_c^*I_c^* + \mu S_c^*\\ r\Gamma &= \beta_m S_f^*I_f^* - \mu S_f^*\\ \kappa_1+\gamma_u+\kappa_5 &= \beta_{fu}S_u^*\\ \kappa_2+\gamma_c+\kappa_5 &= \beta_{fc}S_c^*\\ \kappa_3+\gamma_f+\kappa_5 &= \beta_m S_f^*\end{aligned}\right\} \tag{3.63}$$

Putting (3.63) into (3.62) and simplifying we obtain;

$$\begin{aligned}\frac{dV}{dt} = -\Bigg[&(\kappa+\varphi+\mu)\frac{(S_u-S_u^*)^2}{S_u} + \mu(\kappa+\varphi)\frac{(S_c-S_c^*)^2(S_u-S_u^*)}{S_c} + \mu\frac{\left(S_f-S_f^*\right)^2}{S_f}\Bigg]\\ &+\Bigg[\left(\frac{S_u-S_u^*}{S_u}\right)\left(\beta_{fu}S_u^*I_u^* - \beta_{fu}S_uI_u\right) + \left(\frac{S_c-S_c^*}{S_c}\right)\left(\beta_{fc}S_c^*I_c^* - \beta_{fc}S_cI_c\right)\\ &+\left(\frac{S_f-S_f^*}{S_f}\right)\left(\beta_mS_f^*I_f^* - \beta_mS_fI_f\right) + (I_u-I_u^*)\left(\beta_{fu}S_u - \beta_{fu}S_u^*\right)\\ &+(I_c-I_c^*)\left(\beta_{fc}S_c - \beta_{fc}S_c^*\right) + \left(I_f-I_f^*\right)\left(\beta_mS_f - \beta_mS_f^*\right)\Bigg]\end{aligned} \tag{3.64}$$

Therefore $\frac{dV}{dt}=0$ if and only if $S_u=S_u^*,\ S_c=S_c^*, S_f=S_f^*,\ I_u=I_u^*,\ I_c=I_c^*$ and $I_f=I_f^*$ and this implies that, $\frac{dV}{dt}<0$ if and only if $\left[\left(\frac{S_u-S_u^*}{S_u}\right)\left(\beta_{fu}S_u^*I_u^* - \beta_{fu}S_uI_u\right) + \left(\frac{S_c-S_c^*}{S_c}\right)\left(\beta_{fc}S_c^*I_c^* - \beta_{fc}S_cI_c\right) + \left(\frac{S_f-S_f^*}{S_f}\right)\left(\beta_mS_f^*I_f^* - \beta_mS_fI_f\right) + (I_u-I_u^*)\left(\beta_{fu}S_u - \beta_{fu}S_u^*\right) + (I_c-I_c^*)\left(\beta_{fc}S_c - \beta_{fc}S_c^*\right) + \left(I_f-I_f^*\right)\left(\beta_mS_f - \beta_mS_f^*\right)\right] < \left[(\kappa+\varphi+\mu)\frac{(S_u-S_u^*)^2}{S_u} + \mu(\kappa+\varphi)\frac{(S_c-S_c^*)^2(S_u-S_u^*)}{S_c} + \mu\frac{\left(S_f-S_f^*\right)^2}{S_f}\right]$

Hence $E^*=\left(S_u^*,S_c^*,S_f^*,I_u^*,I_c^*,I_f^*,\ T^*,A^*\right)$ is globally asymptotically stable. ■

CHAPTER 4

RESULTS AND DISCUSSION

4.1 Introduction

In this chapter, the numerical simulations of the two models are done and an exhaustive discussion of the results was done. The dynamics of HIV transmission among truckers and FSWs under the combined influence of funding and circumcision in Kenya is modelled by systems of nonlinear ordinary differential equations (3.1) and (3.34). The numerical schemes coded into MATLAB ode45 was adopted in seeking the solution to the model (see Oke (2017) for other methods of solution) and the behaviours of different state variables over time were extracted as graphs. Default values of the parameters shown in table 4.1 formed the basis of our analysis for both model 1 and model 2.

Table 4.1: Default Values of the Parameters

Parameter	Value	Source
g	0.85	(Cherutich et al., 2012)
r	0.069	(Elmore-Meegan et al., 2004)
Γ	0.3	Estimated
γ	0.34	(UNAIDS, 2020)
σ	0.08	Estimated
μ	0.0539	(UNAIDS, 2020)
δ	0.016	(UNAIDS, 2021)
φ	0.84	(WHO, 2010)
β_{fu}	0.0128	(Doherty et al., 2013)
β_{fc}	0.0051	(Doherty et al., 2013)
β_m	0.049	(Doherty et al., 2013)

4.2 Analysis and Discussion of Results of Model I

The effects are shown in figures (4.1 – 4.5). With any slight increase in the HIV rate of transmission from females to circumcised males, the AIDS population increases and the

circumcised susceptible truckers' population decreases. This is because the interactions between the truckers and the FSWs remain fixed while the rate of transmission to males increases. The infected circumcised population among the truckers rises rapidly between the first and the fourth year, but the infected circumcised population declines slowly after about four years due to the reduction in the total number of the susceptible population. The initial rise is enhanced as the rate of transmission increases. Figure 4.5 shows that increasing HIV rate of transmission from females to uncircumcised males causes an increase in the infected uncircumcised truckers' population. This is because the infected uncircumcised population responds in direct proportion to the rate of transmission from females to the uncircumcised infected population.

