MATHEMATICAL MODELLING OF HIV/AIDS TRANSMISSION DYNAMICS COUPLED WITH AWARENESS AMONG ADOLESCENTS AND YOUNG ADULTS IN KENYA

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DECLARATION

I, the undersigned, declare that this is my original work except where explicitly stated and that it has not been submitted to any other institution for a degree.

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DEDICATION

To God Almighty for His faithfulness in my life. The completion of this research is a true witness of His favor in my life.

To my beloved father, Erick Ronoh Tuimising, who passed away towards the end of this research. He taught me the true meaning of perseverance, dedication and hard work which he evidently showed as he continued discharging his duties when gravely ill. His memory continues to regulate my life and his legacy gave me the much needed strength to continue with this research.

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ABSTRACT

Although HIV/AIDS containment campaigns have recorded substantial success within the past decade, the disease still poses a big health concern to many developing countries, including Kenya. We develop deterministic models to study the effect of HIV/AIDS awareness in the disease transmission dynamics of adolescents aged 10 - 14 years and young adults aged 15-24 years in Kenya. Early sexual debut and risky sexual behavior among the adolescents and young adults play a significant role in determining individual to population level vulnerability to HIV infection. Many mathematical epidemiology models neglect the fact that adolescents and young adults display interesting dynamics and they need to be researched on separately given that majority of all new HIV/AIDS infections in Kenya occur in adolescents and young adults and it is higher in young women than young men. Different regions display distinct differences in HIV/AIDS prevalence largely due to income levels, societal norms, HIV/AIDS status knowledge, health services, exposure risk among others. We study the impact of HIV/AIDS comprehensive knowledge, HIV/AIDS testing levels, condom use, antiretroviral therapy coverage and societal attitudes affecting HIV/AIDS testing, condom use and antiretroviral therapy in the Kenyan youth disease dynamics. We also consider spatial effects of targeted HIV/AIDS combinatory control among the Kenyan youth in high risk Counties. Our findings suggest that increasing comprehensive knowledge of HIV/AIDS among young women has a more direct relationship in decreasing new infection rates among young men, and vice versa. While highly efficacious combinatory control approach significantly reduces HIV/AIDS prevalence rates among adolescent girls and young women, and adolescent boys and young men, the disease remains endemic provided infected unaware sexual interactions persist. Disproportional gender-wise attitudes towards HIV/AIDS controls play a key role in reducing the Kenyan youth HIV/AIDS prevalence trends. Varying departure rates and return rates have little effect in the overall increase of new HIV/AIDS infections among the youth in high risk Counties in Kenya.

Contents

List of Figures

List of Tables

Acronyms

HIV	Human Immunodeficiency Virus
AIDS	Acquired Immunodeficiency Syndrome
AGYW	Adolescent Girls and Young Women
ABYM	Adolescent Boys and Young Men
UN	United Nations
UNAIDS	United Nations Programme on HIV and AIDS
UNICEF	United Nations International Children's Emergency Fund
NACC	National AIDS Control Council
NASCOP	National AIDS and STI Control Program
WHO	World Health Organization
CDC	Center for Disease Control and Prevention
DFE	Disease Free Equilibrium
EE	Endemic Equilibrium
KAIS	Kenya AIDS Indicator Survey
ART	Antiretroviral Therapy

Chapter 1

Introduction

1.1 Background

Human Immunodeficiency Virus (HIV) was first recognized in early 1980 in USA when a number of homosexual men in New York and California suddenly began to develop rare opportunistic infections and cancers that seemed stubbornly resistant to any treatment [5]. Around this time, this mysterious condition did not have a name, but it quickly became evident that all these men were suffering from a common ailment hence the discovery of HIV, the Human Immunodeficiency Virus, and its connection to AIDS [5]. However, what is likely to remain a mystery is who the first person to be infected with HIV/AIDS was or exactly how this disease spread from that initial person. Today, it has penetrated every geographical area and all cultural groups of the world [6]. The first case of HIV/AIDS in Kenya was detected in 1984, and by mid-1990 HIV/AIDS incidence was quite high that it taxed the health care system as well as the country's economy [7]. In Kenya, HIV/AIDS pandemic has generally affected all groups including children, adolescents, young adults, women and men but recent studies reveal that the categories of persons most vulnerable to HIV/AIDS transmission are drug users, sex workers and their clients, homosexuals, adolescents and young adults [8].

1.2 HIV Immunology

Human Immunodeficiency Virus is one of the lentivirus known as retroviruses which targets the human immune system. These viruses act to weaken the immune system by destroying important cells that fight diseases and infection. The name 'lentivirus' means 'slow virus' since they take a long period to produce any adverse effects in the body [5]. Interestingly, HIV resemble other viruses like those that cause common cold among others but unlike these viruses, the human immune system cannot eliminate them, implying that once you are infected with HIV, then it will be a lifelong problem. This virus reproduces itself by taking over the lymphocytes or white blood cells otherwise known as $CD4^+$ T cells importantly known for fighting infections and diseases in the body of its host and uses them to make more copies of it before destroying them. These cells

are also called "helper T cells" as they are known for the secretion of growth and differentiation factors required by other cell populations in the immune system. For a healthy person, the normal $CD4^+$ T cells count is around $1000mm^{-3}$ but $CD4^+$ T cells count in an HIV infected individual is always lower [9]. If there is no intervention like antiretroviral therapy then this virus is capable of destroying a huge number of $CD4^+$ T cells and when the count gets to $200mm^{-3}$ or below then the infected person is said to have a chronic and potentially life-threatening condition known as "Acquired Immunodeficiency Syndrome" or AIDS implying that the immune system is already too weak and a hampered immune system can't hold off any infections and diseases. This is how a person initially infected with HIV progresses to AIDS, which is the final stage of HIV infection [9].

1.3 HIV Transmission

The most common way for the spread of HIV is through sexual transmission but there are other ways of transmission such as mother to child transmission during childbirth or breast-feeding. The specific body fluids that facilitate the spread of HIV are blood, semen, pre-seminal fluid, rectal fluids, vaginal fluids and breast milk. It is important to note that these mucous membranes are mostly found inside the rectum, vagina, opening of the penis and mouth whereas damaged tissue is basically any tissue in which blood vesicles or capillaries may be exposed [9]. HIV can also be introduced by injection thus dirty needles tainted with infected blood used by either medical personnel or drug users can further propagate this pandemic.

1.4 Current Global Picture of the HIV Disease

By the end of 2018, there were about 37.9 million people living with HIV/AIDS globally out of which 4 million were adolescents and young adults aged 15 to 24 years [10]. Due to public awareness, improved HIV/AIDS response in Eastern and Southern Africa which has about 54% of the global population living with HIV/AIDS, there has been a significant decrease in new HIV/AIDS infections from 2.9 million in 1997 to 1.7 million in 2018 [10]. Globally, about 8.1 million people living with HIV/AIDS are not aware of their HIV/AIDS status. In Eastern and Southern Africa, only 19% and 14% of adolescent girls and adolescent boys aged 10 - 19 have tested for HIV/AIDS and been informed of their HIV/AIDS status [11]. Since the onset of the epidemic, about 32 million people have died from AIDS-related illnesses with 1.3 million deaths resulting from adolescents and young adults as a result of delayed treatment, overbearing stigma and discrimination associated with their HIV/AIDS status [10, 12]. Figure 1.1 shows the global prevalence of HIV/AIDS.

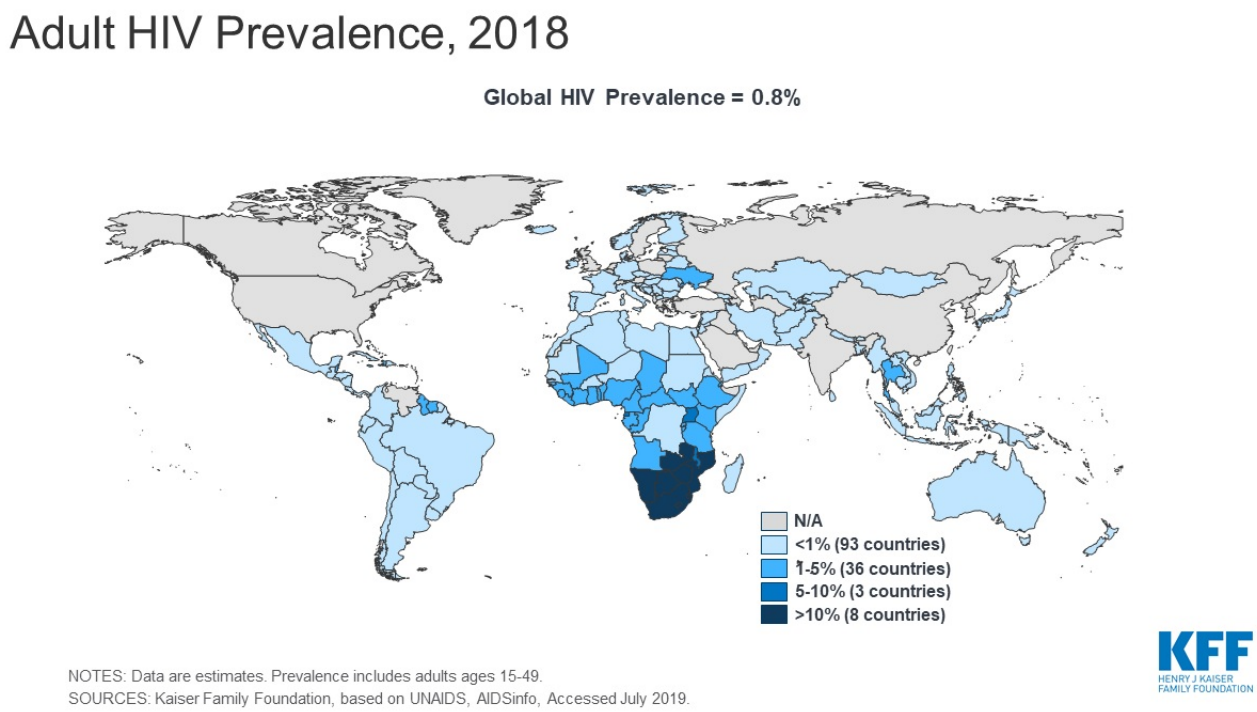
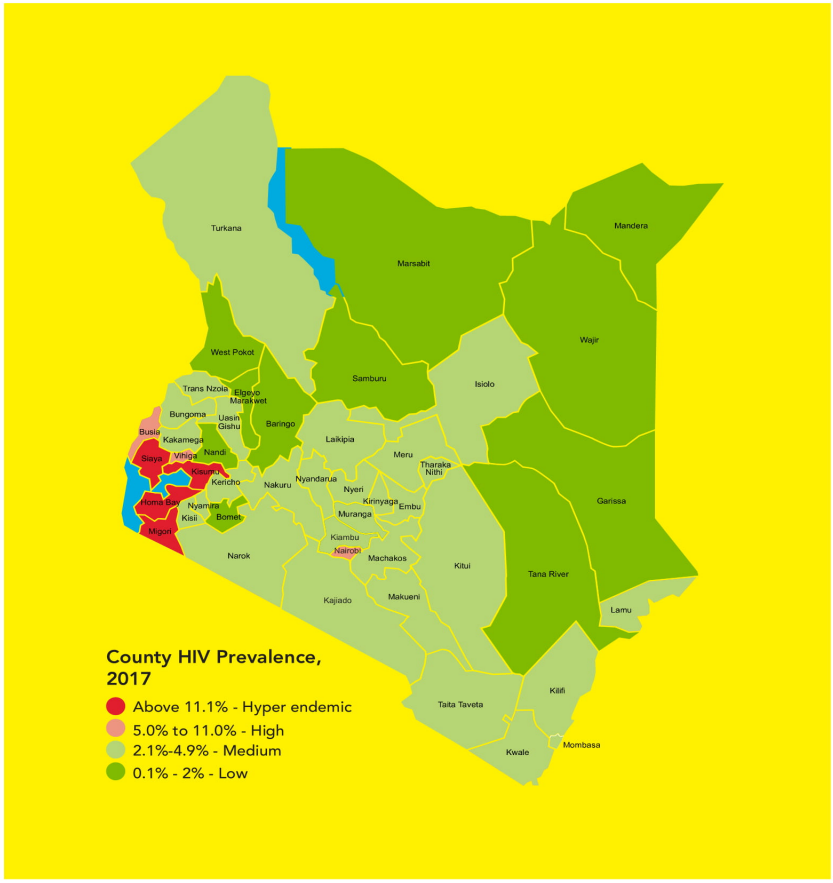


Figure 1.1: Global Prevalence of HIV/AIDS, [1]

1.5 Current HIV State in Kenya

HIV/AIDS is the leading course of morbidity and mortality in Kenya [6]. HIV/AIDS prevalence is quite high in urban areas compared to rural areas in Kenya with Nairobi, Homa Bay and Kisumu Counties having the highest prevalence whereas Wajir County recording the lowest prevalence over the years [2]. As of 2017, the national HIV/AIDS prevalence was about 5% with approximately 1.7 million people living with HIV/AIDS [2]. Adolescent girls and young women and adolescent boys and young men aged 15 to 24 national HIV/AIDS prevalence were 2.61% and 1.34% respectively [2]. There were about 52,800 new HIV/AIDS infections in 2017 with about 9,853 occuring in adolescent and young adults living in high risk Counties in Kenya [2]. Deaths due to AIDS related complications have decreased over the years in Kenya with about 28,200 estimated in 2017 from a high of 43,700 in 2010. AIDS related deaths among the adolescents and young adults in Kenya aged 10-24 dropped from 7500 in 2010 to 4900 in 2017 [2]. Despite the modest progress in HIV/AIDS related mortality in Kenya, majority of the youth are yet to be tested for HIV/AIDS and those who are aware of HIV/AIDS status are yet to be initiated into the treatment programs [2]. Figure 1.2 shows the prevalence of HIV/AIDS in Kenya by County.



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UNAIDS

CDC

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Figure 1.2: Prevalence of HIV/AIDS in Kenya by County, [2]

With the severity of the HIV/AIDS epidemic the Kenyan Government, particularly, the National Aids Control Council (NACC), National AIDS and STI Control Programme (NASCOP) together with other agencies such as Center for Disease Control and Prevention (CDC), the Joint United Nations Programme on HIV and AIDS (UNAIDS), World Health Organization (WHO) and other stakeholders have placed considerable amount of efforts and investments over the years targeting reductions of new HIV/AIDS infections [8]. Recent data review reveals that the cost of the national HIV/AIDS response amounted to over two percent of the annual Gross domestic product (GDP) in the years 2009/2010 to 2011/2012 [8]. Approximately sixty eight percent of the national HIV/AIDS response is externally funded [8]. The Kenyan government allocation towards the HIV/AIDS response has tripled from 57.49 million US dollars in the years 2006/2007 to 174

million US dollars in the years 2015/2016 [8, 13]. However, more investment is still required for effective combat against this disease as these current investments falls way below the country's needs [13].

1.6 Control Measures

Control measures are used to contain HIV epidemics, prevent death and disability (mortality and morbidity), and reduce socioeconomic loss. There is a wide range of HIV control interventions whose efficacy and effectiveness have been repeatedly demonstrated over many years such as use of condoms, prevention of mother to child transmission during pregnancy and childbirth, prompt and effective use of antiretroviral therapy treatment of HIV patients, male circumcision, education campaigns, public rallies and awareness promoting safe sex among adults and abstinence in adolescents. Use of condoms reduces disease transmission by interrupting exchange of vaginal/semen fluids whereas antiretroviral therapy, in combination with HIV/AIDS control programs, have recently played a key role in containing HIV epidemics and reducing new infections in endemic areas, resulting in significant reduction of the geographic range of HIV disease worldwide [14]. A recent HIV treatment guideline detailed by World Health Organization (WHO) recommends offering antiretroviral therapy (ART) to all persons living with HIV/AIDS irrespective of their $CD4^+$ T cell count [15]. This is because recent evidence indicates that administration of antiretroviral therapy increases the lifespan of people with HIV/AIDS and reduces the risk of transmitting HIV/AIDS to others substantially [15, 16]. Further, all people with HIV/AIDS with active tuberculosis or with hepatitis B disease are not to be exempted from antiretroviral therapy [15, 16]. Also the new guidelines recommend that all adults with HIV/AIDS take single fixed-dose combination pill of antiretroviral therapy daily as this is easier to administer and safe compared to the previous alternative combinations [15].

Over the years, however, the spread of new infections among adolescents and young adults has resulted in a re-emergence of HIV/AIDS in Kenya thus, turning back the clock on the disease control efforts [17]. Contributing factors of increasing new HIV/AIDS infections among the youth in Kenya are: denial and fear of their HIV/AIDS status, misinformation or otherwise lack of knowledge entirely of the HIV/AIDS disease, distrust of the medical establishment, fear and lack of belief in the effectiveness of medications, low self-esteem, unstructured and chaotic lifestyles, lack of family and social support, early marriages and sexual violence [17].

1.7 Motivation

More than half of those newly infected with HIV/AIDS in Kenya today are between 15 and 24 years old. In many places, sexual debut begins in adolescence and recent surveys of boys aged 15 to 19 in Kenya confirms that unmarried girls and boys are actually sexually active before the age of 15 [18]. This group is highly at risk of contracting HIV/AIDS as they are likely to have sex with high-risk partners or multiple partners, and are less likely to use condoms due to insufficient

or lack of knowledge of how HIV/AIDS is transmitted or how to adopt protective measures. Also, recent surveys from 40 countries indicate that more than 50 per cent of the young people aged 15 to 24 have serious misconceptions of HIV/AIDS transmission such as contracting HIV/AIDS through mosquito bites or witchcraft or being cured by having sex with a virgin among others [18]. In places where the adolescents and young adults are adequately informed, some will still engage in unprotected sex as they lack the necessary skills to negotiate abstinence or the use of condoms due to fear or embarrassment of talking about sex while others perceive their individual risk to be low [18]. Further, many adolescents experiment with drugs and alcohol at this stage hence, increasing their chances of risky sexual activities including multiple sexual partners [8]. Sadly, young girls in sub-Saharan Africa including Kenya have a higher prevalence than boys of similar age. One of the reasons for this peculiar prevalence is older men sexually preying on young girls. Many girls are disadvantaged economically and therefore easily lured into transactional sex in exchange for school fees, money or personal effeccts whereas on the other hand, many men assume that the younger girls are HIV/AIDS free. Further, many girls, boys and young women are also vulnerable to sexual violence, including abuse and exploitation with their violators less likely to use protection such as condoms hence, exposing them to possible HIV infection [8]. Street children happen to be casualties of war, poverty, domestic violence, physical/mental abuse, and HIV/AIDS. Their struggle for survival often exposes them to transactional sex in exchange for money, personal effects or protection resulting in possible HIV/AIDS infection thus, propagating HIV/AIDS pandemic. Children orphaned by HIV/AIDS are more vulnerable to malnutrition, illness, abuse, child labor and sexual exploitation compared to children orphaned by other reasons thus, increasing their exposure to HIV/AIDS infection [18]. It is vital to pay special attention to vulnerable adolescents and young adults if concerned stakeholders desire to be effective in HIV/AIDS control programs. Mathematical epidemiology offers a low-cost approach for theoretical evaluation of detection, prevention and control programs to help the affected stakeholders understand the effects of different policy decisions.

1.8 Objectives of the Study

General Objective

- To formulate a deterministic model that couples the transmission dynamics of HIV/AIDS in adolescents and young adults with awareness in Kenya.

Specific Objectives

- To show the effects of increasing comprehensive knowledge of HIV/AIDS in the sex-structured adolescents and young adults disease dynamics in Kenya.
- To show the effects of varying HIV/AIDS testing rates, condom use rates and antiretroviral adherence rates on the sex-structured adolescents and young adults disease dynamics in Kenya subject to societal attitudes affecting disease control.
- To investigate varying effects of HIV/AIDS targeted interventions on rural to urban and urban to rural migrating adolescents and young adults in Kenya.

1.9 Outline of this work

This work consists of three manuscripts, one of which is under review and the other two published. In chapter 2, we review the literature on HIV/AIDS mathematical models and metapopulation models. An age and sex-structured model is given in Chapter 3. Comprehensive knowledge of HIV/AIDS is coupled with the disease transmission dynamics of the adolescents and young adults. Mathematical analysis of infection through sub-network analysis was carried out and model simulations given. In Chapter 4, we present a sex-structured deterministic model to study combinatory control of HIV/AIDS together with societal attitudes affecting efficacy of the considered controls among the adolescents and young adults in Kenya. Mathematical analysis and numerical simulations of the single patch model is also presented. We consider a metapopulation model to investigate the role of mobility of the youth in the spread of HIV/AIDS in Kenya in Chapter 5. Simulations on both the control reproduction number and the two County model systems are given. In Chapter 6, a comprehensive conclusion of our results are given.

1.10 Manuscripts

This thesis is built around the following manuscripts.

Chapter 3

- M. Ronoh, F. Chirove, J. Wairimu, W. Ogana, Modeling Disproportional Effects of Educating Infected Kenyan Youth on HIV/AIDS, *Published online in the Journal of Biological Systems, Vol. 28, No. 2 (2020) 1–39, DOI: 10.1142/S0218339020400045.*

Chapter 4

- M. Ronoh, F. Chirove, J. Wairimu, W. Ogana, Evidence-Based Modeling of Combinatory Control on Kenyan Youth HIV/AIDS Dynamics, *Published online in PLoS One Journal, Vol. 15, No. 11 Nov(2020) 1-37, DOI: 10.1371/journal.pone.0242491.*

Chapter 5

- M. Ronoh, F. Chirove, J. Wairimu, W. Ogana, Modeling the Spatial Effects of HIV/AIDS Combinatory Control among the Kenyan Youth in High Risk Counties. *Submitted to African Journal of AIDS Research on 31 May 2020 for review .*

Chapter 2

Literature Review

2.1 Introduction

HIV is quite a recent disease and a number of mathematical models have been used to provide an explicit framework for understanding its transmission dynamics in human populations for over 20 years. Vast literature exists describing a host of modelling approaches. The disease, however, still thrives and constitutes a major source of death and morbidity, especially in developing countries. We review the literature on HIV/AIDS and metapopulation models here.

2.2 HIV/AIDS Mathematical Models

Culshaw et al.[19] considered an in-host two-dimensional model of cell-to-cell spread of HIV-1 in tissue cultures where they assumed that infection spread directly from infected cells to healthy cells. They modeled intracellular incubation period, a system of two differential equations with distributed delay, which included the differential equations model with a discrete delay and the ordinary differential equations model as special cases. Their results indicated that, differing from the cell-to-free virus spread models, the cell-to-cell spread models can produce infective oscillations in typical tissue culture parameter regimes and that the latently infected cells are instrumental in sustaining the infection.

Further, Waema et al. [20] considered an age-structured stochastic approach to model immunodeficiency virus (HIV) transmission epidemics which was specified by stochastic differential equations solved by use of Generating Functions (GF) models based on mother to child transmission (MTCT) (age group 0-5 years), heterosexual transmission (age group 15 and more years) and combined case (incorporating all groups and the two modes of transmission) were developed and the expectations and variances of Susceptible (S) persons, Infected (I) persons and AIDS cases were found. The S(t) susceptible model produced a constant expectation and increasing variance. It was shown that mother to child transmission and heterosexual models are special cases of the combined model. Also, Granich et al. [21] used a stochastic model to explore the effect on the case

reproduction number and a deterministic transmission model to explore the long-term dynamics of the HIV epidemic of testing all people in their test-case community (aged 15 years and older) for HIV every year, starting people on ART immediately after they are diagnosed HIV positive. They used data from South Africa as the test case for a generalized epidemic, and assumed that all HIV transmission was heterosexual. They showed that universal voluntary HIV testing and immediate ART, combined with present prevention approaches, could have a major effect on severe generalized HIV/AIDS epidemics.

Simwa et al.[22] formulated a deterministic model for HIV epidemic which in cooperated the three stages of disease progression among infected patients. They assumed that the patient once infected experienced disease progression up to full-blown AIDS. Further, they used two systems of ordinary differential equations coupled through a delay in one of the systems. Transmission of the HIV disease was limited to heterosexual and vertical transmission. They used numerical integration of the equations to simulate the stage-specific epidemic curves, given the demographic and epidemiological parameters of the model. Their findings from numerical simulation with respect to Uganda's HIV/AIDS epidemic scenario was found to be consistent with the published findings namely that the corresponding prevalence is a non-decreasing function of time for at least 30 years of the epidemic.

Tapadar et al. [23] developed a mathematical model of epidemiology for the spread of HIV where they considered On the basis of extensive analysis they made relevant comments on mutual coexistence of the group infected by HIV and the group not infected by that. They realized an alarming scenario where the group not infected by HIV decreases as well as the group infected by HIV expands with time which they termed as critically epidemic situation. They established the spread of HIV and unlike other infectious diseases they found that it was impossible to localize HIV.

Omondi et al. [24] and Nyabadza et al. [25] used deterministic models coupled with combined controls to study different strategies that would help curb the spread of HIV/AIDS in Kenya and South Africa respectively. The multiple strategies considered were HIV screening, antiretroviral therapy and codom use. These models were further fitted to UNAIDS HIV/AIDS data for Kenya and South Africa respectively. Study by Omondi et al. [24] showed that effective contact rates is the main driver fuelling HIV/AIDS epidemic trends in Kenya whereas Nyabadza et al. [25] was able to determine HIV/AIDS prevalence projections beyond 2007 for various levels of controls in South Africa.

Hussaini et al. [26] developed a nonlinear extended deterministic Susceptible Infected (SI) model for assessing the impact of public health education campaign on curtailing the spread of the HIV pandemic in a population. Rigorous qualitative analysis of the model revealed that, in contrast to the model without education, the full model with education exhibited the phenomenon of backward bifurcation (BB), where a stable disease-free equilibrium coexisted with a stable endemic equilibrium when the effective reproduction number was less than unity. They further derived an explicit threshold value above which, could lead to an increase in disease burden, and below which,

could reduce disease burden in the considered communities. Using data from various countries, they suggested that effective public health education focused on changing risky sexual behavior with a reasonable coverage level could help lower HIV/AIDS transmission risk but not eliminate it.

Mugisha et al. [27] used a continuous age-structured model of McKendrick-von-Foerster type to derive a two-age groups HIV/AIDS epidemic model. They analyzed the model and keenly observed the role of vertical transmission in the dynamics of the spread of the epidemic where they considered two scenarios: the case when the force of infection was a constant and the case when it was given as a mass action. Their results show that the only possible way to ensure a disease-free equilibrium is to bring the force of infection to zero indicating that the epidemic will die out if some effort is put on delivery of HIV-free babies. Also, Bassavarajaiah et al. [28] developed a mathematical model to study HIV/AIDS transmission from mother to child where they considered HIV/AIDS in a population of varying size with treatments and vertical transmission under the assumption that due to sexual interaction of susceptibles with infectives, the infected babies are born to increase the growth of infective population directly. They found that an increase in the rate of vertical transmission leads to increase the population of infectives which in turn increases the pre-AIDS and AIDS population.

2.3 Metapopulation Models

Metapopulation modeling is relatively new in mathematical epidemiology. Arino et al. [29] extended this idea to demonstrate how disease spreads in a metapopulation setting. They used a system of $4p$ ordinary differential equations to describe the disease spread in an environment divided into p patches. They extended the system to include cross infection between several species. They demonstrated that travel can change disease spread in a complicated way i.e., it could either help the disease to persist or aid in disease extinction.

Prosper et al. [30] developed a two-patch metapopulation model inclusive of human migration. They further established that without migration, the disease was endemic in one patch but not in the other, and adding human migration they showed the persistence of the disease in both patches. Their result indicated that regions with low malaria transmission should have an interest in helping to control or eliminate malaria in regions with higher malaria endemicity if human movement connects them. They also showed that control measures targeting the mosquito death rate would be more effective if the extrinsic incubation period was longer than the average mosquito lifespan otherwise, control efforts should focus on mosquito biting rates. Their sensitivity analyses indicated that the slower transmission patch was potentially the better target for malaria control efforts.

Yakob et al. [31] examined the significance of vector dispersal ability, larval habitat stability and productivity on the persistence and extinction of a mosquito population inhabiting a dynamic

network of breeding sites. They used this novel method of vector modelling to show that when dispersal is limited or vector distribution is patchy, the spread and growth of a mosquito population at the onset of a rainy season delayed and extinction through larval habitat destruction was more readily achieved. They also determined the impact of two alternative dry-season survival strategies on mosquito dynamics. Their simulations suggested that if adult vectors remained dormant throughout the dry season, the stage structure of the population was synchronized at the onset of the wet season and its growth was delayed. In contrast, a population that continued to breed throughout the dry season grew more rapidly and was more difficult to control.

Wairimu et al. [32] focused on an age-structured metapopulation model. They considered different ecosystems (or patches) identified as malaria hot spots in the Western Kenya highlands. They classified the hot spots as n patches and they analyzed the model using the theory of triangular system, monotone dynamical systems or anti-monotone non-linear dynamical systems, and Lyapunov-Lasalle invariance principle techniques. They established the existence and stability of disease-free and endemic equilibria. They showed that the age structuring reduced the magnitude of infection. Using actual data they carried out numerical simulation to demonstrate the role played by metapopulation and age structuring on disease-incidence and the reproduction number.

Isodry et al. [33] used a metapopulation model to study the effect of human movement on the HIV/AIDS transmission dynamics in various regions in Kenya. Using real data specifically census data, HIV/AIDS data and mobile phone data, they calibrated their model and the simulation results suggested that human movement between different regions in Kenya increases HIV/AIDS incidences in a relatively small way with regions with initially low HIV/AIDS prevalence most affected.

2.4 Summary

So far we have established the key results from the literature review section on the in-host dynamics of HIV/AIDS and, stochastic, deterministic and metapopulation transmission models on HIV/AIDS and other infectious diseases. Most of these mathematical models on HIV transmission consider homogeneous populations. These models consider that the dynamics of the youth (adolescents and young adults) and the adults are similar whereas that is not the case. These studies did not address the aggregation of age and sex structure in HIV/AIDS transmission dynamics to traverse and quantify the unitary contribution of these factors towards HIV prognosis. Coupling HIV/AIDS social drivers such as comprehensive knowledge, societal attitudes towards the use of condoms and antiretroviral therapy in the Kenyan youth disease dynamics and investigating the gender-wise effects still remains an aspect inadequately addressed in mathematical modeling. In mathematical epidemiology, some models for spatial spread of epidemics have been analyzed as demonstrated in the metapopulation literature review largely on other infectious diseases but hardly none focusing on the interaction of the Kenyan youth among patches connected by travel. Hence, the gap of knowledge which this study will attempt to address.

Chapter 3

Modeling Disproportional Effects of Educating Infected Kenyan Youth on HIV/AIDS

Keywords: HIV/AIDS, AGYW, ABYM, Comprehensive knowledge, Disproportional, Infection spread.

Abstract. We formulate an age and sex-structured deterministic model to assess the effect of increasing comprehensive knowledge of HIV/AIDS disease in the infected Adolescent Girls and Young Women (AGYW) and, Adolescent Boys and Young Men (ABYM) populations in Kenya. Mathematical analysis of infection through sub-network analysis was carried out to trace various infection routes and the veracity of various transmission routes as well as the associated probabilities. Using HIV data in Kenya on our model, disproportional effects were observed when dispensation of comprehensive knowledge of HIV/AIDS was preferred in one population over the other. Effective dispensation of comprehensive knowledge of HIV/AIDS in both the infected AGYW and ABYM populations significantly slows down the infection spread but may not eradicate it.

3.1 Introduction

HIV/AIDS is the leading cause of morbidity and mortality in Kenya [34] whose prevalence has declined over time to about 5.9% [35]. Regional prevalence disparity has been observed with the highest in Homa Bay County at 26% and the lowest in Wajir County at 0.4% [35]. About 18% of people living with HIV in 2015 were adolescents and young adults aged 15-24 years [36] who contributed about 51% of new HIV infections among adults [36]. This group also contributed one tenth of AIDS related deaths [36]. Recent surveys confirms early sexual activity in this age group hence their vulnerability to HIV infection as they are likely to have sex with high-risk partners or multiple partners [37]. This population is less likely to adopt protective measures due to wide gaps in the comprehensive knowledge of HIV/AIDS or otherwise poor/weak negotiation skills in using protective measures due to fear or embarrassment of talking about sex while others perceive their individual risk to be low [37, 38]. For many adolescents and young adults, this is often a time to experiment drugs and alcohol increasing their chances of risky sexual activities including multiple sexual partners [8].

Adolescent girls and young women (AGYW) aged 15-24 in sub-Saharan Africa including Kenya have a higher HIV prevalence than adolescent boys and young men (ABYM) of similar age [39]. Adolescent girls who are disadvantaged economically are easily lured into transactional sex with older men in exchange for school fees, money or goods while the older men are driven by the misconception that the younger girls are not yet infected with HIV/AIDS [18]. The AGYW population is also weak in negotiating protective measures making them an easy target for sexual violation including abuse and exploitation and their violators are less likely to use protection such as condoms exposing them to possible HIV infection [8]. Street children who are casualties of war, poverty, domestic violence, physical or mental abuse and HIV/AIDS in their struggle to survive are often exposed to risky transactional sex in exchange for food, money or protection thereby increasing their chances of contracting the infection. Children orphaned by HIV/AIDS are vulnerable to malnutrition, illness, abuse, child labor and sexual exploitation compared to children orphaned by other reasons and this increases their exposure to HIV infection [18]. The upsurge of "Sponsor" culture among the AGYW aged 18 - 24 years which is now more visible is a significant factor for sexual exploitation in Kenya [40]. A "Sponsor" is an older wealthy adult male who offers young women a glamorous lifestyle in exchange for sex and company [40]. The victims of

"Sponsor" culture are young women from poor backgrounds who lack parental support or those who desire a glamorous lifestyle inspired by socialites or their peers. These inter-generational transactional sexual relationships compound the risk of HIV/AIDS transmission [40].

Sexual violence in Kenya perpetuates the risks of HIV/AIDS transmission especially among the young women who are most affected. Efforts to thwart sexual violence in Kenya is proving to be an uphill task due to the patriarchal nature of the society, deeply entrenched cultural systems, male dominated judiciary and legislative structures among others [41]. While incapacitating the male perpetrators through imprisoning could offer an alternative solution to the HIV/AIDS pandemic, very few bad actors actually face the brunt of the criminal law in Kenya largely due to compromised justice systems which prosecutes about 5% of sex offenders while a large percentage remain at large [42].

With the severity of the HIV/AIDS epidemic, the Kenyan government together with other concerned stakeholders have put in place control measures whose efficacy and effectiveness have been repeatedly demonstrated over many years. Some of these controls are HIV testing and counseling, linkage to antiretroviral therapy treatment of HIV infected patients, media campaigns, safe spaces, family planning, post violence care, care-giving/parenting programmes, education subsidies, cash transfers to families/households, financial literacy training and voluntary medical male circumcision (VMCC) program [43, 44, 45, 46]. While these interventions have recently played a key role in containing Kenya's HIV epidemic, the rise in new HIV infections among the young people in Kenya sets back a decade's effort in disease control. [47].

A number of mathematical models have been used to provide an explicit framework for understanding the transmission dynamics of HIV/AIDS in human populations for over 20 years [19, 20, 21, 22, 23, 26, 48, 49, 50]. While these models have provided some understanding of the HIV/AIDS transmission and possible control measures, they do not address the aggregation of age and sex structure in HIV/AIDS dynamics to traverse and quantify the unitary contribution of these factors towards the HIV infection prognosis. In fact, coupling HIV/AIDS social drivers such as knowledge in the youth disease dynamics and investigating the gender-wise effects still remains an aspect inadequately addressed in mathematical modeling.

We seek to show the effects of increasing comprehensive knowledge of HIV/AIDS in the adolescents and young adults disease dynamics in Kenya. We propose a deterministic model that incorporates the demographic characteristics such as age and gender in the adolescents and young adults populations. HIV/AIDS awareness in Kenya is universal given that this disease is endemic in Kenya [37]. However, comprehensive knowledge of HIV/AIDS is still low at 56.93% among the AGYW and 64.78% among the ABYM in Kenya [38] in comparison to the Millennium Development Goal (MDG) of at least 85% [47] comprehensive knowledge of HIV/AIDS coverage among the AGYW/ABYM populations by the year 2030. An AGYW/ABYM has comprehensive knowledge of HIV/AIDS if he/she is cognizant of the fact that HIV/AIDS risk can be reduced by consistent use of condoms, by having a single partner who is HIV-negative and who has no other partners,

knowing that a healthy-looking person can have HIV/AIDS and rejecting the two most common local misconceptions about HIV/AIDS transmission through mosquitoes and sharing food [38].

3.2 Model Formulation

We formulate a model describing HIV transmission dynamics in the adolescent girls (AG) population aged 10-14, AGYW population aged 15-24, ABYM population aged 15-24 and the male perpetrators (MP) population older than 24 years. The AG, AGYW, ABYM and MP population is each categorized into three classes such that at time $t \geq 0$ there are susceptible AG (S_g), AGYW (S_f), ABYM (S_m) and MP (S_v), infected AG (I_{gu}), AGYW (I_{fu}), ABYM (I_{mu}) and MP (I_{vu}) who do not have comprehensive knowledge of HIV/AIDS and infected AG (I_{ga}), AGYW (I_{fa}), ABYM (I_{ma}) and MP (I_{va}) who have comprehensive knowledge of HIV/AIDS. The total size of the AG, AGYW, ABYM and MP population is given as $N_g = S_g + I_{gu} + I_{ga}$, $N_f = S_f + I_{fu} + I_{fa}$, $N_m = S_m + I_{mu} + I_{ma}$ and $N_v = S_v + I_{vu} + I_{va}$ respectively. C_w is the constant size of fertile women population aged $25 - 49$ making the total size of the fertile women population $N_w = N_f + C_w$ whereas $N_{mv} = N_m + N_v$ We do not include the adolescent girls aged $10 - 14$ in the fertile women population given that most teenage births in Kenya occur in adolescent girls aged $15 - 19$ [51]. Figure 3.1 represents the flow of individuals into different compartments in a single patch model.

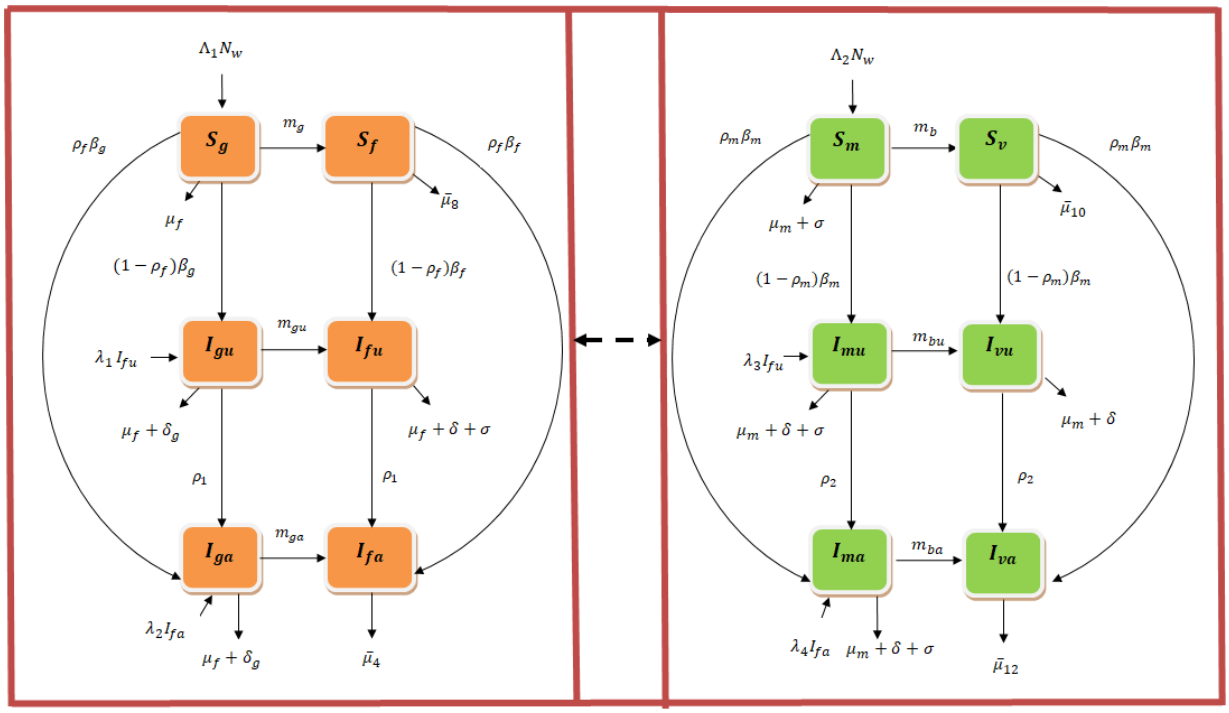


Figure 3.1: Schematics of the Compartmental Model

The adult male population plays a significant role in the AGYW HIV/AIDS transmission dynamics. As a consequence of the Kenyan dynamics, majority of the perpetrators are protected by

the system hence, we deliberately consider educating the male perpetrators infectious classes and investigate the potential impact of this intervention in our model.

We consider the AG population in this model as they are vulnerable to sexual violation from the MP and ABYM populations [52]. Adolescent boys aged 10-14 are not considered in this model as the rate this age group initiate sex is insignificant given that a greater percentage of adolescent boys initiate sex at the ages 15-19 years [37]. Adolescent boys aged 10-14 are coerced into sex mostly by men having sex with men (MSM) compared to older female perpetrators [53]. While there could be evidence of older women sexually exploiting adolescent boys, supporting data is still insufficient in Kenya [53]. Thus, we do not include the older female perpetrators in the model formulation. MSM is not considered as this model limits its mode of HIV/AIDS transmission to only heterosexual and vertical transmission routes.