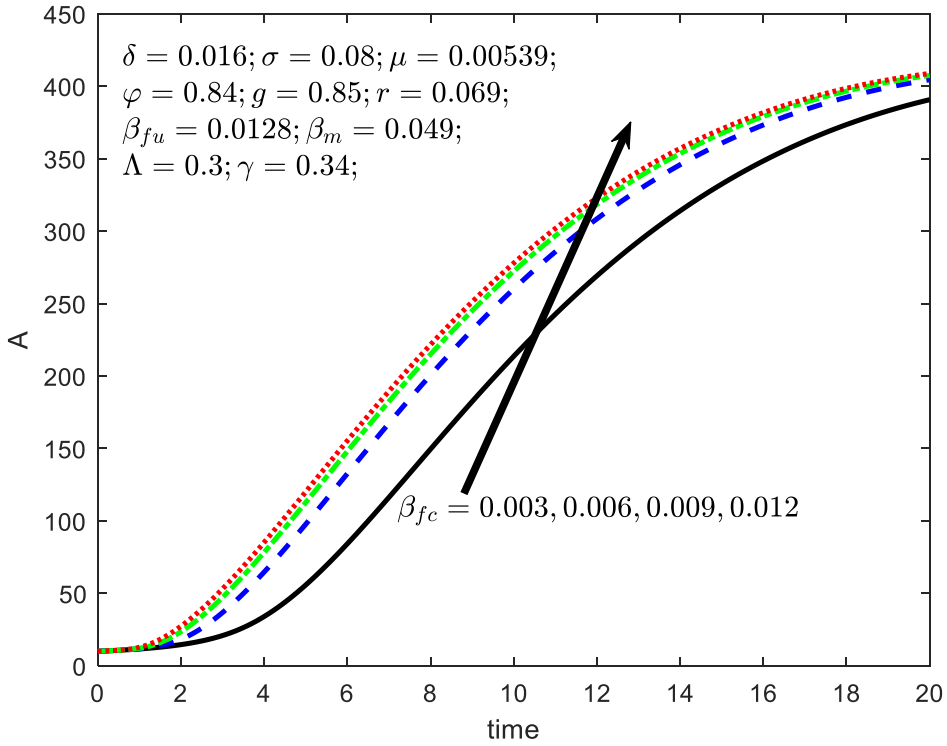

Figure 4.1: Variation of AIDS population with β_{fc}

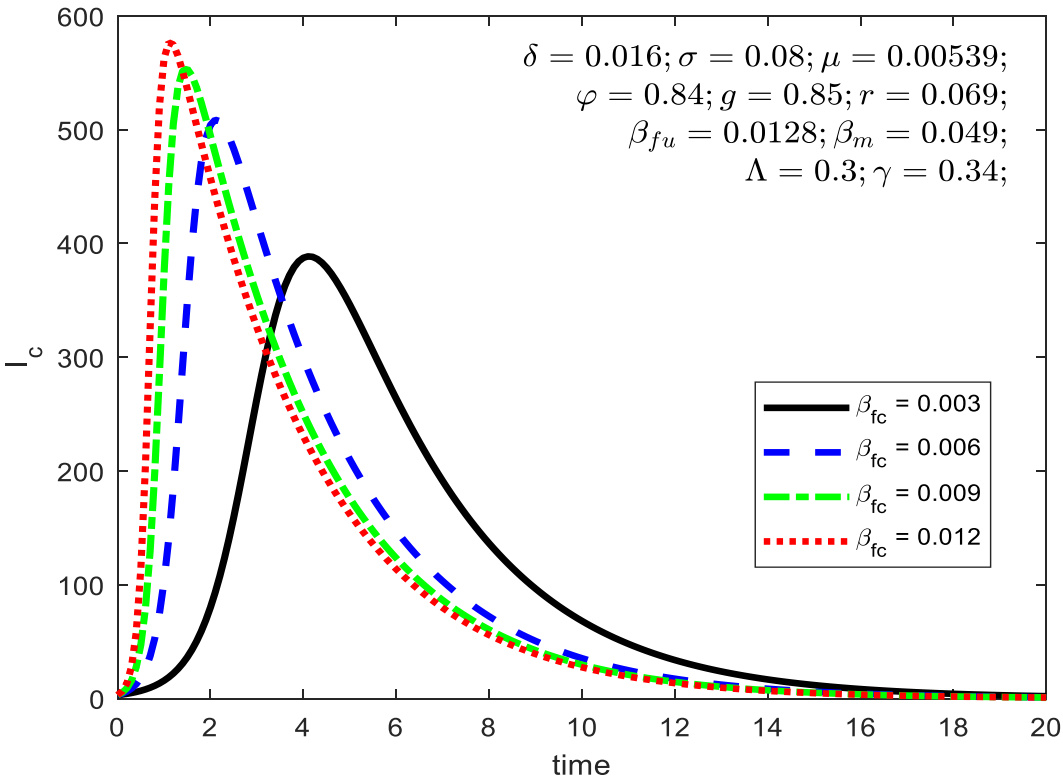

Figure 4.2: Variation of the infected circumcised male population with β_{fc}

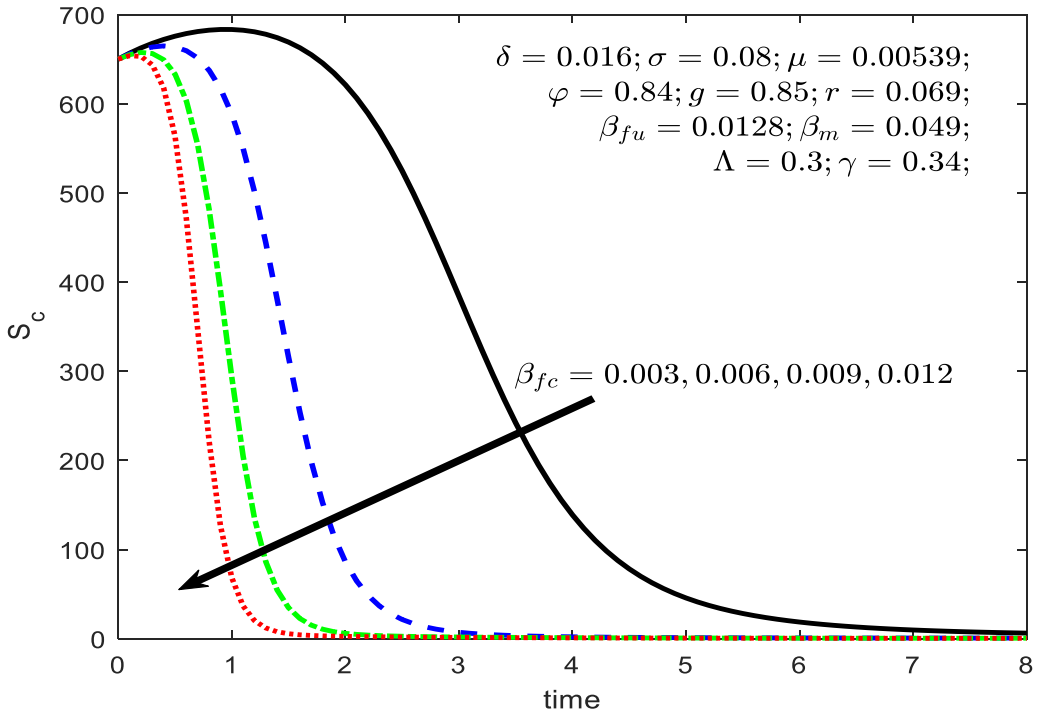

Figure 4.3: Variation of the susceptible circumcised male population with β_{fc}

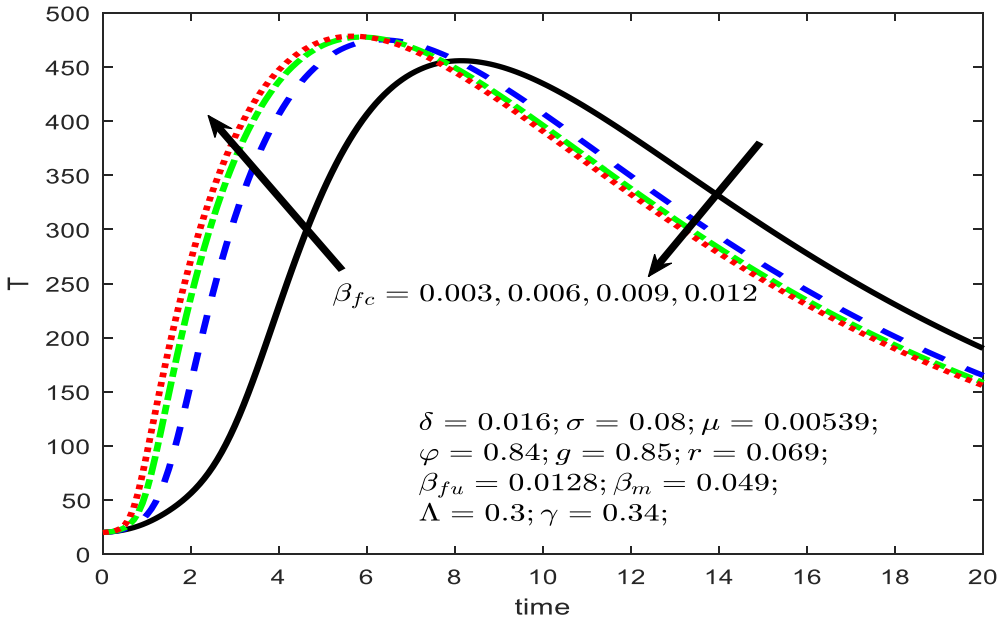

Figure 4.4: Variation of the treated population with β_{fc}

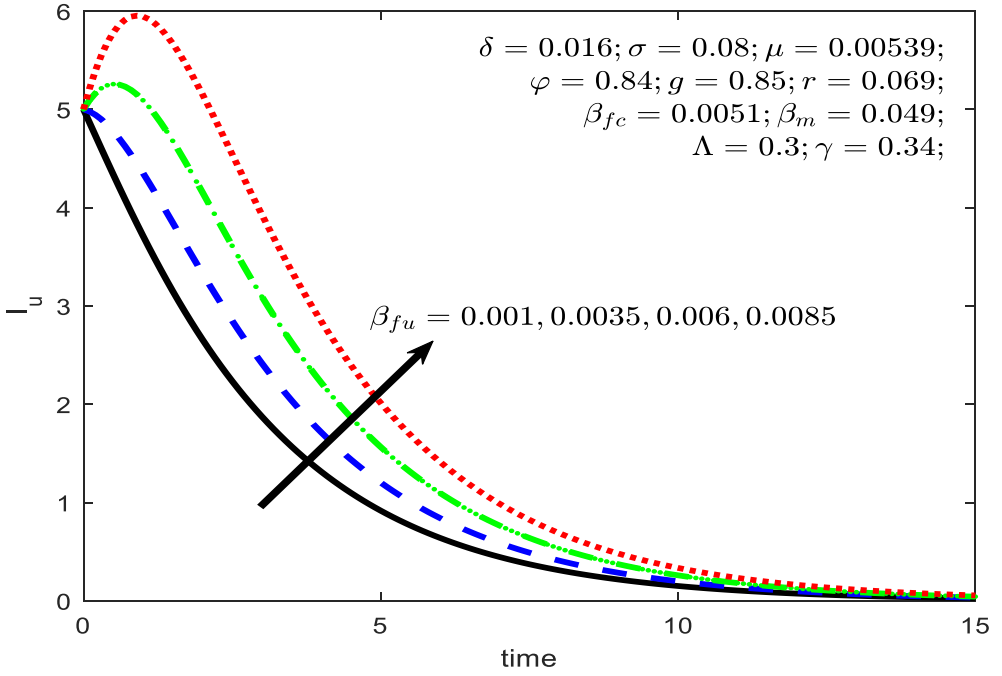

Figure 4.5: Variation of the infected uncircumcised male population with β_{fu}

The effects are shown in figures (4.6 – 4.7). The infected FSWs population responds with high sensitivity to any little change in the rate of transmission from females to males. This is probably because truckers are always on transit and therefore they have the

tendency, once infected, to carry infection from one location to another. The infected female population increases rapidly in the first three years as the rate of transmission from female to male increases. The response is also highly directly proportional to the increase in the rate of transmission β_m. Meanwhile, the susceptible female population reduces drastically as a response to the rise in the infected female population.

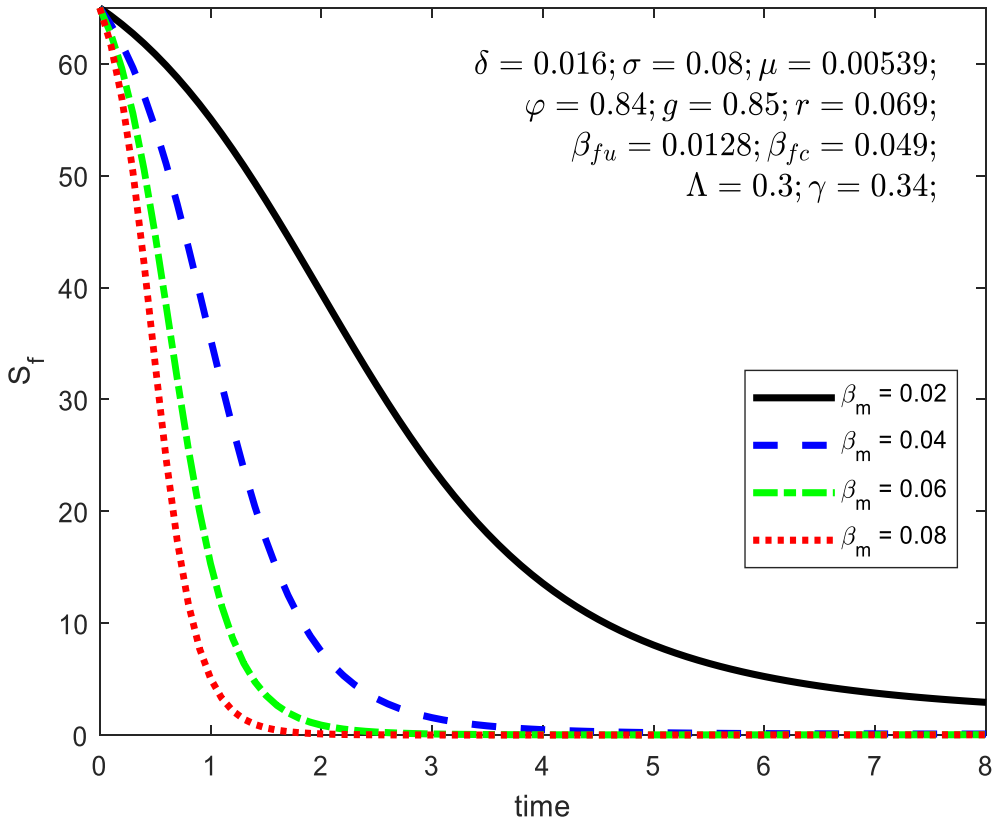

Figure 4.6: Variation of the susceptible female population with β_m

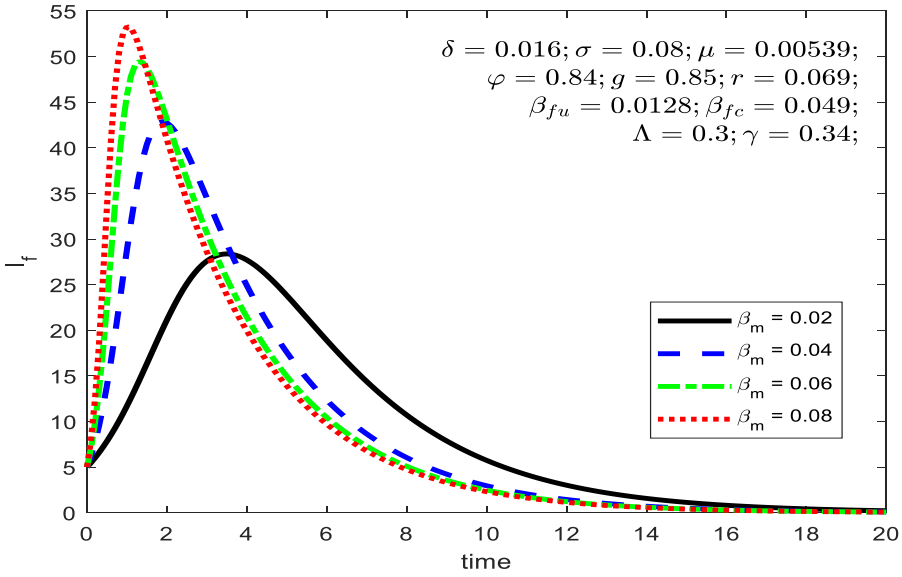

Figure 4.7: Variation of infected females with β_m

Figures (4.8 – 4.12) show the responses of different classes to the increase in the rate of circumcision. Increasing the rate of circumcision consequently increases migration from the susceptible uncircumcised population increases to the susceptible circumcised population. The consequence is an increase in the susceptible circumcised population while the susceptible uncircumcised population decreases. In addition, the infected uncircumcised population decreases with the increasing rate of circumcision since the susceptible uncircumcised population has dropped. This shows that the rate of circumcision reduces the rate at which the truckers get infected.