Susceptible adolescent girls S_g, are free from the HIV infection but are at risk of infection through sexual contact with I_{mu}, I_{ma}, I_{vu} and I_{va}. Infectious classes I_{mu} and I_{vu} infects the S_g class more compared to I_{ma} and I_{va}. This is because the latter population is more cautious given their comprehensive knowledge of the HIV disease compared to the former population. The S_g class is at risk of infection at the rate $\beta_g = \beta_\Theta\, \gamma_g \left(\psi_1 I_{mu} + \psi_2 I_{ma} + \psi_1 I_{vu} + \psi_2 I_{va}\right)$ where β_Θ is the AG sexual contact rate, γ_g is the probability of the AG transmission risk, ψ_1, ψ_2 are dimensionless parameters to give comparison of the infection veracity with $0 < \psi_2 < \psi_1 < 1$. The force of infection, β_g, has a bi-linear incidence since the rate of infection depends on the availability of adolescent girls and male predators.

Susceptible AGYW S_f, are also free from the HIV infection but are at risk of infection through sexual contact with I_{mu}, I_{ma}, I_{vu} and I_{va} at the rate β_f where $\beta_f = \beta_{\text{♀}}\gamma_f \left(\frac{\psi_1 I_{mu} + \psi_2 I_{ma}}{N_{mv}} + \frac{\psi_1 I_{vu} + \psi_2 I_{va}}{N_{mv}}\right)$. $\beta_{\text{♀}}$ is the female sexual contact rate and γ_f is the probability of AGYW transmission risk. The force of infection, β_f, has a proportionate mixing incidence given that the contact rate of the AGYW and the ABYM/older male population is constant as some of the S_f population will have already initiated sex with most of them remaining sexually active with the ABYM and the older male population.

Susceptible ABYM (S_m) and susceptible male predators (S_v) populations are free from the HIV infection but are at risk of infection through sexual contact with I_{gu}, I_{ga}, I_{fu} and I_{fa} population. The adult women population is not considered in this model given their insignificant contribution to the infection in the ABYM population as supporting data still is insufficient in Kenya [53]. Thus, S_m and S_v classes are at risk of infection at the rate β_m where $\beta_m = \gamma_m \left(\beta_{\text{♂}} \left[\frac{\psi_1 I_{fu} + \psi_2 I_{fa}}{N_f}\right] + \beta'_{\text{♂}} N_g \left[\frac{\psi_1 I_{gu} + \psi_2 I_{ga}}{N_g}\right]\right)$. $\beta_{\text{♂}}$ is the male sexual contact rate with AGYW, $\beta'_{\text{♂}} N_g$ is the male sexual contact rate with AG and γ_m is the probability of ABYM transmission risk. The force of infection, β_m, has a combined transmission dynamics [54] of proportionate mixing and bi-linear incidences given the heterogeneities in the contact dynamics of the AGYW and AG populations respectively.

In our model, recruitment into the adolescent girls/boys population is only by natural births and age maturity. $\Lambda_1 N_w$ is the rate adolescent girls who are born free from the HIV infection survives and matures to age 10 so as to be recruited into the S_g population with $\Lambda_1 = \kappa\,(1-\pi)\,\lambda\,\alpha\,e^{-\mu_f \alpha}$. λ is the natural birth rate, κ is the female sex ratio at birth, α is the girls maturity rate, $1-\pi$ is the proportion of the HIV free newborns, $e^{-\mu_f \alpha}$ is the girls survival probability and N_w is the total size of the fertile female population aged $15-49$. $\lambda_1\, I_{fu}$ and $\lambda_2\, I_{fa}$ are the vertical transmission rates of the I_{gu} and I_{ga} respectively with $\lambda_1 = \kappa\,(1-\rho_{fk})\pi\,\lambda\,\alpha\,e^{-\mu_f \alpha}$ and $\lambda_2 = \kappa\rho_{fk}\pi\,\lambda\,\alpha\,e^{-\mu_f \alpha}$. π is the proportion of the HIV infected newborns and ρ_{fk} is the proportion of the HIV infected girls with comprehensive knowledge. $\Lambda_2 N_w$ is the rate adolescent boys who are born free from the HIV infection survives and matures to age 15 so as to be recruited into the S_m population with $\Lambda_2 = \nu(1-\pi)\lambda\zeta\, e^{-\mu_m \zeta}$. ζ is the boys maturity rate, ν is the male sex ratio at birth and $e^{-\mu_m \zeta}$ is the male survival probability. $\lambda_3\, I_{fu}$ and $\lambda_4\, I_{fa}$ are the vertical transmission rates of the I_{mu} and I_{ma} respectively with $\lambda_3 = \nu(1-\rho_{mk})\pi\lambda\zeta e^{-\mu_m \zeta}$ and $\lambda_4 = \nu\rho_{mk}\pi\lambda\zeta e^{-\mu_m \zeta}$. ρ_{mk} is the proportion of the HIV infected boys with comprehensive knowledge.

The AG, AGYW, ABYM and MP susceptible populations are decreased by natural deaths, μ_f, μ_m and infection rates, β_g, β_f, and β_m. Susceptible individuals who get infected and lack comprehensive knowledge of HIV/AIDS move to the classes I_{gu}, I_{fu}, I_{mu}, I_{vu} at rates $(1-\rho_f)\beta_g$, $(1-\rho_f)\beta_f$ and $(1-\rho_m)\beta_m$ respectively resulting in an increase of the I_{gu}, I_{fu}, I_{mu} and I_{vu} classes. ρ_f, ρ_m and, $1-\rho_f$, $1-\rho_m$ are dimensionless quantities representing the proportion of newly infected susceptible individuals with and without comprehensive knowledge of HIV/AIDS respectively. Susceptible individuals who get infected and have the comprehensive knowledge of HIV/AIDS move to the classes I_{ga}, I_{fa}, I_{ma} and I_{va} at rates $\rho_f\beta_g$, $\rho_f\beta_f$ and $\rho_m\beta_m$ respectively resulting in an increase of the I_{ga}, I_{fa}, I_{ma} and I_{va} classes. The infected individuals in the I_{gu}, I_{fu}, I_{mu} and I_{vu} classes upon gaining comprehensive knowledge of HIV/AIDS, move to the I_{ga}, I_{fa}, I_{ma} and I_{va} classes respectively at the rate ρ_1 and ρ_2. The infectious classes are all decreased by natural deaths, μ_f, μ_m and disease induced deaths, δ_g, δ. Upon turning 15 years, the adolescent girls in S_g, I_{gu} and I_{ga} classes move to S_f, I_{fu} and I_{fa} classes at the rates m_g, m_{gu} and m_{ga} respectively. Upon turning 24 years, the AGYW and the ABYM population exit the model at the rate σ whereas a proportion of the ABYM population who prey on the AGYW population joins the older male perpetrators classes S_v, I_{vu} and I_{va} at the rates m_b, m_{bu} and m_{ba} respectively . The state variables and parameters are assumed to be positive given that a population dynamics model is being studied. The system of ordinary differential equations governing the AGYW/ABYM HIV model coupled with comprehensive knowledge of HIV/AIDS is given by the system of equations (3.1) as

$$
\begin{cases}
\dfrac{dS_g}{dt} = \Lambda_1 N_w - (1-\rho_f)\beta_g S_g - \rho_f \beta_g S_g - \bar{\mu}_9 S_g, \\
\dfrac{dS_f}{dt} = m_g S_g - (1-\rho_f)\beta_f S_f - \rho_f \beta_f S_f - \bar{\mu}_8 S_f, \\
\dfrac{dS_m}{dt} = \Lambda_2 N_w - (1-\rho_m)\beta_m S_m - \rho_m \beta_m S_m - \bar{\mu}_7 S_m, \\
\dfrac{dS_v}{dt} = m_b S_m - (1-\rho_m)\beta_m S_v - \rho_m \beta_m S_v - \bar{\mu}_{10} S_v, \\
\dfrac{dI_{gu}}{dt} = \lambda_1 I_{fu} + (1-\rho_f)\beta_g S_g - \bar{\mu}_1 I_{gu}, \\
\dfrac{dI_{ga}}{dt} = \lambda_2 I_{fa} + \rho_f \beta_g S_g + \rho_1 I_{gu} - \bar{\mu}_2 I_{ga}, \\
\dfrac{dI_{fu}}{dt} = m_{gu} I_{gu} + (1-\rho_f)\beta_f S_f - \bar{\mu}_3 I_{fu}, \\
\dfrac{dI_{fa}}{dt} = m_{ga} I_{ga} + \rho_f \beta_f S_f + \rho_1 I_{fu} - \bar{\mu}_4 I_{fa}, \\
\dfrac{dI_{mu}}{dt} = \lambda_3 I_{fu} + (1-\rho_m)\beta_m S_m - \bar{\mu}_5 I_{mu}, \\
\dfrac{dI_{ma}}{dt} = \lambda_4 I_{fa} + \rho_m \beta_m S_m + \rho_2 I_{mu} - \bar{\mu}_6 I_{ma}, \\
\dfrac{dI_{vu}}{dt} = m_{bu} I_{mu} + (1-\rho_m)\beta_m S_v - \bar{\mu}_{11} I_{vu}, \\
\dfrac{dI_{va}}{dt} = m_{ba} I_{ma} + \rho_m \beta_m S_v + \rho_2 I_{vu} - \bar{\mu}_{12} I_{va},
\end{cases}
\tag{3.1}
$$

where $\bar{\mu}_1 = \rho_1 + \delta_g + m_{gu} + \mu_f$, $\bar{\mu}_2 = \delta_g + m_{ga} + \mu_f$, $\bar{\mu}_3 = \rho_1 + \mu_f + \delta + \sigma$, $\bar{\mu}_4 = \mu_f + \delta + \sigma$, $\bar{\mu}_5 = \rho_2 + \mu_m + \delta + \sigma + m_{bu}$, $\bar{\mu}_6 = \mu_m + \delta + \sigma + m_{ba}$, $\bar{\mu}_7 = \mu_m + \sigma + m_b$, $\bar{\mu}_8 = \mu_f + \sigma$, $\bar{\mu}_9 = m_g + \mu_f$, $\bar{\mu}_{10} = \mu_m$, $\bar{\mu}_{11} = \rho_2 + \mu_m + \delta$, $\bar{\mu}_{12} = \mu_m + \delta$.

Tables 3.1 and 3.2 give summaries of the state variables and parameters descriptions used in the model formulation.

Table 3.1: Description of State variables

Variable	Description
S_g	Susceptible AG
S_f	Susceptible AGYW
S_m	Susceptible ABYM
S_v	Susceptible male perpetrators
I_{gu}	Infected AG with no comprehensive knowledge of HIV/AIDS
I_{fu}	Infected AGYW with no comprehensive knowledge of HIV/AIDS
I_{ga}	Infected AG with comprehensive knowledge of HIV/AIDS
I_{fa}	Infected AGYW with comprehensive knowledge of HIV/AIDS
I_{mu}	Infected ABYM with no comprehensive knowledge of HIV/AIDS
I_{ma}	Infected ABYM with comprehensive knowledge of HIV/AIDS
I_{vu}	Infected male perpetrators with no comprehensive knowledge of HIV/AIDS
I_{va}	Infected male perpetrators with comprehensive knowledge of HIV/AIDS

Table 3.2: Description of Parameters

Parameter	Description
ρ_f, ρ_m	Proportion of comprehensive knowledge of the newly infected AGYW and ABYM respectively
λ	Natural birth rate of AGYW and ABYM
μ_f, μ_m	Natural death rate of AGYW and ABYM respectively
γ_g,γ_f, γ_m	Probability of AG, AGYW and ABYM transmission risk
δ_g, δ_m	Disease induced deaths in AGYW and ABYM respectively
α, ζ	Girls and boys maturity rate respectively
π	Proportion of the AGYW/ABYM population born HIV infected
ρ_{fk}, ρ_{mk}	Proportion of HIV infected girls and boys with comprehensive knowledge of HIV/AIDS
κ, ν	Female and male sex ratio at birth respectively
$\beta_{\text{♀}}$, β_{Θ}	AGYW and AG sexual contact rate
$\beta_{\text{♂}}$, $\beta'_{\text{♂}}N_g$	Male sexual contact rate with AGYW and AG respectively
ρ_1, ρ_2	Transition rate of I_{gu}, I_{fu} and, I_{mu}, I_{vu} to I_{ga}, I_{fa} and, I_{ma}, I_{va} respectively
m_g	Transition rate of S_g to S_f
m_{gu}	Transition rate of I_{gu} to I_{fu}
m_{ga}	Transition rate of I_{ga} to I_{fa}
σ	Rate at which the AGYW and ABYM exit the model upon turning 24
m_b	Transition rate of S_m to S_v
m_{bu}	Transition rate of I_{mu} to I_{vu}
m_{ba}	Transition rate of I_{ma} to I_{va}
ψ_1, ψ_2	Dimensionless parameters to give comparison of infection veracity

3.3 Model Analysis

This section presents the mathematical analysis of the formulated model given in section 2. We show that the system of ordinary differential equations (3.1) governing the model is mathematically and epidemiologically well-posed. We calculate the basic reproduction number and give its biological interpretation.

3.3.1 Positivity of Model Solutions

Theorem 3.3.1. *The solution set* $(S_g(t), S_f(t), S_m(t), S_v(t), I_{gu}(t), I_{ga}(t), I_{fu}(t), I_{fa}(t), I_{mu}(t), I_{ma}(t), I_{vu}(t), I_{va}(t))$ *of the model system* (3.1) *is non-negative for all* $t > 0$.

Proof. We use the concept of contradiction to prove that the solutions of the model system (3.1) is strictly positive.

Suppose by contradiction that $t_i, i = 1, 2, ..., 12$ are the initial times when the model solutions $S_g(t), S_f(t), S_m(t), S_v(t), I_{gu}(t), I_{ga}(t), I_{fu}(t), I_{fa}(t), I_{mu}(t), I_{ma}(t), I_{vu}(t), I_{va}(t)$ approach 0 respectively with $t_0 = min\{t_i\}$.

If $t_0 = t_1$, we let $t_1 \neq t_2, t_3, ..., t_{12}$ and $S_g(t)$ be strictly less than 0 in the interval $[0, t_1]$ with $S_g(t_1) = 0$. Let $S_f(t_1) > 0, S_m(t_1) > 0, S_v(t_1) > 0, I_{gu}(t_1) > 0, I_{ga}(t_1) > 0, I_{fu}(t_1) > 0, I_{fa}(t_1) > 0, I_{mu}(t_1) > 0, I_{ma}(t_1) > 0, I_{vu}(t_1) > 0, I_{va}(t_1) > 0 \ \forall t \in [0, t_1]$.

The first equation of the model system (3.1) can be expressed as

$$\frac{dS_g}{dt} = \Lambda_1 N_w - (\beta_g + \bar{\mu}_9) S_g.$$

It is clear that

$$\frac{dS_g}{dt} = \Lambda_1 N_w - (\beta_g + \bar{\mu}_9) S_g \geq -(\beta_g + \bar{\mu}_9) S_g,$$

$$S_g(t) \geq S_g(0)\, e^{\int_0^t -(\beta_g(h) + \bar{\mu}_9) dh}.$$

Now, when $t = t_1$,

$$S_g(t_1) \geq S_g(0)\, e^{\int_0^{t_1} -(\beta_g(h) + \bar{\mu}_9) dh} > 0. \tag{3.2}$$

Equation 3.2 contradicts the fact that $S_g(t_1) = 0$. Thus, $S_g(t)$ is neither strictly negative in the interval $[0, t_1]$ nor equal to 0. This implies that, $S_g(t) > 0$.

If $t_0 = t_2$, and $t_2 \neq t_1, t_3, t_4, ..., t_{12}$ and $S_f(t) < 0$ in the interval $[0, t_2]$ with $S_f(t_2) = 0$. Let $S_g(t_2) > 0, S_m(t_2) > 0, S_v(t_2) > 0, I_{gu}(t_2) > 0, I_{ga}(t_2) > 0, I_{fu}(t_2) > 0, I_{fa}(t_2) > 0, I_{mu}(t_2) > 0, I_{ma}(t_2) > 0, I_{vu}(t_2) > 0, I_{va}(t_2) > 0 \ \forall t \in [0, t_2]$.

The second equation of the model system (3.1) can be written as

$$\frac{dS_f}{dt} = m_g S_g - (\beta_f + \bar{\mu}_8) S_f.$$

It is clear that

$$\frac{dS_f}{dt} = m_g S_g - (\beta_f + \bar{\mu}_8) S_f \geq -(\beta_f + \bar{\mu}_8) S_f,$$

$$S_f(t) \geq S_f(0)\, e^{\int_0^t -(\beta_f(h) + \bar{\mu}_8) dh}.$$

Now, when $t = t_2$,

$$S_f(t_2) \geq S_f(0)\, e^{\int_0^{t_2} -(\beta_f(h) + \bar{\mu}_8) dh} > 0. \tag{3.3}$$

Equation 3.3 contradicts the fact that $S_f(t_2) = 0$. Thus, $S_f(t)$ is neither strictly negative in the interval $[0, t_2]$ nor equal to 0. This implies that, $S_f(t) > 0$.

If $t_0 = t_3$, and $t_3 \neq t_1, t_2, t_4, t_5, ..., t_{12}$ and $S_m(t) < 0$ in the interval $[0, t_3]$ with $S_m(t_3) = 0$. Let $S_g(t_3) > 0, S_f(t_3) > 0, S_v(t_3) > 0, I_{gu}(t_3) > 0, I_{ga}(t_3) > 0, I_{fu}(t_3) > 0, I_{fa}(t_3) > 0, I_{mu}(t_3) > 0, I_{ma}(t_3) > 0, I_{vu}(t_3) > 0, I_{va}(t_3) > 0\ \forall t \in [0, t_3]$.

From the third equation of the model system (3.1),

$$\frac{dS_m}{dt} = \Lambda_2 N_w - (\beta_m + \bar{\mu}_7) S_m \geq -(\beta_m + \bar{\mu}_7) S_m,$$
$$S_m(t) \geq S_m(0)\, e^{\int_0^t -(\beta_m(h) + \bar{\mu}_7) dh}.$$

At $t = t_3$,

$$S_m(t_3) \geq S_m(0)\, e^{\int_0^{t_3} -(\beta_m(h) + \bar{\mu}_7) dh} > 0. \tag{3.4}$$

Equation 3.4 contradicts the fact that $S_m(t_3) = 0$. Thus, $S_m(t)$ is neither strictly negative in the interval $[0, t_3]$ nor equal to 0. This implies that, $S_m(t) > 0$.

If $t_0 = t_4$, and $t_4 \neq t_1, t_2, t_3, t_5, t_6, ..., t_{12}$ and $S_v(t) < 0$ in the interval $[0, t_4]$ with $S_v(t_4) = 0$. Let $S_g(t_4) > 0, S_f(t_4) > 0, S_m(t_4) > 0, I_{gu}(t_4) > 0, I_{ga}(t_4) > 0, I_{fu}(t_4) > 0, I_{fa}(t_4) > 0, I_{mu}(t_4) > 0, I_{ma}(t_4) > 0, I_{vu}(t_4) > 0, I_{va}(t_4) > 0\ \forall t \in [0, t_4]$.

From the fourth equation of the model system (3.1),

$$\frac{dS_v}{dt} = m_b S_m - (\beta_m + \bar{\mu}_{10}) S_v \geq -(\beta_m + \bar{\mu}_{10}) S_v,$$
$$S_v(t) \geq S_v(0)\, e^{\int_0^t -(\beta_m(h) + \bar{\mu}_{10}) dh}.$$

At $t = t_4$,

$$S_v(t_4) \geq S_v(0)\, e^{\int_0^{t_4} -(\beta_m(h) + \bar{\mu}_{10}) dh} > 0. \tag{3.5}$$

Equation 3.5 contradicts the fact that $S_v(t_4) = 0$. Thus, $S_v(t)$ is neither strictly negative in the interval $[0, t_4]$ nor equal to 0. This implies that, $S_v(t) > 0$.

If $t_0 = t_5$, and $t_5 \neq t_1, t_2, t_3, t_4, t_6, t_7..., t_{12}$ and $I_{gu}(t) < 0$ in the interval $[0, t_5]$ with $I_{gu}(t_5) = 0$. Let $S_g(t_5) > 0, S_f(t_5) > 0, S_m(t_5) > 0, S_v(t_5) > 0, I_{ga}(t_5) > 0, I_{fu}(t_5) > 0, I_{fa}(t_5) > 0, I_{mu}(t_5) > 0, I_{ma}(t_5) > 0, I_{vu}(t_5) > 0, I_{va}(t_5) > 0 \; \forall t \in [0, t_5]$.

From the fifth equation of the model system (3.1),

$$\frac{dI_{gu}}{dt} = \lambda_1 I_{fu} + (1 - \rho_f)\beta_g S_g - \bar{\mu}_1 I_{gu} \geq -\bar{\mu}_1 I_{gu},$$
$$I_{gu}(t) \geq I_{gu}(0)\, e^{-\bar{\mu}_1 t}.$$

At $t = t_5$,

$$I_{gu}(t_5) \geq I_{gu}(0)\, e^{-\bar{\mu}_1 t_5} > 0. \tag{3.6}$$

Equation 3.6 contradicts the fact that $I_{gu}(t_5) = 0$. Thus, $I_{gu}(t) \not< 0$ in the interval $[0, t_5]$ and $I_{gu}(t) \neq 0$. This implies that, $I_{gu}(t) > 0$.

If $t_0 = t_6$, and $t_6 \neq t_1, t_2, t_3, t_4, t_5, t_7, t_8..., t_{12}$ and $I_{ga}(t) < 0$ in the interval $[0, t_6]$ with $I_{ga}(t_6) = 0$. Let $S_g(t_6) > 0, S_f(t_6) > 0, S_m(t_6) > 0, S_v(t_6) > 0, I_{gu}(t_6) > 0, I_{fu}(t_6) > 0, I_{fa}(t_6) > 0, I_{mu}(t_6) > 0, I_{ma}(t_6) > 0, I_{vu}(t_6) > 0, I_{va}(t_6) > 0 \; \forall t \in [0, t_6]$.

From the sixth equation of the model system (3.1),

$$\frac{dI_{ga}}{dt} = \lambda_2 I_{fa} + \rho_f \beta_g S_g + \rho_1 I_{gu} - \bar{\mu}_2 I_{ga} \geq -\bar{\mu}_2 I_{ga},$$
$$I_{ga}(t) \geq I_{ga}(0)\, e^{-\bar{\mu}_2 t}.$$

At $t = t_6$,

$$I_{ga}(t_6) \geq I_{ga}(0)\, e^{-\bar{\mu}_2 t_6} > 0. \tag{3.7}$$

Equation 3.7 contradicts the fact that $I_{ga}(t_6) = 0$. Thus, $I_{ga}(t) \not< 0 \in [0, t_6]$ and $I_{ga}(t) \neq 0$. This implies that, $I_{ga}(t) > 0$.

If $t_0 = t_7$, and $t_7 \neq t_1, t_2, t_3, t_4, t_5, t_6, t_8, t_9, t_{10}, t_{11}, t_{12}$ and $I_{fu}(t) < 0$ in the interval $[0, t_7]$ with $I_{fu}(t_7) = 0$. Let $S_g(t_7) > 0, S_f(t_7) > 0, S_m(t_7) > 0, S_v(t_7) > 0, I_{gu}(t_7) > 0, I_{ga}(t_7) > 0, I_{fa}(t_7) > 0, I_{mu}(t_7) > 0, I_{ma}(t_7) > 0, I_{vu}(t_7) > 0, I_{va}(t_7) > 0 \; \forall t \in [0, t_7]$.

From the seventh equation of the model system (3.1),

$$\frac{dI_{fu}}{dt} = m_{gu}I_{gu} + (1-\rho_f)\beta_f S_f - \bar{\mu}_3 I_{fu} \geq -\bar{\mu}_3 I_{fu},$$
$$I_{fu}(t) \geq I_{fu}(0)\, e^{-\bar{\mu}_3 t}.$$

At $t = t_7$,

$$I_{fu}(t_7) \geq I_{fu}(0)\, e^{-\bar{\mu}_3 t_7} > 0. \tag{3.8}$$

Equation 3.8 contradicts the fact that $I_{fu}(t_7) = 0$. Thus, $I_{fu}(t) \not< 0 \in [0, t_7]$ and $I_{fu}(t) \neq 0$. This implies that, $I_{fu}(t) > 0$.

If $t_0 = t_8$, and $t_8 \neq t_1, t_2, t_3, t_4, t_5, t_6, t_7, t_9, t_{10}, t_{11}, t_{12}$ and $I_{fa}(t) < 0$ in the interval $[0, t_8]$ with $I_{fa}(t_8) = 0$. Let $S_g(t_8) > 0, S_f(t_8) > 0, S_m(t_8) > 0, S_v(t_8) > 0, I_{gu}(t_8) > 0, I_{ga}(t_8) > 0, I_{fu}(t_8) > 0, I_{mu}(t_8) > 0, I_{ma}(t_8) > 0, I_{vu}(t_8) > 0, I_{va}(t_8) > 0\ \forall t \in [0, t_8]$.

From the eighth equation of the model system (3.1),

$$\frac{dI_{fa}}{dt} = m_{ga}I_{ga} + \rho_f\beta_f S_f + \rho_1 I_{fu} - \bar{\mu}_4 I_{fa} \geq -\bar{\mu}_4 I_{fa},$$
$$I_{fa}(t) \geq I_{fa}(0)\, e^{-\bar{\mu}_4 t}.$$

At $t = t_8$,

$$I_{fa}(t_8) \geq I_{fa}(0)\, e^{-\bar{\mu}_4 t_8} > 0. \tag{3.9}$$

Equation 3.9 contradicts the fact that $I_{fa}(t_8) = 0$. Thus, $I_{fa}(t) \not< 0 \in [0, t_8]$ and $I_{fa}(t) \neq 0$. This implies that, $I_{fa}(t) > 0$.

If $t_0 = t_9$, and $t_9 \neq t_1, t_2, t_3, t_4, t_5, t_6, t_7, t_8, t_{10}, t_{11}, t_{12}$ and $I_{mu}(t) < 0$ in the interval $[0, t_9]$ with $I_{mu}(t_9) = 0$. Let $S_g(t_9) > 0, S_f(t_9) > 0, S_m(t_9) > 0, S_v(t_9) > 0, I_{gu}(t_9) > 0, I_{ga}(t_9) > 0, I_{fu}(t_9) > 0, I_{fa}(t_9) > 0, I_{ma}(t_9) > 0, I_{vu}(t_9) > 0, I_{va}(t_9) > 0\ \forall t \in [0, t_9]$.

From the ninth equation of the model system (3.1),

$$\lambda_3 I_{fu} + (1-\rho_m)\beta_m S_m - \bar{\mu}_5 I_{mu} \geq -\bar{\mu}_5 I_{mu},$$
$$I_{mu}(t) \geq I_{mu}(0)\, e^{-\bar{\mu}_5 t}.$$

At $t = t_9$,

$$I_{mu}(t_9) \geq I_{mu}(0)\, e^{-\bar{\mu}_5 t_9} > 0. \tag{3.10}$$

Equation 3.10 contradicts the fact that $I_{mu}(t_9) = 0$. Thus, $I_{mu}(t) \not< 0 \in [0, t_9]$ and $I_{mu}(t) \neq 0$. This implies that, $I_{mu}(t) > 0$.

If $t_0 = t_{10}$, and $t_{10} \neq t_1, t_2, t_3, t_4, t_5, t_6, t_7, t_8, t_9, t_{11}, t_{12}$ and $I_{ma}(t) < 0$ in the interval $[0, t_{10}]$ with $I_{ma}(t_{10}) = 0$. Let $S_g(t_{10}) > 0, S_f(t_{10}) > 0, S_m(t_{10}) > 0, S_v(t_{10}) > 0, I_{gu}(t_{10}) > 0, I_{ga}(t_{10}) > 0, I_{fu}(t_{10}) > 0, I_{fa}(t_{10}) > 0, I_{mu}(t_{10}) > 0, I_{vu}(t_{10}) > 0, I_{va}(t_{10}) > 0 \; \forall t \in [0, t_{10}]$.

From the tenth equation of the model system (3.1),

$$\frac{dI_{ma}}{dt} = \lambda_4 I_{fa} + \rho_m \beta_m S_m + \rho_2 I_{mu} - \bar{\mu}_6 I_{ma} \geq -\bar{\mu}_6 I_{ma},$$
$$I_{ma}(t) \geq I_{ma}(0)\, e^{-\bar{\mu}_6 t}.$$

At $t = t_{10}$,

$$I_{ma}(t_{10}) \geq I_{ma}(0)\, e^{-\bar{\mu}_6 t_{10}} > 0. \tag{3.11}$$

Equation 3.11 contradicts the fact that $I_{ma}(t_{10}) = 0$. Thus, $I_{ma}(t) \not< 0 \in [0, t_{10}]$ and $I_{ma}(t) \neq 0$. This implies that, $I_{ma}(t) > 0$.

If $t_0 = t_{11}$, and $t_{11} \neq t_1, t_2, t_3, t_4, t_5, t_6, t_7, t_8, t_9, t_{10}, t_{12}$ and $I_{vu}(t) < 0$ in the interval $[0, t_{11}]$ with $I_{vu}(t_{11}) = 0$. Let $S_g(t_{11}) > 0, S_f(t_{11}) > 0, S_m(t_{11}) > 0, S_v(t_{11}) > 0, I_{gu}(t_{11}) > 0, I_{ga}(t_{11}) > 0, I_{fu}(t_{11}) > 0, I_{fa}(t_{11}) > 0, I_{mu}(t_{11}) > 0, I_{ma}(t_{11}) > 0, I_{va}(t_{11}) > 0 \; \forall t \in [0, t_{11}]$.

From the eleventh equation of the model system (3.1),

$$\frac{dI_{vu}}{dt} = m_{bu} I_{mu} + (1 - \rho_m)\beta_m S_v - \bar{\mu}_{11} I_{vu} \geq -\bar{\mu}_{11} I_{vu},$$
$$I_{vu}(t) \geq I_{vu}(0)\, e^{-\bar{\mu}_{11} t}.$$

At $t = t_{11}$,

$$I_{vu}(t_{11}) \geq I_{vu}(0)\, e^{-\bar{\mu}_{11} t_{11}} > 0. \tag{3.12}$$

Equation 3.12 contradicts the fact that $I_{vu}(t_{11}) = 0$. Thus, $I_{vu}(t) \not< 0 \in [0, t_{11}]$ and $I_{vu}(t) \neq 0$. This implies that, $I_{vu}(t) > 0$.

Lastly, if $t_0 = t_{12}$, and $t_{12} \neq t_1, t_2, t_3, t_4, t_5, t_6, t_7, t_8, t_9, t_{10}, t_{11}$ and $I_{va}(t) < 0$ in the interval $[0, t_{12}]$ with $I_{va}(t_{12}) = 0$. Let $S_g(t_{12}) > 0, S_f(t_{12}) > 0, S_m(t_{12}) > 0, S_v(t_{12}) > 0, I_{gu}(t_{12}) > 0, I_{ga}(t_{12}) > 0, I_{fu}(t_{12}) > 0, I_{fa}(t_{12}) > 0, I_{mu}(t_{12}) > 0, I_{ma}(t_{12}) > 0, I_{vu}(t_{12}) > 0 \; \forall t \in [0, t_{12}]$.

From the last equation of the model system (3.1),

$$\frac{dI_{va}}{dt} = m_{ba} I_{ma} + \rho_m \beta_m S_v + \rho_2 I_{vu} - \bar{\mu}_{12} I_{va} \geq -\bar{\mu}_{12} I_{va},$$
$$I_{va}(t) \geq I_{va}(0)\, e^{-\bar{\mu}_{12} t}.$$

At $t = t_{12}$,

$$I_{va}(t_{12}) \geq I_{va}(0)\, e^{-\bar{\mu}_{12} t_{12}} > 0. \tag{3.13}$$

Equation 3.13 contradicts the fact that $I_{va}(t_{12}) = 0$. Thus, $I_{va}(t) \not< 0 \in [0, t_{12}]$ and $I_{va}(t) \neq 0$. This implies that, $I_{va}(t) > 0$.

The contradiction is true for $\{t_i\}_{i=1,2,\ldots,12} \to \infty$. Thus, there is no such $t_i < 0$ implying that the solution set $(S_g(t), S_f(t), S_m(t), S_v(t), I_{gu}(t), I_{ga}(t), I_{fu}(t), I_{fa}(t), I_{mu}(t), I_{ma}(t), I_{vu}(t), I_{va}(t)) \geq 0\ \forall t > 0$. This proves that the solutions of the model system (3.1) remain in the positive orthant $\forall t > 0$.

□

3.3.2 Boundedness

Theorem 3.3.2. *The model (3.1) solutions are uniformly bounded in a set* $\Omega = \left\{(S_g, S_f, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va}) \in \mathbb{R}^{+}_{12} | N(0) \leq N \leq \frac{\Lambda}{\bar{\mu}_{min}}\right\}$
where $\Lambda = \lambda(1-\pi)(\kappa\alpha e^{-\mu_f \alpha} + \nu\zeta e^{-\mu_m \zeta})$

Proof. Let $(S_g, S_f, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va})$ be the solution to (3.1) and $S_g(0) = S_g^0 \geq 0$, $S_f(0) = S_f^0 \geq 0$, $S_m(0) = S_m^0 \geq 0$, $S_v(0) = S_v^0 \geq 0$, $I_{gu}(0) = I_{gu}^0 \geq 0$, $I_{ga}(0) = I_{ga}^0 \geq 0$, $I_{fu}(0) = I_{fu}^0 \geq 0$, $I_{fa}(0) = I_{fa}^0 \geq 0$, $I_{mu}(0) = I_{mu}^0 \geq 0$, $I_{ma}(0) = I_{ma}^0 \geq 0$, $I_{vu}(0) = I_{vu}^0 \geq 0$, $I_{va}(0) = I_{va}^0 \geq 0$ be the initial conditions. Adding all equations of system (3.1), yields

$$\begin{aligned} &\dot{N} = \Lambda(N - N_g - N_m + C_w) + \kappa\pi\lambda\alpha e^{-\mu_f \alpha}((1-\rho_f) I_{fu} + \rho_f I_{fa}) + \\ &\nu\pi\lambda\zeta e^{-\mu_m \zeta}((1-\rho_m) I_{fu} + \rho_m I_{fa}) - \bar{\mu}_{min}\, N \leq \Lambda N - \bar{\mu}_{min} N \end{aligned} \tag{3.14}$$

where $\bar{\mu}_{min} = min\ \{\bar{\mu}_i\}$, $i = 1, 2, \ldots 12$ which is invariant and attractive for positive starting-point values since $N(0) > 0$. Applying the theory of differential inequality [55], we have

$$\dot{N} \leq \Lambda N - \bar{\mu}_{min} \bar{N},\ \bar{N}(0) = N_0. \tag{3.15}$$

with $N \leq \bar{N}$ and $\bar{N}$ being the solution to the model system (3.1).

Therefore, $\bar{N}(t) \leq \frac{\Lambda}{\bar{\mu}_{min}} + (N_0 - \frac{\Lambda}{\bar{\mu}_{min}}) e^{-\bar{\mu}_{min} t}$.

Considering (3.15), $\bar{N} \leq \frac{\Lambda}{\bar{\mu}_{min}}$ is asymptotically stable.

Thus,

$$\bar{N}(t) \leq \max\left\{N_0, \frac{\Lambda}{\bar{\mu}_{min}}\right\} \quad \text{implies} \quad N(t) \leq \max\left\{N_0, \frac{\Lambda}{\bar{\mu}_{min}}\right\}.$$

All solutions of (3.1) originating in $\mathbb{R}^{12}_{+}$ are confined in Ω. Let M be an upper bound for $S_g, S_f, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va}$. We then conclude that every solution originating

from Ω stays in Ω and is bounded by M. □

3.3.3 Local existence and uniqueness of the model solution.

Lemma 3.3.1. *Let $x = (x_i)_{i=1,..,12}$ and $f : \mathbb{R}_+ \times \mathbb{R}^{12} \to \mathbb{R}^{12}$ be continuous with respect to t, x and Lipschitz continuous. Let $f(t, x)$ be non negative for all $(t, x) \in \mathbb{R}_+ \times \mathbb{R}^{12}$, and $x_i = 0$. For every $x_0 \in \mathbb{R}^{12}_+$, there exists a positive constant T such that $\dot{x} = f(t, x), \quad x(t_0) = x_0$, has a unique, positive and existing solution whose value lies in the interval $[0, T)$ and in $\mathbb{R}^{12}_+$.*

Theorem 3.3.3. *The solution set $\{S_g, S_f, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va}\}$ of the model (3.1) exists, is unique and positive for $t > 0$.*

Proof. Let $x = (S_g, S_f, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va})$ and K be a function such that $K = (K_i)_{i=1...12}$.
The function K is continuous, differentiable and satisfies a Lipschitz condition in Ω with respect to x. There exists $T_0 > 0$ by the Picard's theorem such that a unique local solution to (3.1) exists on any interval containing T_0 in its interior. Applying the maximal forward interval of existence for the model solutions of (3.1) we have

$$
\begin{aligned}
K_1(0, S_f, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va}) &\geq 0,\\
K_2(S_g, 0, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va}) &\geq 0,\\
K_3(S_g, S_f, 0, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va}) &\geq 0,\\
K_4(S_g, S_f, S_m, 0, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va}) &\geq 0,\\
K_5(S_g, S_f, S_m, S_v, 0, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va}) &\geq 0,\\
K_6(S_g, S_f, S_m, S_v, I_{gu}, 0, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va}) &\geq 0,\\
K_7(S_g, S_f, S_m, S_v, I_{gu}, I_{ga}, 0, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va}) &\geq 0,\\
K_8(S_g, S_f, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, 0, I_{mu}, I_{ma}, I_{vu}, I_{va}) &\geq 0,\\
K_9(S_g, S_f, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, 0, I_{ma}, I_{vu}, I_{va}) &\geq 0,\\
K_{10}(S_g, S_f, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, 0, I_{vu}, I_{va}) &\geq 0,\\
K_{11}(S_g, S_f, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, 0, I_{va}) &\geq 0,\\
K_{12}(S_g, S_f, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, 0) &\geq 0.
\end{aligned}
$$

By theorem 3.3.2, the solutions to (3.1) are uniformly bounded on $[0, T)$. By theorem 3.3.3, the solution of (3.1) exists for any finite time. Thus, for any positive initial data in $\mathbb{R}^{12}_+$, the model system (3.1) will possess a unique and positive solution in $\mathbb{R}^{12}_+$. This proves that all feasible solution of the model system (3.1) lies in the feasible region, Ω. □

3.3.4 Equilibria

The model system (3.1) possesses two steady states, namely the disease-free equilibrium (DFE)

$$E_0 = (S_g^0, S_f^0, S_m^0, S_v^0, 0, 0, 0, 0, 0, 0, 0, 0), \tag{3.16}$$

where

$$\begin{aligned} S_g^0 &= \frac{\kappa(1-\pi)\lambda\alpha e^{-\mu_f \alpha} S_f^0}{\bar{\mu}_9}, \\ S_f^0 &= \frac{\kappa(1-\pi)\lambda\alpha e^{-\mu_f \alpha} C_w}{\bar{\mu}_8 - \kappa(1-\pi)\lambda\alpha e^{-\mu_f \alpha}}, \\ S_m^0 &= \frac{\nu(1-\pi)\lambda\zeta e^{-\mu_m \zeta} S_f^0}{\bar{\mu}_7}, \\ S_v^0 &= \frac{\Sigma\nu(1-\pi)\lambda\zeta e^{-\mu_m \zeta} S_f^0}{\bar{\mu}_7\, \bar{\mu}_{10}}, \\ \bar{\mu}_8 &> \kappa(1-\pi)\lambda\alpha e^{-\mu_f \alpha} \end{aligned}$$

and the endemic equilibrium (EE)

$$E_e^* = (S_g^*, S_f^*, S_m^*, S_v^*, I_{gu}^*, I_{ga}^*, I_{fu}^*, I_{fa}^*, I_{mu}^*, I_{ma}^*, I_{vu}^*, I_{va}^*) \tag{3.17}$$

with

$$\begin{cases} S_g^* = \dfrac{\Lambda_1(N_f^* + C_w)}{\beta_g^* + \bar{\mu}_9},\ S_f^* = \dfrac{m_g \Lambda_1 (N_f^* + C_w)}{(\beta_g^* + \bar{\mu}_9)(\beta_f^* + \bar{\mu}_8)},\ S_m^* = \dfrac{\Lambda_2(N_f^* + C_w)}{\beta_m^* + \bar{\mu}_7}, \\ S_v^* = \dfrac{m_b\, \Lambda_2 (N_f^* + C_w)}{(\beta_m^* + \bar{\mu}_7)(\beta_m^* + \bar{\mu}_{10})},\ B_1 = \dfrac{\beta_g^*}{\beta_g^* + \bar{\mu}_9},\ B_2 = \dfrac{\beta_f^*}{(\beta_g^* + \bar{\mu}_9)(\beta_f^* + \bar{\mu}_8)}, \\ A = \dfrac{\beta_m^*}{\beta_m^* + \bar{\mu}_7}, \quad B = \dfrac{1}{\beta_m^* + \bar{\mu}_{10}}, \\ I_{gu}^* = h_{01} B_1 + a_{33} B_2, \quad I_{ga}^* = h_{03} B_1 + h_{04} B_2, \quad I_{fu}^* = a_{32} B_1 + a_{33} B_2, \\ I_{fa}^* = a_{34} B_1 + a_{35} B_2, \quad I_{mu}^* = h_{05} B_1 + h_{06} B_2 + a_{23} A, \quad I_{ma}^* = h_{07} B_1 + h_{08} B_2 + h_{09} A, \\ I_{vu}^* = h_{10} B_1 + h_{11} B_2 + h_{12} A + a_{28} AB, \quad I_{va}^* = h_{13} B_1 + h_{14} B_2 + h_{15} A + h_{16} AB, \\ N_g^* \le g_{11} N_f^*,\ N_f^* \le \dfrac{g_{12}}{g_{11} - g_{12}},\ g_{11} > g_{12},\ N_m^* \le g_{13} N_f^*,\ N_v^* \le g_{14} N_m^*, \\ \beta_g^* = h_{17}\, \beta_f^*,\ \beta_m^* = h_{18} B_1 + h_{19} B_2, \\ \beta_f^* \le \dfrac{h_{40}\beta_f^{*6} + h_{41}\beta_f^{*5} + h_{42}\beta_f^{*4} + h_{43}\beta_f^{*3} + h_{44}\beta_f^{*2} + h_{45}\beta_f}{h_{51}\beta_f^{*6} + h_{52}\beta_f^{*5} + h_{53}\beta_f^{*4} + h_{54}\beta_f^{*3} + h_{55}\beta_f^{*2} + h_{56}\beta_f + h_{57}} \implies \\ C_6\beta_f^{*6} + C_5\beta_f^{*5} + C_4\beta_f^{*4} + C_3\beta_f^3 + C_2\beta_f^{*2} + C_1\beta_f - C_0 \le 0. \end{cases}$$

See appendix A.2.2 for the expressions of a_{11}, a_{12}, ..., a_{35}, g_{11}, g_{12}, ..., g_{14}, h_{01}, h_{02}, ..., h_{57} and C_0, C_1,...C_5.

By Descartes' rule of signs, the polynomial equation $\beta_f^{*6} + C_5\beta_f^5 + C_4\beta_f^{*4} + C_3\beta_f^{*3} + C_2\beta_f^{*2} + C_1\beta_f^* - C_0 \leq 0$ will have at least one positive real root if and only if $C_0 > 0$, $C_1 > 0$, $C_2 > 0$, $C_3 > 0$, $C_4 > 0$, $C_5 > 0$, $C_6 > 0$ given that the sign before C_0 is negative and the sign before C_6 is positive otherwise the polynomial equation will have at most 5 positive real roots. The exact number of positive roots can be determined using Descartes' rule of signs and Euclid's algorithm of the Sturm's theorem. Appendix A.2.3 details the conditions for the global stability of the disease free equilibrium.

3.3.5 Control Reproduction Number and its Biological Interpretation

The control reproduction number, $\mathcal{R}_c$, is defined as the expected number of secondary infections produced by a typical infected individual during its entire period of infectiousness in a population that is not entirely susceptible due to the presence of control efforts [56]. In our model, the control effort present is the dissemination of comprehensive knowledge of HIV/AIDS to the AGYW and ABYM populations. Epidemiologically, $\mathcal{R}_c$ is used to measure the transmission potential of the HIV/AIDS disease among the AGYW and ABYM in the presence of the said control. The threshold criterion states that if $\mathcal{R}_c > 1$, HIV/AIDS disease invades a given population resulting in an epidemic whereas when $\mathcal{R}_c < 1$, the disease reflects the impact of successful comprehensive knowledge dissemination among the AGYW and ABYM consequently curtailing the depletion of susceptible AGYW and ABYM to new HIV/AIDS infection.

We use the next generation matrix approach to compute the control reproduction number for the model system (3.1) [57]. We will use the linearized infected subsystem of model (3.1) to compute $\mathcal{R}_c$ as it has been proved that the reproduction number obtained from the reduced system is similar to that of the original system [58]. Consider the infected subsystem of the model system (1) given as

$$\begin{cases} \frac{dI_{gu}}{dt} = \lambda_1 I_{fu} + (1-\rho_f)\beta_g S_g - \bar{\mu}_1 I_{gu}, \\ \frac{dI_{ga}}{dt} = \lambda_2 I_{fa} + \rho_f \beta_g S_g + \rho_1 I_{gu} - \bar{\mu}_2 I_{ga}, \\ \frac{dI_{fu}}{dt} = m_{gu} I_{gu} + (1-\rho_f)\beta_f S_f - \bar{\mu}_3 I_{fu}, \\ \frac{dI_{fa}}{dt} = m_{ga} I_{ga} + \rho_f \beta_f S_f + \rho_1 I_{fu} - \bar{\mu}_4 I_{fa}, \\ \frac{dI_{mu}}{dt} = \lambda_3 I_{fu} + (1-\rho_m)\beta_m S_m - \bar{\mu}_5 I_{mu}, \\ \frac{dI_{ma}}{dt} = \lambda_4 I_{fa} + \rho_m \beta_m S_m + \rho_2 I_{mu} - \bar{\mu}_6 I_{ma}, \\ \frac{dI_{vu}}{dt} = m_{bu} I_{mu} + (1-\rho_m)\beta_m S_v - \bar{\mu}_{11} I_{vu}, \\ \frac{dI_{va}}{dt} = m_{ba} I_{ma} + \rho_m \beta_m S_v + \rho_2 I_{vu} - \bar{\mu}_{12} I_{va}. \end{cases} \tag{3.18}$$

We then decompose the right hand side of the infected subsystem (3.18) into two parts, F and V where F denotes the transmission part and each F_i represents the new infection. V denotes the transition part and each V_i describes change in state for instance removal through natural deaths, disease induced deaths, aging and comprehensive knowledge gain [58].

$$F = \begin{bmatrix} (1-\rho_f)\beta_{\Theta}\,\gamma_g\left(\psi_1 I_{mu} + \psi_2 I_{ma} + \psi_1 I_{vu} + \psi_2 I_{va}\right) S_g \\ \rho_f \beta_{\Theta}\,\gamma_g(\psi_1 I_{mu} + \psi_2 I_{ma} + \psi_1 I_{vu} + \psi_2 I_{va}) S_g \\ (1-\rho_f)\beta_{♀}\gamma_f \left(\dfrac{\psi_1 I_{mu} + \psi_2 I_{ma}}{N_{mv}} + \dfrac{\psi_1 I_{vu} + \psi_2 I_{va}}{N_{mv}}\right) S_f \\ \rho_f \beta_{♀}\gamma_f \left(\dfrac{\psi_1 I_{mu} + \psi_2 I_{ma}}{N_{mv}} + \dfrac{\psi_1 I_{vu} + \psi_2 I_{va}}{N_{mv}}\right) S_f \\ (1-\rho_m)\gamma_m \left(\beta_{♂}\left[\dfrac{\psi_1 I_{fu} + \psi_2 I_{fa}}{N_f}\right] + \beta'_{♂} N_g \left[\dfrac{\psi_1 I_{gu} + \psi_2 I_{ga}}{N_g}\right]\right) S_m \\ \rho_m \gamma_m \left(\beta_{♂}\left[\dfrac{\psi_1 I_{fu} + \psi_2 I_{fa}}{N_f}\right] + \beta'_{♂} N_g \left[\dfrac{\psi_1 I_{gu} + \psi_2 I_{ga}}{N_g}\right]\right) S_m \\ (1-\rho_m)\gamma_m \left(\beta_{♂}\left[\dfrac{\psi_1 I_{fu} + \psi_2 I_{fa}}{N_f}\right] + \beta'_{♂} N_g \left[\dfrac{\psi_1 I_{gu} + \psi_2 I_{ga}}{N_g}\right]\right) S_v \\ \rho_m \gamma_m \left(\beta_{♂}\left[\dfrac{\psi_1 I_{fu} + \psi_2 I_{fa}}{N_f}\right] + \beta'_{♂} N_g \left[\dfrac{\psi_1 I_{gu} + \psi_2 I_{ga}}{N_g}\right]\right) S_v \end{bmatrix}$$

and

$$V = -\begin{bmatrix} \lambda_1 I_{fu} - \bar{\mu}_1 I_{gu} \\ \lambda_2 I_{fa} + \rho_1 I_{gu} - \bar{\mu}_2 I_{ga} \\ m_{gu} I_{gu} - \bar{\mu}_3 I_{fu} \\ m_{ga} I_{ga} + \rho_1 I_{fu} - \bar{\mu}_4 I_{fa} \\ \lambda_3 I_{fu} - \bar{\mu}_5 I_{mu} \\ \lambda_4 I_{fa} + \rho_2 I_{mu} - \bar{\mu}_6 I_{ma} \\ m_{bu} I_{mu} - \bar{\mu}_{11} I_{vu} \\ m_{ba} I_{ma} + \rho_2 I_{vu} - \bar{\mu}_{12} I_{va} \end{bmatrix}.$$

$\mathcal{F}$ and $\mathcal{V}$ are evaluated as follows:

$$\mathcal{F} = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right] \ \textit{and}\ \mathcal{V} = \left[\frac{\partial V_i(x_0)}{\partial x_j}\right] \tag{3.19}$$

where x_0 is the disease free equilibrium. Computing $\mathcal{F}\mathcal{V}^{-1}$ yields the next generation matrix for the model system (3.1) whose spectral radius is the control reproduction number, $\mathcal{R}_c$. $\mathcal{F}\mathcal{V}^{-1}$ is given as:

$$\mathcal{F}\mathcal{V}^{-1} = \begin{bmatrix} q_1\eta_1 & q_1\eta_2 & q_1\eta_3 & q_1\eta_4 & q_1\eta_5 & q_1\eta_6 & q_1\eta_7 & q_1\eta_8 \\ q_2\eta_1 & q_2\eta_2 & q_2\eta_3 & q_2\eta_4 & q_2\eta_5 & q_2\eta_6 & q_2\eta_7 & q_2\eta_8 \\ q_3\eta_1 & q_3\eta_2 & q_3\eta_3 & q_3\eta_4 & q_3\eta_5 & q_3\eta_6 & q_3\eta_7 & q_3\eta_8 \\ q_4\eta_1 & q_4\eta_2 & q_4\eta_3 & q_4\eta_4 & q_4\eta_5 & q_4\eta_6 & q_4\eta_7 & q_4\eta_8 \\ \varepsilon_{11} & \varepsilon_{12} & \varepsilon_{13} & \varepsilon_{14} & 0 & 0 & 0 & 0 \\ \varepsilon_{15} & \varepsilon_{16} & \varepsilon_{17} & \varepsilon_{18} & 0 & 0 & 0 & 0 \\ \varepsilon_{19} & \varepsilon_{20} & \varepsilon_{21} & \varepsilon_{22} & 0 & 0 & 0 & 0 \\ \varepsilon_{23} & \varepsilon_{24} & \varepsilon_{25} & \varepsilon_{26} & 0 & 0 & 0 & 0 \end{bmatrix}.$$

Appendix A.2.1 gives the components of the new infections Jacobian matrix $\mathcal{F}$. We note that the Jacobian matrix $\mathcal{F}$ has a set of four columns that are constant multiples of each other and another set of four columns that are constant multiples of each other. This implies that the rank of the Jacobian matrix $\mathcal{F}$ is two. Since $\mathcal{V}^{-1}$ is non-singular, it follows that $\mathcal{F}\mathcal{V}^{-1}$ has similar ranking as $\mathcal{F}$. Given that the next generation matrix $\mathcal{F}\mathcal{V}^{-1}$ is a square matrix of order eight, the eigenvalues computed are eight with eigenvalue 0 having multiplicity six and two nonzero eigenvalues, the larger of which is the control basic reproduction number. Using Maple 18 Symbolic Software we compute the control reproduction number, $\mathcal{R}_c$, given as

$$\begin{aligned} \mathcal{R}_c = \frac{1}{2}\left(q_1\eta_1 + q_2\eta_2 + q_3\eta_3 + q_4\eta_4\right) + \frac{1}{2}\Big\{ & {q_1}^2{\eta_1}^2 + {q_2}^2{\eta_2}^2 + {q_3}^2{\eta_3}^2 + {q_4}^2{\eta_4}^2 + \\ & 2\left[q_1\,q_2\,\eta_1\,\eta_2 + q_1\,q_3\,\eta_1\,\eta_3 + q_1\,q_4\,\eta_1\,\eta_4 + q_2\,q_3\,\eta_2\,\eta_3 + q_2\,q_4\,\eta_2\,\eta_4\right. \\ & \left. + q_3\,q_4\,\eta_3\,\eta_4\right] + 4\,q_1\left[\eta_5\,\varepsilon_{11} + \eta_6\,\varepsilon_{15} + \eta_7\,\varepsilon_{19} + \eta_8\,\varepsilon_{23}\right] \\ & + 4\,q_2\left[\eta_5\,\varepsilon_{12} + \eta_6\,\varepsilon_{16} + \eta_7\,\varepsilon_{20} + \eta_8\,\varepsilon_{24}\right] \\ & + 4\,q_3\left[\eta_5\,\varepsilon_{13} + \eta_6\,\varepsilon_{17} + \eta_7\,\varepsilon_{21} + \eta_8\,\varepsilon_{25}\right] \\ & + 4\,q_4\left[\eta_5\,\varepsilon_{14} + \eta_6\,\varepsilon_{18} + \eta_7\,\varepsilon_{22} + \eta_8\,\varepsilon_{26}\right]\Big\}^{\frac{1}{2}} \end{aligned} \tag{3.20}$$

where the expressions for $q_1, q_2, ..., q_4, k_1, k_2, ..., k_8, \eta_1, \eta_2, ..., \eta_8, \varepsilon_{11}, \varepsilon_{12}, ..., \varepsilon_{26}$ are also provided in Appendix A.2.1.

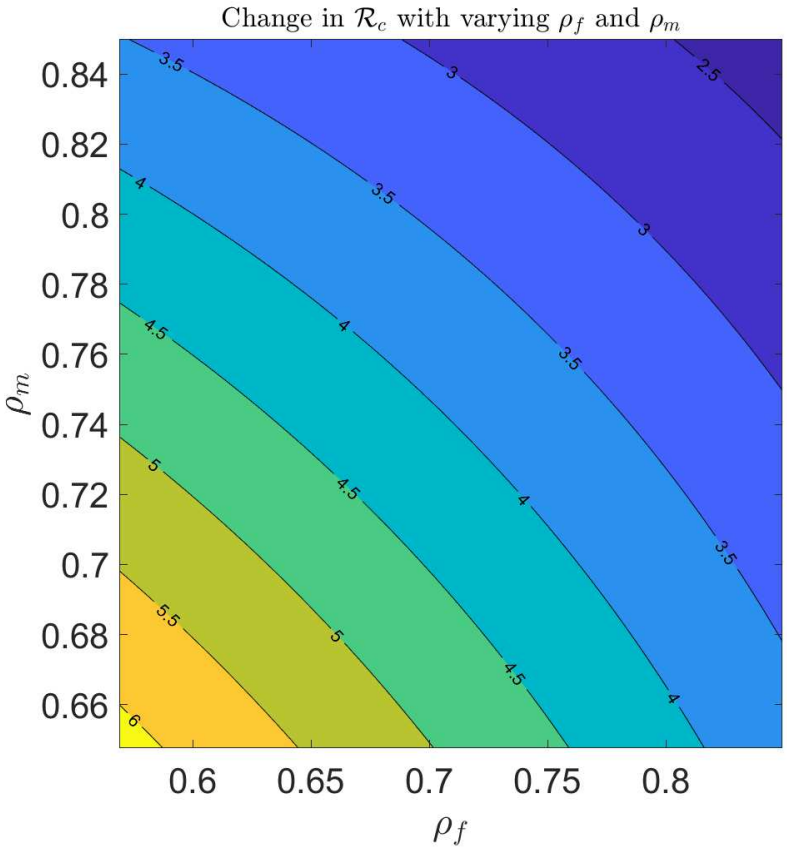


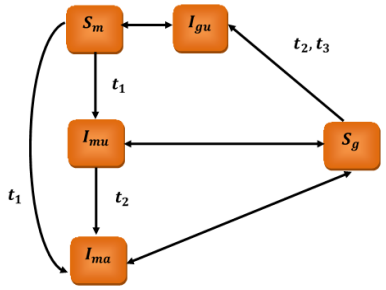
Figure 3.2: Change in the control reproduction number, $\mathcal{R}_c$, as ρ_m, ρ_f varies from the baseline values of 0.5693, 0.6478 respectively to the MDG of 0.85. Table 3.4 gives the parameter values used in $\mathcal{R}_c$ simulation.

The simulations in Figure 3.2 show the effect of increasing comprehensive knowledge on the control reproduction number, $\mathcal{R}_c$. Figure 3.2 reveals that increasing the efficacy of HIV/AIDS comprehensive knowledge in the infectious populations does not reduce the control reproduction number, $\mathcal{R}_c$, below unity even when the millennium development efficacy of 0.85 in all populations is achieved. The reproduction number of HIV/AIDS worldwide is yet to be well defined, Kenya included [59]. Recent studies however provided estimates for the basic reproduction number of HIV/AIDS for sub-Saharan Africa and India [60]. The basic reproduction number of HIV/AIDS for sub-Saharan Africa and Kenya were estimated at 4.6 and 6.3 respectively [60]. Simulations on the control reproduction number show $\mathcal{R}_c$ values quite close to the Kenyan estimates provided by [60] which was based on projections from a sampled antenatal clinic data whereas our $\mathcal{R}_c$ estimates is based on the adolescents and young adults population model. Clearly, increasing the coverage of comprehensive knowledge of HIV/AIDS among the infected Kenyan youth may not guarantee eradication of HIV/AIDS in these populations. [61] reported that progress towards complete HIV/AIDS eradication by year 2030 is proving to be an uphill task as a result of new infections arising from the risk groups.

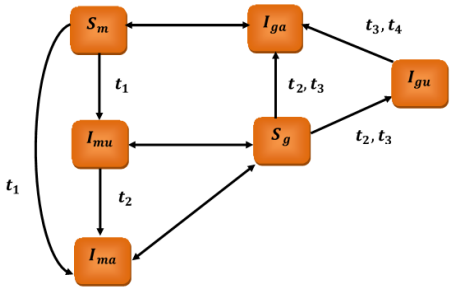
Analysis of the Infection Sub-networks

Figure 3.1 is split into infection sub-networks (see figure 3.3) that result in local secondary infections and we explore the differences in these networks here. Assume for figure 3.3(a) that after S_m and I_{gu} interacts, the infected individual takes time t_1 to move to either I_{mu} or I_{ma}. The new I_{mu} can either interact with S_g to cause an infected S_g to produce a new I_{gu} with time t_2 or progress to I_{ma} with time t_2 who then interacts with an S_g to produce a local secondary infection I_{gu} after time t_3. The new I_{ma} can also interact with S_g to cause an infected S_g to produce a new I_{gu} with time t_2. The local secondary infection generation for the S_m and I_{gu} interactions takes the duration; $d_1 = t_1 + t_2$ which occurs twice and $d_2 = t_1 + t_2 + t_3$.

Assume for figure 3.3(b) that the S_m and I_{ga} interaction results in the infected individual taking time t_1 to move to either I_{mu} or I_{ma}. The new I_{mu} and I_{ma} can either interact with S_g to produce a new I_{gu} or I_{ga} with time t_2. The new I_{mu} can also progress to I_{ma} with time t_2 who then interacts with an S_g to produce a local secondary infection I_{gu} or I_{ga} after time t_3. The new I_{mu} can also interact with an S_g to produce a local secondary infection I_{gu} after time t_2 who then takes time t_3 to move to I_{ga}. The new I_{mu} can also move to I_{ma} after time t_2 who then interacts with an S_g to produce a new I_{gu} after time t_3 who can also move to I_{ga} with time t_4. The duration of local secondary infection generation is hence summarized as follows: $d_1 = t_1 + t_2$, $d_2 = t_1 + t_2 + t_3$ and $d_3 = t_1 + t_2 + t_3 + t_4$. Figures 3.3(a) and 3.3(b) show the time taken for local secondary infection of I_{gu} and I_{ga} to be established respectively.



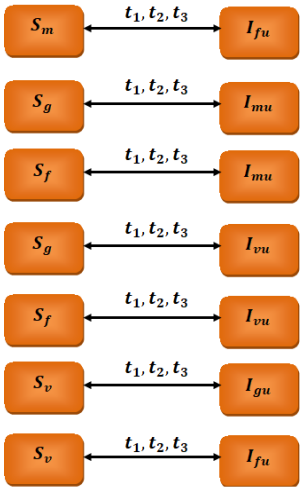
(a) Network 1: Local secondary infection of I_{gu}



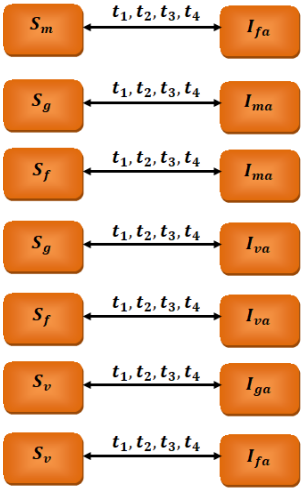
(b) Network 2: Local secondary infection of I_{ga}

Figure 3.3: Infection network of the local secondary infections of I_{gu} and I_{ga}

Similarly, the contacts between S_m and I_{fu}, S_g and I_{mu}, S_f and I_{mu}, S_g and I_{vu}, S_f and I_{vu}, S_v and I_{gu} and S_v and I_{fu} take the same time d_1, d_2 to produce a generation of local secondary infection resulting in sub-networks similar to figure 3.3(a) whereas contacts between S_m and I_{fa}, S_g and I_{ma}, S_f and I_{ma}, S_g and I_{va}, S_f and I_{va}, S_v and I_{ga} and S_v and I_{fa} takes time d_1, d_2, d_3 to produce a generation of local secondary infections resulting in sub-networks similar to figure 3.3(b). These sub-networks are generalized in figure 3.4.



(a) Local secondary infection network for I_{fu}, I_{mu} and I_{vu}



(b) Local secondary infection network for I_{fa}, I_{ma} and I_{va}

Figure 3.4: Infection network for the local secondary infections

Using tables, we summarize the infection duration of the observed infection sub-networks as follows:

Table 3.3: Number of observed infection sub-networks in each duration and their probabilities

Infection duration		d_1	d_2	d_3
Sub-networks observed in network 1	Observations	16	8	0
	Probability P_1	$P_1(d_1) = 2/3$	$P_1(d_2) = 1/3$	$P_1(d_3) = 0$
Sub-networks observed in network 2	Observations	32	24	8
	Probability P_2	$P_2(d_1) = 1/2$	$P_2(d_2) = 3/8$	$P_2(d_3) = 1/8$
Total number of sub-networks	Total	48	32	8
	Probability P_t	$P_t(d_1) = 6/11$	$P_t(d_2) = 4/11$	$P_t(d_3) = 1/11$

If we consider duration d_1, d_2 and d_3 as events, in probability theory sense, then we can say these events are independent. Table 3.3 shows that $P_1(d_1) > P_1(d_2) > P_1(d_3)$, $P_2(d_1) > P_2(d_2) > P_2(d_3)$ and $P_t(d_1) > P_t(d_2) > P_t(d_3)$. The joint probabilities $P_1(d_1 \cap d_2) > P_1(d_1 \cap d_3) > P_1(d_2 \cap d_3) > P_1(d_1 \cap d_2 \cap d_3)$ whereas the joint probabilities $P_2(d_1 \cap d_2) > P_2(d_1 \cap d_3) > P_2(d_2 \cap d_3) > P_2(d_1 \cap d_2 \cap d_3)$. Thus, $P_t(d_1 \cap d_2) > P_t(d_1 \cap d_3) > P_t(d_2 \cap d_3) > P_t(d_1 \cap d_2 \cap d_3)$.

The infection duration with the least joint probabilities involves time d_3 whereas those with higher probabilities are driven by duration d_1 followed by d_2. The local secondary infection driven by duration d_1 results in the production of more local secondary infections, followed by d_2 and a combination of d_1 and d_2. The local secondary infection driven by d_3 alone or in combination

with either d_1 or d_2 will have a lower production of local secondary infections. This is true for each of the networks as well as the combination of both networks. Duration d_2 and d_3 involves the process going through an individual who has comprehensive knowledge of HIV/AIDS and when such are involved in the infection spread, then the probability of local secondary infection is slower compared to an infected adolescent/young adult with no comprehensive knowledge of HIV/AIDS. Infection duration d_2 involves either the AGYW or ABYM gaining comprehensive knowledge of HIV/AIDS whereas infection duration d_3 involves both the AGYW and ABYM gaining comprehensive knowledge of HIV/AIDS. As established earlier, infection duration d_2 has a greater probability of producing local secondary infection compared to infection duration d_3. Thus, comprehensive knowledge of HIV/AIDS disseminated to one gender alone is not as effective in slowing the duration of the infection spread compared to comprehensive knowledge disseminated to both genders. Thus, the longer the duration of infection spread then the lower the probabilities of infection spread.

3.4 Numerical Analysis

3.4.1 Parameter Estimation

This section details how the initial data and parameter values used for the model simulations were obtained. The Kenyan demographic data was used to estimate the death rate. We obtained the HIV parameter values from the National AIDS Control Council (NACC) report of 2015, Kenya AIDS Indicator survey (KAIS) final report of 2012 and the Kenya National Bureau of Statistics (KNBS) report of 2014. The calculations for the estimated parameter values is clearly shown here. The initial values for the state variables were obtained from the Kenyan demographic data and the NACC 2015 report.

In Kenya, the female life expectancy is 65.8 years whereas the male is 61.1 years [62]. We calculate the natural mortality rates, $\mu_f = \frac{1}{65.8} = 0.01519757$ and $\mu_m = \frac{1}{61.1} = 0.01636661$ respectively. The natural birth rate $\lambda = 1.1871$ is an average of the expressions $\lambda = \frac{\bar{\mu}_9 S_g^0}{\kappa(1-\pi)\alpha e^{-\mu_f \alpha} N_f}$, $\lambda = \frac{\bar{\mu}_8 S_f^0}{\kappa(1-\pi)\alpha e^{-\mu_f \alpha} N_f}$, $\lambda = \frac{\bar{\mu}_7 S_m^0}{\nu(1-\pi)\zeta e^{-\mu_m \zeta} N_f}$. National AIDS Control Council reported $35,821$ AIDS related deaths in the year 2015 [36]. Children below 14 years accounted for 14% of the total AIDS related deaths and ABYM/AGYW aged 15 -24 accounted for 11% [36]. Thus, we estimated $\delta_g = 0.14$ and $\delta = 0.11$. In 2015, 60% of the new infections in children was as a result of vertical transmission yielding 3, 967 children infected through mother to child transmission [36]. Approximately one million children are born in Kenya each year [63]. Hence, $\pi = \frac{3967}{1000000}$ is the proportion of the AGYW/ABYM born HIV infected. We estimate the female sexual contact rate in their lifetime as $\beta_{\female} = 0.3$ [64] and the male sexual contact rate in their lifetime as $\beta_{\male} = 0.5$ [64]. We calculate the yearly sexual contact rate for the AGYW and ABYM older than 15 years as follows: $\beta_{\female} = 0.3/50.8 = 0.00590551$, $\beta_{\male} = 0.5/46.1 = 0.01084599$ where 50.8 and 46.1 are the active sexual years for male and females respectively. The yearly sexual contact rate for the AG population is given as $\beta_\Theta = 0.3/5 = 0.06$. The transmission probability for the

AGYW, ABYM/perpetrators and AG populations are estimated as $\gamma_f = 0.0000846667$, $\gamma_m = 0.00004609999$ and $\gamma_g = 0.00000833$. We calculate the adolescent girls maturity rate, $\alpha = \frac{1}{10}$ and the adolescent boys maturity rate $\zeta = \frac{1}{15}$. The estimated values for the transition rates m, m_u and m_a are given in table 3.4. The exit rate through aging, σ is calculated as $\sigma = \frac{1}{24}$.

In the year 2015, the Kenyan population was estimated at $44,000,000$ [63]. The population of the adolescent girls and young women aged $15-24$, N_f, was approximately $4,411,586$ whereas that of the adolescent boys and young men aged $15-24$, N_m, was about $4,398,554$ [63]. The population of reproductive women aged $25-49$ years, C_w, is approximately 7,302,316 [63]. 12% of the Kenyan population comprises of adolescents girls and boys aged $10-14$ [63]. Using the sex ratio at birth, (females, $\kappa = 1/2.02$ and males, $\nu = 1.01/2.02$) [63] yields $2,613,861$ adolescent girls, N_g. Approximately $193,791$ AGYW and $74,797$, ABYM aged $15-24$ are living with HIV/AIDS [36]. Using the ρ_f/ρ_m ratio, we compute $I_{fa} = 110,325$ and $I_{ma} = 48,453$. Thus, $I_{fu} = 83,466$ and $I_{mu} = 23,344$. Using the adolescent girls HIV prevalence of 0.5% [36] and the proportion of newly infected female with comprehensive knowledge, ρ_f yields $I_{ga} = 7440$ and $I_{gu} = 5629$. Table 3.4 gives the parameter values used in the model simulations.

Table 3.4: Parameter Values

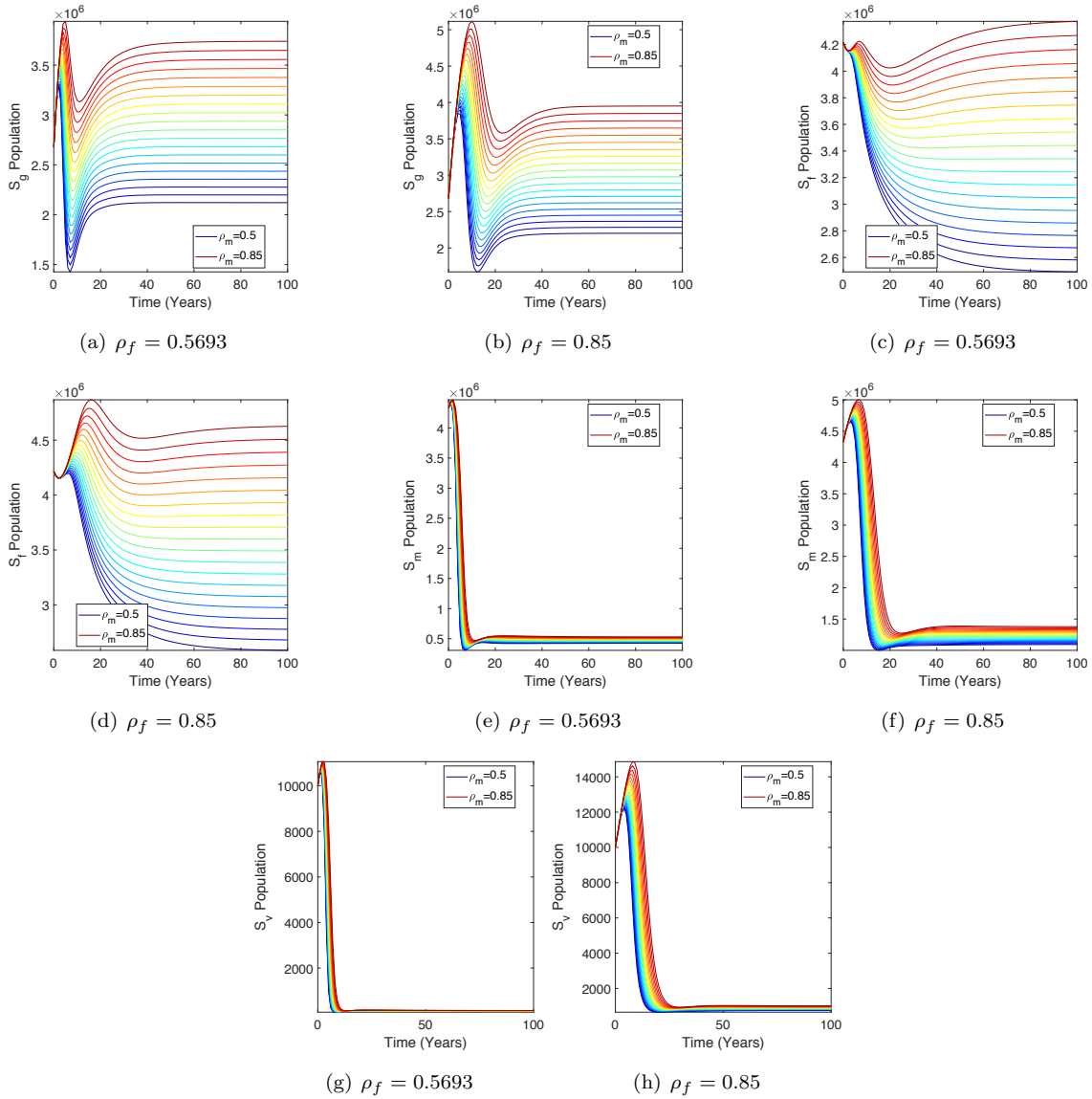
Parameter	Value	Unit	Source
ρ_f	0.5693	-	[38]
ρ_m	0.6478	-	[38]
μ_f	0.01519757	$year^{-1}$	[62]
μ_m	0.01636661	$year^{-1}$	[62]
$\beta_{\female}$, $\beta_{\male}$, β_{Θ}	0.3/50.8, 0.5/46.1, 0.3/5	$year^{-1}$	[64]
$\beta'_{\male}$	0.035	$year^{-1}$	Estimated
δ_g	0.14	$year^{-1}$	[36]
δ	0.11	$year^{-1}$	[36]
λ	1.1871	$year^{-1}$	Calculated
α	0.1	$year^{-1}$	Calculated
κ, ν	1/2.02, 1.02/2.02	$year^{-1}$	[63]
ζ	0.06667	$year^{-1}$	Calculated
m_g	0.06667	$year^{-1}$	Calculated
m_{gu}	0.028713	$year^{-1}$	Calculated
m_{ga}	0.037953	$year^{-1}$	Calculated
σ	0.041667	$year^{-1}$	Calculated
π	0.003967	-	Calculated
γ_f, γ_m, γ_g	0.0000846667, 0.00004609999, 0.00000833	$year^{-1}$	Estimated
ρ_{fk}, ρ_{mk}	0.5693, 0.6478	-	Estimated
m_b, m_{bu}, m_{ba}	0.00023112, 0.00428, 0.00212	$year^{-1}$	Estimated
ρ_1, ρ_2	0.4, 0.5	$year^{-1}$	Estimated
ψ_1, ψ_2	0.92, 0.06	-	Estimated

3.4.2 Simulation Results

Numerical simulations on the model system equations are performed to test the evolution of the HIV/AIDS disease among the AGYW and ABYM populations. The Millennium Development Goal (MDG) is 85% comprehensive knowledge of HIV and AIDS among all AGYW/ABYM [47]. We choose the baseline proportion of the newly infected AGYW/ABYM with comprehensive knowledge as $\rho_f = 0.5693$ and $\rho_m = 0.6478$ respectively, whereas $\rho_f = \rho_m = 0.85$ represents highly efficacious comprehensive knowledge of HIV/AIDS among the newly infected AGYW/ABYM.

Increasing the proportion of the HIV/AIDS comprehensive knowledge of the newly infected female population over the newly infected male population as shown in Figures 3.5, 3.7 and 3.8 increases the S_g, S_f, I_{ga}, I_{fa}, I_{ma} and I_{va} classes with better benefits when $\rho_f = 0.85$. I_{gu}, I_{fu}, I_{mu} and I_{va} classes decrease slightly whereas S_m and S_v increases slightly then decreases significantly with better benefits when $\rho_f = 0.85$. Increasing the efficacy of the HIV/AIDS comprehensive knowledge of the newly infected male population over the newly infected female population as shown in Figures 3.6, 3.9 and 3.10 increases the S_m, S_v, I_{ga}, I_{fa}, I_{ma} and I_{va} classes with better benefits when $\rho_m = 0.85$. I_{gu}, I_{fu}, I_{mu} and I_{vu} classes decrease slightly whereas S_g and S_f increases slightly then decreases significantly with better benefits when $\rho_f = 0.85$.

Increasing the efficacy of HIV/AIDS comprehensive knowledge of only the newly infected ABYM population reduces their infectivity which protects the AGYW susceptible populations ultimately. However, the AGYW infectivity remains high and this reduces the ABYM susceptible population significantly. Similarly, increasing the efficacy of the HIV/AIDS comprehensive knowledge of only the newly infected AGYW population reduces their infectivity which protects the ABYM susceptible populations but the ABYM infectivity remains high hence, reducing the AGYW susceptible population significantly. When knowledge is high in both populations, the infectivity risks are decreased and all populations stabilize at greater population values.

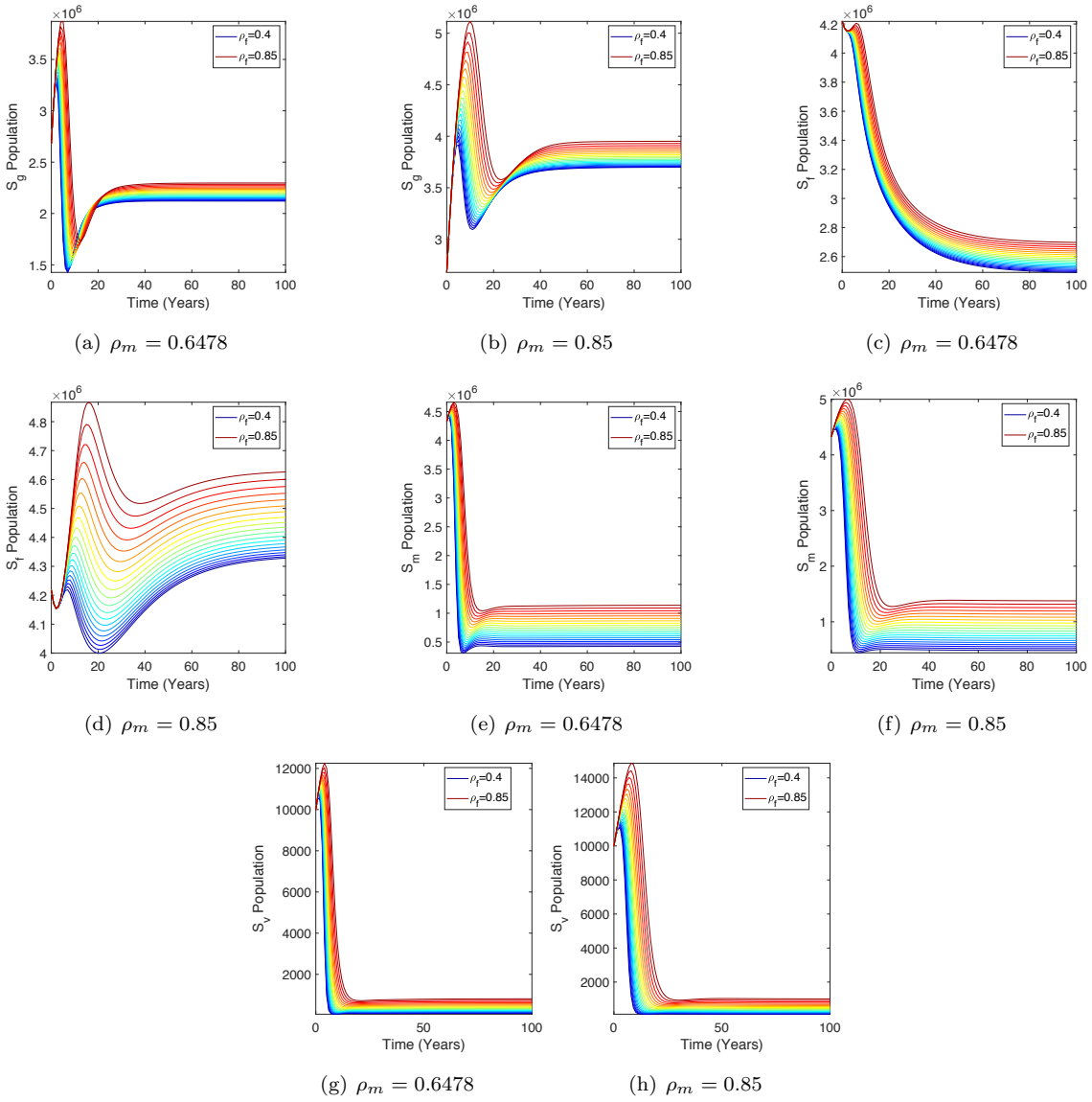


(a) $\rho_f = 0.5693$ (b) $\rho_f = 0.85$ (c) $\rho_f = 0.5693$

(d) $\rho_f = 0.85$ (e) $\rho_f = 0.5693$ (f) $\rho_f = 0.85$

(g) $\rho_f = 0.5693$ (h) $\rho_f = 0.85$

Figure 3.5: Disproportional effects on the Male Susceptible Population when ρ_f is fixed at its baseline value of 0.5693 and at the MDG of 0.85 and while ρ_m is steadily increased from its baseline value of 0.6478 to the MDG of 0.85.

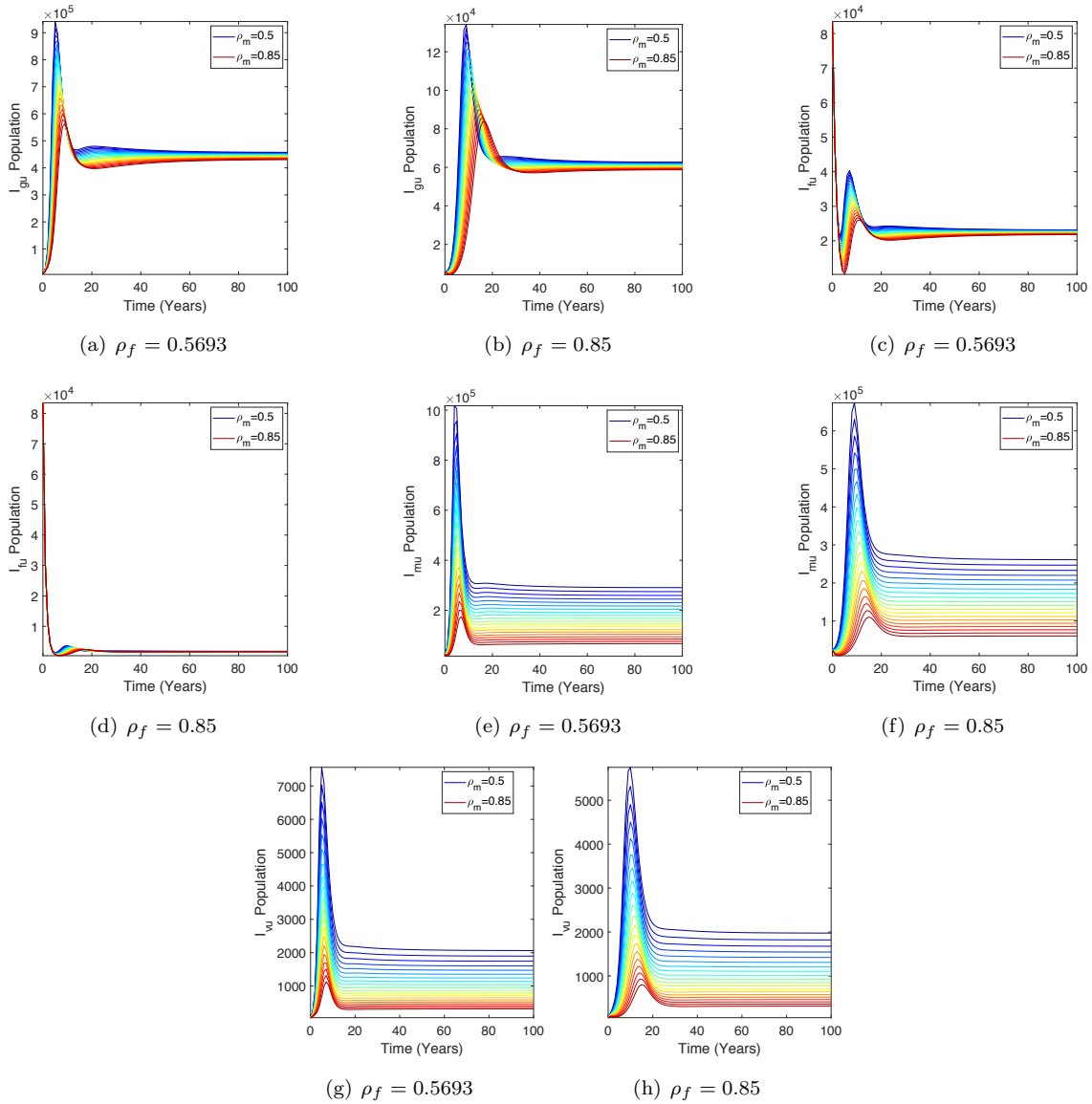


(a) $\rho_m = 0.6478$ (b) $\rho_m = 0.85$ (c) $\rho_m = 0.6478$

(d) $\rho_m = 0.85$ (e) $\rho_m = 0.6478$ (f) $\rho_m = 0.85$

(g) $\rho_m = 0.6478$ (h) $\rho_m = 0.85$

Figure 3.6: Disproportional effects on the AGYW Susceptible Population when ρ_m is fixed at its baseline value of 0.6478 and at the MDG of 0.85 while ρ_f is steadily increased from its baseline value of 0.5693 to the MDG of 0.85.