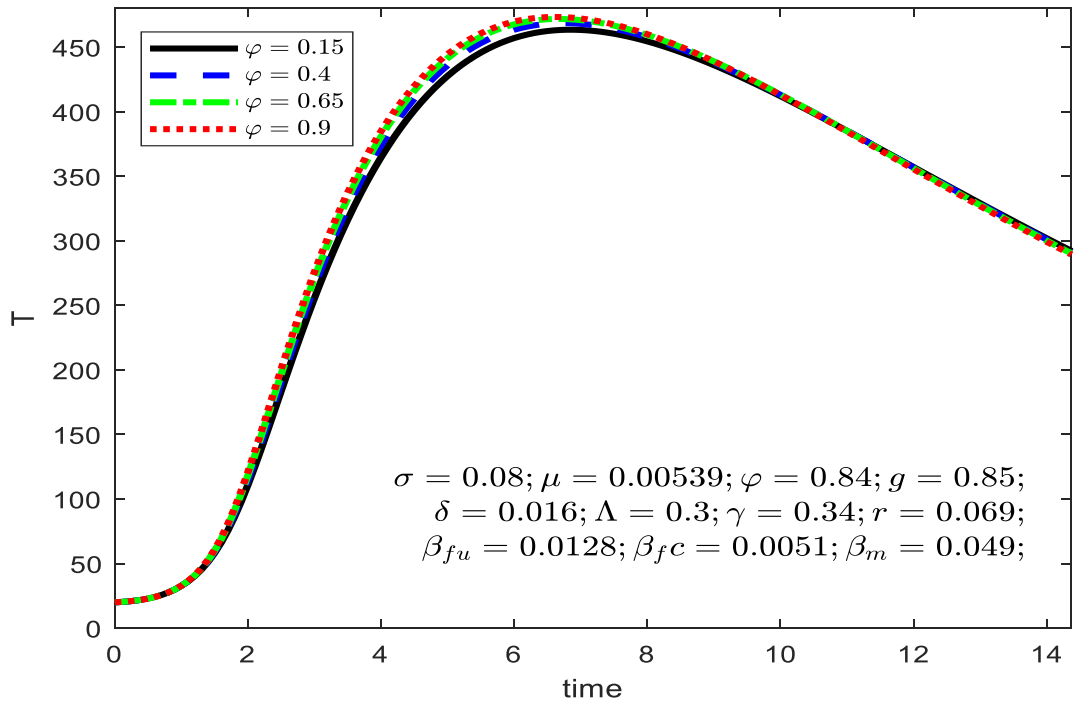

Figure 4.8: Variation of the treated population with φ

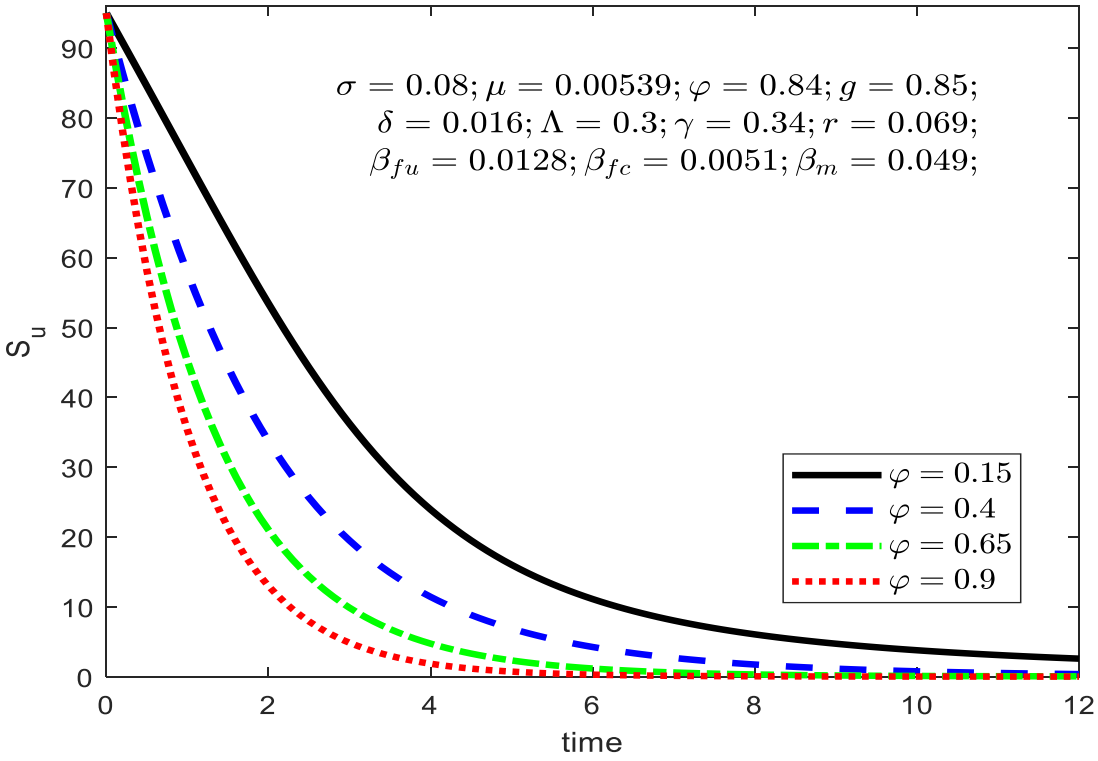

Figure 4.9: Variation of Uncircumcised male susceptible population with φ

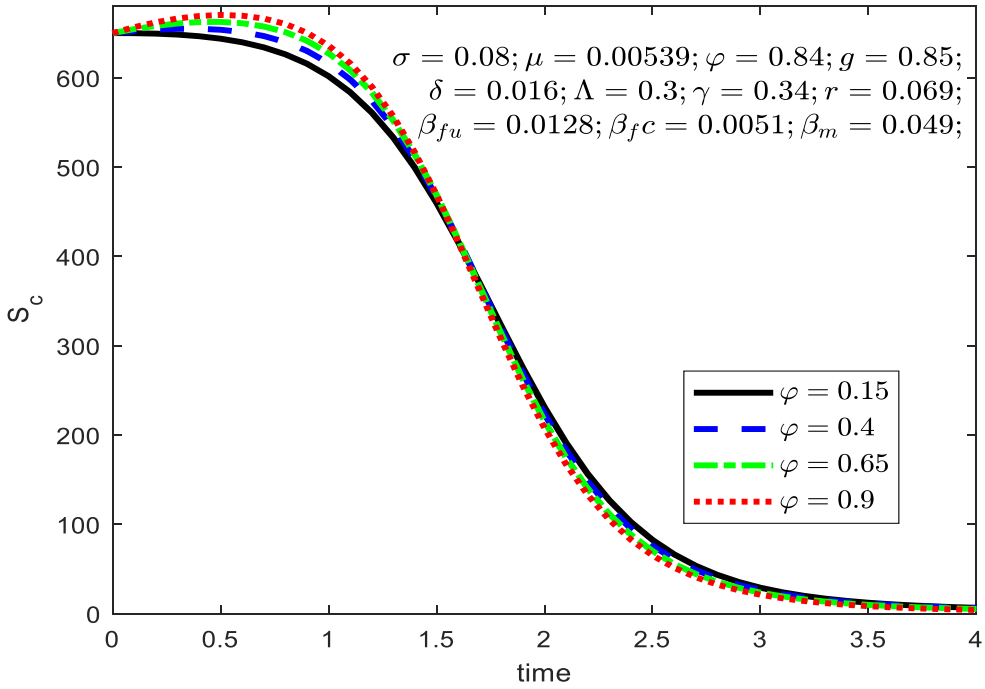

Figure 4.10: Variation of the circumcised male susceptible population with φ

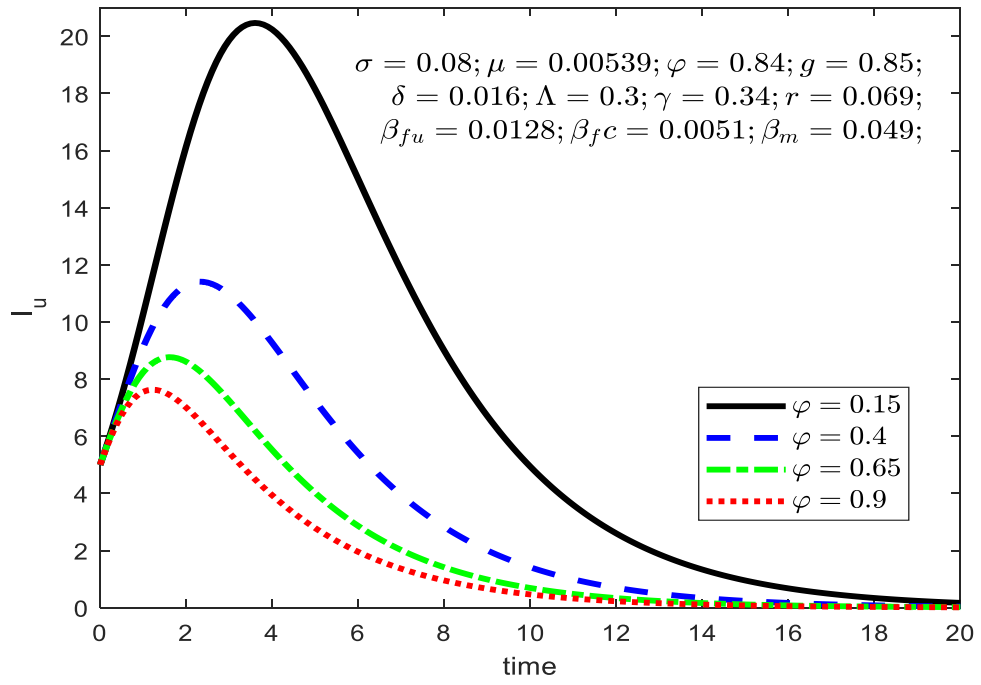

Figure 4.11: Variation of Uncircumcised infected male population with φ

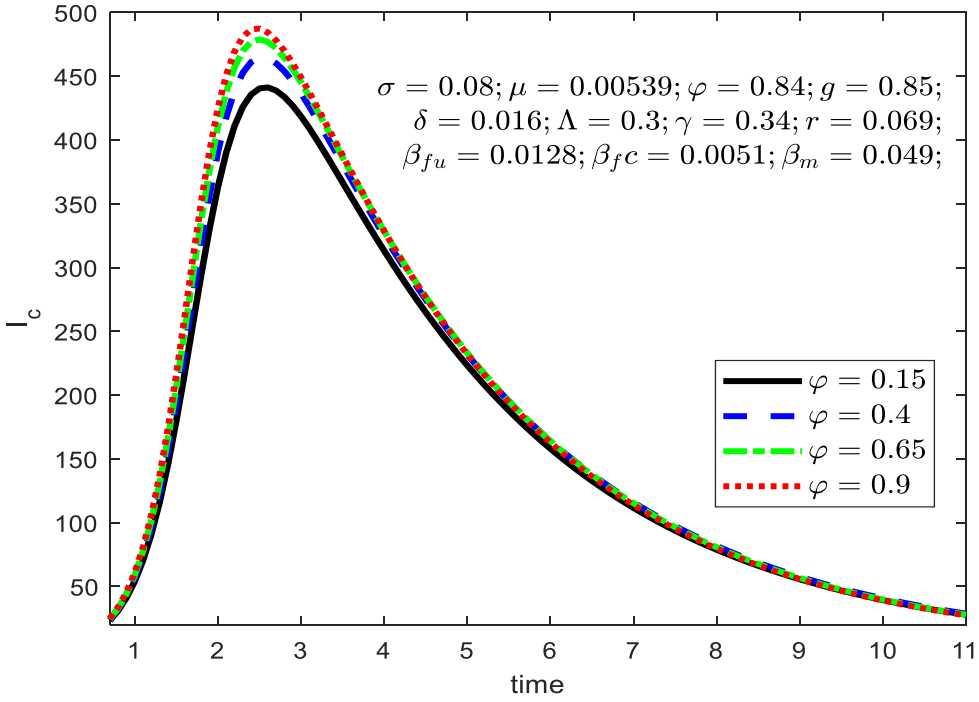

Figure 4.12: Variation of the circumcised infected male population with φ

4.3 Analysis and Discussion of Results of Model 2

The system (3.34) was solved numerically using MATLAB ode45 solver and the effect of funding parameters κ, k_1, k_2 and k_3 were investigated on HIV/AIDS dynamics among truckers/FSWs in Northern corridor highway in Kenya. The parameters used for simulation are shown in Table 5.1 and are chosen to suit the Truckers/FSWs along the Northern corridor highway in Kenya.

Effects of funding of male circumcision κ are shown in Figures (4.13 – 4.15). The value $\kappa = 0$ represents a total removal of circumcision funding while the maximum rate of circumcision funding is 0.14. As circumcision funding increases from zero to maximum, the class of infected circumcised males increases slightly (Figure 4.13), the class of infected uncircumcised males decreases (Figure 4.14) and the class of susceptible

uncircumcised males decrease (Figure 4.15). Funding of male truckers' circumcision increases the chances of migration from the uncircumcised class to the circumcised class. This means an increase in the number of circumcised male truckers susceptible to contracting HIV and a reduction in the number of uncircumcised male truckers susceptible to contracting HIV. Meanwhile, a circumcised male trucker is less likely to contract HIV than the uncircumcised male truckers. The reduction in the uncircumcised class, therefore, means a reduction in the chance for any trucker to migrate to the infected class. Hence, the overall number of male truckers that are recruited to the infected class will decrease overall.