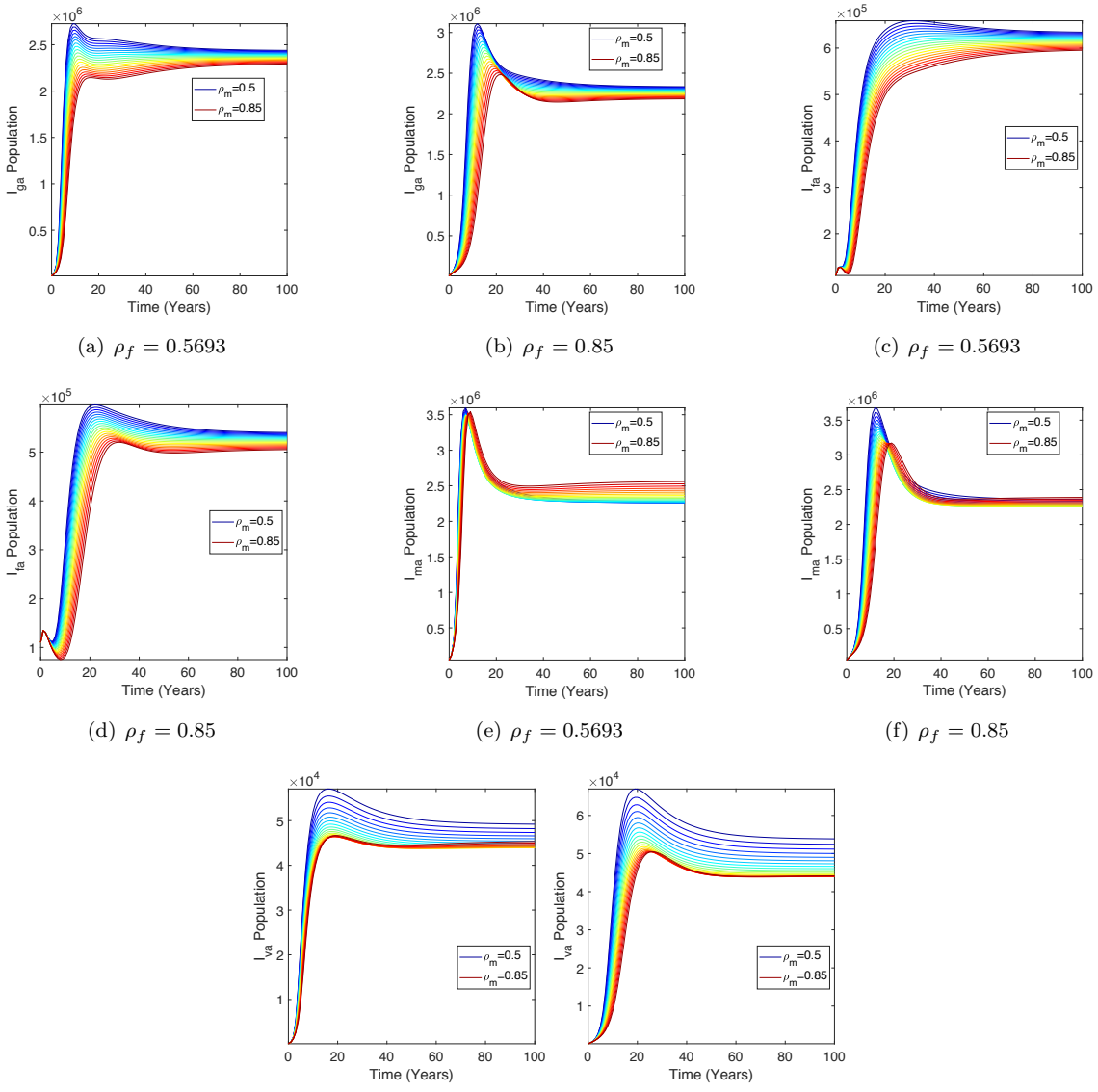


(a) $\rho_f = 0.5693$ (b) $\rho_f = 0.85$ (c) $\rho_f = 0.5693$

(d) $\rho_f = 0.85$ (e) $\rho_f = 0.5693$ (f) $\rho_f = 0.85$

(g) $\rho_f = 0.5693$ (h) $\rho_f = 0.85$

Figure 3.7: Infectious population without comprehensive knowledge of HIV/AIDS when ρ_f is fixed at its baseline value of 0.5693 while ρ_m is steadily increased from its baseline value of 0.6478 to the MDG of 0.85.

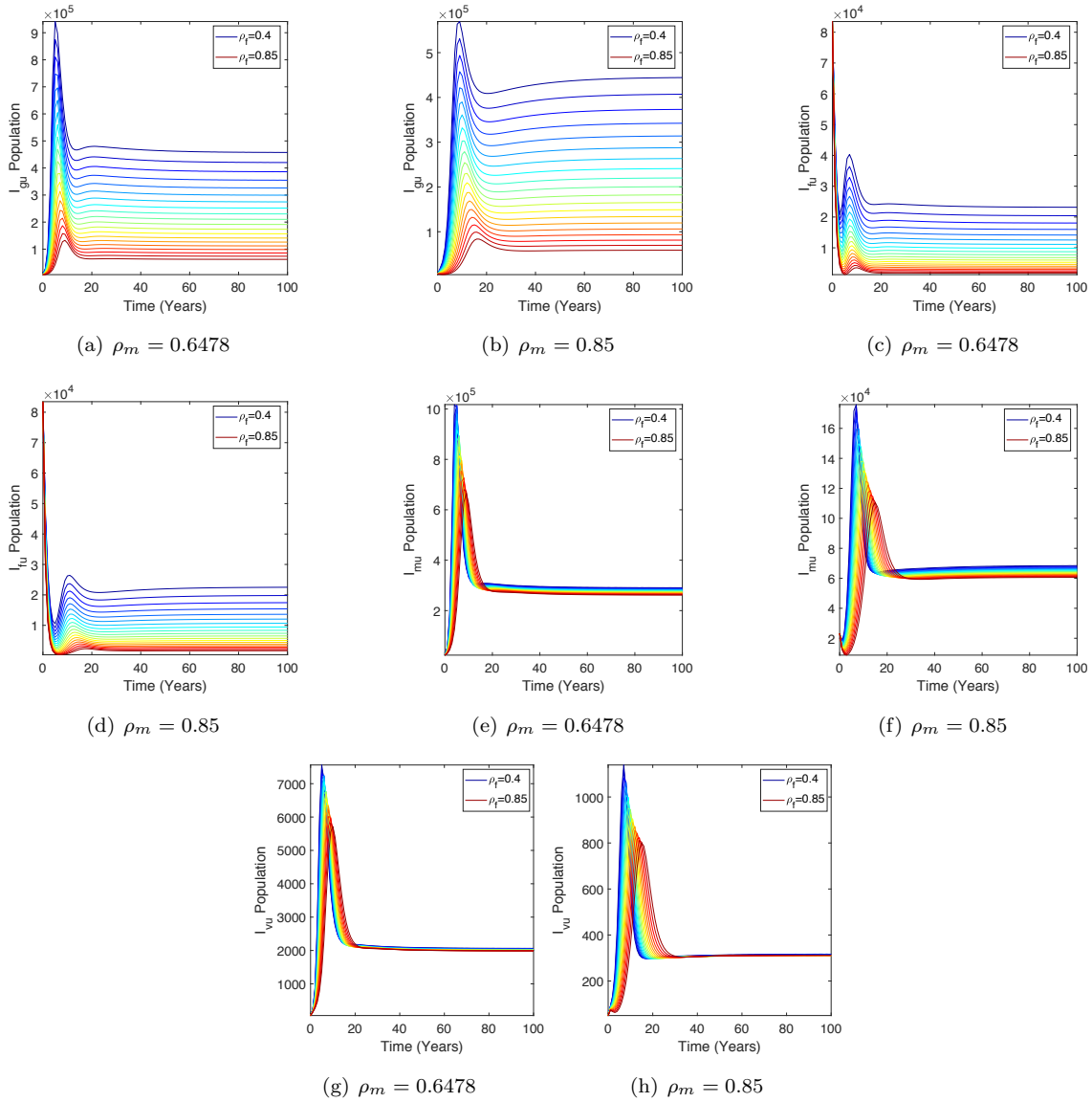


(a) $\rho_f = 0.5693$ (b) $\rho_f = 0.85$ (c) $\rho_f = 0.5693$

(d) $\rho_f = 0.85$ (e) $\rho_f = 0.5693$ (f) $\rho_f = 0.85$

(g) $\rho_f = 0.5693$ (h) $\rho_f = 0.85$

Figure 3.8: Infectious population with comprehensive knowledge of HIV/AIDS when ρ_f is fixed at its baseline value of 0.5693 while ρ_m is steadily increased from its baseline value of 0.6478 to the MDG of 0.85.

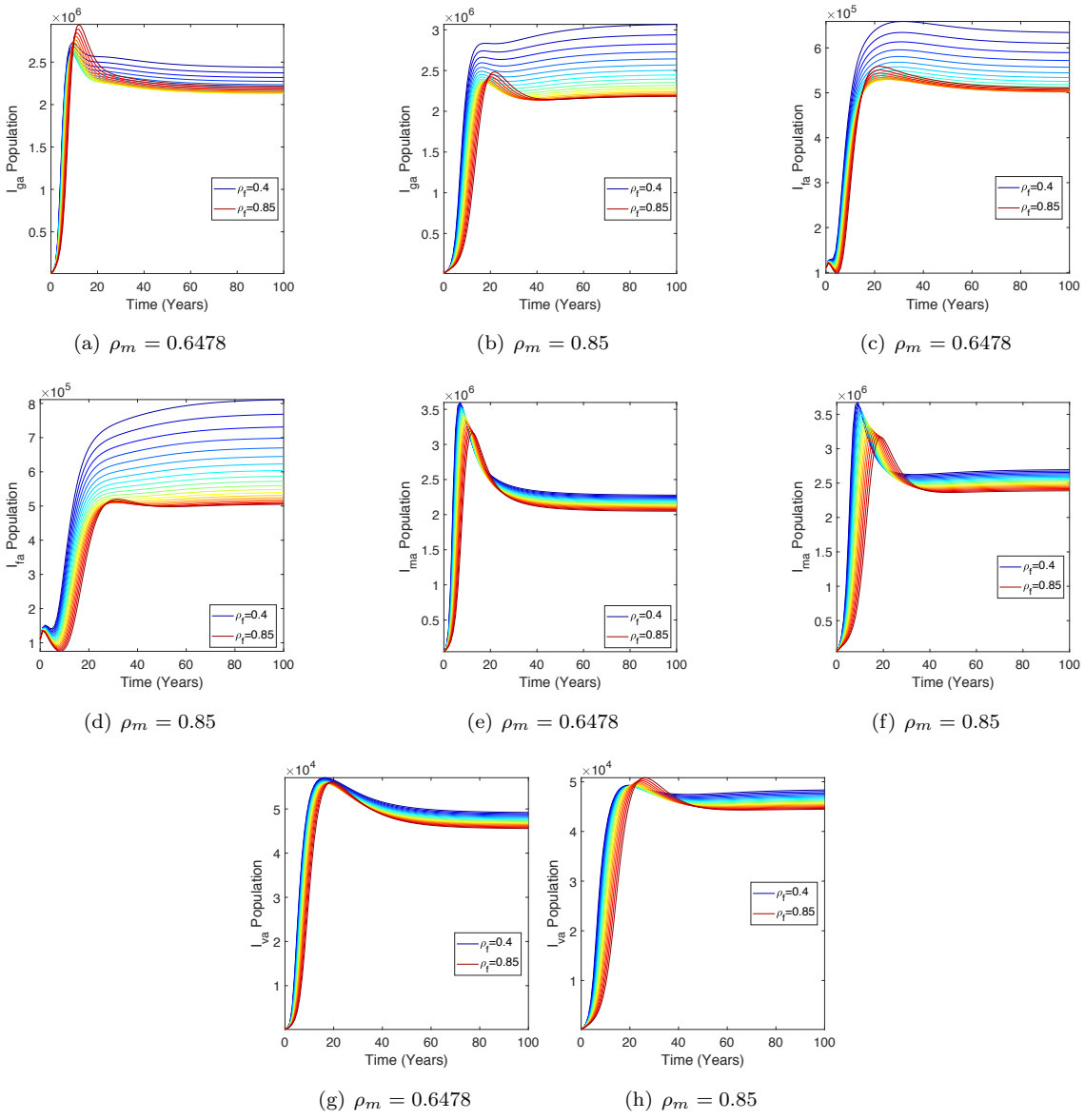


(a) $\rho_m = 0.6478$ (b) $\rho_m = 0.85$ (c) $\rho_m = 0.6478$

(d) $\rho_m = 0.85$ (e) $\rho_m = 0.6478$ (f) $\rho_m = 0.85$

(g) $\rho_m = 0.6478$ (h) $\rho_m = 0.85$

Figure 3.9: Infectious population without comprehensive knowledge of HIV/AIDS when ρ_m is fixed at its baseline value of 0.6478 and at the MDG of 0.85 while ρ_f is steadily increased from its baseline value of 0.5693 to the MDG of 0.85.



(a) $\rho_m = 0.6478$ (b) $\rho_m = 0.85$ (c) $\rho_m = 0.6478$

(d) $\rho_m = 0.85$ (e) $\rho_m = 0.6478$ (f) $\rho_m = 0.85$

(g) $\rho_m = 0.6478$ (h) $\rho_m = 0.85$

Figure 3.10: Infectious population with comprehensive knowledge of HIV/AIDS when ρ_m is fixed at its baseline value of 0.6478 and at the MDG of 0.85 while ρ_f is steadily increased from its baseline value of 0.5693 to the MDG of 0.85.

3.5 Model Results and Discussion

We investigated the effects of increasing HIV/AIDS comprehensive knowledge in the newly infected AG, AGYW and ABYM populations while taking into consideration the critical drivers of infection in these populations. Comprehensive knowledge of HIV/AIDS was varied from the baseline value which was disproportional [38] to the MDG target of 0.85 [47]. We categorized the infectious populations into two broad classes, one with comprehensive knowledge of HIV/AIDS

disease while the other without comprehensive knowledge of the HIV/AIDS disease. The infected populations without comprehensive knowledge of the HIV/AIDS disease could change their knowledge status to infected with comprehensive knowledge of HIV/AIDS once comprehensive knowledge of HIV/AIDS was disseminated to them. Simulations on the control reproduction number revealed that it could only be reduced but not below unity provided appropriate combination levels of comprehensive knowledge of HIV/AIDS disease were achieved among the AGYW and ABYM populations. We also analyzed the infection sub-networks of the local secondary infections and interestingly our model revealed that the longest infection duration associated with slower infection spread involved both the AGYW and ABYM gaining comprehensive knowledge of HIV/AIDS disease. We established the disease-free/endemic equilibria and the conditions for stability of the disease-free equilibrium.

Numerical simulations revealed the knockoff effect on the AGYW/ABYM susceptible populations when either HIV/AIDS comprehensive knowledge of the AGYW population or ABYM population was steadily increased from their baseline values to the MDG target of 85%. The numerical results suggested that protecting susceptible AGYW population against HIV/AIDS infection implied increasing ABYM HIV/AIDS knowledge and also protecting ABYM population against HIV/AIDS infection implied increasing AGYW HIV/AIDS knowledge. Effective dispensation of comprehensive knowledge of HIV/AIDS in all the newly infected populations significantly slowed down infection spread. These numerical results compared to the results of the local secondary infection sub-network analysis. It is therefore imperative that control efforts should target increasing the HIV/AIDS comprehensive knowledge of both newly infected populations as this protects all the susceptible populations.

Further, the simulation results suggests that effective comprehensive knowledge dissemination of 0.85 to both the newly infected AGYW/ABYM populations decreases the infected populations without comprehensive knowledge of HIV/AIDS. This in turn increases the infected populations with comprehensive knowledge of HIV/AIDS who stabilize at high population values posing a significant threat to the AGYW and ABYM susceptible populations. This suggests that even if MDG coverage of comprehensive knowledge of HIV/AIDS is achieved in all AGYW/ABYM populations the disease remains endemic. This result is consistent with the simulations on the control reproduction number which showed that $\mathcal{R}_c > 1$ implying that HIV/AIDS comprehensive knowledge alone is not sufficient in eliminating the disease in these populations.

To the best of our knowledge, there are no existing mathematical models that have addressed the impact of comprehensive knowledge of HIV/AIDS disease dynamics among the adolescents and young adults in Kenya. [26] modeled a homogeneous population which integrated public education campaign against HIV/AIDS. They recommended further investigation to assess the effects of a sex-structured public health education against HIV. While our model was built on this recommendation we however investigated the gender-wise effects of educating the infectious Kenyan youth on HIV/AIDS. Most technical reports [8, 39, 47] agree that these populations are at much higher risk of acquiring HIV/AIDS due to their low HIV/AIDS knowledge, transactional

sex, early sexual debut with multiple partners without the use of protection, sexual violence among others. It is possible that girls are receiving greater attention due to their vulnerability and the fact that they are twice likely to be infected with HIV/AIDS compared to their male counterparts. Our results however suggests that such efforts on AGYW serves to protect the ABYM ultimately but not necessarily the AGYW. The Government of Kenya through their implementing organs of the HIV/AIDS response have strategic plans to increase their efforts in educating the AGYW and ABYM populations against HIV/AIDS. The model results reflects the importance of educating both the infected AGYW and ABYM populations on HIV/AIDS while taking into consideration that effective HIV/AIDS knowledge alone is not enough to eradicate this disease in these populations. [26] model which used data from numerous countries reflected similar conclusions that effective knowledge alone as an intervention strategy does not guarantee eradication of HIV/AIDS in the general population.

It is thus imperative to study the impact of combination control strategies that will significantly reduce the high AGYW/ABYM infected populations. The control strategies that could be incorporated into the model to improve the model predictions could be regular dissemination of comprehensive knowledge to the AGYW/ABYM populations after a specific period of time, awareness through regular HIV testing and inclusion of antiretroviral therapy among others. It will also be interesting to study the role of the comprehensive knowledge of HIV/AIDS disease dynamics among the AGYW/ABYM in each county and its spiraling effects should movement connect these counties in a metapopulation sense in Kenya. The cost benefit analysis of the combination control strategies of the HIV/AIDS disease among the AGYW/ABYM should also be analyzed to ensure maximum benefits while minimizing cost. Using real epidemiological data to analyze all these scenarios would give far much accurate predictions. This in turn will influence relevant policies geared at promoting the well being of the AGYW/ABYM populations, ultimately reducing or completely eradicating the new HIV/AIDS infections in these populations.

Chapter 4

Evidence-Based Modeling of Combinatory Control on Kenyan Youth HIV/AIDS Dynamics

Keywords: HIV/AIDS, AGYW, ABYM, HIV Testing, Condom Use, Antiretroviral therapy, Combinatory control.

Abstract. We formulate a sex-structured deterministic model to study the effects of varying HIV testing rates, condom use rates and ART adherence rates among Adolescent Girls and Young Women (AGYW) and, Adolescent Boys and Young Men (ABYM) populations in Kenya. Attitudes influencing the Kenyan youth HIV/AIDS control measures both positively and negatively were considered. Using the 2012 Kenya AIDS Indicator Survey (KAIS) microdata we constructed our model, which we fitted to the UNAIDS-Kenya youth prevalence estimates to understand factors influencing Kenyan youth HIV/AIDS prevalence trends. While highly efficacious combination control approach significantly reduces HIV/AIDS prevalence rates among the youth, the disease remains endemic provided infected unaware sexual interactions persist. Disproportional gender-wise attitudes towards HIV/AIDS control measures play a key role in reducing the Kenyan youth HIV/AIDS prevalence trends. The female youth HIV/AIDS prevalence trend seems to be directly linked to increased male infectivity with decreased female infectivity while the male youth prevalence trend seems to be directly associated with increased female infectivity and reduced male infectivity.

4.1 Introduction

Kenya's HIV epidemic ranks fourth worldwide with its general population affected most alongside risk groups such as sex workers, people who inject drugs, men who have sex with men and the youth population [39, 47]. Two decades of successful combination control measures such as HIV testing, public health education campaigns, condom usage, antiretroviral therapy (ART) among others has resulted in the country's significant reduction of the HIV/AIDS prevalence from 10.5% in 1996 to 5.9% in 2015 [65].

Integral to the ongoing fight against HIV/AIDS in Kenya is the component of HIV Counseling and Testing (HCT) with the Government of Kenya and International Development Partners substantially increasing voluntary counseling and testing (VCT) services in the country [66]. Under the Adolescent Reproductive Health Development policy in the 2005-2015 Plan of Action the Government of Kenya sought to establish adolescent friendly voluntary counseling and testing services in a bid to improve and promote accessibility of youth friendly sexual and reproductive health services [67]. Scale up in innovative approaches to HIV testing in the country include community based HIV testing, door to door testing campaigns and self-testing kits [68, 69]. Despite these great progress in increasing HIV testing centers and new approaches to HIV testing, combined effects of inadequate health services, poverty, sociodemographic characteristics, HIV testing behavior, difficult socio-cultural and psycho-social conditions heavily impact the youth volunteering to HIV testing [70, 71, 72]. There is significant gender disparity in factors associated with HIV testing among the youth in Kenya with pregnant female youth required to test for HIV/AIDS due to advanced prevention of mother-to-child transmission(PMTCT) in the country compared to their male counterparts leading to female youth reporting higher HIV testing rates in comparison to male youth of a similar age cohort [47, 51, 65].

The youth in Kenya often engage in unprotected and unplanned sexual intercourse often resulting in sexually transmitted infections, pregnancies and HIV infections [51, 65, 73, 74]. While condom

use offers dual protection against unplanned pregnancies and protection against HIV/AIDS infection, there is increasing decline in condom use among the youth in Kenya [51, 74]. Some of the factors influencing condom use among the Kenyan youth include perceived individual's risk, peer influence, partner betrayal and socio-cultural factors such as religion, communities, schools and families [65, 73, 74, 75, 76]. The youth are easily influenced with their peers negative attitudes to condom use with male peers highly affected compared to female peers [77, 78]. Incorrect use of condoms in these population group places them at a higher risk of HIV/AIDS infection as many of them are experimenting with sex or under the influence of drugs or alcohol [73, 74]. While condom use among the youth remains inconsistent, condom use is generally higher among male youth compared to female youth due to the patriarchal society in Kenya where the male condom is the most preferred method with female youth reporting pressure from male partner not to use condoms [73, 74, 76]. External funding was responsible for most of the free condoms distribution in Kenya and cuts in donor funding has affected majority of the sexually active youth in Kenya who cannot afford to purchase condoms. [79].

Universal Test and Treat strategy by the World Health Organization (WHO) requires that all persons testing positive for HIV/AIDS be initiated on ART immediately irrespective of their CD4+ T cell count so as to achieve 90% diagnosis of all HIV positive persons with 90% of those positively diagnosed initiated on ART so as to achieve 90% viral load suppression [16]. Unfortunately, the adherence rates to ART is proving to be an uphill task among the youth in Kenya [80]. Factors influencing non adherence to ART among the youth in Kenya include stigma associated with disclosure of HIV/AIDS status, lack of adequate support from primary care givers and health workers, treatment fatigue, lack of adequate support structures in schools for youth living with HIV/AIDS, confidentiality breaches by health providers leading to disclosure of patients status to the community, fear of gossip and ridicule, financial constraints leading to failure to honor medical appointments or collect ART drugs and physical and emotional violence meted to orphaned perinatally infected youth by their care givers prompting them to fend for themselves or forcing them to street life [71, 72, 80, 81].

In Kenya, changing key HIV/AIDS control measures among the youth like HIV testing, condom use and ART adherence has faced significant challenges mostly due to societal attitudes towards the uptake of these control measures by the youth [82]. There is significant disparity in societal attitudes by gender towards the youth using some of these HIV/AIDS control measures [82]. On one hand, community norms and structural barriers directly affect condom use among the youth in Kenya with some communities advocating harsh punishment towards the youth using condoms [65, 73, 74, 75, 76, 82]. On the other hand, HIV knowledge, HIV-related stigma, income and social support from family and religious affiliations, mental health (depression, anxiety, stress) and substance use directly affect HIV test-seeking and treatment adherence among the youth [83, 84, 85]. These social drivers directly influencing HIV testing, condom use and ART adherence are rarely addressed in mathematical modelling.

Models formulated for HIV/AIDS dynamics have so far informed strategic planning, implemen-

tation and evaluation of control programs [86, 87, 88, 89, 90]. As of 2000, HIV/AIDS models have coupled interventions such as screening, anti-retroviral therapy (ART) treatment, Prep uptake and condom use [91, 92, 93, 94, 95, 96, 97]. Few of these models considered combination control strategies [25]. Real epidemiological data was used in [24, 25, 98, 99, 100, 101] to predict HIV/AIDS prevalence subject to the considered control measures.

We seek to show the effects of varying HIV testing rates, condom use rates and antiretroviral adherence rates on the sex-structured AGYW/ABYM disease dynamics in Kenya subject to attitudes influencing disease control such as psycho-social conditions, sociodemographic and socio-cultural characteristics described earlier. In this study, the positive/negative attitudes towards the use of HIV/AIDS control measures are designed to allow HIV testing, condom use and ART adherence to change over time. Using the 2019 UNAIDS-Kenya HIV Surveillance data we fit the AGYW/ABYM model prevalence under the three combination control measures to their respective prevalence data for reliable prevalence predictions and model parameter estimation. HIV/AIDS prevalence among the Adolescent Girls and Young Women (AGYW) population aged 15-24 is high at 5.7% whereas the Adolescent Boys and Young Men (ABYM) population is low at 2.2% [47]. About 73.6% of adolescent girls and young women aged 15-24 tested for HIV/AIDS in 2015 [47]. Similarly, 56% of adolescent boys aged and young men aged 15-24 reported to have tested for HIV/AIDS that year [47]. Approximately 89% of the AGYW reported not using condoms in trusted sexual relations whereas 57.6% of ABYM used condoms at their first sexual encounter [47]. Out of the 268, 586 youth living with HIV/AIDS, 16% are yet to access anti-retroviral therapy (ART) [65]. This model formulation provides a low cost approach to identify key areas for intervention in the real world that could help in reducing new HIV/AIDS infections among the youth in Kenya.

4.2 Methods

4.2.1 Data Description

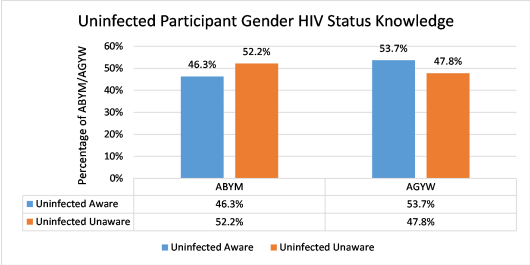
This section details the 2012 Kenya AIDS Indicator Survey description which was used to inform the model formulation described in section 4.2.2 and the UNAIDS-Kenya National Survey prevalence data description used for the model prevalence fit given in section 4.3.2.

Kenya AIDS Indicator Survey (KAIS) Data Description

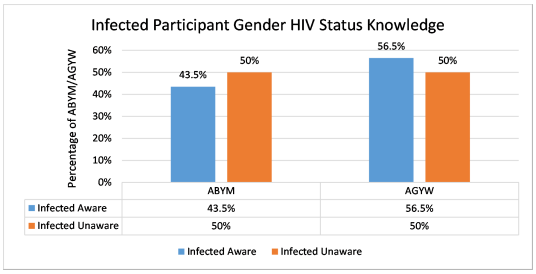
We used the 2012 Kenya AIDS Indicator Survey (KAIS) micro-data obtained from the Kenya National Bureau of Statistics website [102] to construct our model as it included data on HIV testing, sexual behavior and HIV care and treatment of children and adults. Given our interest in HIV testing, sexual behavior and HIV care and treatment of youth, we concentrated only on the all adults and sexual partners data sets. The all adults data set comprised of adolescents and adults aged 15-64 years totaling to 10, 811 with 5,211 males and 5,600 females. The sex partner data set had information regarding sex partner's gender, sexual behavior and HIV/AIDS status. We considered the sex partner data set as we were interested in heterosexual partners. We combined the all adults data set with the sex partners data set and extracted the youth aged

15-24 years. Thus, the combined data set comprised of 3,278 sexually active youth aged 15-24 years with 1,597 ABYM and 1,681 AGYW.

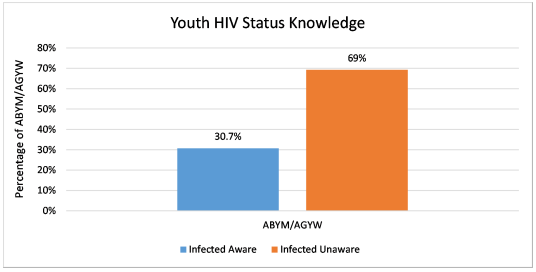
We generated a new variable for HIV/AIDS status knowledge from the combined data set based on HIV testing and it's structure included uninfected unaware, uninfected aware, infected unaware and infected aware. The self-reported status referred to the respondents self-reported HIV status whereas KAIS confirmed HIV status referred to the respondents HIV status based on laboratory results from the survey [102]. The KAIS confirmed HIV status took into account the viral load testing which we compared to the self-reported status thus adjusting the HIV/AIDS status knowledge of the youth [102]. Uninfected aware population comprised of individuals who reported negative HIV/AIDS status and were KAIS confirmed negative and those who reported negative having tested for HIV/AIDS elsewhere. Uninfected unaware were individuals who reported never tested for HIV/AIDS and were KAIS confirmed negative and those who reported positive HIV/AIDS status and were KAIS confirmed negative. Infected aware included those AGYW / ABYM who reported positive HIV/AIDS status and were KAIS confirmed positive and those who self-reported positive having tested for HIV/AIDS elsewhere. We classified the infected unaware as those who were HIV infected but reported negative and those who reported never tested for HIV/AIDS. Figures 4.1(a) and 4.1(b) gives the data summary for participant gender HIV status knowledge of the youth. HIV/AIDS status knowledge is highest among AGYW at 53.7% and 56.5% among susceptible and infected AGYW in comparison to ABYM. This is consistent with literature findings described in section 5.1 (see figures 4.1(a) and 4.1(b)). Infected unaware youth are 38.6% more compared to infected aware youth. (see figure 4.1(c)).



(a) Susceptible AGYW and ABYM HIV Status Knowledge



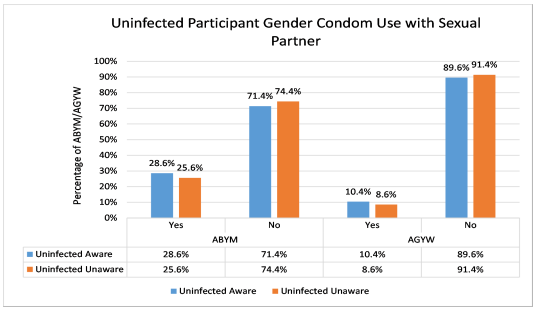
(b) Infected AGYW and ABYM HIV Status Knowledge



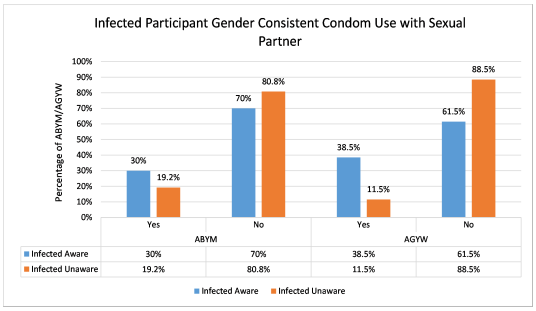
(c) AGYW/ABYM HIV Status Knowledge

Figure 4.1: Participant Gender HIV Status Knowledge

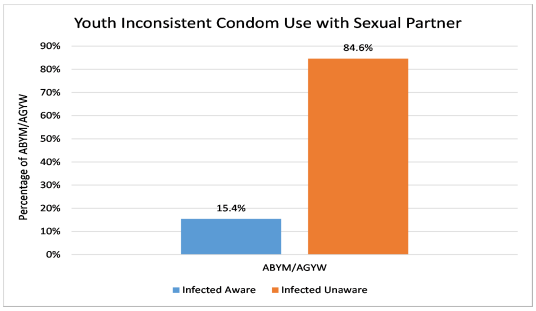
The question around the use of condom every time with sexual partner was used to determine condom use patterns among the youth and this was tabulated against their HIV status knowledge [102]. Figures 4.2(a) and 4.2(b) gives the data summary for participant gender condom use patterns with the youth sexual partners.



(a) Susceptible AGYW and ABYM Condom Use Patterns with Sexual Partner



(b) Infected AGYW and ABYM Condom Use Patterns with Sexual Partner

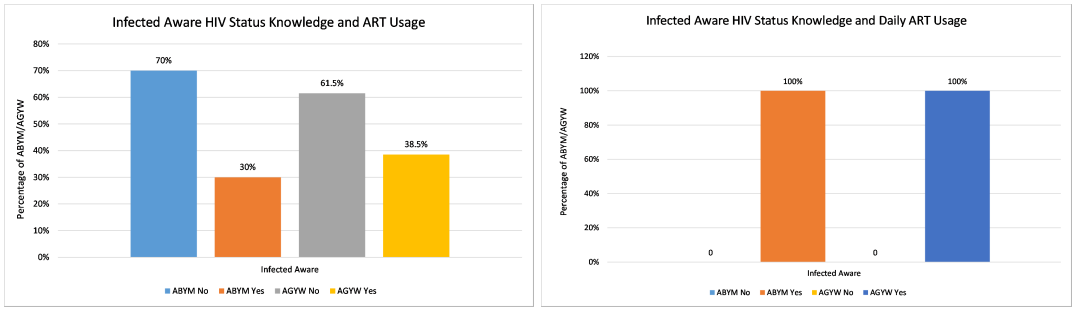


(c) Infected AGYW/ABYM Inconsistent Condom Use Patterns with Sexual Partner

Figure 4.2: Participant Gender Condom Use Patterns with Sexual Partner

Consistent condom use patterns among the uninfected aware ABYM is 18.2% higher in comparison to uninfected aware AGYW (see figure 4.2(a)). However, most of the uninfected aware youth fail to use condoms consistently with sexual partners with uninfected aware AGYW ranking highest at 89.6% (see figure 4.2(a)). Infected unaware youth inconsistent condom use with sexual partners is 69.2% higher compared to infected aware AGYW/ABYM populations (see figure 4.2(c)).

On ART adherence, the questions around currently using ART and daily ART usage were used to determine ART adherence among the infected AGYW/ABYM and this was also tabulated against their HIV status knowledge [102]. Figures 4.3(a) and 4.3(b) gives the data summary for participant gender HIV status knowledge and ART usage.



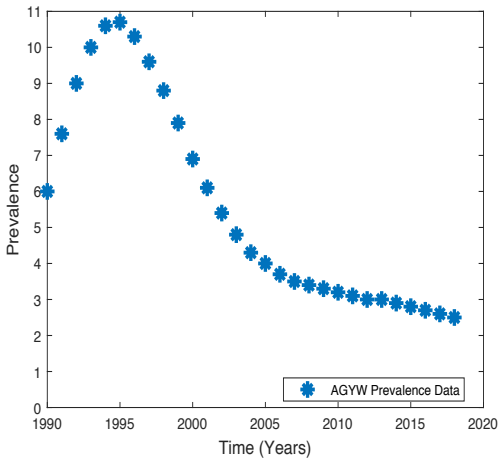
(a) Infected AGYW and ABYM on ART (b) Infected AGYW and ABYM Daily ART Usage

Figure 4.3: HIV/AIDS Infected Participant Gender ART Usage

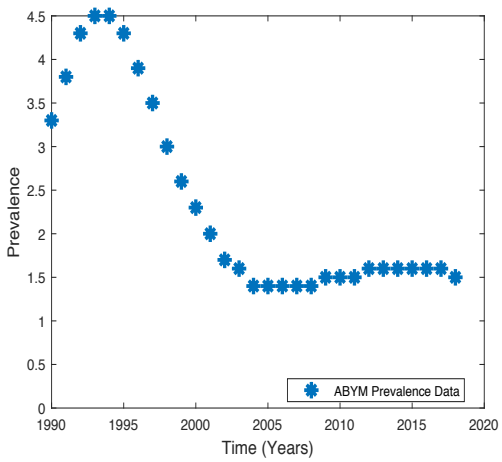
About 38.5% and 30% of infected aware AGYW and ABYM are on ART (see figure 4.3(a)). Figure 4.3(b) shows AGYW/ABYM initiated on ART with daily use, which implies adherence to ART. However, 61.5% and 70% of the infected aware AGYW and ABYM are yet to be initiated on ART (see figures 4.3(a)).

UNAIDS-KENYA HIV Surveillance Data Description

The National AIDS Control Council in Kenya partners with Avenir Health, UNAIDS, public health professionals, demographers, global epidemiologists and monitoring and evaluation experts to annually provide Kenya's HIV/AIDS estimates [2, 103]. These experts use the Spectrum tools endorsed by UNAIDS to provide these estimates which are based on data from five national surveys (2003 Kenya Demographic and Health Survey, 2007 Kenya AIDS Indicator Survey, 2008/2009 Kenya Demographic and Health Survey, 2012 Kenya AIDS Indicator Survey and 2014 Kenya Demographic and Health Survey) and, data from HIV Sentinel Surveillance among pregnant women, national census and data from various programmes [2]. Hence, Kenya's annual HIV/AIDS prevalence estimates provided by UNAIDS reflect the existing HIV epidemic in the country [2]. For this reason we use the UNAIDS-Kenya HIV Surveillance data on Kenyan youth prevalence to fit the model prevalence for AGYW and ABYM populations. The model fit was also used to estimate the best parameter estimates for some of the model parameters and predict the AGYW and ABYM prevalence for the years 2019 - 2023. Tables 4.3 - 4.5 give the AGYW/ABYM UNAIDS-Kenya prevalence estimates and figures 4.4(a), 4.4(b) show the 1990 - 2018 UNAIDS-Kenya prevalence estimates for the Kenyan youth [3]. In South Africa, [25] fitted their mathematical model to UNAIDS HIV prevalence data to study the country's HIV epidemic trends. Hence, we used the 2012 KAIS data to inform the model formulation described in section 4.2.2 and some state variables initial conditions and, the UNAIDS-Kenya HIV Surveillance data to fit the model and estimate some of the model parameters.



(a) AGYW UNAIDS-Kenya 1990 - 2018 Prevalence Estimates [3]



(b) ABYM UNAIDS-Kenya 1990 - 2018 Prevalence Estimates [3]

Figure 4.4: AGYW and ABYM UNAIDS-Kenya 1990-2018 Percentage Prevalence Estimates [3]

4.2.2 Model Formulation

We formulate a model describing HIV transmission dynamics in the AGYW and ABYM populations aged 15-24 with most of the state variables derived from the 2012 KAIS data described in section 4.2.1 [102]. While all the infected aware on ART treatment remained adherent in section 4.2.1 and figure 4.3, the model formulation considers the infected aware AGYW and ABYM populations on ART but are not adherent so as to make our model adaptable to non-adherence as the ART adherence rates among the infected aware youth in the KAIS data set was only for the 2012 data point. Section 5.1 highlights the need to model this population group as some of the infected aware youth on ART in general are not adherent to ART. Hence, we include this population group in the model formulation. We do not include the male population older than 24 years in this formulation as transactional sex in the 2012 KAIS population based survey was not common [104]. Hence, we primarily focus on the sexual behavior and use of HIV/AIDS control measures among the sexually active youth. In this study, the youth are defined as persons between the ages of 15 and 24 [105, 106].

The AGYW and ABYM populations are each categorized into six classes such that at time $t \geq 0$ there are susceptible AGYW, ABYM (S_{fu}, S_{mu}), infected AGYW, ABYM (I_{fu}, I_{mu}) who are not aware of their HIV status, susceptible AGYW, ABYM (S_{fa}, S_{ma}), infected AGYW, ABYM (I_{fa}, I_{ma}) who have tested for HIV/AIDS and are aware of their HIV status and use condoms consistently but are yet to be initiated on ART, infected AGYW, ABYM (T_{fu}, T_{mu}) who have tested for HIV/AIDS and are aware of their HIV status but use ART and condoms inconsistently and infected AGYW, ABYM (T_{fa}, T_{ma}) who have tested for HIV/AIDS and are aware of their HIV status and are adherent to ART and use condoms consistently. The total size of the AGYW and ABYM populations is given as $N_f = S_{fu} + S_{fa} + I_{fu} + I_{fa} + T_{fu} + T_{fa}$,

$N_m = S_{mu} + S_{ma} + I_{mu} + I_{ma} + T_{mu} + T_{ma}$ respectively. $N = N_f + N_m$ is the total AGYW and ABYM population. Figure 5.2 represents the flow of individuals into different compartments in a single patch model.