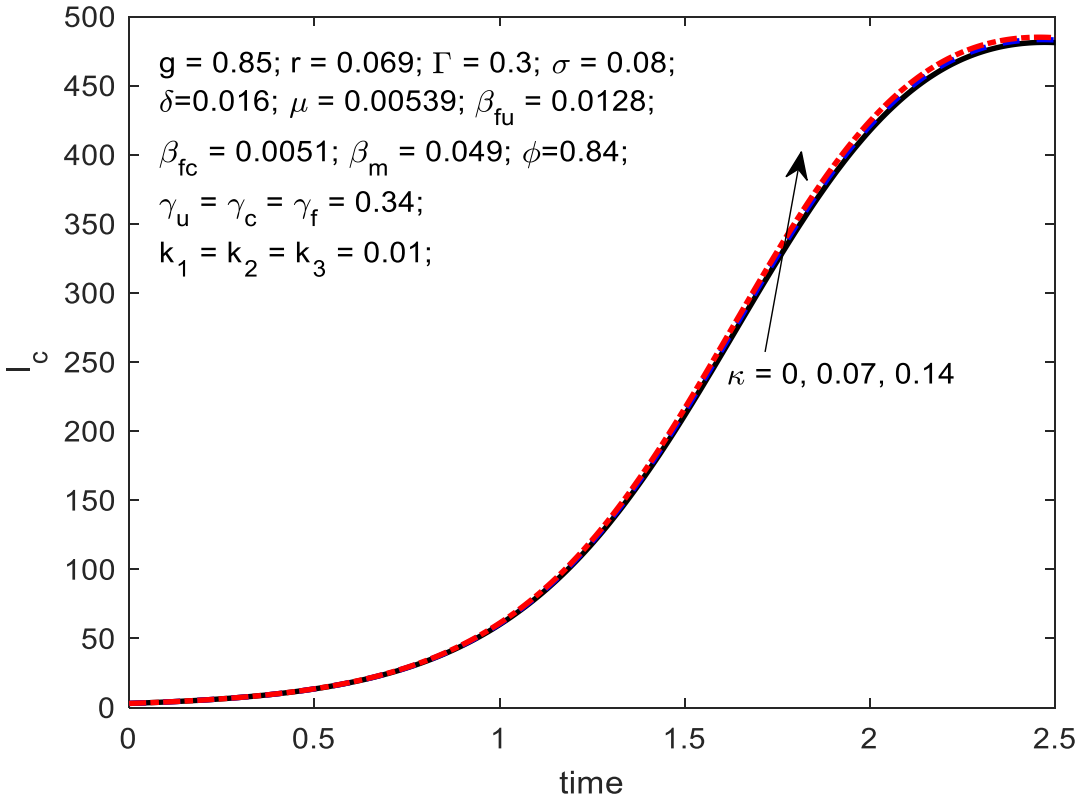

Figure 4.13: Variation of Infected Circumcised Male with circumcision funding parameter

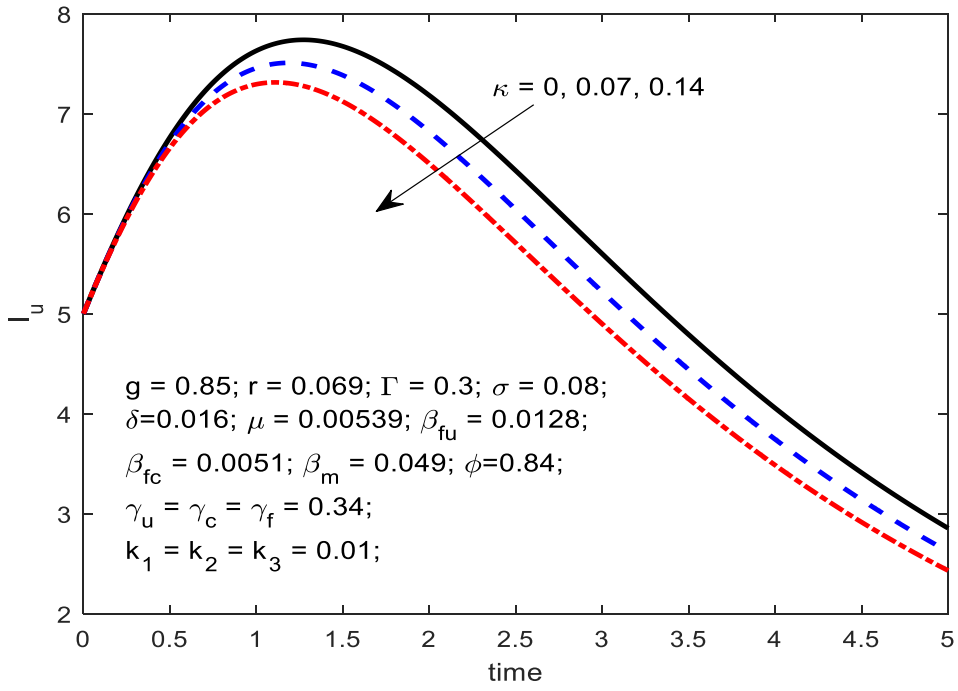

Figure 4.14: Variation of Infected uncircumcised Male with circumcision funding parameter

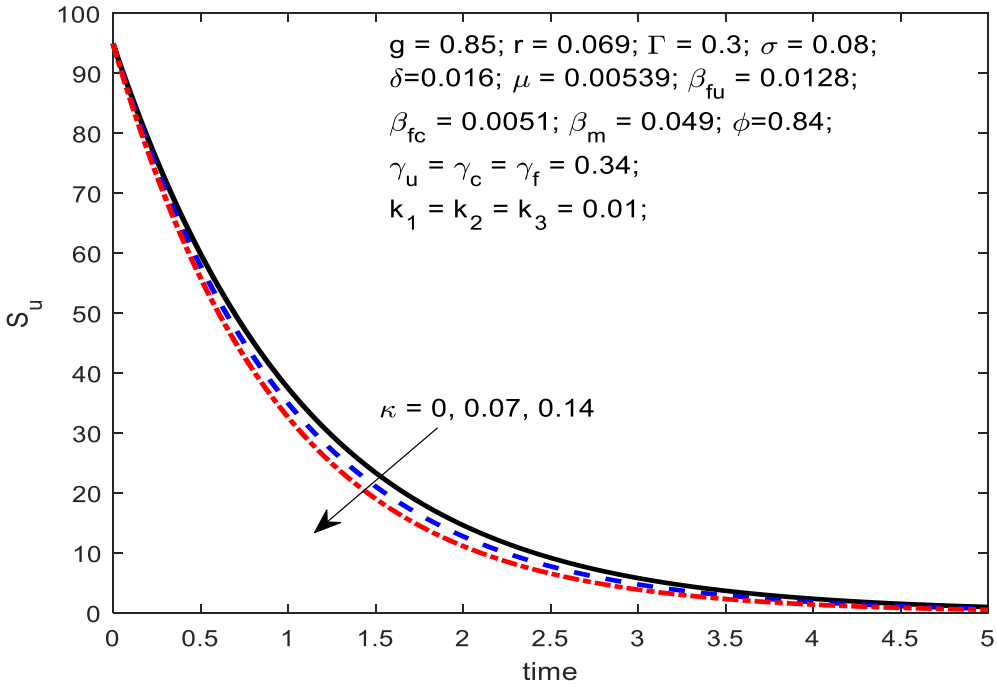

Figure 4.15: Susceptible uncircumcised Male with circumcision funding parameter

Effects of funding of treatment for the infected uncircumcised males are shown in Figures (4.16 – 4.17). $k_1 = 0$ represents the absence of treatment funding for the infected uncircumcised males and $k_1 = 0.6$ represents the maximum funding possible for the infected uncircumcised males. As funding increases for the treatment of infected uncircumcised males, the class of infected uncircumcised males decreases (Figure 4.16) and the class of susceptible uncircumcised males increases (Figure 4.17). Investigating the effects of funding of treatment for the infected uncircumcised males shows that increasing funding leads to a decrease in the class of infected uncircumcised males while the class of susceptible uncircumcised males increases. Increasing funding of treatment for the infected uncircumcised males means more infected uncircumcised male truckers can assess treatment more readily and thereby many migrate to the Treatment class.

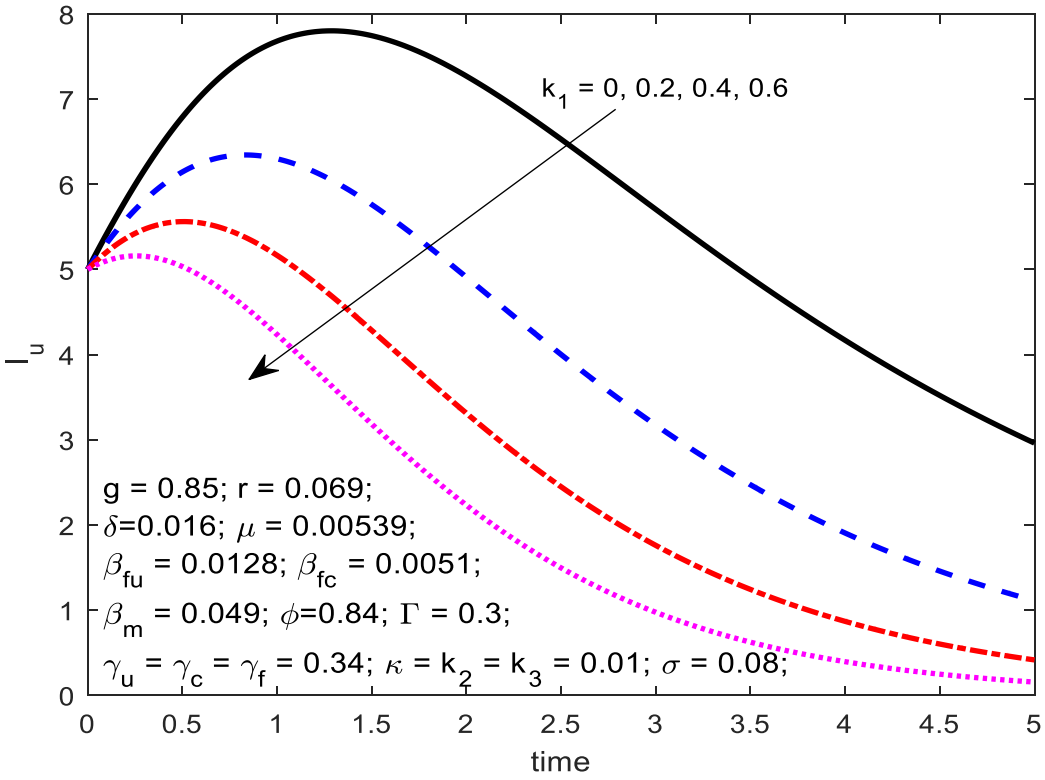

Figure 4.16: Infected uncircumcised male with treatment funding for the uncircumcised male

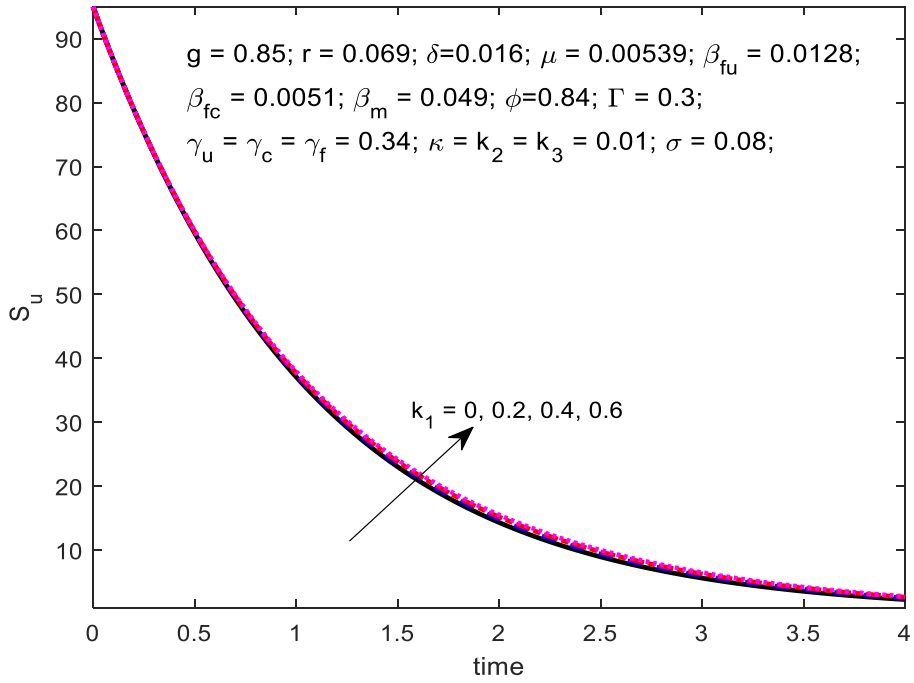

Figure 4.17: Variation of Susceptible uncircumcised male with treatment funding for the uncircumcised male

Effects of funding of treatment for the circumcised males are shown in Figures (4.18 – 4.21). $k_2 = 0$ represents the absence of treatment funding for the circumcised males and $k_1 = 0.6$ represents the maximum funding possible for circumcised males. As funding increases for the treatment of infected circumcised males, the AIDS population increases (Figure 4.18), Treated class increases (Figure 4.19), class of infected circumcised males decreases (Figure 4.20) and the class of susceptible circumcised males increases (Figure 4.21). Increasing funding of treatment for the infected circumcised males means more infected circumcised male truckers can assess treatment more readily and thereby many migrate to the Treatment class. An increase in funding for treating infected circumcised makes truckers brings about an increase in the number of Treated individuals.