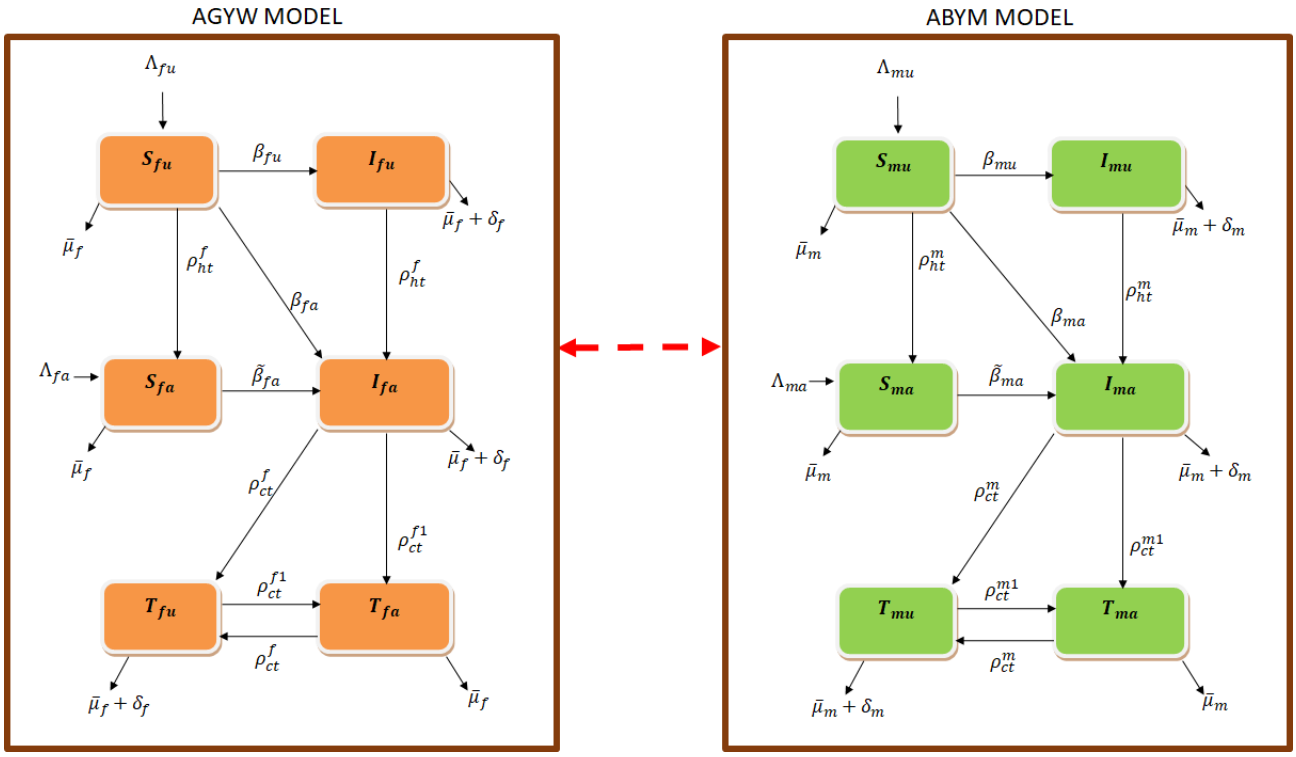


Figure 4.5: Schematics of the Compartmental Model. The AGYW and ABYM model describes the AGYW and ABYM transitions and interactions respectively.

The susceptibles females S_{fu}, S_{fa}, are free from the HIV infection but are at risk of infection through sexual contact with I_{mu}, I_{ma} and T_{mu} whereas the susceptibles males S_{mu}, S_{ma}, are free from the HIV infection but are at risk of infection through sexual contact with I_{fu}, I_{fa} and T_{fu}. Infectivity in I_{fu}, I_{mu} is much higher compared to I_{fa}, I_{ma} and T_{fu}, T_{mu} as the latter populations are more cautious given their infection status awareness compared to I_{fu}, I_{mu} populations. Also, T_{fu}, T_{mu} infectivity is further reduced given their partial use of condoms and ART compared to I_{fa}, I_{ma} who partially use condoms for either pregnancy or HIV/AIDS protection. Perfect adherence of T_{fa}, T_{ma} to condom use and ART reduces their viral load significantly such that they cannot sexually transmit HIV/AIDS given that undetectable viral load equals untransmittable [107]. Hence, we do not consider T_{fa}, T_{ma} populations infectious in this model as their infectivity risks are negligible. The susceptible classes S_{fu}, S_{mu} are at risk of infection at the incidence rates β_{fu}, β_{mu}, β_{fa}, β_{ma} whereas S_{fa}, S_{ma} are at risk of infection at the incidence rates $\tilde{\beta}_{fa}$, $\tilde{\beta}_{ma}$. The rates β_{fu}, β_{mu}, β_{fa}, β_{ma}, $\tilde{\beta}_{fa}$ and $\tilde{\beta}_{ma}$ are given in equations (5.1) as

$$
\begin{cases}
\beta_{fu} = \dfrac{c_f \gamma_f}{N_m} \left[I_{mu} + \alpha_c^m \rho_c I_{ma} + (\alpha_c^m \rho_c + \alpha_t^m \rho_t) T_{mu} \right], \\
\beta_{fa} = \dfrac{c_f \gamma_f}{N_m} \left[I_{mu} + \alpha_c^m \rho_c I_{ma} + (\alpha_c^m \rho_c + \alpha_t^m \rho_t) T_{mu} \right] \alpha_{ht}^m \, \rho_{ht}, \\
\tilde{\beta}_{fa} = \dfrac{c_f \gamma_f}{N_m} \left[I_{mu} + \alpha_c^m \rho_c I_{ma} + (\alpha_c^m \rho_c + \alpha_t^m \rho_t) T_{mu} \right] \alpha_{ht}^{m1} \, \rho_{ht}, \\
\beta_{mu} = \dfrac{c_m \gamma_m}{N_f} \left[I_{fu} + \alpha_c^f \rho_c I_{fa} + (\alpha_c^f \rho_c + \alpha_t^f \rho_t) T_{fu} \right], \\
\beta_{ma} = \dfrac{c_m \gamma_m}{N_f} \left[I_{fu} + \alpha_c^f \rho_c I_{fa} + (\alpha_c^f \rho_c + \alpha_t^f \rho_t) T_{fu} \right] \alpha_{ht}^f \, \rho_{ht}, \\
\tilde{\beta}_{ma} = \dfrac{c_m \gamma_m}{N_f} \left[I_{fu} + \alpha_c^f \rho_c I_{fa} + (\alpha_c^f \rho_c + \alpha_t^f \rho_t) T_{fu} \right] \alpha_{ht}^{f1} \, \rho_{ht}.
\end{cases} \tag{4.1}
$$

Contacts c_f, c_m are the average number of sexual interactions by AGYW/ABYM with individuals of the opposite sex per unit time whereas γ_f, γ_m are the probabilities that a susceptible AGYW/ABYM coming into proper contact with an infected individual of the opposite sex per unit time will contract the disease. Condom use rate (ρ_c) decreases the disease spread by I_{fa}, I_{ma} whereas condom use and ART adherence rate (ρ_t) reduces the infection risk by T_{fu}, T_{mu}. HIV/AIDS status disclosure (ρ_{ht}) by newly HIV/AIDS tested I_{fu}, I_{mu} and already tested populations I_{fa}, I_{ma}, T_{fu}, T_{mu} further reduces the disease spread to the susceptible populations.

When each of the HIV/AIDS control measures ρ_{ht}, ρ_c, ρ_t in the AGYW/ABYM populations is 1 we have perfect adherence otherwise, $0 \leq \rho_{ht}$, ρ_c, $\rho_t < 1$. The rates α_{ht}^f, α_{ht}^m represent negative attitudes affecting the efficacy of HIV testing rate ρ_{ht} in the AGYW and ABYM populations such as poor health services, poverty, psycho-social conditions, socio-demographic characteristics among others [70, 71, 72]. Rates α_c^f, α_c^m represent negative attitudes affecting the efficacy of condom use rate in the AGYW and ABYM populations such as religion, peer influence, perceived individual's risk among others [65, 73, 74, 75, 76]. Also, α_t^f, α_t^m represent negative attitudes affecting the efficacy of ART usage rate among the infected AGYW and ABYM such as stigma, poverty, caregivers waning support, confidentiality breaches by health workers among others [71, 72, 80, 81]. Section 5.1 highlights how societal attitudes affect HIV testing rates, condom use and adherence to ART among the youth in Kenya. The rates $\alpha_c^f \, \rho_c$, $\alpha_c^m \, \rho_c$ acts on I_{fa}, I_{ma} to reduce their infectivity as condom use serves to protect susceptible AGYW and ABYM from acquiring new HIV/AIDS infection. In addition to condom use, T_{fu}, T_{mu} partially uses ART which works to reduce their HIV/AIDS viral load. The combined effects of condom use and ART usage ($\alpha_c^f \, \rho_c + \alpha_t^f \, \rho_t$, $\alpha_c^m \, \rho_c + \alpha_t^m \, \rho_t$) further reduces the infectivity of T_{fu}, T_{mu} as $0 < \alpha_c^f, \alpha_c^m, \alpha_t^f, \alpha_t^m < 1$. Thus, T_{fu}, T_{mu} infectivity is less than I_{fa}, I_{ma} which is less than I_{fu}, I_{mu}.

Incidence rates by untested AGYW/ABYM with individuals of the opposite sex per unit time are given as β_{fu}, β_{mu} respectively . The incidence rates β_{fa}, β_{ma} are given by HIV/AIDS tested

AGYW/ABYM but not under ART treatment with individuals of the opposite sex per unit time. The incidence rates $\tilde{\beta}_{fa}$, $\tilde{\beta}_{ma}$ results from HIV/AIDS tested youth who are not perfectly adherent to consistent condom use and ART treatment with individuals of the opposite sex per unit time. The incidence rates β_{fu}, β_{mu}, β_{fa}, β_{ma}, $\tilde{\beta}_{fa}$ and $\tilde{\beta}_{ma}$ have proportionate mixing incidences since some of the youth aged 15-24 will have already initiated sex with most of them remaining sexually active.

Uninfected unaware S_{fu}, S_{mu} who know their HIV/AIDS status through HIV testing moves to S_{fa}, S_{ma} at the rates ρ_{ht}^{f}, ρ_{ht}^{m} with $\rho_{ht}^{f} = \alpha_{ht}^{f}\,\rho_{ht}$ and $\rho_{ht}^{m} = \alpha_{ht}^{m}\,\rho_{ht}$. A newly infected S_{fu}, S_{mu} through interaction with infected I_{mu}, I_{ma} or T_{mu} who fail to disclose their HIV/AIDS status will move to I_{fu}, I_{mu} at the rates β_{fu}, β_{mu}. Also, a newly infected S_{fu}, S_{mu} through sexual contact with infected aware populations of the opposite sex will move to I_{fa}, I_{ma} at the rates β_{fa}, β_{ma} given that status disclosure by the infected aware populations results in HIV/AIDS awareness of the newly infected S_{fu}, S_{mu}. A newly infected S_{fa}, S_{ma} moves to I_{fa}, I_{ma} at the rates $\tilde{\beta}_{fa}$, $\tilde{\beta}_{ma}$. Infected unaware I_{fu}, I_{mu} can move to I_{fa}, I_{ma} at the rates ρ_{ht}^{f}, ρ_{ht}^{m} through HIV testing. Also, I_{fa}, I_{ma} and T_{fu}, T_{mu} who consistently use condoms and adhere to ART treatment moves to T_{fa}, T_{ma} at the rates ρ_{ct}^{f1}, ρ_{ct}^{m1} whereas an I_{fa}, I_{ma} or T_{fa}, T_{ma} who fail to use condoms consistently or adhere to ART treatment moves to T_{fu}, T_{mu} at the rates ρ_{ct}^{f}, ρ_{ct}^{m} respectively with $\rho_{ct}^{f1} = \alpha_{c}^{f1}\,\rho_{c} + \alpha_{t}^{f1}\,\rho_{t}$, $\rho_{ct}^{m1} = \alpha_{c}^{m1}\rho_{c} + \alpha_{t}^{m1}\,\rho_{t}$, $\rho_{ct}^{f} = \alpha_{c}^{f}\rho_{c} + \alpha_{t}^{f}\,\rho_{t}$ and $\rho_{ct}^{m} = \alpha_{c}^{m}\rho_{c} + \alpha_{t}^{m}\rho_{t}$ respectively. α_{ht}^{f1}, α_{ht}^{m1}, α_{c}^{f1}, α_{t}^{f1}, α_{c}^{m1}, α_{t}^{m1} and α_{ht}^{f}, α_{ht}^{m}, α_{c}^{f}, α_{t}^{f}, α_{c}^{m}, α_{t}^{m} are parameters representing negative/positive attitudes influencing HIV/AIDS control measures (ρ_{ht}, ρ_{c}, ρ_{t}) but not to zero given that in the Kenyan HIV/AIDS youth dynamics some control measures are in place [2]. The rates α_{ht}^{f1}, α_{ht}^{m1}, α_{c}^{f1}, α_{t}^{f1}, α_{c}^{m1} represent attitudes affecting the efficacy of ρ_{ht}, ρ_{c}, ρ_{t} positively such as confidentiality by health workers, adequate support structure at home and the community at large, improved financial status among others whereas α_{ht}^{f}, α_{ht}^{m}, α_{c}^{f}, α_{t}^{f}, α_{c}^{m} represent negative attitudes, which was explained earlier, influencing the said control measures. The rates ρ_{ct}^{f}, ρ_{ct}^{m} represent combined condom use and ART use coupled with negative attitudes whereas ρ_{ct}^{f1}, ρ_{ct}^{m1} represent combined condom use and ART use coupled with positive attitudes among the AGYW and ABYM respectively. Thus,

$$0 < \alpha_{ht}^{f1}, \alpha_{ht}^{m1}, \alpha_{c}^{f1}, \alpha_{t}^{f1}, \alpha_{c}^{m1}, \alpha_{t}^{m1}, \alpha_{ht}^{f}, \alpha_{ht}^{m}, \alpha_{c}^{f}, \alpha_{t}^{f}, \alpha_{c}^{m}, \alpha_{t}^{m} < 1$$

with

$$\alpha_{ht}^{f1}, \alpha_{ht}^{m1}, \alpha_{c}^{f1}, \alpha_{t}^{f1}, \alpha_{c}^{m1}, \alpha_{t}^{m1} > \alpha_{ht}^{f}, \alpha_{ht}^{m}, \alpha_{c}^{f}, \alpha_{t}^{f}, \alpha_{c}^{m}, \alpha_{t}^{m}.$$

Recruitment rates into susceptible populations S_{fu}, S_{mu}, S_{fa}, S_{ma} is by natural births and maturity to 15 years and are given as Λ_{fu}, Λ_{mu}, Λ_{fa}, Λ_{ma} respectively. The susceptible classes are all reduced by natural deaths μ_{f}, μ_{m} whereas the infectious classes are all decreased by natural deaths and disease induced deaths, δ_{f}, δ_{m}. Upon turning 24 years, the AGYW and the ABYM population exit the model at the rate σ. The state variables and parameters are assumed

to be positive given that a population dynamics model is being studied. Tables 4.1 and 4.2 gives the summary description for the state variables and model parameters respectively.

The system of ordinary differential equations governing the AGYW/ABYM HIV model is given by the system of equations (5.3) as

$$
\begin{cases}
\dfrac{dS_{fu}}{dt} = \Lambda_{fu} - \beta_{fu}\, S_{fu} - \beta_{fa}\, S_{fu} - \mu_{f1}\, S_{fu}, \\
\dfrac{dS_{fa}}{dt} = \Lambda_{fa} + \rho_{ht}^{f}\, S_{fu} - \tilde{\beta}_{fa}\, S_{fa} - \mu_{f2}\, S_{fa}, \\
\dfrac{dI_{fu}}{dt} = \beta_{fu}\, S_{fu} - \mu_{f3}\, I_{fu}, \\
\dfrac{dI_{fa}}{dt} = \tilde{\beta}_{fa}\, S_{fa} + \beta_{fa}\, S_{fu} + \rho_{ht}^{f}\, I_{fu} - \mu_{f4}\, I_{fa}, \\
\dfrac{dT_{fu}}{dt} = \rho_{ct}^{f}\, I_{fa} + \rho_{ct}^{f}\, T_{fa} - \mu_{f5}\, T_{fu}, \\
\dfrac{dT_{fa}}{dt} = \rho_{ct}^{f1} I_{fa} + \rho_{ct}^{f1}\, T_{fu} - \mu_{f6}\, T_{fa}, \\
\dfrac{dS_{mu}}{dt} = \Lambda_{mu} - \beta_{mu}\, S_{mu} - \beta_{ma}\, S_{mu} - \mu_{m1}\, S_{mu}, \\
\dfrac{dS_{ma}}{dt} = \Lambda_{ma} + \rho_{ht}^{m}\, S_{mu} - \tilde{\beta}_{ma}\, S_{ma} - \mu_{m2}\, S_{ma}, \\
\dfrac{dI_{mu}}{dt} = \beta_{mu}\, S_{mu} - \mu_{m3}\, I_{mu}, \\
\dfrac{dI_{ma}}{dt} = \tilde{\beta}_{ma}\, S_{ma} + \beta_{ma}\, S_{mu} + \rho_{ht}^{m}\, I_{mu} - \mu_{m4}\, I_{ma}, \\
\dfrac{dT_{mu}}{dt} = \rho_{ct}^{m}\, I_{ma} + \rho_{ct}^{m}\, T_{ma} - \mu_{m5}\, T_{mu}, \\
\dfrac{dT_{ma}}{dt} = \rho_{ct}^{m1}\, I_{ma} + \rho_{ct}^{m1}\, T_{mu} - \mu_{m6}\, T_{ma}.
\end{cases}
\tag{4.2}
$$

where
$\bar{\mu}_f = \mu_f + \sigma$, $\mu_{f1} = \rho_{ht}^{f} + \bar{\mu}_f$, $\mu_{f2} = \bar{\mu}_f$, $\mu_{f3} = \rho_{ht}^{f} + \bar{\mu}_f + \delta_f$, $\mu_{f4} = \rho_{ct}^{f} + \rho_{ct}^{f1} + \bar{\mu}_f + \delta_f$, $\mu_{f5} = \rho_{ct}^{f1} + \bar{\mu}_f + \delta_f$, $\mu_{f6} = \rho_{ct}^{f} + \bar{\mu}_f$, $\bar{\mu}_m = \mu_m + \sigma$, $\mu_{m1} = \rho_{ht}^{m} + \bar{\mu}_m$, $\mu_{m2} = \bar{\mu}_m$, $\mu_{m3} = \rho_{ht}^{m} + \bar{\mu}_m + \delta_m$, $\mu_{m4} = \rho_{ct}^{m} + \rho_{ct}^{m1} + \bar{\mu}_m + \delta_m$, $\mu_{m5} = \rho_{ct}^{m1} + \bar{\mu}_m + \delta_m$, $\mu_{m6} = \rho_{ct}^{m} + \bar{\mu}_m$.

Table 4.1: Summary Description of State variables

Variable	Description
S_{fu}, S_{mu}	Susceptible AGYW & ABYM who have never tested for HIV/AIDS
S_{fa}, S_{ma}	Susceptible AGYW & ABYM who have ever tested for HIV/AIDS
I_{fu}, I_{mu}	Infected AGYW & ABYM who have never tested for HIV/AIDS
I_{fa}, I_{ma}	Infected AGYW & ABYM who have ever tested for HIV/AIDS
T_{fu}, T_{mu}	Infected aware AGYW & ABYM who are not adherent to ART or consistent condom use
T_{fa}, T_{ma}	Infected aware AGYW & ABYM who are adherent to ART and use condoms consistently

Table 4.2: Summary Description of Parameters

Parameter	Description
Λ_{fu}, Λ_{mu}	Natural birth and maturity rates of susceptible AGYW and ABYM unaware of their HIV status
Λ_{fa}, Λ_{ma}	Natural birth and maturity rates of susceptible AGYW and ABYM aware of their HIV status
ρ_{ht}	AGYW/ABYM HIV testing rate
ρ_t	AGYW/ABYM adherence rate to anti-retroviral therapy treatment
ρ_c	AGYW/ABYM condom use rate
μ_f, μ_m	Natural death rates of AGYW and ABYM respectively
γ_f, γ_m	Probabilities of AGYW and ABYM transmission risk
δ_f, δ_m	Disease induced deaths in AGYW and ABYM respectively
c_f, c_m	AGYW and ABYM sexual contact rates
α_{ht}^f, α_{ht}^m, α_{ht}^{f1}, α_{ht}^{m1}	Negative and positive attitude rates influencing HIV testing rates among the AGYW and ABYM respectively
α_c^f, α_c^m, α_c^{f1}, α_c^{m1}	Negative and positive attitude rates influencing condom use rates among the AGYW and ABYM respectively
α_t^f, α_t^m, α_t^{f1}, α_t^{m1}	Negative and positive attitude rates influencing ART adherence rates among the AGYW and ABYM respectively
σ	Exit rate of AGYW and ABYM upon turning 24 years

4.2.3 Model Properties

Mathematical analysis of the formulated model system (4.2) is presented here. We show that the compact system of ordinary differential equations (5.3) governing the model of biological interest is well-posed and control reproduction number with its biological interpretation given. The conditions for stability of the model steady states are determined.

4.2.4 Positivity

Theorem 4.2.1.
The model solutions $S_{fu}(t)$, $S_{fa}(t)$, $I_{fu}(t)$, $I_{fa}(t)$, $T_{fu}(t)$, $T_{fa}(t)$, $S_{mu}(t)$, $S_{ma}(t)$, $I_{mu}(t)$, $I_{ma}(t)$, $T_{mu}(t)$, $T_{ma}(t)$ *of the model system* (4.2) *are positive for all* $t > 0$.

Proof. Using the contradiction concept, we show that the model solutions of the differential equation system (3.1) are strictly non-negative.

Suppose by contradiction that $t_i, i = 1, 2, ..., 12$ are the respective first times when the model trajectories $S_{fu}(t)$, $S_{fa}(t)$, $I_{fu}(t)$, $I_{fa}(t)$, $T_{fu}(t)$, $T_{fa}(t)$, $S_{mu}(t)$, $S_{ma}(t)$, $I_{mu}(t)$, $I_{ma}(t)$, $T_{mu}(t)$, $T_{ma}(t)$ reach 0 with $t_0 = min\{t_i\}$.

If $t_0 = t_1$, with $t_1 \neq t_2, t_3, ..., t_{12}$ and $S_{fu}(t)$ be strictly less than $0 \in [0, t_1]$ with $S_f(t_1) = 0$. We let $S_{fa}(t_1) > 0$, $I_{fu}(t_1) > 0$, $I_{fa}(t_1) > 0$, $T_{fu}(t_1) > 0$, $T_{fa}(t_1) > 0$, $S_{mu}(t_1) > 0$, $S_{ma}(t_1) > 0$, $I_{mu}(t_1) > 0$, $I_{ma}(t_1) > 0$, $T_{mu}(t_1) > 0$, $T_{ma}(t_1) > 0$ $\forall t \in [0, t_1]$.

From the first equation of the model system (4.2)

$$\frac{dS_{fu}}{dt} = \Lambda_{fu} - (\beta_{fu} + \beta_{fa} + \mu_{f1})S_{fu} \geq -(\beta_{fu} + \beta_{fa} + \mu_{f1})S_{fu},$$

$$S_{fu}(t) \geq S_{fu}(0)\, e^{\int_0^t -(\beta_{fu}(s)+\beta_{fa}(s)+\mu_{f1})ds}.$$

At $t = t_1$,

$$S_{fu}(t_1) \geq S_{fu}(0)\, e^{\int_0^{t_1} -(\beta_{fu}(s)+\beta_{fa}(s)+\mu_{f1})ds} > 0. \tag{4.3}$$

Equation 4.3 is a contradiction to the fact that $S_{fu}(t_1) = 0$. Therefore, $S_{fu}(t) \not< 0 \in [0, t_1]$ and $S_{fu}(t) \neq 0$ implying that, $S_{fu}(t) > 0$.

If $t_0 = t_2$, and $t_2 \neq t_1, t_3, t_4, ..., t_{12}$ and $S_f(t) < 0$ in the interval $[0, t_2]$ with $S_{fa}(t_2) = 0$. Let $S_{fu}(t_2) > 0$, $I_{fu}(t_2) > 0$, $I_{fa}(t_2) > 0$, $T_{fu}(t_2) > 0$, $T_{fa}(t_2) > 0$, $S_{mu}(t_2) > 0$, $S_{ma}(t_2) > 0$, $I_{mu}(t_2) > 0$, $I_{ma}(t_2) > 0$, $T_{mu}(t_2) > 0$, $T_{ma}(t_2) > 0$ $\forall t \in [0, t_2]$.

From second equation of the model system (4.2) it is clear that

$$\frac{dS_{fa}}{dt} = \Lambda_{fa} + \rho_{ht}^f S_{fu} - (\tilde{\beta}_{fa} + \mu_{f2})S_{fa} \geq -(\tilde{\beta}_{fa} + \mu_{f2})S_{fa},$$
$$S_{fa}(t) \geq S_{fa}(0)\, e^{\int_0^t -(\tilde{\beta}_{fa}(s)+\mu_{f2})ds}.$$

Now, when $t = t_2$,

$$S_{fa}(t_2) \geq S_{fa}(0)\, e^{\int_0^{t_2} -(\tilde{\beta}_{fa}(s)+\mu_{f2})ds} > 0. \tag{4.4}$$

Equation 4.4 contradicts the fact that $S_{fa}(t_2) = 0$. Thus, $S_{fa}(t)$ is neither strictly negative in the interval $[0, t_2]$ nor equal to 0. This implies that, $S_{fa}(t) > 0$.

If $t_0 = t_3$, and $t_3 \neq t_1, t_2, t_4, t_5, ..., t_{12}$ and $S_m(t) < 0$ in the interval $[0, t_3]$ with $S_m(t_3) = 0$. Let $S_{fu}(t_3) > 0$, $S_{fa}(t_3) > 0$, $I_{fa}(t_3) > 0$, $T_{fu}(t_3) > 0$, $T_{fa}(t_3) > 0$, $S_{mu}(t_3) > 0$, $S_{ma}(t_3) > 0$, $I_{mu}(t_3) > 0$, $I_{ma}(t_3) > 0$, $T_{mu}(t_3) > 0$, $T_{ma}(t_3) > 0$ $\forall t \in [0, t_3]$.

From the third equation of the model system (4.2),

$$\frac{dI_{fu}}{dt} = \beta_{fu} S_{fu} - \mu_{f3} I_{fu} \geq -\mu_{f3} I_{fu},$$
$$I_{fu}(t) \geq I_{fu}(0) e^{-\mu_{f3} t}.$$

At $t = t_3$,

$$I_{fu}(t_3) \geq I_{fu}(0) e^{-\mu_{f3} t_3} > 0. \tag{4.5}$$

Equation 4.5 contradicts the fact that $I_{fu}(t_3) = 0$. Thus, $I_{fu}(t)$ is neither strictly negative in the interval $[0, t_3]$ nor equal to 0. This implies that, $I_{fu}(t) > 0$.

If $t_0 = t_4$, and $t_4 \neq t_1, t_2, t_3, t_5, t_6, ..., t_{12}$ and $S_v(t) < 0$ in the interval $[0, t_4]$ with $S_v(t_4) = 0$. Let $S_{fu}(t_4) > 0$, $S_{fa}(t_4) > 0$, $I_{fu}(t_4) > 0$, $T_{fu}(t_4) > 0$, $T_{fa}(t_4) > 0$, $S_{mu}(t_4) > 0$, $S_{ma}(t_4) > 0$, $I_{mu}(t_4) > 0$, $I_{ma}(t_4) > 0$, $T_{mu}(t_4) > 0$, $T_{ma}(t_4) > 0$ $\forall t \in [0, t_4]$.

From the fourth equation of the model system (4.2),

$$\frac{dI_{fa}}{dt} = \tilde{\beta}_{fa} S_{fa} + \beta_{fa} S_{fu} + \rho^f_{ht} I_{fu} - \mu_{f4} I_{fa} \geq -\mu_{f4} I_{fa},$$
$$I_{fa}(t) \geq I_{fa}(0) e^{-\mu_{f4} t}.$$

At $t = t_4$,

$$I_{fa}(t_4) \geq I_{fa}(0) e^{-\mu_{f4} t_4} > 0. \tag{4.6}$$

Equation 4.6 contradicts the fact that $I_{fa}(t_4) = 0$. Thus, $I_{fa}(t)$ is neither strictly negative in the interval $[0, t_4]$ nor equal to 0. This implies that, $I_{fa}(t) > 0$.

If $t_0 = t_5$, and $t_5 \neq t_1, t_2, t_3, t_4, t_6, t_7 ..., t_{12}$ and $I_{gu}(t) < 0$ in the interval $[0, t_5]$ with $I_{gu}(t_5) = 0$. Let $S_{fu}(t_5) > 0$, $S_{fa}(t_5) > 0$, $I_{fu}(t_5) > 0$, $I_{fa}(t_5) > 0$, $T_{fa}(t_5) > 0$, $S_{mu}(t_5) > 0$, $S_{ma}(t_5) > 0$, $I_{mu}(t_5) > 0$, $I_{ma}(t_5) > 0$, $T_{mu}(t_5) > 0$, $T_{ma}(t_5) > 0$ $\forall t \in [0, t_5]$.

From the fifth equation of the model system (4.2),

$$\frac{dT_{fu}}{dt} = \rho^f_{ct} I_{fa} + \rho^f_{ct} T_{fa} - \mu_{f5} T_{fu} \geq -\mu_{f5} T_{fu},$$
$$T_{fu}(t) \geq T_{fu}(0) e^{-\mu_{f5} t}.$$

At $t = t_5$,

$$T_{fu}(t_5) \geq T_{fu}(0)\, e^{-\mu_{f5} t_5} > 0. \tag{4.7}$$

Equation 4.7 contradicts the fact that $T_{fu}(t_5) = 0$. Thus, $T_{fu}(t) \not< 0$ in the interval $[0, t_5]$ and $T_{fu}(t) \neq 0$. This implies that, $T_{fu}(t) > 0$.

If $t_0 = t_6$, and $t_6 \neq t_1, t_2, t_3, t_4, t_5, t_7, t_8 ..., t_{12}$ and $I_{ga}(t) < 0$ in the interval $[0, t_6]$ with $I_{ga}(t_6) = 0$. Let $S_{fu}(t_6) > 0$, $S_{fa}(t_6) > 0$, $I_{fu}(t_6) > 0$, $I_{fa}(t_6) > 0$, $T_{fu}(t_6) > 0$, $S_{mu}(t_6) > 0$, $S_{ma}(t_6) > 0$, $I_{mu}(t_6) > 0$, $I_{ma}(t_6) > 0$, $T_{mu}(t_6) > 0$, $T_{ma}(t_6) > 0$ $\forall t \in [0, t_6]$.

From the sixth equation of the model system (4.2),

$$\frac{dT_{fa}}{dt} = \rho_{ct}^{f1} I_{fa} + \rho_{ct}^{f1} T_{fu} - \mu_{f6} T_{fa} \geq -\mu_{f6} T_{fa},$$
$$T_{fa}(t) \geq T_{fa}(0)\, e^{-\mu_{f6} t}.$$

At $t = t_6$,

$$T_{fa}(t_6) \geq T_{fa}(0)\, e^{-\mu_{f6} t_6} > 0. \tag{4.8}$$

Equation 4.8 contradicts the fact that $T_{fa}(t_6) = 0$. Thus, $T_{fa}(t) \not< 0 \in [0, t_6]$ and $T_{fa}(t) \neq 0$. This implies that, $T_{fa}(t) > 0$.

Similar proofs of contradiction can be used to show that at the initial conditions $t_0 = t_7, t_8, t_9, t_{10}, t_{11}, t_{12}$, the model solutions for the male populations $S_{mu}(t) > 0$, $S_{ma}(t) > 0$, $I_{mu}(t) > 0$, $I_{ma}(t) > 0$, $T_{mu}(t) > 0$, $T_{ma}(t) > 0$ respectively. This contradiction is also true for large $\{t_i\}_{i=1,2,...,12}$. Hence, there is no such $t_i < 0$ implying that the solutions for the model system (4.2) $S_{fu}(t)$, $S_{fa}(t)$, $I_{fu}(t)$, $I_{fa}(t)$, $T_{fu}(t)$, $T_{fa}(t)$, $S_{mu}(t)$, $S_{ma}(t)$, $I_{mu}(t)$, $I_{ma}(t)$, $T_{mu}(t)$, $T_{ma}(t) \geq 0$ $\forall t > 0$. This proves that the solutions of the model system (4.2) are restricted in the non-negative region $\forall t > 0$.

□

Boundedness

Theorem 4.2.2. *The model (4.2) solutions are uniformly bounded in a set*

$$\Omega = \left\{ (S_{fu}, S_{fa}, I_{fu}, I_{fa}, T_{fu}, T_{fa}, S_{mu}, S_{ma}, I_{mu}, I_{ma}, T_{mu}, T_{ma}) \in \mathbb{R}_{12}^{+} \; | N(0) \leq N \leq \frac{\tilde{\Lambda}}{\mu_f + \mu_m} \right\}.$$

Proof. Given that system (4.2) is a finite dimensional dynamical system, its initial conditions and boundary conditions need to be constrained to Ω. Let
$(S_{fu}, S_{fa}, I_{fu}, I_{fa}, T_{fu}, T_{fa}, S_{mu}, S_{ma}, I_{mu}, I_{ma}, T_{mu}, T_{ma})$ be the solution to (4.2) and $S_{fu}(0) = S_{fu}^0 \geq 0$, $S_{fa}(0) = S_{fa}^0 \geq 0$, $I_{fu}(0) = I_{fu}^0 \geq 0$, $I_{fa}(0) = I_{fa}^0 \geq 0$, $T_{fu}(0) = T_{fu}^0 \geq 0$, $T_{fa}(0) = T_{fa}^0 \geq 0$ $S_{mu}(0) = S_{mu}^0 \geq 0$, $S_{ma}(0) = S_{ma}^0 \geq 0$, $I_{mu}(0) = I_{mu}^0 \geq 0$, $I_{ma}(0) = I_{ma}^0 \geq 0$, $T_{mu}(0) = T_{mu}^0 \geq 0$, $T_{ma}(0) = T_{ma}^0 \geq 0$ be the initial conditions. Adding all equations of system

(4.2), yields

$$\begin{aligned}\dot{N} &= (\tilde{\Lambda}) - \bar{\mu}_f\, N_f - \bar{\mu}_m\, N_m - \delta_f\,(N_f - \tilde{N}_f) - \delta_m\,(N_m - \tilde{N}_m)\\ &\leq \tilde{\Lambda} - (\bar{\mu}_f + \delta_f)N_f - (\bar{\mu}_m + \delta_m)N_m - \delta_f\tilde{N}_f - \delta_m\tilde{N}_m\\ &\leq \tilde{\Lambda} - \tilde{\mu}N\end{aligned}$$

where $\tilde{\Lambda} = \Lambda_{fu} + \Lambda_{fa} + \Lambda_{mu} + \Lambda_{ma}$, $\tilde{N}_f = S_{fu} + S_{fa} + T_{fa}$, $\tilde{N}_m = S_{mu} + S_{ma} + T_{ma}$, $\tilde{\mu} = min(\bar{\mu}_f + \delta_f, \bar{\mu}_m + \delta_m)$. Thus, Ω is a compact attracting non-negatively invariant for positive starting-point values since $N(0) > 0$. This can easily be proved using the theory of differential inequality [55]. All solutions of (4.2) originating in $\mathbb{R}^{12}_+$ are confined in Ω. Let M be an upper bound for S_{fu}, S_{fa}, I_{fu}, I_{fa}, T_{fu}, T_{fa}, S_{mu}, S_{ma}, I_{mu}, I_{ma}, T_{mu}, T_{ma}. We then conclude that every solution originating from Ω stays in Ω and is bounded by M. □

Local existence and uniqueness

Lemma 4.2.1. *Let $x = (x_i)_{i=1,2,\ldots,12}$ and $f : \mathbb{R}_+ \times \mathbb{R}^{12} \to \mathbb{R}^{12}$ be continuous with respect to t, x and Lipschitz continuous. Let $f(t,x)$ be non negative for all $(t,x) \in \mathbb{R}_+ \times \mathbb{R}^{12}$, and $x_i = 0$. For every $x_0 \in \mathbb{R}^{12}_+$, there exists a positive constant T such that $\dot{x} = f(t,x), \quad x(t_0) = x_0$, has a unique, positive and existing solution whose value lies in the interval $[0,T)$ and in $\mathbb{R}^{12}_+$.*

Theorem 4.2.3. *The solution set $\{S_{fu}, S_{fa}, I_{fu}, I_{fa}, T_{fu}, T_{fa}, S_{mu}, S_{ma}, I_{mu}, I_{ma}, T_{mu}, T_{ma}\}$ of the model (4.2) exists, is unique and positive for $t > 0$.*

Remark

We can easily show the local existence and uniqueness of solutions of model system (4.2) using similar proof ideas given in section 3.3.3. By theorem 4.2.2, the solutions to (4.2) are uniformly bounded on $[0,T)$. By theorem 4.2.3, the solution of (4.2) exists for any finite time. Thus, for any positive initial data in $\mathbb{R}^{12}_+$, the model system (4.2) will possess a unique and positive solution in $\mathbb{R}^{12}_+$ and it will be contained in the feasible region, Ω.

Equilibria

To find equilibrium solutions we set right hand side of model system (4.2) to 0 and solve for the state variables which yields a unique disease-free equilibrium (DFE)

$$E^0 = (S^0_{fu}, S^0_{fa}, 0, 0, 0, 0, S^0_{mu}, S^0_{ma}, 0, 0, 0, 0)$$

and possibly an endemic equilibrium (EE)

$$E^* = (S^*_{fu}, S^*_{fa}, I^*_{fu}, I^*_{fa}, T^*_{fu}, T^*_{fa}, S^*_{mu}, S^*_{ma}, I^*_{mu}, I^*_{ma}, T^*_{mu}, T^*_{ma})$$

with

$$
\begin{cases}
S^0_{fu} = \dfrac{\Lambda_{fu}}{\mu_{f1}}, \quad S^0_{fa} = \dfrac{\Lambda_{fa}\,\mu_{f1} + \rho^f_{ht}\Lambda_{fu}}{\mu_{f1}\,\mu_{f2}}, \\
S^0_{mu} = \dfrac{\Lambda_{mu}}{\mu_{m1}}, \quad S^0_{ma} = \dfrac{\Lambda_{ma}\,\mu_{m1} + \rho^m_{ht}\Lambda_{mu}}{\mu_{m1}\,\mu_{m2}}, \\
S^*_{fu} = \dfrac{\Lambda_{fu}}{g_{02}\beta^*_{fu} + \mu_{f1}}, \; S^*_{fa} = \dfrac{\Lambda_{fa}}{\rho^{m1}_{ht}\beta_{fu}* + \mu_{f2}} + \dfrac{\rho^f_{ht}\,\Lambda_{fu}}{(\rho^{m1}_{ht}\beta^*_{fu} + \mu_{f2})(g_{02}\,\beta^*_{fu} + \mu_{f1})}, \\
I^*_{fu} = \dfrac{\Lambda_{fu}\,\beta^*_{fu}}{\mu_{f3}\,(g_{02}\beta^*_{fu} + \mu_{f1})}, \; I^*_{fa} = \dfrac{q_{02}\,\beta^{*2}_{fu} + q_{03}\,\beta^*_{fu} + q_{04}}{q_{05}\,\beta^{*2}_{fu} + q_{06}\,\beta^*_{fu} + q_{07}}, \; T^*_{fu} = g_{01}\,I^*_{fa}, \; T^*_{fa} = g_{00}\,I^*_{fa}, \\
S^*_{mu} = \dfrac{\Lambda_{mu}}{g_{08}\beta^*_{mu} + \mu_{m1}}, \; S^*_{ma} = \dfrac{\Lambda_{ma}}{\rho^{f1}_{ht}\beta_{mu}* + \mu_{m2}} + \dfrac{\rho^m_{ht}\,\Lambda_{mu}}{(\rho^{f1}_{ht}\beta^*_{mu} + \mu_{m2})(g_{08}\,\beta^*_{mu} + \mu_{m1})}, \\
I^*_{mu} = \dfrac{\Lambda_{mu}\,\beta^*_{mu}}{\mu_{m3}\,(g_{08}\beta^*_{mu} + \mu_{m1})}, \; I^*_{ma} = \dfrac{h_{02}\,\beta^{*2}_{mu} + h_{03}\,\beta^*_{mu} + h_{04}}{h_{05}\,\beta^{*2}_{mu} + h_{06}\,\beta^*_{mu} + h_{07}}, \; T^*_{mu} = g_{07}\,I^*_{ma}, \\
T^*_{ma} = g_{06}\,I^*_{ma}, \; N^*_f = \dfrac{\Lambda_{fu} + \Lambda_{fa} + \delta_f\,\tilde{N}^*_f}{\bar{\mu}_f + \delta_f}, \; \tilde{N}^*_f = S^*_{fu} + S^*_{fa} + T^*_{fa}, \\
N^*_m = \dfrac{\Lambda_{mu} + \Lambda_{ma} + \delta_m\,\tilde{N}^*_m}{\bar{\mu}_m + \delta_m}, \; \tilde{N}^*_m = S^*_{mu} + S^*_{ma} + T^*_{ma}, \\
\beta^{*5}_{fu} + C_1\,\beta^{*4}_{fu} + C_2\,\beta^{*3}_{fu} + C_3\,\beta^{*2}_{fu} + C_4\,\beta^*_{fu} - C_5 = 0, \\
\beta^{*5}_{mu} + C_{11}\,\beta^{*4}_{mu} + C_{21}\,\beta^{*3}_{mu} + C_{31}\,\beta^{*2}_{mu} + C_{41}\,\beta^*_{mu} - C_{51} = 0.
\end{cases}
\tag{4.9}
$$

Refer to A.26 for the expressions of g_{00}, $g_{01}, ..., g_{11}$, q_{01}, $q_{02}, ..., q_{20}$, h_{01}, $h_{02}, ..., h_{20}$, C_1, $C_2, ..., C_5$ and C_{11}, $C_{21}, ..., C_{51}$.