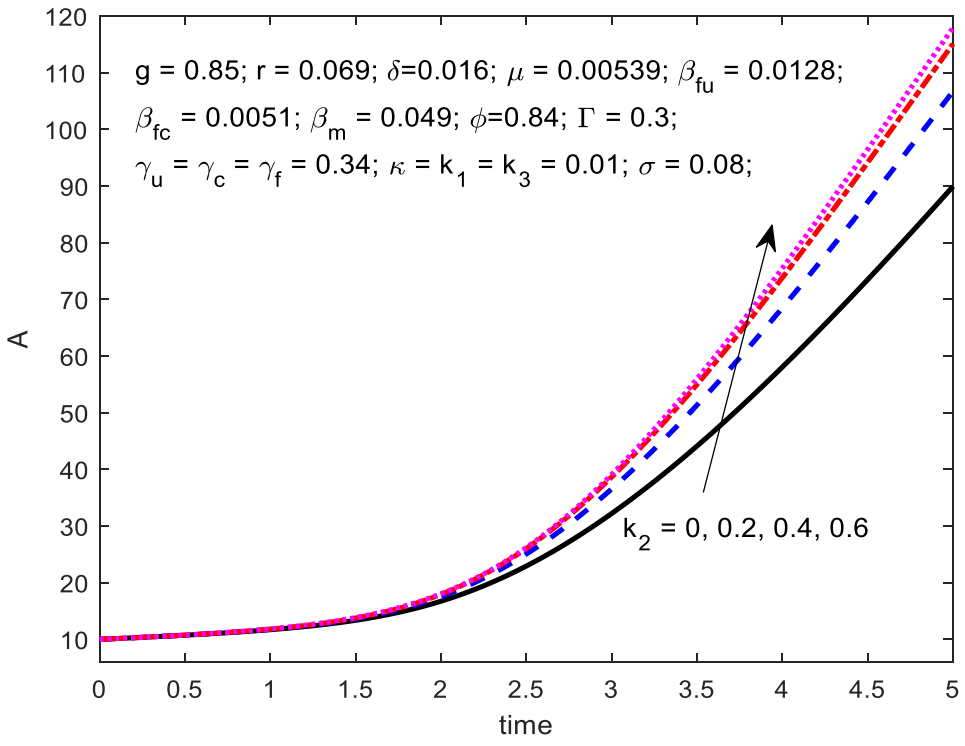

Figure 4.18: Variation of the AIDS population with treatment funding for the circumcised male

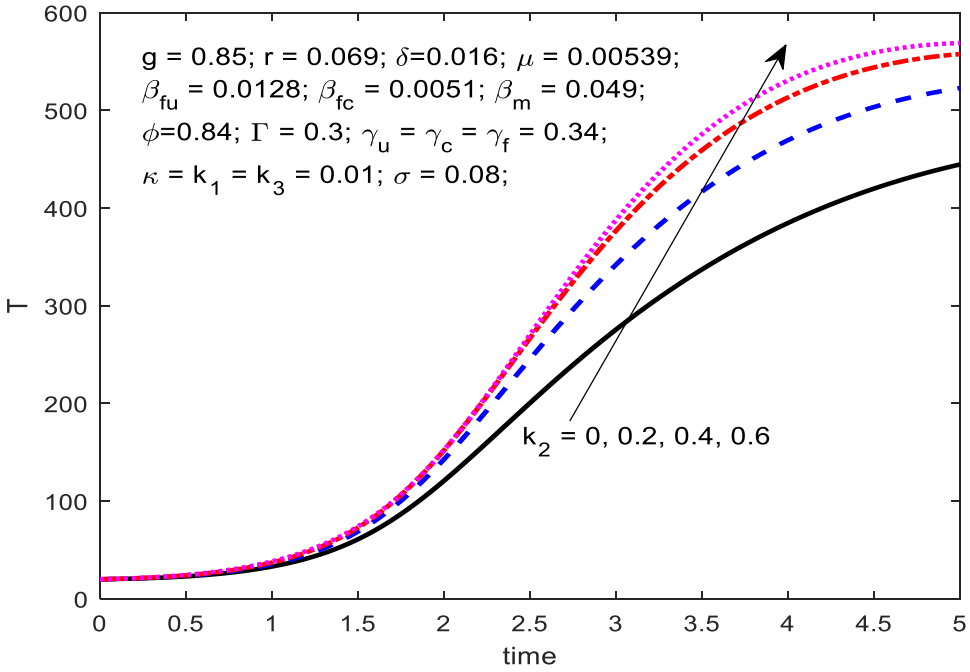

Figure 4.19: Variation of Treatment class with treatment funding for the circumcised male

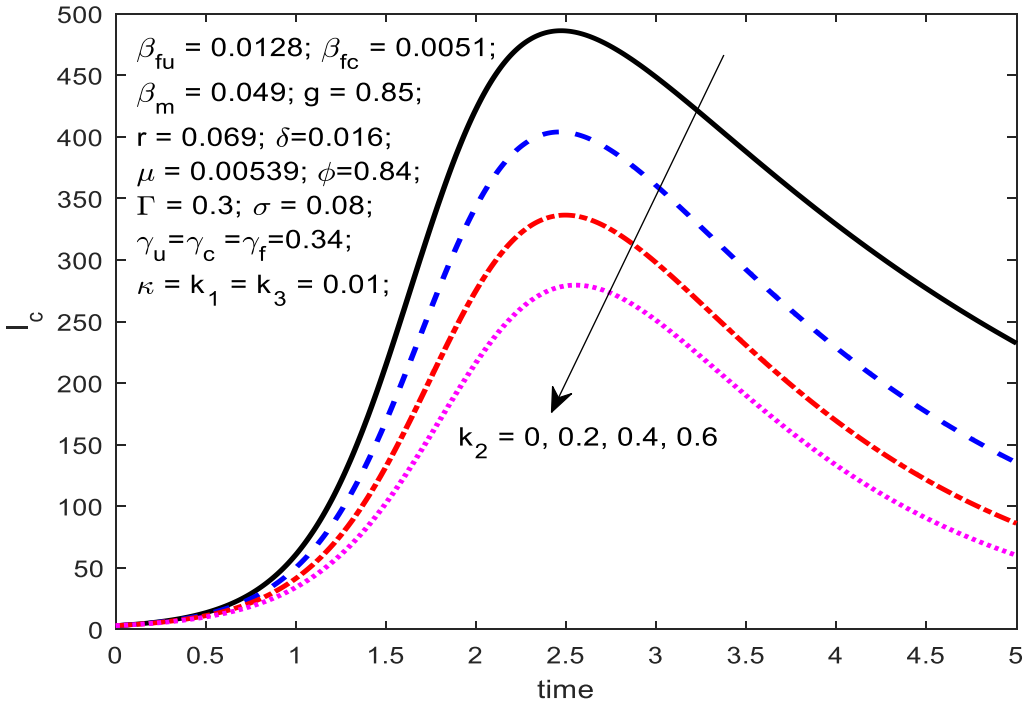

Figure 4.20: Variation of Infected circumcised male with treatment funding for the circumcised male

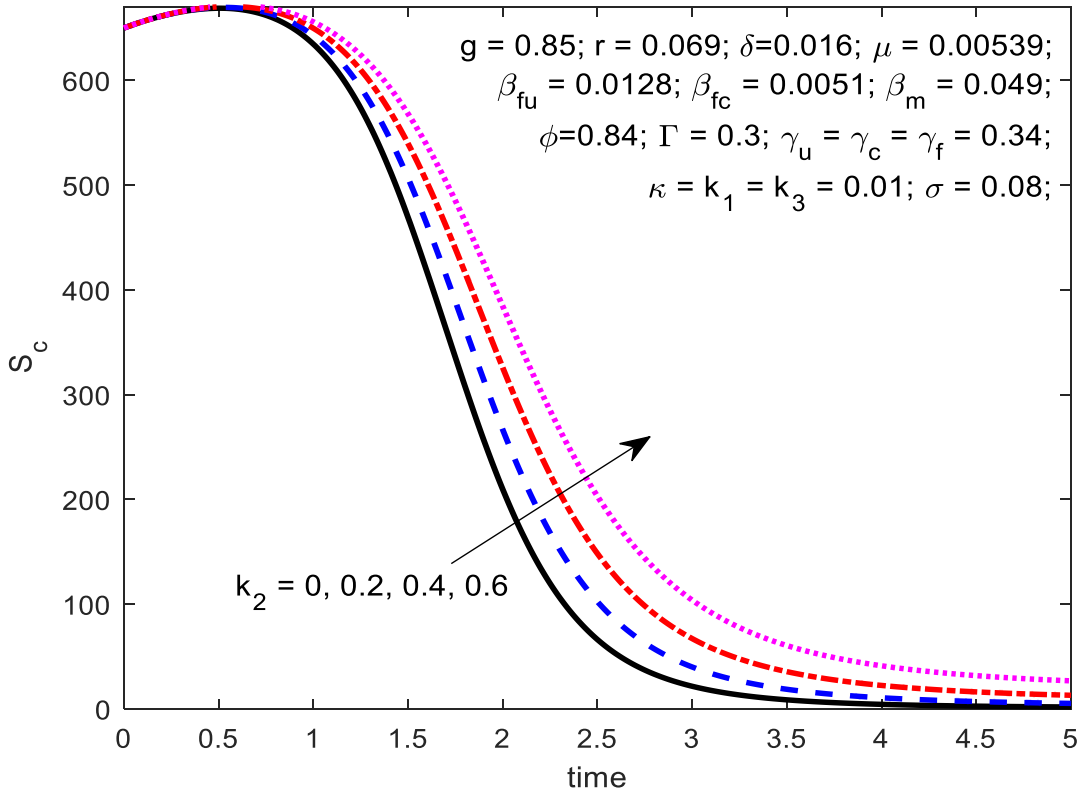

Figure 4.21: Variation of Susceptible circumcised male with treatment funding for the circumcised male

Effects of treatment funding for FSWs are shown in Figures (4.22 – 4.25). $k_3 = 0$ represents the absence of treatment funding for FSWs and $k_3 = 0.6$ represents the maximum funding possible for FSWs. As funding increases for the treatment of FSWs, the AIDS population increases (Figure 4.25), the Treated class increases (Figure 4.23), the class of infected FSWs decreases (Figure 4.24) and the class of susceptible FSWs increases (Figure 4.25). Funding treatment for FSWs increases the number of infected FSWs that get treatment and thereby reduces the number of individuals entering the AIDS class. The Treated class will also increase because of the increased funding and thereby reduced the number of new migrations into the AIDS population.

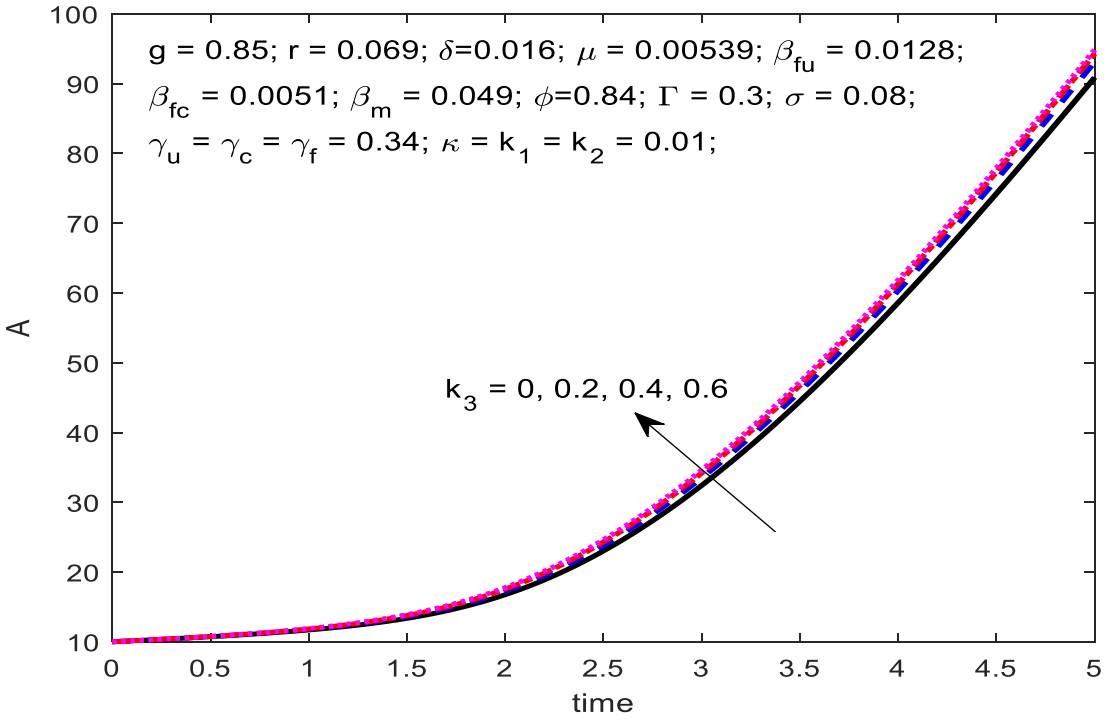

Figure 4.22: Variation of the AIDS population with treatment funding for FSWs

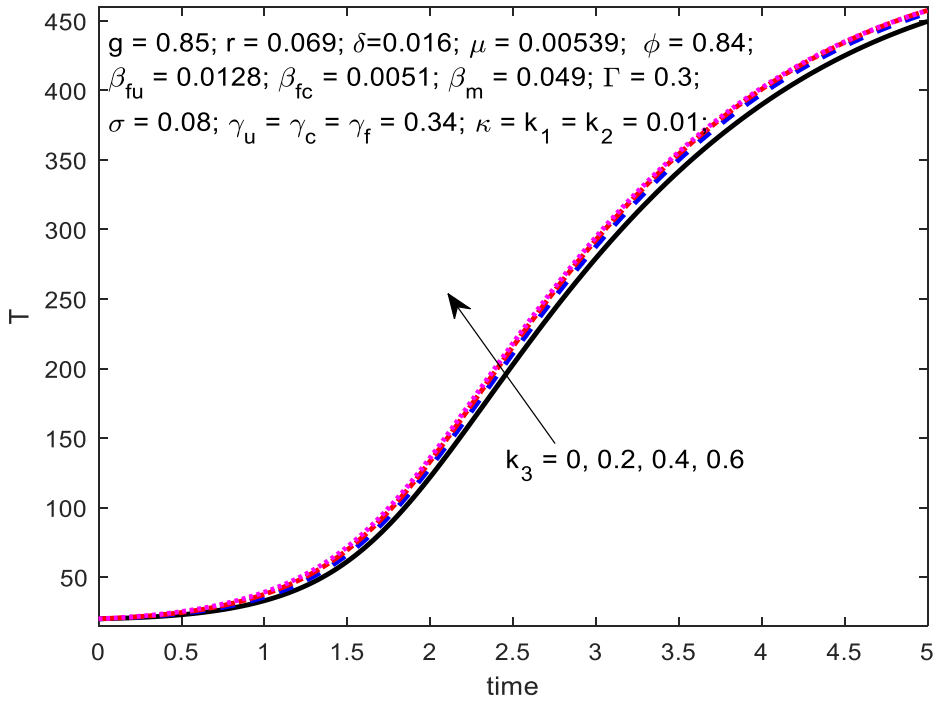

Figure 4.23: Variation of Treatment class with treatment funding for FSWs

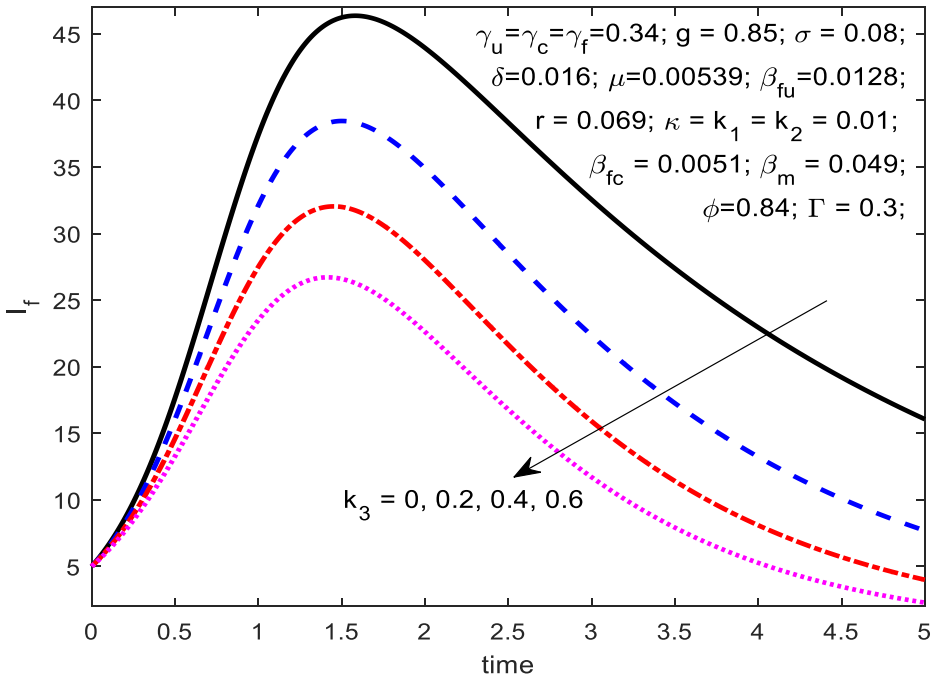

Figure 4.24: Variation of Infected FSWs with treatment funding for FSWs

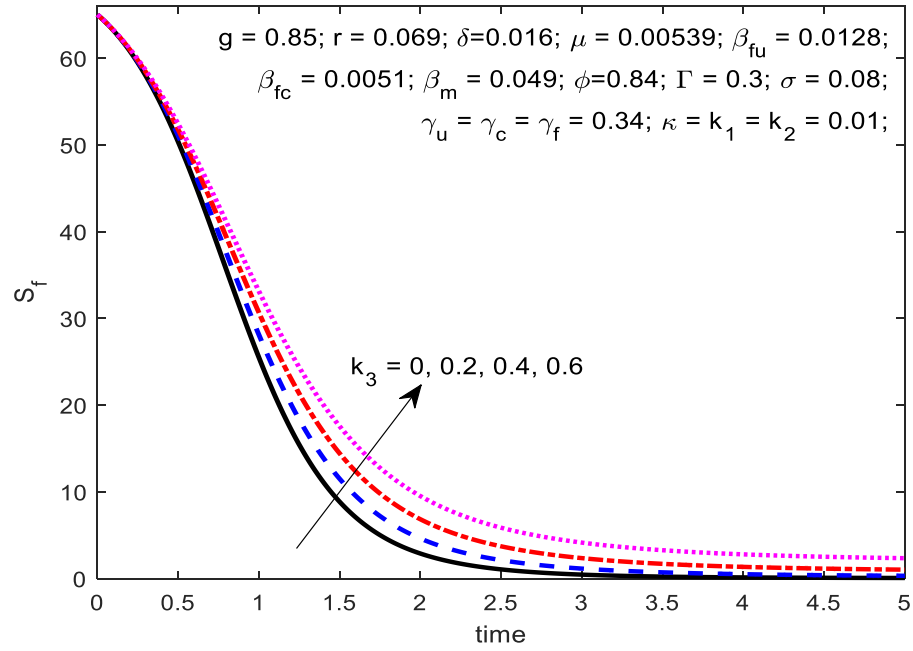

Figure 4.25: Variation of Susceptible FSWs with treatment funding for FSWs

Effects of increasing overall funding are shown in Figures (4.26 – 4.33). In this case, overall funding refers to combined funding of male truckers' circumcision and funding of

treatment of all classes of infected individuals. Increasing overall funding leads to a decrease in susceptible uncircumcised male truckers (shown in Figure 4.26), an increase in susceptible circumcised male truckers (shown in Figure 4.27), an increase in susceptible FSWs (shown in Figure 4.28), a decrease in infected uncircumcised male truckers (shown in Figure 4.29), an increase in infected circumcised male truckers (shown in Figure 4.30), an increase in infected FSWs (shown in Figure 4.31), a decrease in Treated (shown in Figure 4.32, and an increase in the AIDS population (shown in Figure 4.33).