By the fundamental theorem of algebra, the polynomial equations $\beta^{*5}_{fu} + C_1\,\beta^{*4}_{fu} + C_2\,\beta^{*3}_{fu} + C_3\,\beta^{*2}_{fu} + C_4\,\beta^*_{fu} - C_5 = 0$ and $\beta^{*5}_{mu} + C_{11}\,\beta^{*4}_{mu} + C_{21}\,\beta^{*3}_{mu} + C_{31}\,\beta^{*2}_{mu} + C_{41}\,\beta^*_{mu} - C_{51} = 0$, of odd degree, have at least one real root each. By Descartes' rule of signs, the polynomial equations will each have at least one non-negative real root if and only if $C_1 > 0$, $C_2 > 0$, $C_3 > 0$, $C_4 > 0$, $C_5 > 0$ and $C_{11} > 0$, $C_{21} > 0$, $C_{31} > 0$, $C_{41} > 0$, $C_{51} > 0$, given that the sign before C_5 and C_{51} is negative and the sign before β^{*5}_{fu} and β^{*5}_{mu} is non-negative otherwise each of the polynomial equation will have at most four (4) non-negative real roots. The exact number of non-negative roots can be determined using Descartes' rule of signs and Euclid's algorithm of the Sturm's theorem.

4.2.5 Control Reproduction Number,$\mathcal{R}_c$

The control reproduction number, $\mathcal{R}_c$, is defined as the expected number of secondary infections produced by a typical infected individual during its entire period of infectiousness in a population that is not entirely susceptible due to the presence of control measures [56]. The control measures present in our model are HIV testing (ρ_{ht}), condom use (ρ_c) and ART adherence (ρ_t).

The global dynamics for many disease models is determined by the sharp threshold criterion given by the basic reproduction number and this is true for our model system (4.2) [57]. Model system (4.2) possesses a sharp threshold if the control reproduction number $\mathcal{R}_c$ given by equation 4.13 is

such that E^0 is globally attractive for $\mathcal{R}_c \leq 1$ and there is a unique endemic equilibrium E^* that is globally attractive in the feasible region for $\mathcal{R}_c > 1$. Biologically, $\mathcal{R}_c$ is used to measure the transmission potential of the HIV/AIDS disease among the AGYW and ABYM in the presence of the said control measures [57]. The threshold property states that if $\mathcal{R}_c > 1$, HIV/AIDS disease persists in the youthful population hence becoming endemic whereas when $\mathcal{R}_c < 1$, the disease mirrors the effects of successful combination control measures to the AGYW and ABYM consequently protecting the susceptible youth from acquiring new HIV/AIDS infection.

The next generation matrix approach is used to compute the control reproduction number for the model system (4.2) [57]. Consider the infected subsystem of the model system (4.2) given as

$$\begin{cases} \dfrac{dI_{fu}}{dt} = \beta_{fu}\, S_{fu} - \mu_{f3}\, I_{fu}, \\ \dfrac{dI_{fa}}{dt} = \tilde{\beta}_{fa}\, S_{fa} + \beta_{fa}\, S_{fu} + \rho^f_{ht}\, I_{fu} - \mu_{f4}\, I_{fa}, \\ \dfrac{dT_{fu}}{dt} = \rho^f_{ct}\, I_{fa} + \rho^f_{ct}\, T_{fa} - \mu_{f5}\, T_{fu}, \\ \dfrac{dI_{mu}}{dt} = \beta_{mu}\, S_{mu} - \mu_{m3}\, I_{mu}, \\ \dfrac{dI_{ma}}{dt} = \tilde{\beta}_{ma}\, S_{ma} + \beta_{ma}\, S_{mu} + \rho^m_{ht}\, I_{mu} - \mu_{m4}\, I_{ma}, \\ \dfrac{dT_{mu}}{dt} = \rho^m_{ct}\, I_{ma} + \rho^m_{ct}\, T_{ma} - \mu_{m5}\, T_{mu}. \end{cases} \tag{4.10}$$

The right hand side of the infected subsystem (4.10) is decomposed into two parts, F and V where F denotes the transmission part and each F_i represents new infection. V denotes the transition part and each V_i describes change in state for instance removal through natural deaths, disease induced deaths, aging, HIV/AIDS status knowledge, condom use and ART adherence [58].

$$F = \begin{bmatrix} \left(\dfrac{c_f \gamma_f}{N_m}\left[I_{mu} + \alpha^m_c \rho^m_c I_{ma} + (\alpha^m_c \rho^m_c + \alpha^m_t \rho^m_t) T_{mu}\right]\right) S_{fu} \\ \rho^m_{ht}\left(\dfrac{c_f \gamma_f}{N_m}\left[I_{mu} + \alpha^m_c \rho^m_c I_{ma} + (\alpha^m_c \rho^m_c + \alpha^m_t \rho^m_t) T_{mu}\right]\right)(S_{fu} + \alpha^m_{ht} S_{fa}) \\ 0 \\ \left(\dfrac{c_m \gamma_m}{N_f}\left[I_{fu} + \alpha^f_c \rho^f_c I_{fa} + (\alpha^f_c \rho^f_c + \alpha^f_t \rho^f_t) T_{fu}\right]\right) S_{mu} \\ \rho^f_{ht}\left(\dfrac{c_m \gamma_m}{N_f}\left[I_{fu} + \alpha^f_c \rho^f_c I_{fa} + (\alpha^f_c \rho^f_c + \alpha^f_t \rho^f_t) T_{fu}\right]\right)(S_{mu} + \alpha^f_{ht} S_{ma}) \\ 0 \end{bmatrix}$$

and

$$V = -\begin{bmatrix} -\mu_{f3}\, I_{fu} \\ \rho^{f}_{ht}\, I_{fu} - \mu_{f4}\, I_{fa} \\ \rho^{f}_{ct}\, I_{fa} + \rho^{f}_{ct}\, T_{fa} - \mu_{f5}\, T_{fu} \\ -\mu_{m3}\, I_{mu} \\ \rho^{m}_{ht}\, I_{mu} - \mu_{m4}\, I_{ma} \\ \rho^{m}_{ct}\, I_{ma} + \rho^{m}_{ct}\, T_{ma} - \mu_{m5}\, T_{mu} \end{bmatrix}.$$

$\mathcal{F}$ and $\mathcal{V}$ are computed as:

$$\mathcal{F} = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right] \; and \; \mathcal{V} = \left[\frac{\partial V_i(x_0)}{\partial x_j}\right] \tag{4.11}$$

where x_0 is the disease free state. Evaluating $\mathcal{F}\mathcal{V}^{-1}$ yields the next generation matrix for the model system (4.2) whose largest non-negative eigenvalue is the reproduction number, $\mathcal{R}_c$. $\mathcal{F}\mathcal{V}^{-1}$ and $\mathcal{R}_c$ are given as follows:

$$\mathcal{F}\mathcal{V}^{-1} = \begin{bmatrix} 0 & 0 & 0 & \omega_1\,\eta_1 & \omega_1\,\eta_2 & \omega_1\,\eta_3 \\ 0 & 0 & 0 & \omega_2\,\eta_1 & \omega_2\,\eta_2 & \omega_2\,\eta_3 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \omega_3\,\varepsilon_1 & \omega_3\,\varepsilon_2 & \omega_3\,\varepsilon_3 & 0 & 0 & 0 \\ \omega_4\,\varepsilon_1 & \omega_4\,\varepsilon_2 & \omega_4\,\varepsilon_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \tag{4.12}$$

$$\mathcal{R}_c = \sqrt{\mathcal{R}_{uf}\,\mathcal{R}_{um} + \mathcal{R}_{af}\,\mathcal{R}_{am} + \mathcal{R}_{uf}\,\mathcal{R}_{am} + \mathcal{R}_{af}\,\mathcal{R}_{um}} \tag{4.13}$$

with

$$
\begin{cases}
\mathcal{R}_{uf} = \omega_1\,\epsilon_1, \quad \mathcal{R}_{um} = \omega_3\,\eta_1, \quad \mathcal{R}_{af} = \omega_2\,\epsilon_2, \quad \mathcal{R}_{am} = \omega_4\,\eta_2, \\
\mathcal{R}_u = \mathcal{R}_{uf}\,\mathcal{R}_{um}, \quad \mathcal{R}_a = \mathcal{R}_{af}\,\mathcal{R}_{am}, \quad \mathcal{R}_{mm} = \mathcal{R}_{uf}\,\mathcal{R}_{am}, \quad \mathcal{R}_{mf} = \mathcal{R}_{af}\,\mathcal{R}_{um}, \\
\omega_1 = \dfrac{c_f\,\gamma_f\,S^0_{fu}}{S^0_{mu}+S^0_{ma}}, \quad \omega_2 = \dfrac{\rho^m_{ht}\,c_f\,\gamma_f\,(S^0_{fu}+\alpha^m_{ht}\,S^0_{fa})}{S^0_{mu}+S^0_{ma}}, \\
\omega_3 = \dfrac{c_m\,\gamma_m\,S^0_{mu}}{S^0_{fu}+S^0_{fa}}, \quad \omega_4 = \dfrac{\rho^f_{ht}\,c_m\,\gamma_m\,(S^0_{mu}+\alpha^f_{ht}\,S^0_{ma})}{S^0_{fu}+S^0_{fa}}, \\
\eta_1 = \dfrac{1}{\mu_{m3}} + \dfrac{\alpha^m_c\rho_c\rho^m_{ht}}{\mu_{m3}\,\mu_{m4}} + \dfrac{(\alpha^m_c\rho_c+\alpha^m_t\rho_t)\,\rho^m_{ct}\rho^m_{ht}}{\mu_{m3}\,\mu_{m4}\,\mu_{m5}}, \\
\eta_2 = \dfrac{\alpha^m_c\rho_c}{\mu_{m4}} + \dfrac{(\alpha^m_c\rho_c+\alpha^m_t\rho_t)\,\rho^m_{ct}}{\mu_{m4}\,\mu_{m5}}, \quad \eta_3 = \dfrac{(\alpha^m_c\rho_c+\alpha^m_t\rho_t)}{\mu_{m5}}, \\
\varepsilon_1 = \dfrac{1}{\mu_{f3}} + \dfrac{\alpha^f_c\rho_c\rho^f_{ht}}{\mu_{f3}\,\mu_{f4}} + \dfrac{\left(\alpha^f_c\rho_c+\alpha^f_t\rho_t\right)\rho^f_{ct}\rho^f_{ht}}{\mu_{f4}\,\mu_{f3}\,\mu_{f5}}, \\
\varepsilon_2 = \dfrac{\alpha^f_c\rho_c}{\mu_{f4}} + \dfrac{\left(\alpha^f_c\rho_c+\alpha^f_t\rho_t\right)\rho^f_{ct}}{\mu_{f4}\,\mu_{f5}}, \quad \varepsilon_3 = \dfrac{\left(\alpha^f_c\rho_c+\alpha^f_t\rho_t\right)}{\mu_{f5}}.
\end{cases}
\tag{4.14}
$$

$\mathcal{R}_{uf}$, $\mathcal{R}_{um}$ gives the average number of the newly infected unaware AGYW and ABYM whereas $\mathcal{R}_{af}$, $\mathcal{R}_{am}$ gives the average number of the newly infected aware AGYW and ABYM. Newly infected youth generated by individuals with same status is given by $\mathcal{R}_{uf}\,\mathcal{R}_{um}$ and $\mathcal{R}_{af}\,\mathcal{R}_{am}$ whereas newly infected youth generated by mixed status interaction is given by $\mathcal{R}_{uf}\,\mathcal{R}_{am}$ and $\mathcal{R}_{af}\,\mathcal{R}_{um}$. In the absence of HIV testing, condom use and ART control, the control reproduction number $\mathcal{R}_c$ reduces to the basic reproduction number $\mathcal{R}_0$ and this is given as:

$$
\mathcal{R}_0 = \sqrt{\mathcal{R}_{0f}\,\mathcal{R}_{0m}} \tag{4.15}
$$

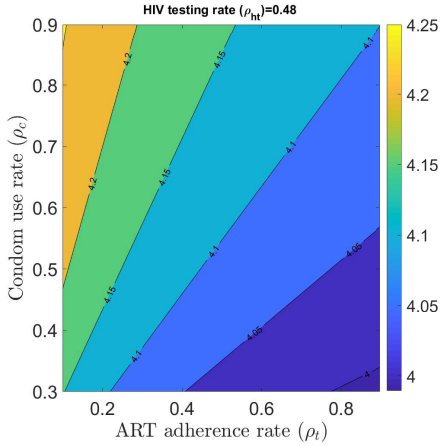
with

$$
\mathcal{R}_{0f} = \frac{c_f\,\gamma_f\,S^0_{fu}}{\mu_{f3}\,(S^0_{mu}+S^0_{ma})} \quad \text{and} \quad \mathcal{R}_{0m} = \frac{c_m\,\gamma_m\,S^0_{mu}}{\mu_{m3}(S^0_{fu}+S^0_{fa})}.
$$

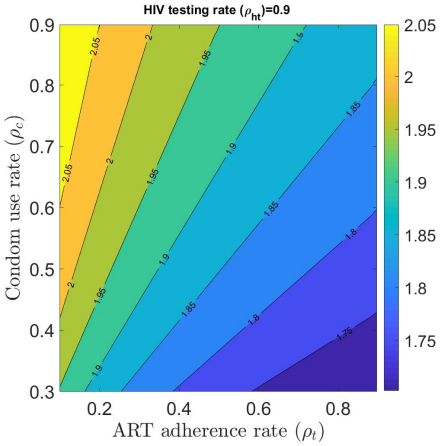
4.3 Results

4.3.1 Control Reproduction Number Simulations

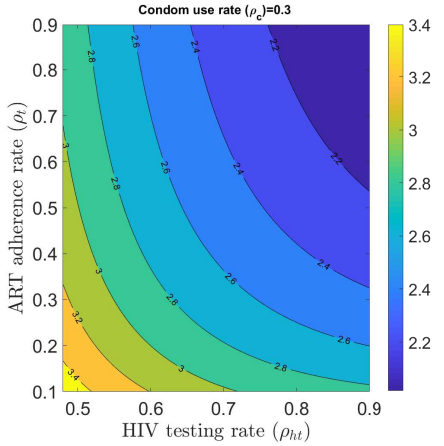
Using the parameter estimates for our model system given in Tables 4.6, 4.7 and 4.8, $\mathcal{R}_0$ is estimated at 20.4409 with $\mathcal{R}_{0f} = 22.9550$ and $\mathcal{R}_{0m} = 18.2021$. $\mathcal{R}_{0f} > \mathcal{R}_{0m}$ implies that the adolescent girls and young women have a greater susceptibility to HIV/AIDS infection compared to their male counterparts which is consistent with Kenyan youth HIV/AIDS disease dynamics [39]. The Kenyan reproduction number $\mathcal{R}_0$ was derived from early prevalence antenatal clinic data which was estimated at 6.34 [60]. The presence of combination control measures, however low, has played a key role in reducing new HIV infections among the youthful population with our model control reproduction number $\mathcal{R}_c$ estimated at 4.1003 when $\rho_{ht} = 0.48$, $\rho_c = 0.3$ and $\rho_t = 0.1$ and control attitude rates for the low control simulations given in Table 4.7.



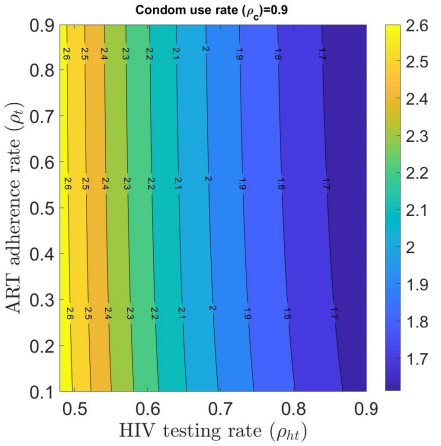
(a) Change in $\mathcal{R}_c$ with low ρ_{ht} and varying ρ_c and ρ_t



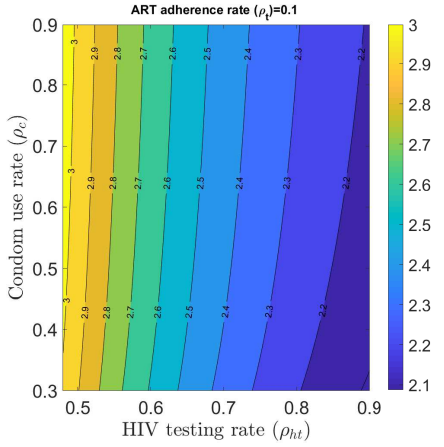
(b) Change in $\mathcal{R}_c$ with high ρ_{ht} and varying ρ_c and ρ_t



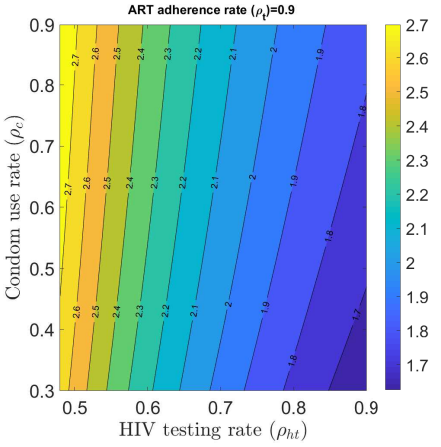
(c) Change in $\mathcal{R}_c$ with low ρ_c and varying ρ_{ht} and ρ_t



(d) Change in $\mathcal{R}_c$ with high ρ_c and varying ρ_{ht} and ρ_t



(e) Change in $\mathcal{R}_c$ with low ρ_t and varying ρ_{ht} and ρ_c



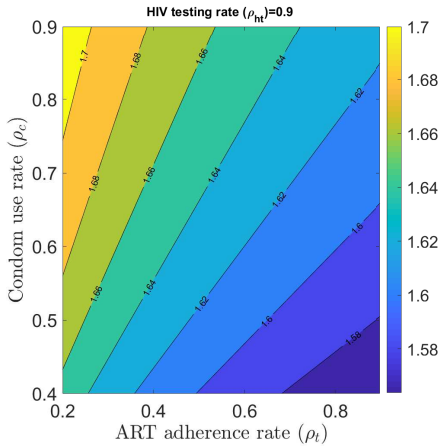
(f) Change in $\mathcal{R}_c$ with high ρ_t and varying ρ_{ht} and ρ_c

Figure 4.6: Change in the local control reproduction number $\mathcal{R}_c$ with varying ρ_{ht}, ρ_c and ρ_t.

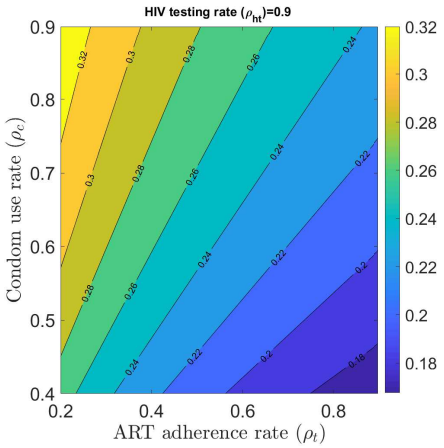
Figures 4.6(a) -4.6(f)) show the change in control reproduction number with fixed HIV/AIDS control measures and varying HIV/AIDS control measures. The control measures are varied from an estimated baseline rate to a 90% efficacy rate. Figures 4.6(a) -4.6(b)) show the change in the local control reproduction number when HIV testing is fixed at 0.48 and 0.9 respectively while condom use and ART adherence rates are varied from $0.3 - 0.9$ and $0.1 - 0.9$ efficacy rates. Similarly, figures 4.6(c) -4.6(d)) show the change in the local control reproduction number when condom use rate is fixed at 0.3 and 0.9 respectively while HIV testing and ART adherence rates are varied from $0.48 - 0.9$ and $0.1 - 0.9$ efficacy rates. Figures 4.6(e) -4.6(f)) show the change in the local control reproduction number when ART adherence is fixed at 0.1 and 0.9 respectively while HIV testing and condom use rates are varied from $0.48 - 0.9$ and $0.3 - 0.9$ efficacy rates.

Figures 4.6(b), 4.6(d) and 4.6(f) generally reflect the impact of reduced transmission potential of the control reproduction number when fixed control measures are at a high efficacy rate of 0.9. The greatest reduction in the control reproduction number is realized when HIV testing rate is fixed at 0.9 with condom use and ART adherence rates increasing from their respective baseline values to 0.9 efficacy rate (see figure 4.6(b)). This suggests that fixed higher HIV testing rates in all populations coupled with increased condom use and ART adherence rates work well to reduce the control reproduction number but not below unity for the Kenyan youth. This implies that the current sexual interactions among the various states will sustain the HIV epidemic even when efficacy rate of 90% is achieved.

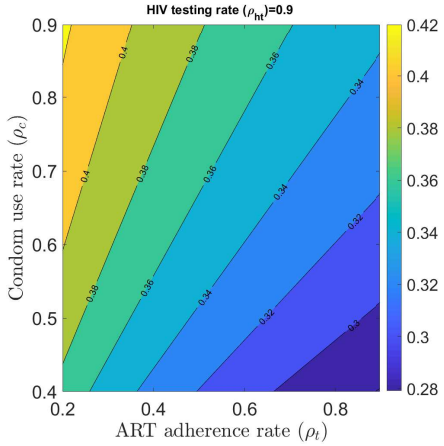
Taking the best scenario of reduced transmission potential of the control reproduction number described earlier, we unpack the unitary contributors to the control reproduction number to find the best case scenarios that could significantly reduce the control reproduction number (see figure 4.7). $\mathcal{R}_u$ contribution will sustain HIV/AIDS at endemic levels among the Kenyan youth population whereas $\mathcal{R}_a$ contribution will result in significant disease reduction among the AGYW and ABYM populations (see figures 4.7(a), 4.7(b)). Further, any interaction between aware male/female youth with unaware male/female youth yields good result that could lead to significant disease reduction among the Kenyan youth (see figures 4.7(c), 4.7(d)). Mixed status sexual interaction brings the control reproduction number down in our model as a result of HIV/AIDS status disclosure by the aware AGYW/ABYM. Any sexual relationship fostered with HIV/AIDS tested youth using condoms and adherent to ART promises hope for new HIV/AIDS infection reduction among the Kenyan youth.



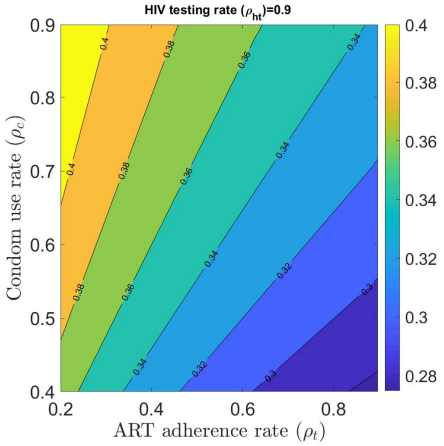
(a) Change in $\mathcal{R}_u$ with high ρ_{ht} and varying ρ_c and ρ_t



(b) Change in $\mathcal{R}_a$ with high ρ_{ht} and varying ρ_c and ρ_t



(c) Change in $\mathcal{R}_{mf}$ with high ρ_{ht} and varying ρ_c and ρ_t



(d) Change in $\mathcal{R}_{mm}$ with high ρ_{ht} and varying ρ_c and ρ_t

Figure 4.7: Change in $\mathcal{R}_u$,$\mathcal{R}_a$, $\mathcal{R}_{mf}$ and $\mathcal{R}_{mm}$ with fixed $\rho_{ht} = 0.9$ and varying ρ_c and ρ_t.

4.3.2 Data Fitting and Parameter Estimation

The UNAIDS Kenyan data for HIV/AIDS prevalence was used to fit the AGYW and ABYM model prevalence for both the sex-structured formulation described in section 4.2.2 and the single-sex formulation given in section 4.8. We considered the gender-wise annual HIV prevalence data for the years 1990 to 2018. Table 4.3 gives the UNAIDS HIV prevalence data summary for the AGYW and ABYM populations respectively [3].

Table 4.3: 1990-2001 AGYW and ABYM UNAIDS-Kenya's Prevalence Data [3]

Year	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000	2001
AGYW Prevalence	6.0	7.6	9.0	10.0	10.6	10.7	10.3	9.6	8.8	7.9	6.9	6.1
ABYM Prevalence	3.3	3.8	4.3	4.5	4.5	4.3	3.9	3.5	3.0	2.6	2.3	2.0

Table 4.4: 2002-2013 AGYW and ABYM UNAIDS-Kenya's Prevalence Data [3]

Year	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013
AGYW Prevalence	5.4	4.8	4.3	4.0	3.7	3.5	3.4	3.3	3.2	3.1	3.0	3.0
ABYM Prevalence	1.7	1.6	1.4	1.4	1.4	1.4	1.4	1.5	1.5	1.5	1.6	1.6

Table 4.5: 2014-2018 AGYW and ABYM UNAIDS-Kenya's Prevalence Data [3]

Year	2014	2015	2016	2017	2018
AGYW Prevalence	2.9	2.8	2.7	2.6	2.5
ABYM Prevalence	1.6	1.6	1.6	1.6	1.5

We define the AGYW and ABYM model prevalence as follows:

$$\text{AGYW Model Prevalence} = \frac{\text{Total number of infected AGYW}}{\text{Total AGYW population}} = \frac{I_{fu} + I_{fa} + T_{fu}}{N_f}, \tag{4.16}$$

$$\text{ABYM Model Prevalence} = \frac{\text{Total number of infected ABYM}}{\text{Total ABYM population}} = \frac{I_{mu} + I_{ma} + T_{mu}}{N_m}. \tag{4.17}$$

The AGYW and ABYM model prevalence described in equations 4.16 and 4.17 are fitted to the UNAIDS HIV prevalence data given in Table 4.3 to estimate parameters in Tables 4.6 and 4.7. Using MATLAB built in functions 'ODE45' and 'fminsearch' we estimated the parameters in Tables 4.6 and 4.7 by minimizing the sum of square difference of the AGYW and ABYM model prevalence solution and the HIV prevalence data for the AGYW and ABYM populations given in equations 4.18 and 4.19 as

$$SS^f = \sum_{k=1}^{29} \left(\frac{\left[\frac{I_{fu}^k + I_{fa}^k + T_{fu}^k}{S_{fu}^k + S_{fa}^k + I_{fu}^k + I_{fa}^k + T_{fu}^k + T_{fa}^k} - \tilde{Q}_1^k \right]^2}{\left[Max(\tilde{Q}_2^k, \tilde{Q}_3^k) \right]^2} \right), \tag{4.18}$$

$$SS^m = \sum_{k=1}^{29} \left(\frac{\left[\frac{I_{mu}^k + I_{ma}^k + T_{mu}^k}{S_{mu}^k + S_{ma}^k + I_{mu}^k + I_{ma}^k + T_{mu}^k + T_{ma}^k} - \tilde{Q}_4^k \right]^2}{\left[Max(\tilde{Q}_5^k, \tilde{Q}_6^k) \right]^2} \right). \tag{4.19}$$

To estimate parameters with little uncertainty, the 'fminsearch' algorithm in MATLAB software computes the goodness of fit by calculating the minimum sum of squares due to error (SSE). The minimum value of sum of squares due to error that is closer to 0 implies that the model has a smaller random error component and the resulting fit can be used for prediction [108]. This approach of fitting has also been used successfully elsewhere [109, 110, 111]. The higher the minimum value of SSE, the greater the variation from the prevalence data. For our model fit, the SSE prevalence fit for the AGYW was found to be 0.0167 whereas that for the ABYM was 0.0450. Given that the minimum SSE values we obtained are close to 0, the estimated parameters can be trusted and used for the time series model simulations.

The time length for the years 1990 to 2018 is given as k with $\tilde{Q}_1^k$, $\tilde{Q}_4^k$ being the yearly AGYW/ABYM UNAIDS prevalence data, $\tilde{Q}_2^k$, $\tilde{Q}_5^k$ the maximum yearly AGYW/ABYM model prevalence solutions and $\tilde{Q}_3^k$, $\tilde{Q}_6^k$ the maximum yearly AGYW/ABYM UNAIDS prevalence data. S_{fu}^k, S_{fa}^k, I_{fu}^k, I_{fa}^k, T_{fu}^k, T_{fa}^k, S_{mu}^k, S_{ma}^k, I_{mu}^k, I_{ma}^k, T_{mu}^k, T_{ma}^k are numerically computed solutions at each time k.

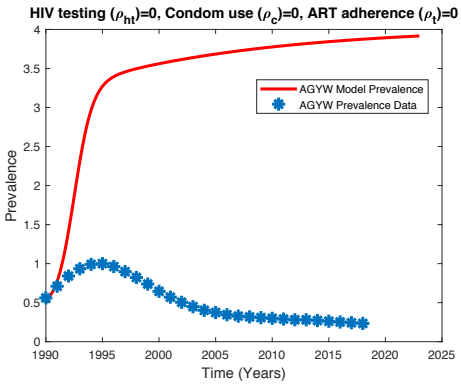
Attitudes affecting efficacy of HIV testing rate ρ_{ht}, condom use rate ρ_c and ART adherence rate ρ_t negatively α_{ht}^f, α_c^f, α_t^f, α_{ht}^m, α_c^m, α_t^m and positively α_{ht}^{f1}, α_c^{f1}, α_t^{f1}, α_{ht}^{m1}, α_c^{m1}, α_t^{m1} are estimated whereas the exit parameter σ is calculated as $1/24$ given that the AGYW and ABYM exit the model at the age of 24 years. The best parameters estimated by model fitting and calculated parameter are given in Tables 4.6 and 4.7 with $\tilde{\gamma}_f = c_f\,\gamma_f$ and $\tilde{\gamma}_m = c_m\,\gamma_m$.

Table 4.6: Parameter Values

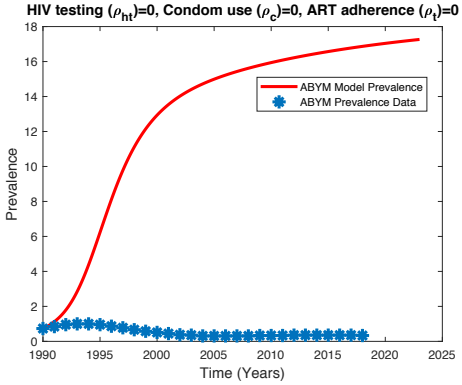
Parameter	Value	Unit	Source
Λ_{mu}, Λ_{ma}	60.7685, 100.9858	$year^{-1}$	Data Estimated
μ_m	0.0101	$year^{-1}$	Data Estimated
$\tilde{\gamma}_m$	2.617	$year^{-1}$	Data Estimated
δ_m	0.0090	$year^{-1}$	Data Estimated
Λ_{fu}, Λ_{fa}	61.1842, 118.1215	$year^{-1}$	Data Estimated
μ_f	0.0004	$year^{-1}$	Data Estimated
$\tilde{\gamma}_f$	3.97580754	$year^{-1}$	Data Estimated
δ_f	0.0285	$year^{-1}$	Data Estimated
σ	0.041667	$year^{-1}$	Calculated
ρ_{ht}	0.48	$year^{-1}$	Data Estimated
ρ_c	0.3	$year^{-1}$	Data Estimated
ρ_t	0.1	$year^{-1}$	Data Estimated

We used the 2012 KAIS data described in section 4.2.1 to estimate the initial population for the state variables $S_{fu}(0) = 636$, $S_{fa}(0) = 1006$, $T_{fa}(0) = 5$, $S_{mu}(0) = 694$, $S_{ma}(0) = 867$ and $T_{ma}(0) = 3$. We estimated the initial infected population for our model as $I_{fu}(0) = 54$, $I_{fa}(0) = 76$, $T_{fu}(0) = 10$, $I_{mu}(0) = 13$, $I_{ma}(0) = 26$ and $T_{mu}(0) = 5$.

In the absence of control measures, the Kenyan youth model prevalence trends increases with time (see figures 4.8(a), 4.8(b)). Interestingly, the ABYM model prevalence exceeds the AGYW model prevalence when intervention is absent (see figures 4.8(a), 4.8(b)). The Kenyan youth model prevalence without control measures only fits the initial rise of the HIV/AIDS epidemic.



(a) AGYW model prevalence with no control

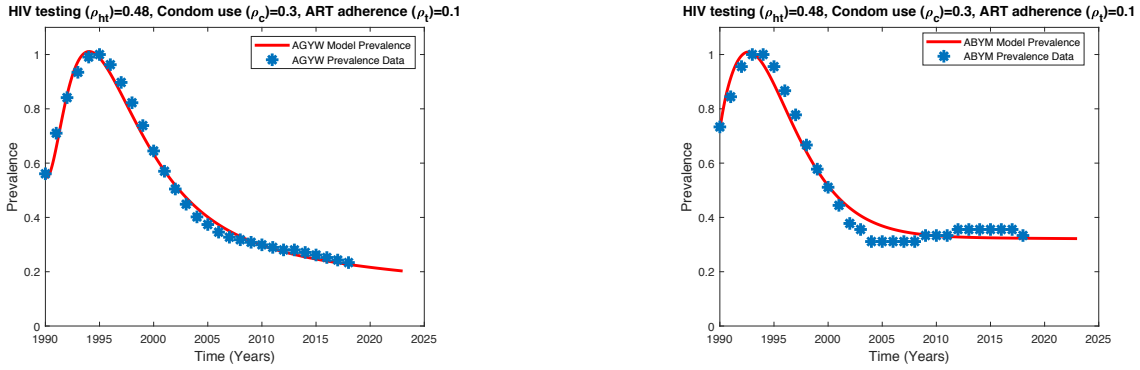


(b) ABYM model prevalence with no control

Figure 4.8: AGYW and ABYM model prevalence with no control fitted to UNAIDS AGYW and ABYM percent prevalence data respectively.

While the earliest cases of HIV/AIDS in Kenya were reported in the 1980's, it was only until

the late 1990's that the HIV/AIDS epidemic increased from 5.3% in 1990 to a peak prevalence of 10.5% in the years 1995-1996 and by 2003, the HIV/AIDS prevalence had declined to about 6.7% [7]. A combination of factors such as higher mortality rates, sexual behaviour change, lower incidences, delay in sexual debut among others contributed to the dramatic decline in Kenya's HIV/AIDS epidemic [7]. It is possible that even the Kenyan youth adopted safer sexual behaviors including condom use, reduction of multiple sexual partners and delay in first sex. Thus, fitting the AGYW and ABYM model prevalence to the Kenyan youth UNAIDS HIV/AIDS data subject to the estimated HIV testing, condom use and ART adherence control measures with disproportional AGYW/ABYM attitudes affecting the mentioned control measures efficacy resulted in a good fit (see figures 4.9(a), 4.9(b)).



(a) AGYW model prevalence with low control

(b) ABYM model prevalence with low control

Figure 4.9: AGYW and ABYM model prevalence with low control fitted to UNAIDS AGYW and ABYM percent prevalence data respectively.

AGYW HIV/AIDS model prevalence fits well to the Kenyan UNAIDS female youth HIV/AIDS prevalence when negative attitudes towards HIV testing, condom use and ART adherence are lower in AGYW population at 18% and higher in ABYM population at 30% with positive attitudes towards the three HIV/AIDS control measures greater in AGYW population at 86% compared to ABYM population which is at 69%. Similarly, ABYM model prevalence fits well when negative attitudes towards HIV/AIDS control measures are greater in AGYW population at 33.7% and positive attitudes greater in ABYM population at 96%. Our results project a decrease in the AGYW prevalence trend from 2.5 in 2018 to about 2.17745 in 2023 (see Figures 4.4(a) and 4.9(a)). Similarly, our model predicts a decrease in the ABYM prevalence trend from 1.5 in 2018 to about 1.44855 in 2023 (see Figures 4.4(b) and 4.9(b)). These results hold assuming the control measures and the constant negative/positive attitudes towards the control measures remain the same.

We used the parameter values given in Table 4.6 to perform the numerical simulations for the model system (4.2) and the control reproduction number in section 4.2.5 with low control attitude rates given in Table 4.7

Table 4.7: Estimated negative/positive attitude rates towards HIV/AIDS controls for low control simulations

Parameter	Value	Unit	Source
α_{ht}^{m}, α_{c}^{m}, α_{t}^{m}	0.15, 0.36, 0.38	$year^{-1}$	Data Estimated
α_{ht}^{m1}, α_{c}^{m1}, α_{t}^{m1}	0.99, 0.95, 0.95	$year^{-1}$	Data Estimated
α_{ht}^{f}, α_{c}^{f}, α_{t}^{f}	0.25, 0.2, 0.1	$year^{-1}$	Data Estimated
α_{ht}^{f1}, α_{c}^{f1}, α_{t}^{f1}	0.97, 0.8, 0.8	$year^{-1}$	Data Estimated

and high control attitude rates given in Table 4.8.

Table 4.8: Estimated negative/positive attitude rates towards HIV/AIDS controls for high control simulations

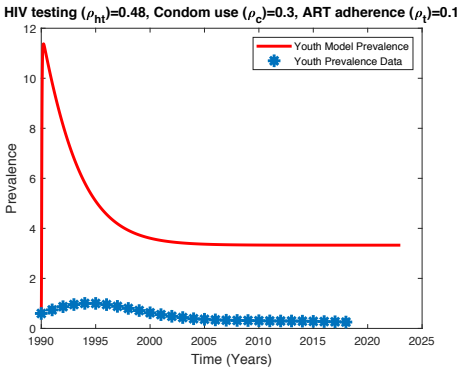
Parameter	Value	Unit	Source
α_{ht}^{m}, α_{c}^{m}, α_{t}^{m}	0.1, 0.1, 0.1	$year^{-1}$	Assumed
α_{ht}^{m1}, α_{c}^{m1}, α_{t}^{m1}	0.9, 0.9, 0.9	$year^{-1}$	Assumed
α_{ht}^{f}, α_{c}^{f}, α_{t}^{f}	0.1, 0.1, 0.1	$year^{-1}$	Assumed
α_{ht}^{f1}, α_{c}^{f1}, α_{t}^{f1}	0.9, 0.9, 0.9	$year^{-1}$	Assumed

Single-Sex Youth Model Fit

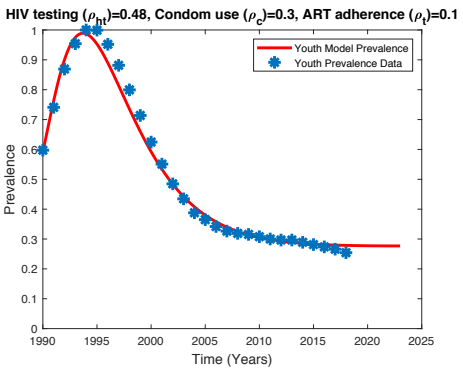
We considered the single-sex youth model given in model system (4.20) to understand factors influencing its model fit. The incidence rates β_u, β_a, $\tilde{\beta}_a$ and exit rates μ_1, μ_2, ..., μ_6 are given in equation A.27. See A.1 and A.2 for the single-sex model state variables and parameters description.

$$\begin{cases} \dfrac{dS_u}{dt} = \Lambda_u - \beta_u\, S_u - \beta_a\, S_u - \mu_1\, S_u, \\ \dfrac{dS_a}{dt} = \Lambda_a + \rho_{ht}\, S_u - \tilde{\beta}_a\, S_a - \mu_2\, S_a, \\ \dfrac{dI_u}{dt} = \beta_u\, S_u - \mu_3\, I_u, \\ \dfrac{dI_a}{dt} = \tilde{\beta}_a\, S_a + \beta_a\, S_u + \rho_{ht}\, I_u - \mu_4\, I_a, \\ \dfrac{dT_u}{dt} = \rho_{ct}\, I_a + \rho_{ct}\, T_a - \mu_5\, T_u, \\ \dfrac{dT_a}{dt} = \rho_{ct}^{1} I_a + \rho_{ct}^{1}\, T_u - \mu_6\, T_a. \end{cases} \tag{4.20}$$

We fitted the single-sex model to the averaged AGYW/ABYM UNAIDS-Kenya HIV/AIDS prevalence data given in Table 4.3. Using AGYW/ABYM averaged initial conditions in section 4.3.2 and parameter values given in A.3 yields the model fit given in figure 4.10(a). Adjusting the transmission risk and contact rates (see A.4) results in a good fit (see figure 4.10(b)).



(a) Single-sex model prevalence with high transmission risk and high contact rate



(b) Single-Sex model prevalence with reduced transmission risk and reduced contact rate

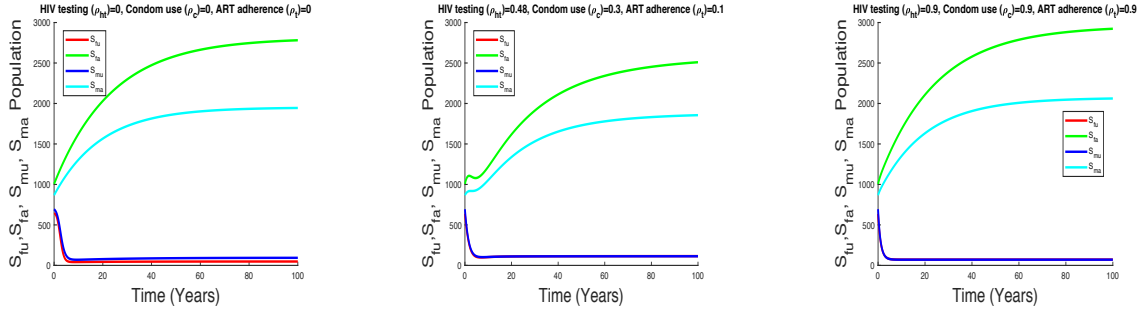
Figure 4.10: Single-sex model prevalence with varying transmission risk and contact rate fitted to averaged UNAIDS AGYW and ABYM percent prevalence data.

The single sex-structured model fits well to data (SSE=0.0232) when HIV testing rate, condom use rate and ART adherence rates are 0.48, 0.3 and 0.1 respectively with the product of probability of transmission risk (γ) and contact rate (c) reduced from 3.17245525 to 0.03022869. This seems to suggest that for the single-sex structured model, change in contact behavior could have influenced the change in HIV/AIDS prevalence trends among the youth. When we reduced the contact rate and probability of transmission risk in the sex-structured model, the resultant prevalence fit was poor and only a good fit was realized when the gender-wise attitudes towards HIV/AIDS control measures were disproportional. The sex-structured model further revealed that disproportional gender-wise attitudes towards HIV/AIDS control measures could have also influenced the Kenyan youth HIV/AIDS prevalence trends.

4.3.3 Model Simulations Results

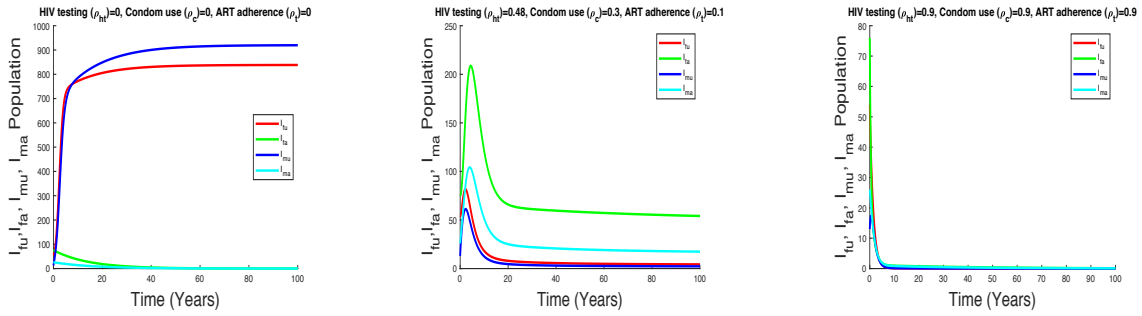
Numerical simulations on the model system equations (4.2) are carried out to test the AGYW and ABYM HIV/AIDS epidemic behavior. The 2020 UNAIDS 90-90-90 HIV/AIDS eradication plan aims to have at least 90% HIV testing coverage for all persons living with HIV with at least 90% initiated on ART achieving a 90% viral load suppression [16]. This informed the 90% HIV testing and ART efficacy rates for our high control simulations. Male condoms when used correctly and consistently in every sexual intercourse is estimated to have at least 90% efficacy against HIV/AIDS transmission whereas female condoms offer at least 94% protection [112]. In Kenya, male condoms are most preferred as described in section 5.1. Hence, we used the male condom efficacy of 90% to model high control cases. The baseline rates for HIV testing $\rho_{ht} = 0.48$, condom use $\rho_c = 0.3$ and ART adherence $\rho_t = 0.1$ were estimated by model fitting as described in section 4.3.2. Estimated constant negative/positive attitudes towards HIV/AIDS control measures for the low control and high control simulations are given in Tables 4.7 and 4.8 respectively.

Figures 4.11(a), 4.12(a), 4.13(a), 4.14(a) suggest that with time the Kenyan youth HIV/AIDS epidemic matures and attains stability without any intervention. However, the prevalence doesn't decline after attaining stability in the absence of HIV/AIDS control measures (see figure 4.14(a)). Low control use ($\rho_{ht} = 0.48$, $\rho_c = 0.3$, $\rho_t = 0.1$) with estimated attitude rates given in Table 4.7 seems to reduce the infected populations and the AGYW/ABYM model prevalence with better benefits in the ABYM population (see figures 4.12(b), 4.13(b), 4.14(b)).



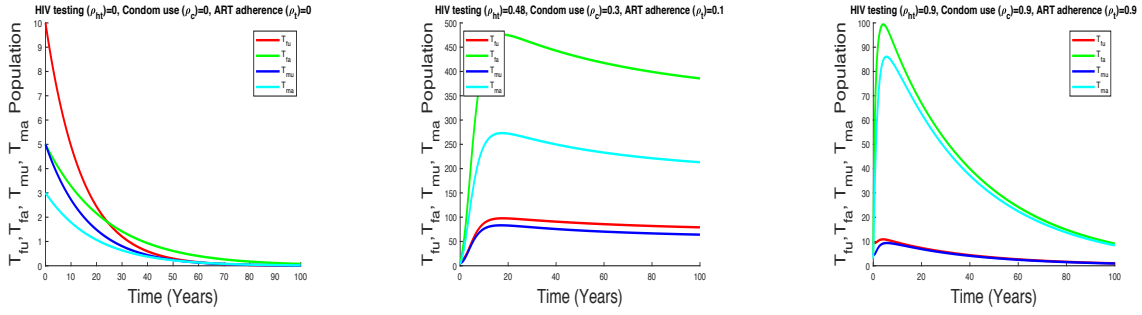
(a) (b) (c)

Figure 4.11: Transmission Dynamics of S_{fu}, S_{fa}, S_{mu} and S_{ma} populations with varying control.



(a) (b) (c)

Figure 4.12: Transmission Dynamics of I_{fu}, I_{fa}, I_{mu} and I_{ma} population with varying control.

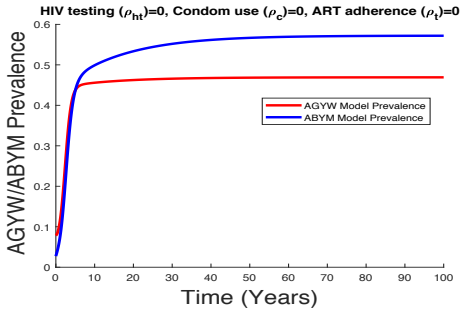


(a) (b) (c)

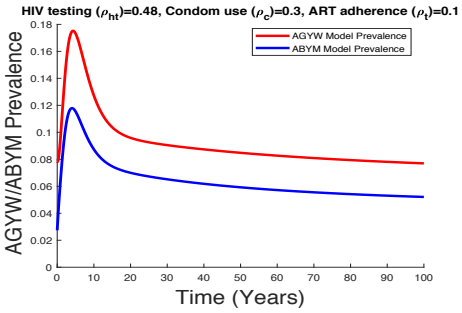
Figure 4.13: Transmission Dynamics of T_{fu}, T_{fa}, T_{mu} and T_{ma} population with varying control.

High control rates, $\rho_{ht} = 0.9$, $\rho_c = 0.9$, $\rho_t = 0.9$, with reduced negative control attitudes and in-

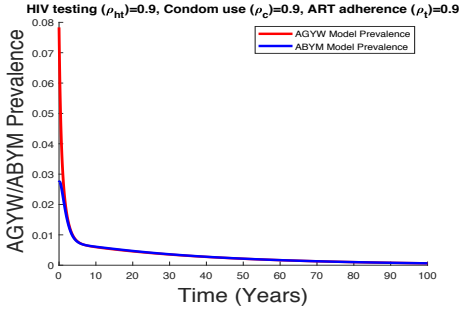
creased positive control attitudes in all populations seems to have a significant effect in HIV/AIDS disease decline among the AGYW and ABYM populations as the infected populations are reduced significantly with similar trends observed in the youth prevalence (see figures 4.12(c), 4.13(c), 4.14(c)). Interestingly, when the negative attitudes towards condom use and ART adherence among the AGYW and ABYM population are slightly increased when HIV/AIDS control measures are low, the youth HIV/AIDS model prevalence begins to increase despite the initial decline (see figure 4.14(d)).



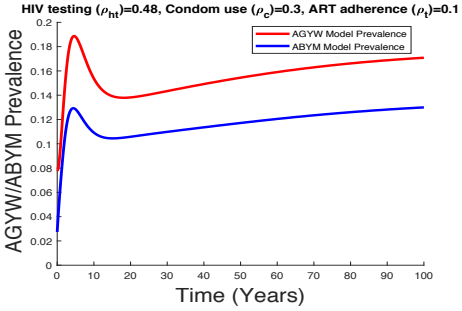
(a) No control



(b) Low control paired with Table 4.7 attitudes



(c) High control paired with Table 4.8 attitudes



(d) Low control with increased negative attitudes towards condom use and ART adherence

Figure 4.14: AGYW and ABYM model prevalence with varying control.

We investigated the effects of varying HIV testing rates, condom use rates and ART adherence rates among the adolescent girls and young women (AGYW) and, adolescent boys and young men (ABYM) populations aged 15-24. We considered constant negative and positive attitudes influencing the uptake of HIV/AIDS control measures in these populations. HIV testing rates, condom use rates and ART adherence rates were varied from their estimated low baseline rates of 0.48, 0.3, 0.1 respectively to the estimated efficacy rates of 0.9 each. Low control simulations were associated with increased constant negative attitudes towards HIV/AIDS control measures whereas high control simulations were associated with reduced negative attitudes towards HIV/AIDS control measures and increased constant positive attitudes towards HIV/AIDS control measures among the AGYW/ABYM populations and the Kenyan society/cultural groups. The susceptible and infected AGYW/ABYM populations were each differentiated into two broad categories according

to their HIV/AIDS status knowledge. That is, uninfected aware or uninfected unaware and infected aware or infected unaware. Infected aware populations were further differentiated into two categories based on their condom use and ART adherence. Unaware populations could change their status and move to aware populations through HIV testing, condom use or ART initiation. This model structure was largely informed by the 2012 Kenya AIDS Indicator Survey (KAIS) [102].

We fitted both the single-sex model and the sex-structured model to UNAIDS-Kenya HIV Surveillance prevalence data for the young males and young females aged 15-24. The sex-structured HIV/AIDS model prevalence seems to fit to each of their estimated UNAIDS-Kenya HIV/AIDS prevalence data reasonably well when negative/positive attitudes towards HIV/AIDS control measures were disproportional in the AGYW/ABYM populations whereas the single-sex model prevalence trend seemed sensitive to transmission risk and contact rate. The single sex-structured model suggests that reduced transmission risk and sexual contact rate in the presence of low control measures could have resulted in reduced HIV/AIDS prevalence among the youth in Kenya. The sex-structured model seemed to reveal further the effects of disproportional gender-wise attitudes towards HIV/AIDS control measures affecting uptake of control measures in the youth populations. Increased ABYM infectivity and reduced AGYW infectivity resulted in the female youth model good fit whereas increased AGYW infectivity and reduced ABYM infectivity resulted in the male youth good model fit. In addition to reduced transmission risk and contact rate, it seems that gender-wise attitudes towards HIV/AIDS control measures played a role in reducing HIV/AIDS prevalence among the youth in Kenya. The AGYW/ABYM model fit estimated the best parameters for model simulations.

Simulations on the control reproduction number revealed the impact of reduced transmission potential of the control reproduction number but not below unity when HIV testing rate was fixed at a high efficacy rate of 0.9 with increasing condom use and ART adherence to high efficacy rates. This was as a result of the complex sexual structure among the Kenyan youth with the HIV/AIDS disease being sustained at endemic levels by the unaware youth. The simulations suggest that significant HIV/AIDS reduction among the Kenyan youth will only be possible if for each sexual relationship established, there is at least one partner who is willing to disclose his/her HIV/AIDS status to his/her sex partner as well as use protection consistently. Numerical simulations on our model system revealed the impact of successful combination control approach in drastically reducing new HIV/AIDS infection. Low combination control approach has a positive effect in reducing youth disease prevalence with better benefits in the ABYM population provided the negative attitudes towards HIV/AIDS control are kept in check. Slight increase in negative attitudes towards AGYW/ABYM condom use or ART adherence can easily increase the youth disease prevalence even after the initial disease decline. Significant HIV/AIDS disease reduction is achieved only when positive attitudes towards HIV/AIDS control measures are increased in all AGYW/ABYM populations with decreasing negative attitudes.

4.4 Discussion

Globally, male and female youth are central in the HIV/AIDS action plans due to the high numbers of youth unaware of their HIV/AIDS status [18, 47]. The 2012 Kenya AIDS Indicator Survey (KAIS) also revealed a worrying trend of many infected male and female youth unaware of their HIV/AIDS status and this is consistent with the global trends [18, 102]. The social attitudes influencing HIV testing, condom use and ART adherence efficacy cannot be downplayed as they play a critical role in either fueling the HIV/AIDS epidemic or curtailing its spread in this population group as evidenced by the model results. The female youth HIV/AIDS prevalence trend seems to be associated with increased male infectivity with decreased female infectivity while the male youth prevalence trend seems to be associated with increased female infectivity and reduced male infectivity.

The annual increase of new HIV infections among the youth exceeds HIV/AIDS related deaths which in turn increases the net size of HIV/AIDS infected population in the country [2]. This remains a huge concern since, as the HIV/AIDS infected youth population continues to increase, the risk of HIV/AIDS transmission increases too. Kenya's HIV/AIDS response is quite dynamic and there is increased efforts in scaling up HIV testing, condom use and ART adherence among the AGYW and ABYM populations. Our model results reflect the importance of addressing the social attitudes inhibiting efficacy of HIV testing, condom use and ART adherence among the Kenyan youth. While combination control measures play a huge role in reducing HIV/AIDS prevalence trends among the youth in Kenya, the disease may still remain endemic provided the infected unaware populations' sexual interactions exist. Our results suggest that it is necessary to scale up HIV testing among the youth while at the same time addressing factors affecting its efficacy such as perceived individual's risk to HIV infection, HIV/AIDS knowledge, education, inadequate health services among others. It is also necessary to address the societal norms, psycho-social conditions, stigma, socio-cultural factors associated with condom use and ART adherence among the youth in Kenya. Their negative influence is possibly one of the significant drivers for the reversal of decades of successful control measures geared at reducing HIV/AIDS prevalence in Kenya.

The 2014/2015 – 2018/2019 Kenya AIDS Strategic Framework (KASF) by the Ministry of Health goal was to significantly reduce new HIV infections, AIDS-related mortality, HIV/AIDS related stigma and discrimination and, significantly increase domestic financing of HIV/AIDS response programmes [113]. KASF plan ties together with Kenya's 2030 vision of an economically transformed nation where health plays a key role in realizing this goal. HIV/AIDS epidemic in Kenya significantly increases the disease burden in the country and part of Kenya's Vision 2030 is to have a country free of HIV infections, HIV-related stigma and AIDS-related deaths. Despite the considerable progress in reducing new HIV infections among the youth in Kenya since the KASF initiation, challenges surrounding policy implementation and community response continue to affect effective HIV/AIDS response [114, 115]. The time series model predictions from our study suggested that Kenya's Vision 2030 of a country free of new HIV infections might not be realized given the low HIV/AIDS control measures and societal attitudes hampering the uptake

of HIV/AIDS control measures by the youth who are a priority population targeted in Kenya's HIV response.

Since Kenya became a low middle income country in 2014, the progress towards HIV/AIDS control slowed down [113]. The country's income status drastically reduced international donor support on HIV/AIDS policy implementation and monitoring of key prevention areas such as HIV testing, condom use and ART adherence [113, 115]. Reduced HIV/AIDS funding has significantly affected programs addressing social drivers of HIV/AIDS such as societal attitudes, which directly influence the uptake of HIV/AIDS control measures by the youth [114, 115]. Low funding has also affected Community Based Organizations and Community Leadership who play a key role in addressing the societal attitudes directly affecting the uptake of HIV/AIDS control measures hence, increasing the youths vulnerability to HIV infection [113]. Our study suggested that for significant reduction of new HIV infections and possible elimination of new HIV infections among the youth, key intervention areas such as HIV screening, condom use and ART adherence needs to be significantly increased and societal negative attitudes directly affecting the uptake of these control measures significantly reduced. Hence, it will be necessary to address challenges affecting HIV/AIDS funding and empower Community Based Organizations and Community Leadership so as to successfully combat the root cause (societal attitudes) affecting the uptake of HIV/AIDS control measures by the Kenyan youth.

As far as we know, there are no existing mathematical models that have addressed the impact of combination control measures and their influence among the youth HIV/AIDS disease dynamics in Kenya with differentiated HIV/AIDS status knowledge using two-sex structured models. Multiple control strategies such as HIV screening, ARV drug treatment and condom use in a single sex-structured model was considered by [25] to understand the potential impact on the current HIV/AIDS control measures. Their results reflected the projections of HIV/AIDS epidemic trends when HIV/AIDS control measures and multiple sex partners varied. Our work presented similar results using a single-sex structured model which further revealed the effects of transmission risk and contact rate in informing the Kenyan youth HIV/AIDS prevalence trends. The limitations of a single-sex structured model was evident in our work where the single-sex structured model could not fit the HIV/AIDS prevalence when control measures were influenced by gender-wise societal attitudes that were incorporated into the model. The two-sex structured model in this study resolved this weakness. The importance of two-sex model speaks to increased mathematical complexity but provided an appropriate tool to explain the associated drivers of the Kenyan youth HIV/AIDS dynamics.

Having studied the impact of combination control strategies and constant negative/positive attitudes influencing the efficacy of the HIV/AIDS control measures among the youth infected populations in a single patch model, it will be interesting to study the effects of combination control in a metapopulation model in Kenya given that this population group is highly mobile. Dynamic attitudes towards HIV/AIDS control measures should also be considered. We used the UNAIDS-Kenya HIV Surveillance data to fit our model which is not exempt from biases due to

insufficient nationally representative HIV/AIDS prevalence data. For accurate model fitting to national prevalence trends, nationally representative HIV/AIDS surveillance need to be increased so as to create a larger prevalence data pool. While this study focused on population dynamics of the AGYW/ABYM, it will be interesting to study the individual based model for this AGYW/ABYM formulation. Given the behavior heterogeneity among the youth, studying each individual behavior explicitly to population level could give deeper insights in understanding the social drivers of HIV among the Kenyan youth. This in turn will help influence relevant policies geared at eradicating new HIV infections among the youth in Kenya.

Chapter 5

Modeling the Spatial Effects of HIV/AIDS Combinatory Control among the Kenyan Youth in High Risk Counties

Keywords: Spatial, HIV/AIDS, Youth, HIV Testing, Condom Use, Antiretroviral therapy(ART), Combinatory control.

Abstract. We formulate a homogeneous deterministic model to study the impact of migration on HIV/AIDS disease dynamics among the youth in Homa Bay and Nairobi Counties in Kenya. Targeted HIV/AIDS controls such as HIV/AIDS testing, condom use and ART use coupled with County attitudes promoting or negating their efficacy were considered. Varying departure rates and return rates have little effect in increasing new HIV/AIDS infections in the two high risk Counties. Fewer HIV/AIDS tested youth who are aware of their HIV/AIDS status in Homa Bay and Nairobi Counties are reduced to new HIV/AIDS infection. Significant reduction of new HIV/AIDS infections among the mobile youth is possible if HIV/AIDS testing, condom use and ART adherence are highly efficacious and County attitudes on these targeted controls are highly positive. While migration has little effect on the overall increase of new HIV/AIDS infection in the considered high risk Counties, increase in new HIV/AIDS infections is propagated by sexual interactions with youth who are unaware of their HIV/AIDS status.

5.1 Introduction

A large proportion of Kenya's population are youth and children with about 7.9 million adolescent and young adults aged 15 - 24 years [63, 116]. The Country's HIV/AIDS epidemic is largely driven by sex workers, injecting drug users, homosexuals, discordant couples, truck drivers, prisoners and young people [117, 118, 119]. Adolescents and young adults health remain central in the country's development agenda dubbed 'Kenya's Vision 2030 Plan' [120]. Kenya's progress towards 'Zero New Infections', 'Zero AIDS Related Deaths' and 'Zero Discrimination' is commendable [2]. Increased investments in HIV/AIDS response has resulted in HIV/AIDS prevalence halved in just over a decade but, in 2015 the country's HIV/AIDS epidemic was still high at 5.9% [65].

In 2017, the National HIV/AIDS prevalence among the adolescent girls and young women (AGYW) was approximately 2.61% and about 1.34% among the adolescent boys and young men (ABYM) [2]. New HIV/AIDS infections in 2017 were estimated at 52,767 with adolescents and young adults aged 15-24 contributing 33% [2]. The burden of new HIV/AIDS infection in this population group was largely reported in Counties with leading HIV/AIDS incidence rates with 2,857 in Nairobi County, 1,852 in Homa Bay County, 1,641 in Siaya County, 1,630 in Kisumu County, 1,143 in Migori County, 730 in Kiambu County, 596 in Kakamega County and 562 reported in Mombasa County [2]. Sadly, HIV/AIDS related deaths is the leading cause of morbidity and mortality among the AGYW/ABYM in Kenya and about 9,720 HIV/AIDS related deaths in 2014 were adolescents and young adults aged 15-24 [47].

Evidence based interventions specific to Kenya's youth sub-populations such as orphans, sexually active adolescents, injecting drug users, homosexuals, survivors of sexual violence, disabled youth among others prioritize adolescents and young adults in high risk Counties such as Nairobi, Homa Bay, Siaya, Kisumu, Migori, Kiambu, Kakamega and Mombasa [47]. These interventions include retention of adolescents and young adults in school until completion of studies, sex and HIV education to delay sex debut, HIV/AIDS testing, condom use and reduced sexual partners, mass media and social marketing campaigns to aid in risky sexual behavior change, training health workers to offer adequate services to the adolescents and young adults among others [47].

Since the onset of HIV/AIDS epidemic globally, many researchers have formulated HIV/AIDS disease models to understand its dynamics. Few of these models considered spatial spread of HIV/AIDS disease. To model spatial spread, metapopulation modeling tool is used. A metapopulation is a group of populations that are separated by space, but consist of homogeneous species [56]. These spatially separated populations interact as individual members move from one population to another to create networks connected by routes of travel [56]. The main factor determining spatial spread is contact between infected and susceptible individuals from diverse geographic regions which eventually escalates the epidemic. Study by [33] showed that migration in different regions in Kenya increases HIV/AIDS incidences in a relatively small way. Similarly, [121] study suggested that mobility paired with risky sexual behaviour casually increases South Africa's HIV/AIDS pandemic whereas [122] metapopulation study advocated for equal resource distribution in all regions globally to eradicate HIV/AIDS epidemic. Results by [123] suggested that China's mainland HIV/AIDS epidemic could be decreased by mobility of HIV/AIDS infected individuals. Using individual based modeling, [124] considered the relationship of female sex workers mobility in Western Kenya and high HIV/AIDS prevalence. Standard HIV/AIDS deterministic formulations rarely consider heterogeneities such as socio-cultural, economic, demographic, geographical diversity among others that potentially play a role in driving the HIV/AIDS epidemic.

We seek to investigate varying effects of HIV/AIDS targeted interventions on rural to urban and urban to rural migrating adolescents and young adults in Kenya such as HIV testing, condom use and antiretroviral adherence. We consider constant factors influencing the targeted controls efficacy in high risk Kenyan Counties such as health services, community norms, culture, poverty among others. On one hand, some persistent risks faced by vulnerable adolescents and young adults in Kenya include unemployment, sexual violence, poverty, marginalization, inability to access health and education services forcing them to move to different regions in the country in search for a better life. On the other hand, privileged adolescents and young adults in Kenya would move to different parts of the country to advance their studies in higher centers of learning. We calibrate the model using 2012-2014 Kenya AIDS Indicator Survey (KAIS), 2018 HIV/AIDS prevalence estimates by National AIDS Control Council (NACC) and the most recent Kenyan Counties demographic data. Kenya's fast track plan to end HIV/AIDS among adolescents and young adults report recommends dispensing HIV/AIDS interventions to specific populations while prioritizing high risk geographical regions so as to work towards UNAIDS goal of ending new HIV/AIDS infections by 2030 [47, 125].

5.2 Model Construction

We construct a model describing HIV/AIDS transmission dynamics among the mobile adolescents and young adults population aged 15-24 in high risk Counties in Kenya specifically, Homa Bay and Nairobi Counties. Nairobi and Homa Bay high risk Counties are considered as these Counties

lead in new HIV/AIDS infections with 2,857 reported in Nairobi County and 1,852 reported in Homa Bay County [2]. The 2012 Kenya AIDS Indicator survey informed most of the state variables [102]. Section 5.1 highlights the need to model targeted populations such as adolescents and young adults while prioritizing targeted HIV/AIDS interventions in high risk regions.

Homa Bay County is set in the rural part of Kenya whereas Nairobi County is the Capital City of Kenya hence, urban [126, 127]. The main drivers of migration from Homa Bay County to Nairobi County are inadequate infrastructure, ill-equipped health facilities, high poverty levels, education opportunities, employment opportunities among others [126]. The greatest percentage of individuals migrating from Homa Bay County to Cities in Kenya are youth aged $15-29$ with male departure rates greater than female [126]. A recent interview of migrant youth from Western Kenya where Homa Bay County is situated revealed that 41% of male migrant youth in Nairobi County were willing to return to their home County whereas females were less likely to return to their home County [128]. Youths driven from Homa Bay County in search for employment opportunities settle in informal settlements in Nairobi County hence exposed to poor sanitation, insecurity, poor housing, HIV/AIDS risk among others [129]. Some youths upon failing to secure employment in Nairobi County return to their home County while others choose to stay in the City [129].

Migration from Nairobi County to Homa Bay County is driven by university education with two major public universities located in Homa Bay County [126]. Also, some of the poor Nairobi residents prefer to send their children to rural Counties like Homa Bay to continue their education at cheaper primary schools, secondary schools or technical institutions while under the care of their aging grandparents [130] . Driving distance between Nairobi and Homa Bay Counties is about 408.9 kilometers and driving time between the two Counties is about seven hours hence temporary residence in between travel is not considered. This formulation considers rural (Homa Bay) to urban (Nairobi) migration and urban (Nairobi) to rural (Homa Bay) migration. Migrant youth are reported to foster new relationships with opposite sex partners in Counties different from their origin [129]. Married migrant youth are known to either abandon their relationships at home in preference of their new found relationships or add to their existing spouse(s) as polygamy remains a strong tradition in Kenya furthering the risk of HIV/AIDS transmission in this population [129].

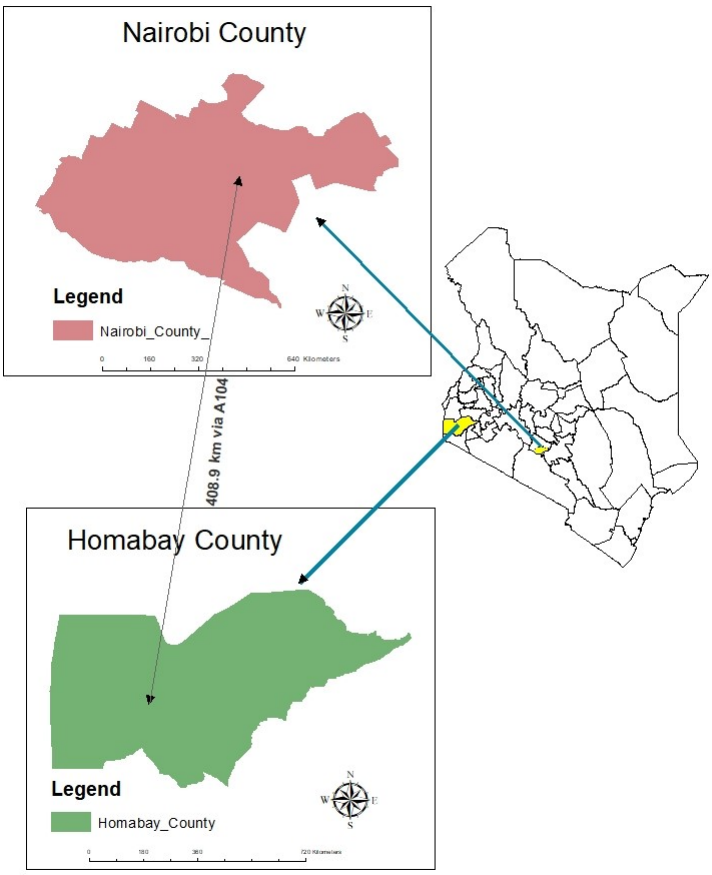


Figure 5.1: Geographic Location of Homa Bay and Nairobi Counties in Kenya [4].

Subscript 11 represents youth born and living in Homa Bay County, subscript 12 represents youth residing in Homa Bay County but originally from Nairobi County, subscript 21 represents youth living in Nairobi County but originally from Homa Bay County and subscript 22 represents youth born and living in Nairobi County. The youth population in Homa Bay County are decomposed into six classes such that at time $t \geq 0$ there uninfected S_{11}^u who have not tested for HIV/AIDS , uninfected S_{11}^a who have tested for HIV/AIDS and are aware of their HIV/AIDS status, infected unaware I_{11}^u who are yet to test for HIV/AIDS, infected aware I_{11}^a who know their HIV/AIDS status, infected aware not consistent with condom use and ART adherence T_{11}^u and infected aware consistent with condom use and ART adherence T_{11}^a. The total youth population in Homa Bay County is given as $N_{11} = S_{11}^u + S_{11}^a + I_{11}^u + I_{11}^a + T_{11}^u + T_{11}^a$. Similar state variables definitions follows for migrant youth in Homa Bay or Nairobi Counties and youth born and living in Nairobi County. We consider only sexually active youth aged $15 - 24$ in all high risk Counties as this formulation limits its HIV/AIDS transmission route to only heterosexual mode. Also, some of the adolescent and young adults aged 15-24 will have already initiated sex with most of them remaining sexually active in this age cohort. We do not include older male population greater than 24 years as results from a nationally representative population-based

survey which largely informed this formulation state variables considered transactional sex insignificant [102, 104]. Homogeneous mixing occurs between the youth with sexual partners of opposite sex in high risk Counties. Parameter d_{21} is the annual departure rate per youth from Homa Bay County to Nairobi County whereas r_{21} is the annual return rate per youth from Nairobi County to Homa Bay County. Travel and stay between Counties is assumed to be sufficient enough for HIV/AIDS transmission to occur which could happen with even a single exposure [131].

Susceptible youth in Homa Bay County, S_{11}^u and S_{11}^a are HIV free but risk infection through sexual contact with I_{12}^u, I_{12}^a and T_{12}^u. Population I_{12}^u is more infectious compared to I_{12}^a and T_{12}^u given the HIV/AIDS infection status awareness of the latter populations in comparison to I_{12}^u who are unaware of their HIV/AIDS status. The combinatory use of condoms and ART reduces the infectivity of T_{12}^u populations further in comparison to I_{12}^a who only use condoms. ART use works to reduce the viral load of T_{12}^u partially given their inconsistent use. Population T_{12}^a adhere perfectly to ART and use condoms consistently in every sexual contact which works to reduce their viral loads to undetectable levels [107]. Hence, we consider T_{12}^a not infectious in this model construction given that they are no longer engaging in HIV/AIDS disease transmission. The susceptible class in Homa Bay County, S_{11}^u is at risk of infection at the incidence rates β_{12}^u, β_{12}^a whereas S_{11}^a risks infection at the incidence rate $\tilde{\beta}_{12}^a$. We consider disease transmission driven by migrant youth with the incidence rates β_{12}^u, β_{12}^a and $\tilde{\beta}_{12}^a$ given in equation (5.1) as

$$\begin{cases} \beta_{12}^u = \dfrac{c_{12}\,\gamma_{12}\,[I_{12}^u + \alpha_{12}^c\,\rho_{12}^c\,I_{12}^a + (\alpha_{12}^c\,\rho_{12}^c + \alpha_{12}^t\,\rho_{12}^t)T_{12}^u]}{N_{12}}, \\ \beta_{12}^a = \dfrac{\alpha_{12}^{ht}\,\rho_{12}^{ht}\,c_{12}\,\gamma_{12}\,[I_{12}^u + \alpha_{12}^c\,\rho_{12}^c\,I_{12}^a + (\alpha_{12}^c\,\rho_{12}^c + \alpha_{12}^t\,\rho_{12}^t)T_{12}^u]}{N_{12}}, \\ \tilde{\beta}_{12}^a = \dfrac{\tilde{\alpha}_{12}^{ht}\,\rho_{12}^{ht}\,c_{12}\gamma_{12}\,[I_{12}^u + \alpha_{12}^c\,\rho_{12}^c\,I_{12}^a + (\alpha_{12}^c\,\rho_{12}^c + \alpha_{12}^t\,\rho_{12}^t)T_{12}^u]}{N_{12}}. \end{cases} \tag{5.1}$$

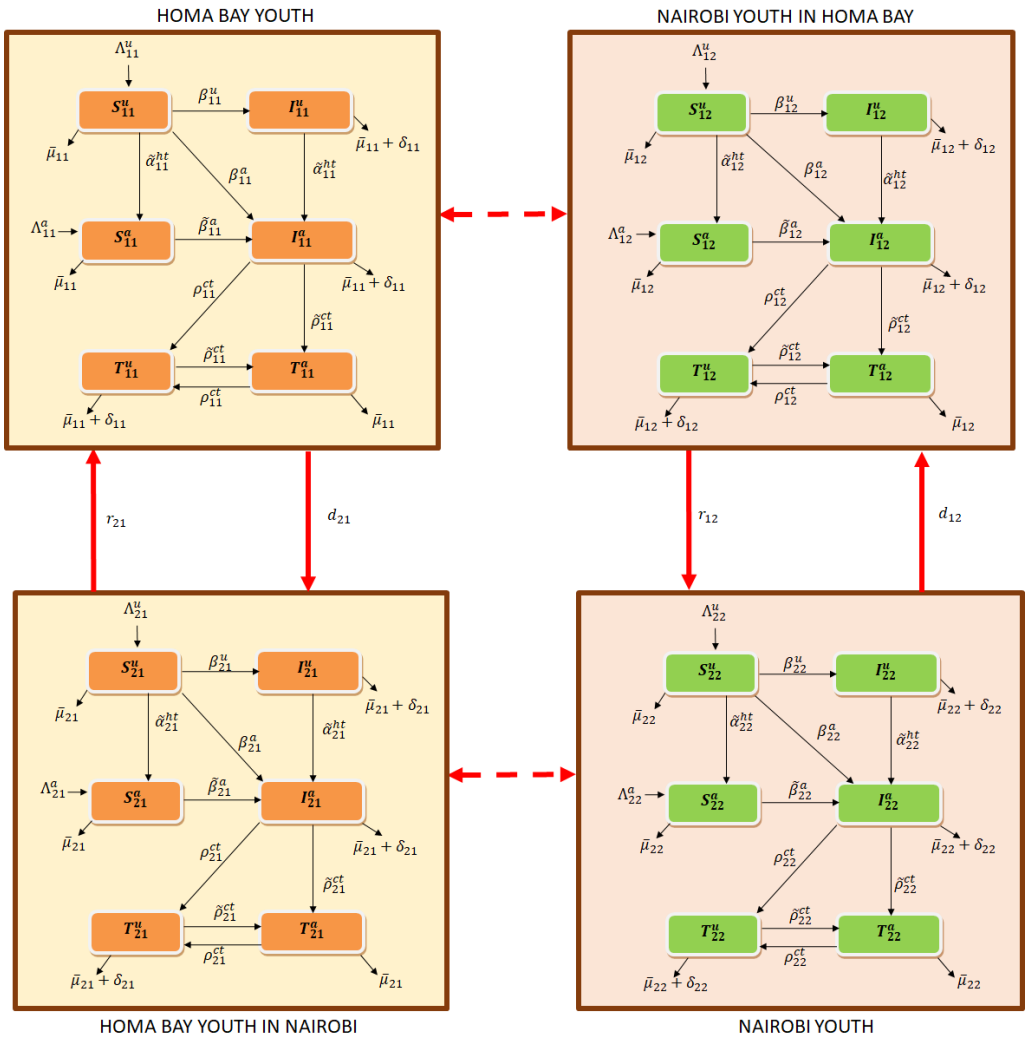


Figure 5.2: Schematics of the Spatial Model. Transitions and interactions between migrant youth and youth of County of origin.

Similarly, susceptible class in Nairobi County, S^u_{22} is at risk of HIV/AIDS infection at the incidence rates β^u_{21}, β^a_{21} whereas S^a_{22} risks infection at the incidence rate $\tilde{\beta}^a_{21}$ given in equation 5.2 as

$$\begin{cases} \beta^u_{21} = \dfrac{c_{21}\,\gamma_{21}\,[I^u_{21} + \alpha^c_{21}\,\rho^c_{21}\,I^a_{21} + (\alpha^c_{21}\,\rho^c_{21} + \alpha^t_{21}\,\rho^t_{21})T^u_{21}]}{N_{12}}, \\ \beta^a_{21} = \dfrac{\alpha^{ht}_{21}\,\rho^{ht}_{21}\,c_{21}\,\gamma_{21}\,[I^u_{21} + \alpha^c_{21}\,\rho^c_{21}\,I^a_{21} + (\alpha^c_{21}\,\rho^c_{21} + \alpha^t_{21}\,\rho^t_{21})T^u_{21}]}{N_{21}}, \\ \tilde{\beta}^a_{21} = \dfrac{\tilde{\alpha}^{ht}_{21}\,\rho^{ht}_{21}\,c_{21}\gamma_{21}\,[I^u_{21} + \alpha^c_{21}\,\rho^c_{21}\,I^a_{21} + (\alpha^c_{21}\,\rho^c_{21} + \alpha^t_{21}\,\rho^t_{21})T^u_{21}]}{N_{21}}. \end{cases} \tag{5.2}$$

This formulation assumes different basic parameters governing the Kenyan youth disease dynamics in the considered high risk Counties. Contact rate c_{12} denotes the the average number of sexual contacts by the youth with opposite sex partners per unit time whereas γ_{12}

denotes the probability of disease spread by the youth with opposite sex partners per unit time. Condom use rate is given as ρ_{12}^{c} whereas ρ_{12}^{t} denotes ART adherence rate. HIV/AIDS status disclosure rate by newly HIV/AIDS tested I_{12}^{u} and already tested populations I_{12}^{a}, T_{12}^{u} is given by ρ_{12}^{ht}.

Perfect adherence is attained when each of the HIV/AIDS targeted intervention ρ_{12}^{ht}, ρ_{12}^{c}, ρ_{12}^{t} is 1 otherwise, $0 \leq \rho_{12}^{ht}, \rho_{12}^{c}, \rho_{12}^{t} \leq 1$. The rate α_{12}^{ht} denotes County attitudes affecting HIV testing rate ρ_{12}^{ht} negatively in the youth populations such as poor health services, HIV testing behavior, low education levels among others. Similarly, α_{12}^{c} denotes negative County attitudes affecting condom use rate in the youth populations such as religion, community norms, perceived individual's risk among others. Further, α_{12}^{t} denotes County attitudes affecting ART usage rate negatively among the infected adolescents and young adults such as stigma, poverty, lack of caregiver(s) support, health officials disclosing youth HIV/AIDS status in the community among others. The rates $\alpha_{12}^{c}\,\rho_{12}^{c}$ reduces I_{12}^{a} infectiousness as condom use shields the uninfected youth from new HIV/AIDS infection. In addition to condom use, T_{12}^{u} partial use of ART reduces their HIV/AIDS viral load making them less infectious. Thus, $0 < \alpha_{12}^{c}, \alpha_{12}^{t} < 1$. T_{12}^{u} infectivity is less than I_{12}^{a} which is less than I_{12}^{u}.

The incidence rate β_{12}^{u} is driven by the Kenyan youth unaware of their HIV/AIDS status with their opposite sex partners per unit time whereas β_{12}^{a} is the incidence rate by HIV/AIDS tested youth who are yet to be initiated on ART with their opposite sex partners per unit time. Also, $\tilde{\beta}_{12}^{a}$ is the incidence rate by youth who are aware of their HIV/AIDS status but fail to use condoms and ART treatment consistently with their opposite sex partners per unit time. The incidence rates β_{12}^{u}, β_{12}^{a} and $\tilde{\beta}_{12}^{a}$ have frequency dependent incidences given that most of the adolescents and young adults aged 15-24 will have already initiated sex with most of them maintaining regular sexual contacts with their sex partners.