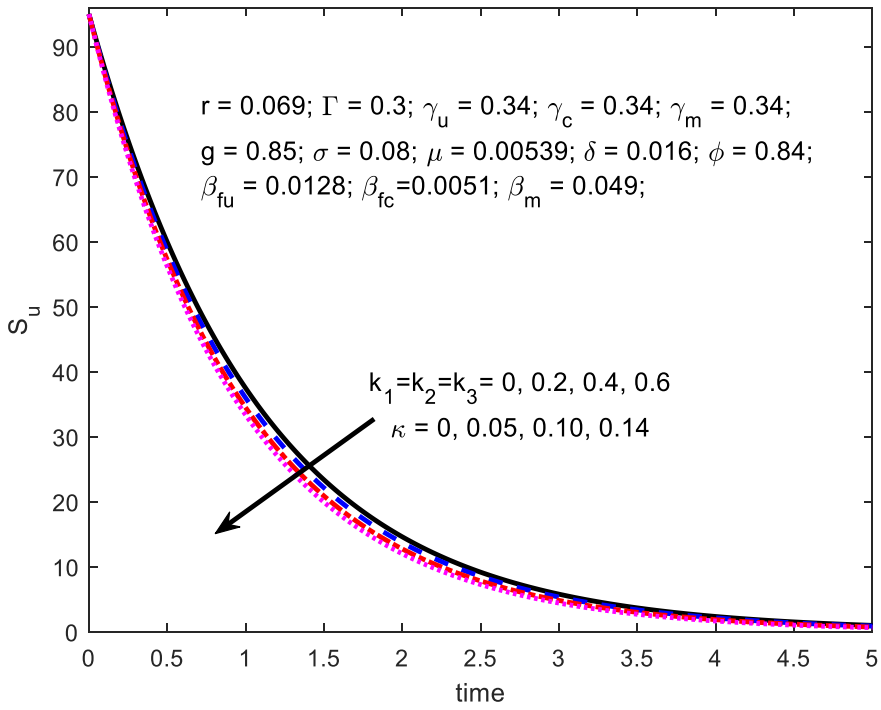

Figure 4.26: Variation of Susceptible uncircumcised male with funding

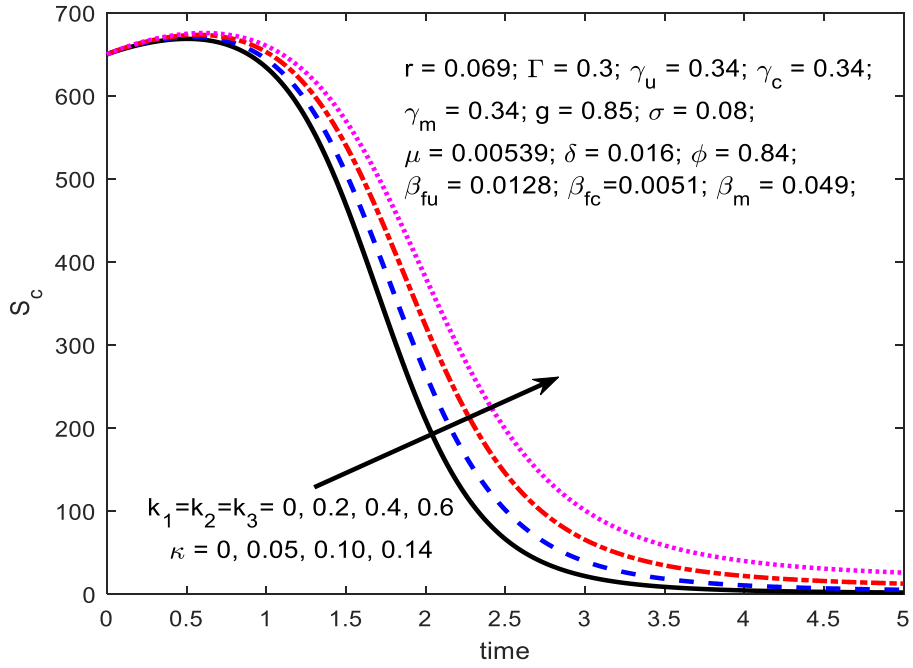

Figure 4.27: Variation of Susceptible circumcised male with funding

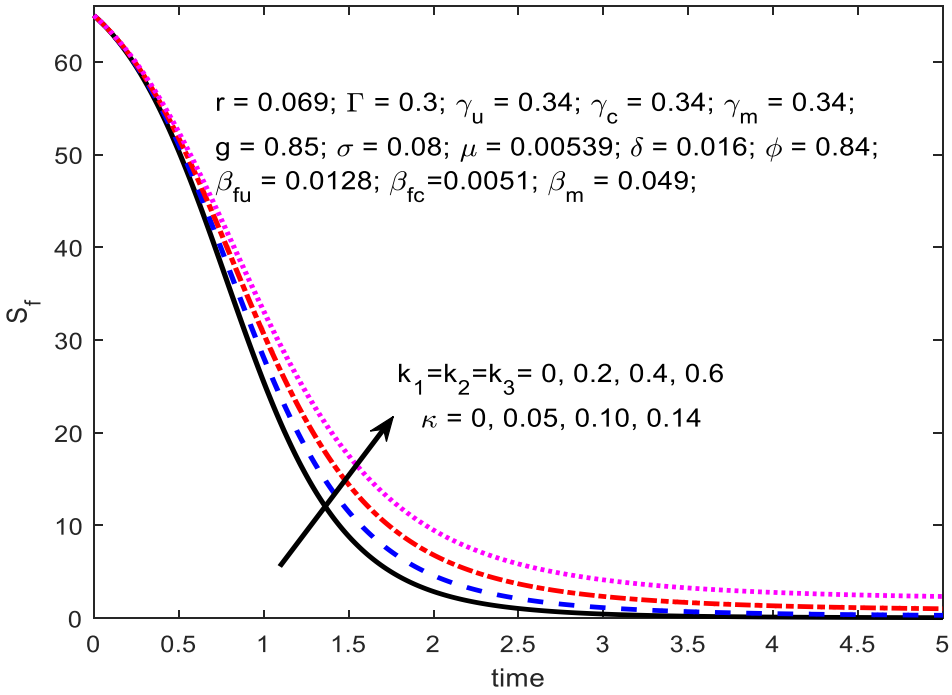

Figure 4.28: Variation of infected FSWs with funding

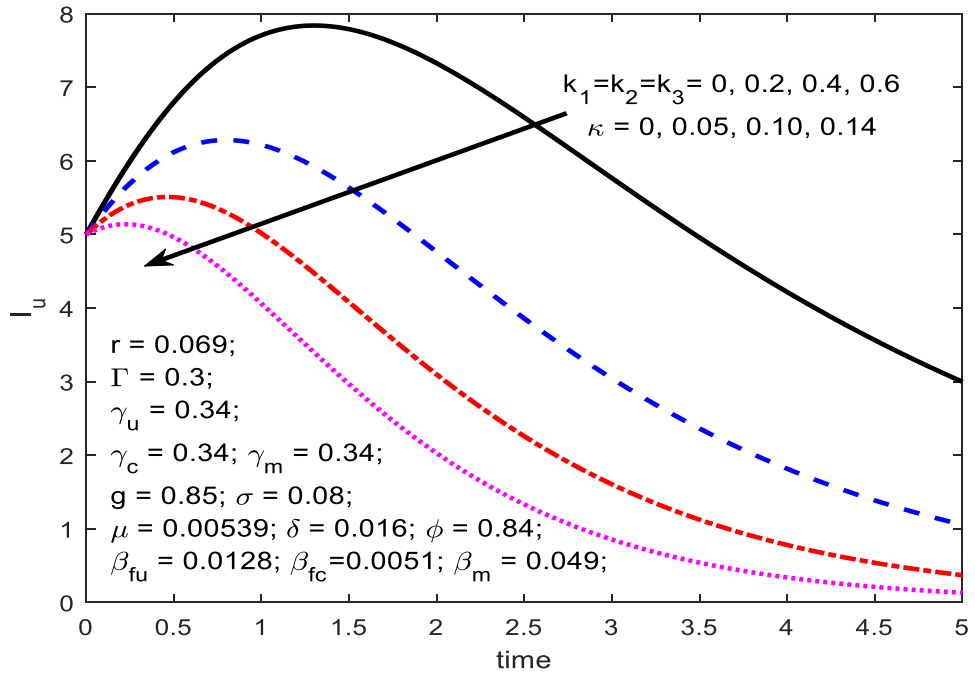

Figure 4.29: Variation of infected uncircumcised male with funding

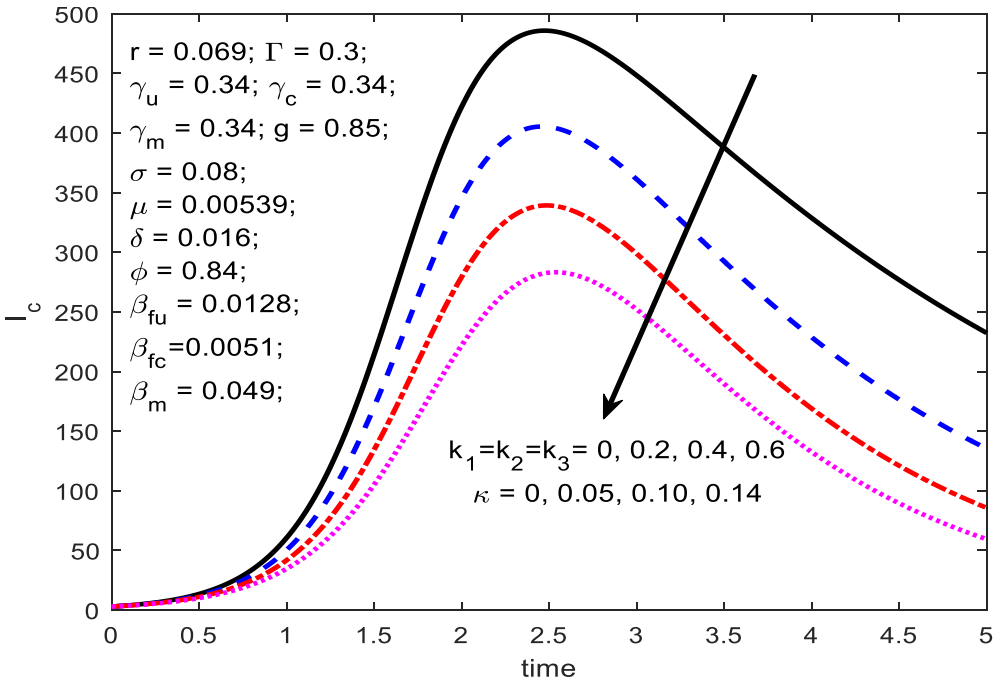

Figure 4.30: Variation of infected circumcised male with funding

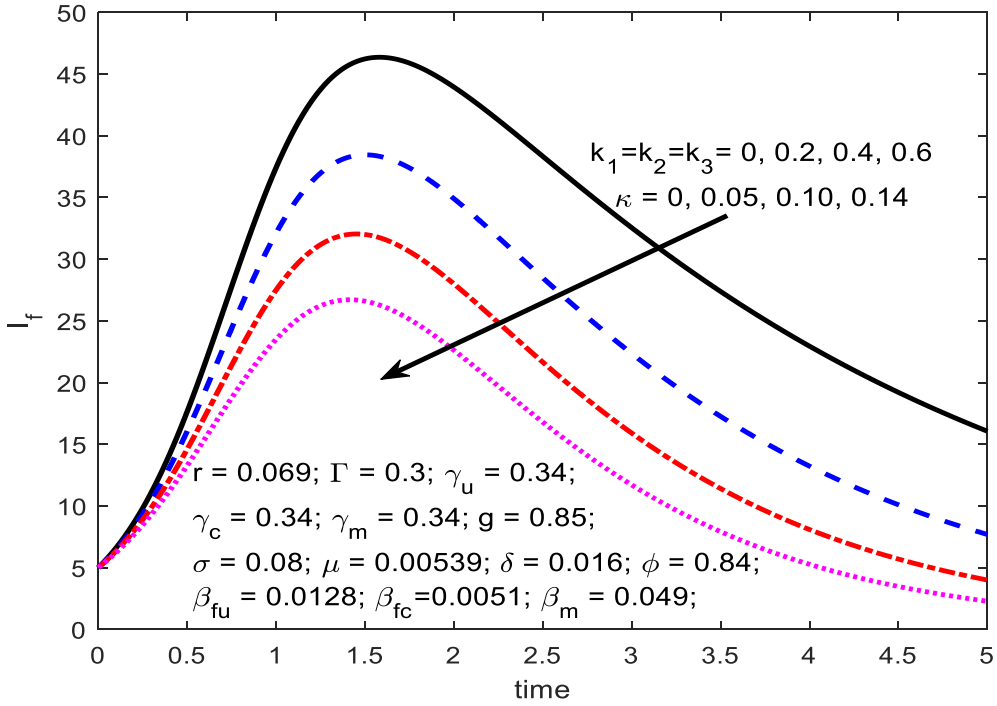

Figure 4.31: Variation of infected FSWs with funding

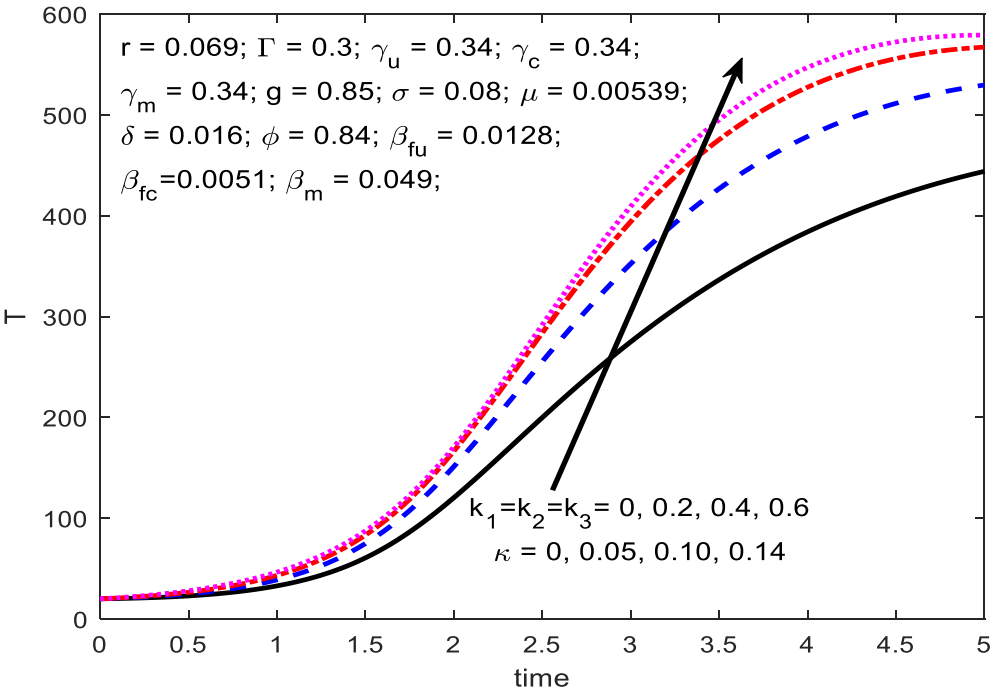

Figure 4.32: Variation of Treated class with funding

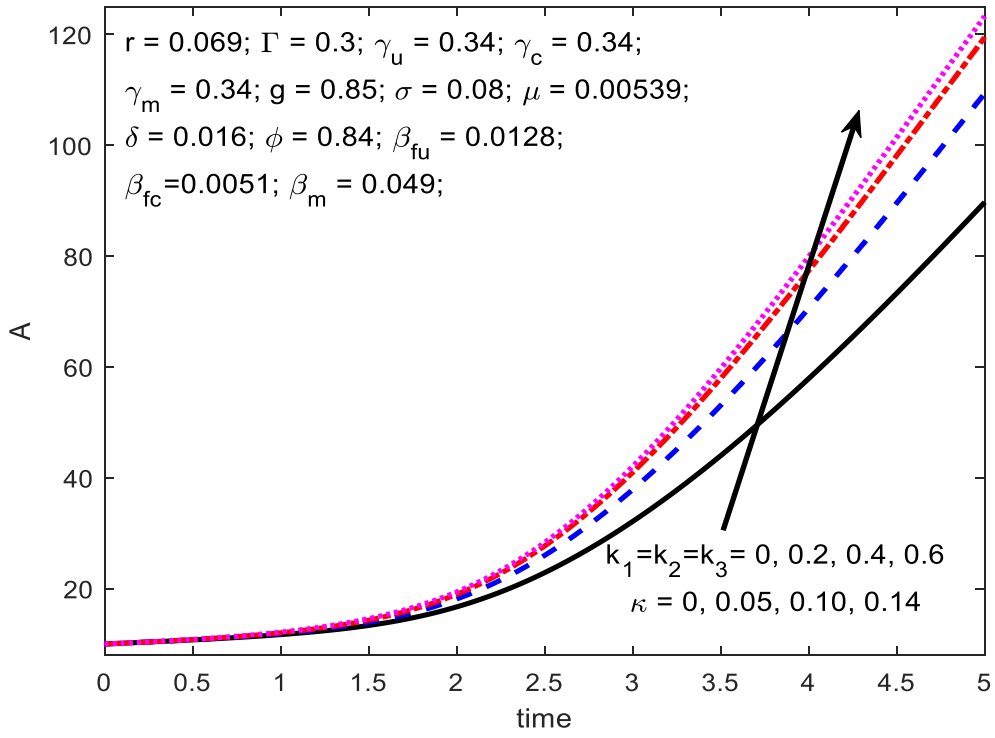

Figure 4.33: Variation of the AIDS population with funding

In general, if the circumcision is funded at a higher rate and all infected classes are funded for treatment, then the overall funding improves. Studying the effects of the improved overall funding on the entire truckers/FSWs population. Due to improved circumcision, more percentage of the susceptible population enters into the susceptible circumcised male. Due to the reduced chance of infection for the susceptible circumcised male truckers, it means the total number of infected individuals will drop.

CHAPTER 5

CONCLUSIONS AND RECOMMENDATIONS

5.1 Conclusions

This study tried to model HIV AIDS dynamics with funding along the Northern Corridor Highway in Kenya by first formulating two deterministic models incorporating circumcision and funding for circumcision and treatment; model 1 which studied the effect of circumcision on HIV transmission dynamics between truckers and female sex workers and Model 2 investigated the effect of both funding and circumcision on the dynamics of HIV/AIDS among Kenyan truckers and FSWs on the Northern corridor highway. Three reproduction numbers R_{0u}, R_{0c} and R_{0f} were obtained and they were used to explain the behaviour of the pandemic as time went on

The local stability of Disease Free Equilibrium and Endemic Equilibrium Point were determined which was important in predicting whether the pandemic will be endemic with time or not. . It was noted that when $R_{0u} > 1, R_{0c} > 1$ and $R_{0f} > 1$, the pandemic would persist in the population with time and dies out with time when $R_{0u} < 1, R_{0c} < 1$ and $R_{0f} < 1$. The systems of equations were solved numerically using MATLAB ode45 solver.

For Model 1, Analysis of the model indicated that an increase in the transmission rates β_{fu}, β_{fc} and β_m produced more infections among the truckers and FSWs and circumcision can be used as a method of minimizing HIV infections among this population as it produces a drastic decrease in I_u and insignificant increase in I_c.

For Model 2, increasing funding for circumcision reduces the rate of migration from the Susceptible class to the Infected class. Increasing funding for treatment of any class increases the Treatment class and reduces the overall number of AIDS-related.

In conclusion, funding circumcision (such as subsidizing the cost of circumcision, providing quality free post-circumcision services, and/or setting up circumcision clinics along the Kenya Northern highway corridors) can reduce the rate at which truckers contract HIV/AIDS in Kenya. Also, increasing funding for treatment of the infected classes (such as providing free or subsidized anti-retroviral drugs, free quality condoms, providing free counselling to infected individuals, etc.) can help ameliorate the overall number of AIDS-related death.

5.2 Recommendations

Using the results obtained then we recommend the following to the policy makers;

i. Government to introduce awareness programs on circumcision to truckers as well as the entire population through the social media platforms and broadcasting stations since it reduces HIV infections.
ii. The Ministry of Health to provide subsidized circumcision or free circumcision by providing quality post-circumcision services.
iii. The government to provide skills to FSWs which can provide an income to them as an alternative to female sex work. This can be done by admitting them government sponsored TVET programs.
iv. Kenya government to increase funding for HIV treatment by sourcing for internal means because of drastic decrease of donor funding on HIV. This can be done by

increasing domestic allocations from internal sources for HIV and AIDS and also monitoring to ensure proper spending.

For further studies, we recommend that;

i. Collection of data to do data fitting to the deterministic models formulated and confirm the validity of the results obtained.

ii. Collect data on the population of truckers and FSWs along the Northern corridor highway in Kenya since no such concentrated data exists.

iii. Research on the applicability of the formulated models on other key populations like men who inject drugs, male sex workers and prison communities.

iv. Develop a stochastic mathematical model and compare the results with this of the deterministic model.

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APPENDICES

A. MATLAB code for Model 1

```
%%Main Execution Code
clc
clear all
global g r Gamma phi mu gamma delta sigma beta_fu beta_fc beta_m
g=0.85;
r=0.069;Gamma=0.3;phi=0.84;mu=0.00539;gamma=0.34;delta=0.016;sigma=0.08
;
beta_fu=0.0128;beta_fc=0.0051;beta_m=0.049;
tspan=0:0.1:20; xzero=[95 650 65 5 3 5 20 10]; i=1;
for phi = [0.15,0.4, 0.65,0.9]
    if i==1
        txt1 = 'k-'; txt2 = 'r-'; txt3 = 'b-';
    elseif i==2
        txt1 = 'b--'; txt2 = 'r-.'; txt3 = 'b-.';
    elseif i==3
        txt1 = 'g-.'; txt2 = 'r.'; txt3 = 'b.';
    elseif i==4
        txt1 = 'r:'; txt2 = 'r.'; txt3 = 'b.';
    end
    [t,x] = ode45(@HIV_transmission,tspan,xzero);
    figure(1)
    plot(t,x(:,1),txt1,'LineWidth',2)
    xlabel('time'),ylabel('S_u')
    hold on
    figure(2)
    plot(t,x(:,2),txt1,'LineWidth',2)
    xlabel('time'),ylabel('S_c')
    hold on
    figure(3)
    plot(t,x(:,3),txt1,'LineWidth',2)
    xlabel('time'),ylabel('S_f')
    hold on
    figure(4)
    plot(t,x(:,4),txt1,'LineWidth',2)
    xlabel('time'),ylabel('I_u')
    hold on
    figure(5)
    plot(t,x(:,5),txt1,'LineWidth',2)
    xlabel('time'),ylabel('I_c')
    hold on
    figure(6)
    plot(t,x(:,6),txt1,'LineWidth',2)
    xlabel('time'),ylabel('I_f')
    hold on
    figure(7)
    plot(t,x(:,7),txt1,'LineWidth',2)
    xlabel('time'),ylabel('T')
    hold on
    figure(8)
    plot(t,x(:,8),txt1,'LineWidth',2)
    xlabel('time'),ylabel('A')
```

```
    hold on
    figure(9)
    plot(t,x(:,1)+x(:,2)+x(:,3),txt1,'LineWidth',2)
    xlabel('time'),ylabel('S')
    hold on
    figure(10)
    plot(t,x(:,4)+x(:,5)+x(:,6),txt1,'LineWidth',2)
    xlabel('time'),ylabel('I')
    hold on
    i=i+1;
end



%%The function Execution Code

function dx=HIV_transmission(t,x)
global g r Gamma phi mu gamma delta sigma beta_fu beta_fc beta_m

dx1= (1-g)*(1-r)*Gamma - beta_fu*x(1)*x(4) - (phi+mu)*x(1);
dx2= g*(1-r)*Gamma +phi*x(1)- beta_fc*x(2)*x(5) - mu*x(2);
dx3= r*Gamma - beta_m*x(3)*x(6) - mu*x(3);
dx4= beta_fu*x(1)*x(4) - (mu+gamma+delta)*x(4);
dx5= beta_fc*x(2)*x(5) - (mu+gamma+delta)*x(5);
dx6= beta_m*x(3)*x(6) - (mu+gamma+delta)*x(6);
dx7= (x(4)+x(5)+x(6))*gamma - (mu+delta+sigma)*x(7);
dx8= sigma*x(7) - (mu+delta)*x(8);
dx = [dx1;dx2;dx3;dx4;dx5;dx6;dx7;dx8];
end
```

B. MATLAB code for Model 2

```
clc
format compact
clear all
global g r Gamma phi mu gamma delta sigma beta_fu beta_fc beta_m k kap
%Parameters from previous studies
g=0.85; r=0.069; Gamma=0.3; gamma=[0.34,0.34,0.34]; sigma=0.08;
mu=0.00539;
delta=0.016; phi=0.84; beta_fu=0.0128; beta_fc=0.0051; beta_m = 0.049;

%Parameters that can be varied.
k = [0.01,0.01,0.01]; kap = 0.01;

%initial conditions
tspan=0:0.01:5; xzero=[95 650 65 5 3 5 20 10]; i=1;
kappa =[0,0.05,0.10,0.14];

%simulations
for k_val = [0,0.2,0.4,0.6]
    k(1)=k_val; k(2)=k_val; k(3)=k_val;
    kap = kappa(i);
    aaa=[kap + phi + beta_fu; beta_fu + mu; beta_m + mu; k(1) +
gamma(1) + mu + delta;...
        k(2) + gamma(2) + mu + delta; k(3) + gamma(3) + mu + delta;
sigma + mu + delta]'
    if i==1
```

```
        txt1 = 'k-'; txt2 = 'r-'; txt3 = 'b-';
    elseif i==2
        txt1 = 'b--'; txt2 = 'r-.'; txt3 = 'b-.';
    elseif i==3
        txt1 = 'r-.'; txt2 = 'r.'; txt3 = 'b.';
    elseif i==4
        txt1 = 'm:'; txt2 = 'r.'; txt3 = 'b.';
    end

    [t,x] = ode45(@HIV_transmission,tspan,xzero);
    figure(1), plot(t,x(:,1),txt1,'LineWidth',2)
    xlabel('time'),ylabel('S_u')
    hold on
    figure(2), plot(t,x(:,2),txt1,'LineWidth',2)
    xlabel('time'),ylabel('S_c')
    hold on
    figure(3), plot(t,x(:,3),txt1,'LineWidth',2)
    xlabel('time'),ylabel('S_f')
    hold on
    figure(4), plot(t,x(:,4),txt1,'LineWidth',2)
    xlabel('time'),ylabel('I_u')
    hold on
    figure(5), plot(t,x(:,5),txt1,'LineWidth',2)
    xlabel('time'),ylabel('I_c')
    hold on
    figure(6), plot(t,x(:,6),txt1,'LineWidth',2)
    xlabel('time'),ylabel('I_f')
    hold on
    figure(7), plot(t,x(:,7),txt1,'LineWidth',2)
    xlabel('time'),ylabel('T')
    hold on
    figure(8), plot(t,x(:,8),txt1,'LineWidth',2)
    xlabel('time'),ylabel('A')
    hold on
    i=i+1;
end

dim1 = [0.2, 0.1, 0.3, 0.3]; dim2 = [0.2, 0.5, 0.3, 0.3];
str1 = 'k_1 = k_2 = k_3 = 0, 0.2, 0.4, -1.4; \kappa = 0, 0.05, 0.10,
0.14 ';
str21 = 'r=0.069; \Gamma=0.3; \gamma_u=0.34; \gamma_c = 0.34;  \gamma_m
=0.34;';
str22 = 'g=0.85; \sigma=0.08; \mu=0.00539; \delta=0.016; phi=0.84;
beta_fu=0.0128;';
str23 = 'beta_fc=0.0051; beta_m = 0.049; k_1=0.01; k_2=0.01; k_3=0.01;
\kappa = 0.01;';
str2 = append(str21,str22,str23);

for i=1:8
    figure(i)
    annotation('textbox',dim1,'String',str1,'FitBoxToText','on',
'LineStyle','none','FontSize',11);
    annotation('textbox',dim2,'String',str2,'FitBoxToText','on',
'LineStyle','none','FontSize',11);

annotation('arrow',[dim1(1),dim1(2)],[dim1(3),dim1(4)],'LineWidth',2)
end
```

```
%% The function code
function dx=HIV_transmission(t,x)
global g r Gamma phi mu gamma delta sigma beta_fu beta_fc beta_m k kap

dx1= (1-g)*(1-r)*Gamma - beta_fu*x(1)*x(4) - (kap+phi+mu)*x(1);
dx2= g*(1-r)*Gamma +(kap+phi)*x(1)- beta_fc*x(2)*x(5) - mu*x(2);
dx3= r*Gamma - beta_m*x(3)*x(6) - mu*x(3);
dx4= beta_fu*x(1)*x(4) - (k(1)+mu+gamma(1)+delta)*x(4);
dx5= beta_fc*x(2)*x(5) - (k(2)+mu+gamma(2)+delta)*x(5);
dx6= beta_m*x(3)*x(6) - (k(3)+mu+gamma(3)+delta)*x(6);
dx7= sum((k+gamma).*[x(4), x(5), x(6)]) - (mu+delta+sigma)*x(7);
dx8= sigma*x(7) - (mu+delta)*x(8);
dx = [dx1;dx2;dx3;dx4;dx5;dx6;dx7;dx8];
end
```