Susceptibles S_{11}^{u} who are aware of their HIV/AIDS status through HIV/AIDS testing and is willing to disclose their HIV/AIDS status to sex partner moves to S_{11}^{a} at the rates $\alpha_{11}^{ht}\,\rho_{11}^{ht}$. A newly infected S_{11}^{u} through sexual contact with either infected unaware I_{12}^{u} or infected aware I_{12}^{a}, T_{12}^{u} who are secretive about their HIV/AIDS status will move to I_{11}^{u} at the rate β_{12}^{u}. Also, a newly infected S_{11}^{u}, through sexual interaction with infected aware populations of the opposite sex who are open about their HIV/AIDS status will move to I_{11}^{a} at the rate β_{12}^{a} given that HIV/AIDS status openness by infected aware population results in HIV/AIDS awareness of the newly infected S_{11}^{u}. A newly infected S_{11}^{a} moves to I_{11}^{a} at the rate $\tilde{\beta}_{12}^{a}$. Population I_{11}^{u} in high risk County i can move to I_{11}^{a} at the rate $\alpha_{11}^{ht}\,\rho_{11}^{ht}$ through HIV/AIDS testing. Similarly, I_{11}^{a} and T_{11}^{u} who use condoms and ART consistently moves to T_{11}^{a} at the rate $\tilde{\rho}_{11}^{ct}$ whereas an I_{11}^{a} or T_{11}^{a} who use condoms and ART inconsistently moves to T_{11}^{u} at the rate ρ_{11}^{ct} respectively with $\tilde{\rho}_{11}^{ct} = \tilde{\alpha}_{11}^{c}\,\rho_{11}^{c} + \tilde{\alpha}_{11}^{t}\,\rho_{11}^{t}$ and $\rho_{11}^{ct} = \alpha_{11}^{c}\rho_{11}^{c} + \alpha_{11}^{t}\rho_{11}^{t}$ respectively. The rates $\tilde{\alpha}_{11}^{ht}$, $\tilde{\alpha}_{11}^{c}$, $\tilde{\alpha}_{11}^{t}$ and α_{11}^{ht}, α_{11}^{c}, α_{11}^{t} denote County attitudes affecting the efficacy of HIV/AIDS interventions ρ_{11}^{ht}, ρ_{11}^{c}, ρ_{11}^{t} but not to zero given that these targeted interventions have kicked off in Kenyan HIV/AIDS youth dynamics. Rates $\tilde{\alpha}_{11}^{ht}$, $\tilde{\alpha}_{11}^{c}$, $\tilde{\alpha}_{11}^{t}$ represent County attitudes promoting the efficacy of ρ_{11}^{ht}, ρ_{11}^{c}, ρ_{11}^{t} in Homa Bay County whereas α_{11}^{ht}, α_{11}^{c}, α_{11}^{t} represent County attitudes negating the efficacy of the mentioned

targeted interventions in Homa Bay County. Thus,

$$0 < \tilde{\alpha}_{11}^{ht},\, \tilde{\alpha}_{11}^{c},\, \tilde{\alpha}_{11}^{t},\, \alpha_{11}^{ht},\, \alpha_{11}^{c},\, \alpha_{11}^{t} < 1,$$

with

$$\tilde{\alpha}_{11}^{ht},\, \tilde{\alpha}_{11}^{c},\, \tilde{\alpha}_{11}^{t} > \alpha_{11}^{ht},\, \alpha_{11}^{c},\, \alpha_{11}^{t}.$$

Recruitment rates into susceptible populations S_{11}^{u}, S_{11}^{a} in Homa Bay County are by natural births and maturity to 15 years of age and are given as Λ_{11}^{u}, Λ_{11}^{a} respectively whereas Λ_{12}^{u}, Λ_{12}^{a} are the recruitment rates of migrant Nairobi youth in Homa Bay County. Recruitment rate Λ_{11}^{u} denotes adolescents boys and girls who have never tested for HIV/AIDS from birth until age 15 whereas Λ_{11}^{a} denotes adolescents boys and girls who have tested for HIV/AIDS by age 15. Recruitment rate for migrant youth in Counties different from their origin is assumed to be less than the recruitment rate of youth born and living in their home County. The susceptible classes in Homa Bay County are all decreased by natural mortality μ_{11} whereas the infectious classes are all reduced by natural mortality and HIV/AIDS related deaths, δ_{11}. Adolescents and young adults populations in Homa Bay County exit the model at the rate σ_{11} after their 24th birthday. The model variables and parameters in migrant youth and Nairobi County residents take similar definitions as Homa Bay County and are assumed to be non-negative given that a population dynamics model is under consideration.

The youth rate of change in Homa Bay and Nairobi Counties are governed by equation systems (5.3) and (5.4) as

$$\begin{cases} \dot{S}_{11}^{u} = \Lambda_{11}^{u} - \beta_{12}^{u}\, S_{11}^{u} - \beta_{12}^{a}\, S_{11}^{u} - \mu_{11}^{1}\, S_{11}^{u} - d_{21} S_{11}^{u} + r_{21} S_{21}^{u}, \\ \dot{S}_{11}^{a} = \Lambda_{11}^{a} + \alpha_{11}^{ht} \rho_{11}^{ht}\, S_{11}^{u} - \tilde{\beta}_{12}^{a}\, S_{11}^{a} - \mu_{11}^{2}\, S_{11}^{a} - d_{21} S_{11}^{a} + r_{21} S_{21}^{a}, \\ \dot{I}_{11}^{u} = \beta_{12}^{u}\, S_{11}^{u} - \mu_{11}^{3}\, I_{11}^{u} - d_{21} I_{11}^{u} + r_{21} I_{21}^{u}, \\ \dot{I}_{11}^{a} = \tilde{\beta}_{12}^{a}\, S_{11}^{a} + \beta_{12}^{a}\, S_{11}^{u} + \alpha_{11}^{ht}\, \rho_{11}^{ht}\, I_{11}^{u} - \mu_{11}^{4}\, I_{11}^{a} - d_{21} I_{11}^{a} + r_{21} I_{21}^{a}, \\ \dot{T}_{11}^{u} = \rho_{11}^{ct}\, I_{11}^{a} + \rho_{11}^{ct}\, T_{11}^{a} - \mu_{11}^{5}\, T_{11}^{u} - d_{21} T_{11}^{u} + r_{21} T_{21}^{u}, \\ \dot{T}_{11}^{a} = \tilde{\rho}_{11}^{ct} I_{11}^{a} + \tilde{\rho}_{11}^{ct}\, T_{11}^{u} - \mu_{11}^{6}\, T_{11}^{a} d_{21} T_{11}^{a} + r_{21} T_{21}^{a}, \end{cases} \tag{5.3}$$

$$\begin{cases} \dot{S}^u_{22} = \Lambda^u_{22} - \beta^u_{21}\, S^u_{22} - \beta^a_{21}\, S^u_{22} - \mu^1_{22}\, S^u_{22} - d_{12} S^u_{22} + r_{12} S^u_{12}, \\ \dot{S}^a_{22} = \Lambda^a_{22} + \alpha^{ht}_{22}\rho^{ht}_{22}\, S^u_{22} - \tilde{\beta}^a_{21}\, S^a_{22} - \mu^2_{22}\, S^a_{22} - d_{12} S^a_{22} + r_{12} S^a_{12}, \\ \dot{I}^u_{22} = \beta^u_{21}\, S^u_{22} - \mu^3_{22}\, I^u_{22} - d_{12} I^u_{22} + r_{12} I^u_{12}, \\ \dot{I}^a_{22} = \tilde{\beta}^a_{21}\, S^a_{22} + \beta^a_{21}\, S^u_{22} + \alpha^{ht}_{22}\,\rho^{ht}_{22}\, I^u_{22} - \mu^4_{22}\, I^a_{22} - d_{12} I^a_{22} + r_{12} I^a_{12}, \\ \dot{T}^u_{22} = \rho^{ct}_{22}\, I^a_{22} + \rho^{ct}_{22}\, T^a_{22} - \mu^5_{22}\, T^u_{22} - d_{12} T^u_{22} + r_{12} T^u_{12}, \\ \dot{T}^a_{22} = \tilde{\rho}^{ct}_{22} I^a_{22} + \tilde{\rho}^{ct}_{22}\, T^u_{22} - \mu^6_{22}\, T^a_{22} - d_{12} T^a_{22} + r_{12} T^a_{12}. \end{cases} \tag{5.4}$$

where
$\bar{\mu}_{11} = \mu_{11} + \sigma_{11}$, $\mu^1_{11} = \alpha^{ht}_{11}\,\rho^{ht}_{11} + \bar{\mu}_{11}$, $\mu^2_{11} = \bar{\mu}_{11}$, $\mu^3_{11} = \alpha^{ht}_{11}\,\rho^{ht}_{11} + \bar{\mu}_{11} + \delta_{11}$, $\mu^4_{11} = \rho^{ct}_{11} + \tilde{\rho}^{ct}_{11} + \bar{\mu}_{11} + \delta_{11}$, $\mu^5_{11} = \tilde{\rho}^{ct}_{11} + \bar{\mu}_{11} + \delta_{11}$, $\mu^6_{11} = \rho^{ct}_{11} + \bar{\mu}_{11}$,

$\bar{\mu}_{22} = \mu_{22} + \sigma_{22}$, $\mu^1_{22} = \alpha^{ht}_{22}\,\rho^{ht}_{22} + \bar{\mu}_{22}$, $\mu^2_{22} = \bar{\mu}_{22}$, $\mu^3_{22} = \alpha^{ht}_{22}\,\rho^{ht}_{22} + \bar{\mu}_{22} + \delta_{22}$, $\mu^4_{22} = \rho^{ct}_{22} + \tilde{\rho}^{ct}_{22} + \bar{\mu}_{22} + \delta_{22}$, $\mu^{22}_{f5} = \tilde{\rho}^{ct}_{22} + \bar{\mu}_{22} + \delta_{22}$, $\mu^6_{22} = \rho^{ct}_{22} + \bar{\mu}_{22}$.

Equation systems (5.5) and (5.6) governs the rate of change for migrant youth in Counties different from their home Counties and are given as

$$\begin{cases} \dot{S}^u_{12} = \Lambda^u_{12} - \beta^u_{12}\, S^u_{12} - \beta^a_{12}\, S^u_{12} - \mu^1_{12}\, S^u_{12} - d_{12} S^u_{22} + r_{12} S^u_{12}, \\ \dot{S}^a_{12} = \Lambda^a_{12} + \alpha^{ht}_{12}\rho^{ht}_{12}\, S^u_{12} - \tilde{\beta}^a_{12}\, S^a_{12} - \mu^2_{12}\, S^a_{12} - d_{12} S^a_{22} + r_{12} S^a_{12}, \\ \dot{I}^u_{12} = \beta^u_{12}\, S^u_{12} - \mu^3_{12}\, I^u_{12} - d_{12} I^u_{22} + r_{12} I^u_{12}, \\ \dot{I}^a_{12} = \tilde{\beta}^a_{12}\, S^a_{12} + \beta^a_{12}\, S^u_{12} + \alpha^{ht}_{12}\,\rho^{ht}_{12}\, I^u_{12} - \mu^4_{12}\, I^a_{12} - d_{12} I^a_{22} + r_{12} I^a_{12}, \\ \dot{T}^u_{12} = \rho^{ct}_{12}\, I^a_{12} + \rho^{ct}_{12}\, T^a_{12} - \mu^5_{12}\, T^u_{12} - d_{12} T^u_{22} + r_{12} T^u_{12}, \\ \dot{T}^a_{12} = \tilde{\rho}^{ct}_{12} I^a_{12} + \tilde{\rho}^{ct}_{12}\, T^u_{12} - \mu^6_{12}\, T^a_{12} - d_{12} T^a_{22} + r_{12} T^a_{12}, \end{cases} \tag{5.5}$$

$$\begin{cases} \dot{S}^u_{21} = \Lambda^u_{21} - \beta^u_{21}\, S^u_{21} - \beta^a_{21}\, S^u_{21} - \mu^1_{21}\, S^u_{21} - d_{21} S^u_{11} + r_{21} S^u_{21}, \\ \dot{S}^a_{21} = \Lambda^a_{21} + \alpha^{ht}_{21}\rho^{ht}_{21}\, S^u_{21} - \tilde{\beta}^a_{21}\, S^a_{21} - \mu^2_{21}\, S^a_{21} - d_{21} S^a_{11} + r_{21} S^a_{21}, \\ \dot{I}^u_{21} = \beta^u_{21}\, S^u_{21} - \mu^3_{21}\, I^u_{21} - d_{21} I^u_{11} + r_{21} I^u_{21}, \\ \dot{I}^a_{21} = \tilde{\beta}^a_{21}\, S^a_{21} + \beta^a_{21}\, S^u_{21} + \alpha^{ht}_{21}\,\rho^{ht}_{21}\, I^u_{21} - \mu^4_{21}\, I^a_{21} - d_{21} I^a_{11} + r_{21} I^a_{21}, \\ \dot{T}^u_{21} = \rho^{ct}_{21}\, I^a_{21} + \rho^{ct}_{21}\, T^a_{21} - \mu^5_{21}\, T^u_{21} - d_{21} T^u_{11} + r_{21} T^u_{21}, \\ \dot{T}^a_{21} = \tilde{\rho}^{ct}_{21} I^a_{21} + \tilde{\rho}^{ct}_{21}\, T^u_{21} - \mu^6_{21}\, T^a_{21} - d_{21} T^a_{11} + r_{21} T^a_{21}. \end{cases} \tag{5.6}$$

Tables 5.1 and 5.2 gives the summary description for the state variables and model parameters

respectively.

Table 5.1: State Variables Description

Variable	Description
S_{11}^u, S_{12}^u, S_{21}^u, S_{22}^u	Susceptible adolescents and young adults who have never tested for HIV/AIDS
S_{11}^a, S_{12}^a, S_{21}^a, S_{22}^a	Susceptible adolescents and young adults who have ever tested for HIV/AIDS and are aware of their HIV/AIDS status
I_{11}^u, I_{12}^u, I_{21}^u, I_{22}^u	Infected adolescents and young adults who have never tested for HIV/AIDS
I_{11}^a, I_{12}^a, I_{21}^a, I_{22}^a	Infected adolescents and young adults who have ever tested for HIV/AIDS and are aware of their HIV/AIDS status
T_{11}^u, T_{12}^u, T_{21}^u, T_{22}^u	Infected aware adolescents and young adults who are not adherent to ART or consistent condom use
T_{11}^a, T_{12}^a, T_{21}^a, T_{22}^a	Infected aware adolescents and young adults who are adherent to ART and use condoms consistently

Table 5.2: Parameters Description

Parameter	Description
c_{12}, c_{21}	Youth contact rate with partners of opposite sex
γ_{12}, γ_{21}	HIV/AIDS transmission risk rate
δ_{11}, δ_{12}, δ_{21}, δ_{22}	HIV/AIDS disease induced death rate
μ_{11}, μ_{12}, μ_{21}, μ_{22}	Natural mortality rate
Λ_{11}^u, Λ_{12}^u, Λ_{21}^u, Λ_{22}^u	Natural birth and survival rate to 15 years of HIV/AIDS untested adolescents
Λ_{11}^a, Λ_{12}^a, Λ_{21}^a, Λ_{22}^a	Natural birth and survival rate to 15 years of HIV/AIDS tested adolescents
d_{21}, d_{12}	Departure rates from County of origin
r_{21}, r_{12}	Return rates to County of origin
σ_{11}, σ_{12}, σ_{21}, σ_{22}	Youth exit rates upon turning 24 years
ρ_{11}^{ht}, ρ_{12}^{ht}, ρ_{21}^{ht}, ρ_{22}^{ht}	Youth HIV/AIDS testing rates
ρ_{11}^{c}, ρ_{12}^{c}, ρ_{21}^{c}, ρ_{22}^{c}	Youth condom use rates
ρ_{11}^{t}, ρ_{12}^{t}, ρ_{21}^{t}, ρ_{22}^{t}	HIV/AIDS infected youth adherence rate to ART
α_{11}^{ht}, α_{12}^{ht}, α_{21}^{ht}, α_{22}^{ht}	County attitudes negating HIV/AIDS testing
$\tilde{\alpha}_{11}^{ht}$, $\tilde{\alpha}_{12}^{ht}$, $\tilde{\alpha}_{21}^{ht}$, $\tilde{\alpha}_{22}^{ht}$	County attitudes promoting HIV/AIDS testing
α_{11}^{c}, α_{12}^{c}, α_{21}^{c}, α_{22}^{c}	County attitudes negating condom use
$\tilde{\alpha}_{11}^{c}$, $\tilde{\alpha}_{12}^{c}$, $\tilde{\alpha}_{21}^{c}$, $\tilde{\alpha}_{22}^{c}$	County attitudes promoting condom use
α_{11}^{t}, α_{12}^{t}, α_{21}^{t}, α_{22}^{t}	County attitudes negating ART adherence
$\tilde{\alpha}_{11}^{t}$, $\tilde{\alpha}_{12}^{t}$, $\tilde{\alpha}_{21}^{t}$, $\tilde{\alpha}_{22}^{t}$	County attitudes promoting ART adherence

5.3 Model Analysis

5.3.1 Positivity and Boundedness of Model Trajectories

Proposition 5.3.1. *The positive orthant $\mathbb{R}^{24}_{+}$ is positively invariant in the solution region for model systems (5.3) - (5.6) $\forall\ t > 0$, $S^u_{11} > 0$, $S^a_{11} > 0$, $S^u_{22} > 0$, $S^a_{22} > 0$, $S^u_{12} > 0$, $S^a_{12} > 0$, $S^u_{21} > 0$ and $S^a_{21} > 0$ provided the departure rates $d_{12} > 0$ and $d_{21} > 0$. Also, all trajectories of model systems are bounded [132].*

Proof. Given that the model systems (5.3) - (5.6) state initial conditions and model parameters are considered non-negative, positivity of all model trajectories follows. Consider the first differential equation in model system (5.3). Suppose $S^u_{11} = 0$ at $t = 0$, then $\dot{S}^u_{11} > 0$ and this implies $S^u_{11} > 0$ $\forall\ t > 0$. Also, consider the first equation of model system (5.5). $\forall\ t > 0$, $S^u_{12} > 0$ for $2 \neq 1$. Given the HIV/AIDS related mortality, we use the theory of differential inequality by [55]. Thus, every trajectory originating from the region of solution remains in the solution region and is bounded. See [132] for details on the proof ideas of positivity and boundedness of spatial models. □

5.3.2 Equilibria

The model systems (5.3) - (5.6) are at equilibria when the state derivatives in (5.3) - (5.6) are 0. Homa Bay County model given in equation system (5.3) is at disease free state if

$$I^u_{21} = I^a_{21} = T^u_{21} = 0 \quad \text{and,} \quad S^{u0}_{21} = \frac{\Lambda^u_{11}\, d_{21} - \Lambda^u_{21}(\mu^1_{11} + d_{21})}{(\mu^1_{11} + d_{21})(r_{21} - \mu^1_{21}) - d_{21}\, r_{21}},$$

$$S^{a0}_{21} = \frac{d_{21}(\Lambda^a_{11} + \tilde{\alpha}^{ht}_{11}\, S^{u0}_{11}) - (\mu^2_{11} + d_{21})(\Lambda^a_{21} + \tilde{\alpha}^{ht}_{21}\, S^{u0}_{21})}{(\mu^2_{11} + d_{21})(r_{21} - \mu^2_{21}) - d_{21}\, r_{21}}, \quad S^{u0}_{11} = \frac{\Lambda^u_{21} - (\mu^1_{21} - r_{21})S^{u0}_{21}}{d_{21}}$$

$$\text{provided } \Lambda^u_{21} < \frac{\Lambda^u_{11}\, d_{21}}{\mu^1_{11} + d_{21}},\ \mu^1_{21} < \frac{\mu^1_{11}\, r_{21}}{\mu^1_{11} + d_{21}},\ \mu^2_{21} < \frac{\mu^2_{11}\, d_{21}}{\mu^2_{11} + d_{21}},\ S^{u0}_{21} < \frac{\Lambda^u_{21}}{\mu^1_{21} - r_{21}},$$

$$\mu^1_{21} > r_{21} \text{ and } d_{21} > 0.$$

Nairobi County model presented in equation system (5.6) is at disease free state if

$$I^u_{12} = I^a_{12} = T^u_{12} = 0 \quad \text{and,} \quad S^{u0}_{12} = \frac{\Lambda^u_{22}\, d_{12} - \Lambda^u_{12}(\mu^1_{22} + d_{12})}{(\mu^1_{22} + d_{12})(r_{12} - \mu^1_{12}) - d_{12}\, r_{12}},$$

$$S^{a0}_{12} = \frac{d_{12}(\Lambda^a_{22} + \tilde{\alpha}^{ht}_{22}\, S^{u0}_{22}) - (\mu^2_{22} + d_{12})(\Lambda^a_{12} + \tilde{\alpha}^{ht}_{12}\, S^{u0}_{12})}{(\mu^2_{22} + d_{12})(r_{12} - \mu^2_{12}) - d_{12}\, r_{12}}, \quad S^{u0}_{22} = \frac{\Lambda^u_{12} - (\mu^1_{12} - r_{12})S^{u0}_{12}}{d_{12}}$$

$$\text{provided } \Lambda^u_{12} < \frac{\Lambda^u_{22}\, d_{12}}{\mu^1_{22} + d_{12}},\ \mu^1_{12} < \frac{\mu^1_{22}\, r_{12}}{\mu^1_{22} + d_{12}},\ \mu^2_{12} < \frac{\mu^2_{22}\, d_{12}}{\mu^2_{22} + d_{12}},\ S^{u0}_{12} < \frac{\Lambda^u_{12}}{\mu^1_{12} - r_{12}},$$

$$\mu^1_{12} > r_{12} \text{ and } d_{12} > 0.$$

Theorem 5.3.1. *If model systems (5.3) - (5.6) are at equilibria and that HIV/AIDS is endemic among the youth in Homa Bay County , then HIV/AIDS will be endemic in all Counties that are accessible from Homa Bay County.*

See [132] for proof ideas of theorem 5.3.1.

5.3.3 Control Reproduction Number Computation

Consider the infected subsystem of model systems (5.3) - (5.6) which includes infected youth in Homa Bay and Nairobi Counties and those infected youth commuting between these Counties given in model systems (5.5) - (5.6). The infected subsystem in equation (5.7) is further decomposed into two matrices, F, representing the appearance rate of new HIV/AIDS infections and V, representing the transition rate of HIV/AIDS infected AGYW/ABYM in the model world. Matrices V and F are given in equations 5.8 and 5.9 respectively.

$$\begin{cases}
\dot{I}^u_{11} = \beta^u_{12}\, S^u_{11} - \mu^3_{11}\, I^u_{11} - d_{21} I^u_{11} + r_{21} I^u_{21}, \\
\dot{I}^u_{12} = \beta^u_{12}\, S^u_{12} - \mu^3_{12}\, I^u_{12} - d_{12} I^u_{22} + r_{12} I^u_{12}, \\
\dot{I}^u_{21} = \beta^u_{21}\, S^u_{21} - \mu^3_{21}\, I^u_{21} - d_{21} I^u_{11} + r_{21} I^u_{21}, \\
\dot{I}^u_{22} = \beta^u_{21}\, S^u_{22} - \mu^3_{22}\, I^u_{22} - d_{12} I^u_{22} + r_{12} I^u_{22}, \\
\dot{I}^a_{11} = \tilde{\beta}^a_{12}\, S^a_{11} + \beta^a_{12}\, S^u_{11} + \alpha^{ht}_{11}\, \rho^{ht}_{11}\, I^u_{11} - \mu^4_{11}\, I^a_{11} - d_{21} I^a_{11} + r_{21} I^a_{21}, \\
\dot{I}^a_{12} = \tilde{\beta}^a_{12}\, S^a_{12} + \beta^a_{12}\, S^u_{12} + \alpha^{ht}_{12}\, \rho^{ht}_{12}\, I^u_{12} - \mu^4_{12}\, I^a_{12} - d_{12} I^a_{22} + r_{12} I^a_{12}, \\
\dot{I}^a_{21} = \tilde{\beta}^a_{21}\, S^a_{21} + \beta^a_{21}\, S^u_{21} + \alpha^{ht}_{21}\, \rho^{ht}_{21}\, I^u_{21} - \mu^4_{21}\, I^a_{21} - d_{21} I^a_{11} + r_{21} I^a_{21}, \\
\dot{I}^a_{22} = \tilde{\beta}^a_{21}\, S^a_{22} + \beta^a_{21}\, S^u_{22} + \alpha^{ht}_{22}\, \rho^{ht}_{22}\, I^u_{22} - \mu^4_{22}\, I^a_{22} - d_{12} I^a_{22} + r_{12} I^a_{12}, \\
\dot{T}^u_{11} = \rho^{ct}_{11}\, I^a_{11} + \rho^{ct}_{11}\, T^a_{11} - \mu^5_{11}\, T^u_{11} - d_{21} T^u_{11} + r_{21} T^u_{21}, \\
\dot{T}^u_{12} = \rho^{ct}_{12}\, I^a_{12} + \rho^{ct}_{12}\, T^a_{12} - \mu^5_{12}\, T^u_{12} - d_{12} T^u_{22} + r_{12} T^u_{12}, \\
\dot{T}^u_{21} = \rho^{ct}_{21}\, I^a_{21} + \rho^{ct}_{21}\, T^a_{21} - \mu^5_{21}\, T^u_{21} - d_{21} T^u_{11} + r_{21} T^u_{21}, \\
\dot{T}^u_{22} = \rho^{ct}_{22}\, I^a_{22} + \rho^{ct}_{22}\, T^a_{22} - \mu^5_{22}\, T^u_{22} - d_{12} T^u_{22} + r_{12} T^u_{12}.
\end{cases} \tag{5.7}$$

$$V = -\begin{bmatrix}
-\mu^3_{11}\, I^u_{11} - d_{21} I^u_{11} + r_{21} I^u_{21} \\
-\mu^3_{12}\, I^u_{12} - d_{12} I^u_{22} + r_{12} I^u_{12} \\
-\mu^3_{21}\, I^u_{21} - d_{21} I^u_{11} + r_{21} I^u_{21} \\
-\mu^3_{22}\, I^u_{22} - d_{12} I^u_{22} + r_{12} I^u_{12} \\
\alpha^{ht}_{11}\, \rho^{ht}_{11}\, I^u_{11} - \mu^4_{11}\, I^a_{11} - d_{21} I^a_{11} + r_{21} I^a_{21} \\
\alpha^{ht}_{12}\, \rho^{ht}_{12}\, I^u_{12} - \mu^4_{12}\, I^a_{12} - d_{12} I^a_{22} + r_{12} I^a_{12} \\
\alpha^{ht}_{21}\, \rho^{ht}_{21}\, I^u_{21} - \mu^4_{21}\, I^a_{21} - d_{21} I^a_{11} + r_{21} I^a_{21} \\
\alpha^{ht}_{22}\, \rho^{ht}_{22}\, I^u_{22} - \mu^4_{22}\, I^a_{22} - d_{12} I^a_{22} + r_{12} I^a_{12} \\
\rho^{ct}_{11}\, I^a_{11} + \rho^{ct}_{11}\, T^a_{11} - \mu^5_{11}\, T^u_{11} - d_{21} T^u_{11} + r_{21} T^u_{21} \\
\rho^{ct}_{12}\, I^a_{12} + \rho^{ct}_{12}\, T^a_{12} - \mu^5_{12}\, T^u_{12} - d_{12} T^u_{22} + r_{12} T^u_{12} \\
\rho^{ct}_{21}\, I^a_{21} + \rho^{ct}_{21}\, T^a_{21} - \mu^5_{21}\, T^u_{21} - d_{21} T^u_{11} + r_{21} T^u_{21} \\
\rho^{ct}_{22}\, I^a_{22} + \rho^{ct}_{22}\, T^a_{22} - \mu^5_{22}\, T^u_{22} - d_{12} T^u_{22} + r_{12} T^u_{12}
\end{bmatrix} \tag{5.8}$$

and

$$F = \begin{bmatrix} \frac{c_{12}\gamma_{12}\left[I_{12}^{u} + \alpha_{12}^{c}\rho_{12}^{c}I_{12}^{a} + (\alpha_{12}^{c}\rho_{12}^{c} + \alpha_{12}^{t}\rho_{12}^{t})T_{12}^{u}\right]S_{11}^{u}}{N_{12}} \\ \frac{c_{12}\gamma_{12}\left[I_{12}^{u} + \alpha_{12}^{c}\rho_{12}^{c}I_{12}^{a} + (\alpha_{12}^{c}\rho_{12}^{c} + \alpha_{12}^{t}\rho_{12}^{t})T_{12}^{u}\right]S_{12}^{u}}{N_{12}} \\ \frac{c_{21}\gamma_{21}\left[I_{21}^{u} + \alpha_{21}^{c}\rho_{21}^{c}I_{21}^{a} + (\alpha_{21}^{c}\rho_{21}^{c} + \alpha_{21}^{t}\rho_{21}^{t})T_{21}^{u}\right]S_{21}^{u}}{N_{21}} \\ \frac{c_{21}\gamma_{21}\left[I_{21}^{u} + \alpha_{21}^{c}\rho_{21}^{c}I_{21}^{a} + (\alpha_{21}^{c}\rho_{21}^{c} + \alpha_{21}^{t}\rho_{21}^{t})T_{21}^{u}\right]S_{22}^{u}}{N_{21}} \\ \frac{\rho_{12}^{ht}c_{12}\gamma_{12}\left[I_{12}^{u} + \alpha_{12}^{c}\rho_{12}^{c}I_{12}^{a} + (\alpha_{12}^{c}\rho_{12}^{c} + \alpha_{12}^{t}\rho_{12}^{t})T_{12}^{u}\right]\left[S_{11}^{u} + \alpha_{12}^{ht}S_{11}^{a}\right]}{N_{12}} \\ \frac{\rho_{12}^{ht}c_{12}\gamma_{12}\left[I_{12}^{u} + \alpha_{12}^{c}\rho_{12}^{c}I_{12}^{a} + (\alpha_{12}^{c}\rho_{12}^{c} + \alpha_{12}^{t}\rho_{12}^{t})T_{12}^{u}\right]\left[S_{12}^{u} + \alpha_{12}^{ht}S_{12}^{a}\right]}{N_{12}} \\ \frac{\rho_{21}^{ht}c_{21}\gamma_{21}\left[I_{21}^{u} + \alpha_{21}^{c}\rho_{21}^{c}I_{21}^{a} + (\alpha_{21}^{c}\rho_{21}^{c} + \alpha_{21}^{t}\rho_{21}^{t})T_{21}^{u}\right]\left[S_{21}^{u} + \alpha_{21}^{ht}S_{21}^{a}\right]}{N_{21}} \\ \frac{\rho_{21}^{ht}c_{21}\gamma_{21}\left[I_{21}^{u} + \alpha_{21}^{c}\rho_{21}^{c}I_{21}^{a} + (\alpha_{21}^{c}\rho_{21}^{c} + \alpha_{21}^{t}\rho_{21}^{t})T_{21}^{u}\right]\left[S_{22}^{u} + \alpha_{21}^{ht}S_{22}^{a}\right]}{N_{21}} \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}. \tag{5.9}$$

We use the next generation matrix method to compute the control reproduction number $\mathcal{R}_c$. $\mathcal{F}$ and $\mathcal{V}$ are evaluated as:

$$\mathcal{F} = \left[\frac{\partial F(x_0)}{\partial X}\right] = \left[\begin{array}{c|c|c} \mathcal{F}_{11} & \mathcal{F}_{12} & \mathcal{F}_{13} \\ \hline \mathcal{F}_{21} & \mathcal{F}_{22} & \mathcal{F}_{23} \\ \hline \mathcal{F}_{31} & \mathcal{F}_{32} & \mathcal{F}_{33} \end{array}\right], \tag{5.10}$$

$$\mathcal{V} = \left[\frac{\partial V(x_0)}{\partial X}\right] = \left[\begin{array}{c|c|c} \mathcal{V}_{11} & \mathcal{V}_{12} & \mathcal{V}_{13} \\ \hline \mathcal{V}_{21} & \mathcal{V}_{22} & \mathcal{V}_{23} \\ \hline \mathcal{V}_{31} & \mathcal{V}_{32} & \mathcal{V}_{33} \end{array}\right], \tag{5.11}$$

where x_0 is the disease free equilibrium. The next generation matrix $\mathcal{F}\mathcal{V}^{-1}$ is given as:

$$\mathcal{F}\mathcal{V}^{-1} = \left[\begin{array}{c|c|c} \mathcal{F}_{11}\,\mathcal{V}_{11}^{-1} + \mathcal{F}_{12}\,\mathcal{V}_{21}^{-1} + \mathcal{F}_{13}\,\mathcal{V}_{31}^{-1} & \mathcal{F}_{12}\,\mathcal{V}_{22}^{-1} + \mathcal{F}_{13}\,\mathcal{V}_{32}^{-1} & \mathcal{F}_{13}\,\mathcal{V}_{33}^{-1} \\ \hline \mathcal{F}_{21}\,\mathcal{V}_{11}^{-1} + \mathcal{F}_{22}\,\mathcal{V}_{21}^{-1} + \mathcal{F}_{23}\,\mathcal{V}_{31}^{-1} & \mathcal{F}_{22}\,\mathcal{V}_{22}^{-1} + \mathcal{F}_{23}\,\mathcal{V}_{32}^{-1} & \mathcal{F}_{23}\,\mathcal{V}_{33}^{-1} \\ \hline \mathcal{F}_0 & \mathcal{F}_0 & \mathcal{F}_0 \end{array}\right]. \tag{5.12}$$

The control reproduction number $\mathcal{R}_c$ is the dominant eigenvalue of $\mathcal{F}\mathcal{V}^{-1}$ denoted as $\rho\left(\mathcal{F}\mathcal{V}^{-1}\right)$

and is given as follows:

$$\rho\left(\mathcal{F}\mathcal{V}^{-1}\right) = max\left\{\mathcal{R}_c^1, \mathcal{R}_c^2, \mathcal{R}_c^3, \mathcal{R}_c^4, \mathcal{R}_c^5, \mathcal{R}_c^6, \mathcal{R}_c^7, \mathcal{R}_c^8, \mathcal{R}_c^9, \mathcal{R}_c^{10}, \mathcal{R}_c^{11}, \mathcal{R}_c^{12}\right\}. \tag{5.13}$$

See appendix A.4.1 for the matrix entries of $\mathcal{F}\mathcal{V}^{-1}$ and appendix A.5 for the expressions of $\mathcal{R}_c^1$, $\mathcal{R}_c^2$,..., $\mathcal{R}_c^{12}$.

5.4 Parameter Estimation and Simulations

5.4.1 Parameter Estimation

The Kenya Demographic Health Survey (KDHS) of 2014 estimated 21.25% and 24.9% youth aged 15-24 from Homa Bay and Nairobi Counties to be sexually active [38]. A study done by [64] revealed that 20.5% of youth aged $18-24$ in the former Nyanza Province where Homa Bay County is located, reported $5-14$ sexual contacts per month whereas 10% reported 15 or more monthly sexual contacts. Averaging the lower frequency estimate and the upper frequency estimate we calculated the monthly sex contacts for each sexually active Homa Bay youth to be approximately 10. Thus, the yearly sex frequency for each sexually active Homa Bay youth is about 120. Given that Nairobi County has a slightly higher percentage of sexually active youth compared to Homa Bay County, we estimate the annual sexual contacts for each sexually active Nairobi youth to be 125 which is calculated by increasing the annual sex frequency of Homa Bay Youth by 3.65%. Given that mobility influences sexual behavior as youth living in counties different from their origin tend to be influenced into the social norms and cultural practices of their new environment we adjust the annual sexual contact rates for the migrant youth in Homa Bay County and Nairobi County c_{12} and c_{21} to 122 and 123 respectively [133].

Increased sexual contacts increases the overall risk of HIV/AIDS transmission. A single exposure can easily result in HIV/AIDS transmission [131]. Transmission risk is even higher if a HIV/AIDS infected youth has a high viral load and biological factors such as weakened immunity or sexually transmitted diseases among others present in either partners compounds the risk of HIV/AIDS transmission. Study done by [131] estimated the male-female HIV/AIDS transmission risk in low income countries to be 0.38% whereas the female-male transmission risk in low income countries was approximately 0.38%. Given that HIV/AIDS is hyper-endemic in Homa Bay and Nairobi Counties, the annual HIV/AIDS transmission risk in migrant Homa Bay and Nairobi Counties youth γ_{12}, γ_{21} are approximately 2% and 0.34%. This is obtained by averaging the male-female and the female-male transmission risk in low income countries and taking into consideration that the youth in Nairobi County have a greater susceptibility to new HIV/AIDS infections.

Homa Bay County's life expectancy is 57 years and Nairobi County's life expectancy is estimated

at 69.3 years [134]. Hence, the annual natural mortality rates μ_{11}, μ_{22} for residents in Homa Bay and Nairobi Counties are calculated as 1/57 and 1/69.3 respectively. The mortality rates for youth living in Counties different from their origin, μ_{12}, μ_{21} are adjusted from the mortality rates of their County of origin and estimated at 0.0168, 0.0165. This is because most of the migrants adopt new lifestyles practiced in their new environments which in turn affects their mortality rates [133]. HIV/AIDS infected youth continue to lose their lives due to HIV/AIDS related complications with 246 and 294 deaths in Homa Bay and Nairobi Counties respectively in 2017 [2]. Disease induced deaths for youths born and living in their Counties of origin δ_{11}, δ_{22} is calculated as 246/19050 and 294/24918 with 19,050 and 24,918 being the estimated HIV/AIDS infected youth aged $15-24$ in Homa Bay and Nairobi Counties in 2017 [2]. Considering availability and accessibility to health services in the mentioned Counties, the disease induced death rate for migrant HIV/AIDS infected youth δ_{12}, δ_{21} is estimated at 0.018, 0.0119 respectively.

The $15-24$ age cohort in Kenya has the highest mobility rates [133, 135]. Most of the youth migrating from rural environments such as Homa Bay County in search for job opportunities in Urban areas such as Nairobi County settle in informal settlements exposing them to diverse disadvantages such as morbidity, mortality, risky sexual behaviors and inadequate health services [136]. Return migration rate from urban to rural environments is low among the youth [133]. Using the migration patterns of the youth in Kenya, that is, high departure rates and low return rates, we estimate the departure rate from Homa Bay County to Nairobi County d_{21} at 0.1016% whereas the return rate from Nairobi County to Homa Bay County r_{21} is approximately 0.01016%. Also, the departure rate from Nairobi County to Homa Bay County d_{12} is approximated at 0.09% whereas the return rate from Homa Bay County to Nairobi County r_{12} is about 0.05%. Using the disease free state expressions given in section 5.3.2 we estimate recruitment rates Λ_{11}^u, Λ_{11}^a, Λ_{12}^u, Λ_{12}^a, Λ_{21}^u, Λ_{21}^a , Λ_{22}^u and Λ_{22}^a whose values are given in table 5.3. The youth exit the model system at the age of 24 hence, the exit rates σ_{11}, σ_{12}, σ_{21}, σ_{22} are calculated as 1/24.

The total population of adolescents and young adults aged $15-24$ in Homa Bay and Nairobi Counties are estimated at 279,862 and 749,369 respectively [38, 137]. The 2018 HIV/AIDS estimate by National AIDS Control Council approximated HIV/AIDS infected youth aged $15-24$ to be about 19,050 and 24,918 respectively [2]. Subtracting the infected populations from the total populations and using the National youth HIV/AIDS testing rate of 65.25% and departure rates d_{12}, d_{21} yields the initial conditions $S_{11}^u = 83,490$, $S_{22}^u = 229,088$, $S_{11}^a = 156,770$, $S_{22}^a = 430,161$, $S_{12}^u = 22,658$, $S_{21}^u = 9442$, $S_{12}^a = 42,544$, and $S_{21}^a = 17,729$ [47]. We use the National HIV/AIDS testing rate for the adolescents and young adults to further compute the infected unaware and infected aware populations. About 31.95% of infected aware populations are initiated into ART [138]. After two years, approximately 34% of HIV/AIDS infected youth initiated on ART are lost to follow up [138]. Using the National HIV/AIDS testing rate, initiation to ART rate, lost to follow-up rate and departure rates yields $I_{11}^u = 5946$, $I_{22}^u = 7879$, $I_{11}^a = 7599$, $I_{22}^a = 10068$, $I_{12}^u = 780$, $I_{21}^u = 673$, $I_{12}^a = 996$, $I_{21}^a = 860$, $T_{11}^u = 1213$, $T_{22}^u = 1605$, $T_{11}^a = 2354$, $T_{22}^a = 3122$, $T_{12}^u = 159$, $T_{21}^u = 138$, $T_{12}^a = 309$ and $T_{21}^a = 267$. Table 5.3 gives the parameter values used in the calibrations.

Table 5.3: Parameter Values

Parameter	Value	Unit	Source
c_{12}, c_{21}	122, 123	$year^{-1}$	[38, 64]
γ_{12}, γ_{21}	0.02, 0.0034	$year^{-1}$	[131]
δ_{11}, δ_{12}, δ_{21}, δ_{22}	0.01291339, 0.018, 0.0119, 0.0117987	$year^{-1}$	[2, 133]
μ_{11}, μ_{12}, μ_{21}, μ_{22}	0.01754386, 0.0168, 0.0165, 0.01587302	$year^{-1}$	[134]
Λ^u_{11}, Λ^a_{11}	10,008, 20,389	$year^{-1}$	Calculated
Λ^u_{12}, Λ^a_{12}	1008, 2389	$year^{-1}$	Calculated
Λ^u_{21}, Λ^a_{21}	908, 1900	$year^{-1}$	Calculated
Λ^u_{22}, Λ^a_{22}	9008, 19,000	$year^{-1}$	Calculated
d_{12}, d_{21}	0.0009, 0.001016	$year^{-1}$	Estimated
r_{12}, r_{21}	0.0005, 0.0001016	$year^{-1}$	Estimated
σ_{11}, σ_{12}	0.04166667, 0.04166667	$year^{-1}$	Calculated
σ_{21}, σ_{22}	0.04166667, 0.04166667	$year^{-1}$	Calculated

The parameter values given in table 5.3 together with negative and positive County attitudes affecting HIV/AIDS controls rates given in tables 5.4 and 5.5 are used to calibrate the model systems (5.3) - (5.6) and the control reproduction number given in section 5.3.3 for the low and high efficacy controls respectively.

Table 5.4: Estimates for negative County attitudes and low HIV/AIDS control rates

Parameter	Value	Unit	Source
ρ^{ht}_{11}, ρ^{ht}_{12}, ρ^{ht}_{21}, ρ^{ht}_{22}	0.48, 0.48, 0.48, 0.48	$year^{-1}$	Estimated
ρ^{11}_{c}, ρ^{c}_{12}, ρ^{c}_{21}, ρ^{c}_{22}	0.3, 0.3, 0.3, 0.3	$year^{-1}$	Estimated
ρ^{t}_{11}, ρ^{t}_{12}, ρ^{t}_{21}, ρ^{t}_{22}	0.1, 0.1, 0.1, 0.1	$year^{-1}$	Estimated
α^{ht}_{11}, α^{ht}_{12}, α^{ht}_{21}, α^{ht}_{22}	0.6, 0.65, 0.7, 0.55	$year^{-1}$	Estimated
$\tilde{\alpha}^{ht}_{11}$, $\tilde{\alpha}^{ht}_{12}$, $\tilde{\alpha}^{ht}_{21}$, $\tilde{\alpha}^{ht}_{22}$	0.7, 0.8, 0.8, 0.7	$year^{-1}$	Estimated
α^{c}_{11}, α^{c}_{12}, α^{c}_{21}, α^{c}_{22}	0.6, 0.65, 0.7, 0.55	$year^{-1}$	Estimated
$\tilde{\alpha}^{c}_{11}$, $\tilde{\alpha}^{c}_{12}$, $\tilde{\alpha}^{c}_{21}$, $\tilde{\alpha}^{c}_{22}$	0.7, 0.8, 0.8, 0.7	$year^{-1}$	Estimated
α^{t}_{11}, α^{t}_{12}, α^{t}_{21}, α^{t}_{22}	0.6, 0.65, 0.7, 0.55	$year^{-1}$	Estimated
$\tilde{\alpha}^{t}_{11}$, $\tilde{\alpha}^{t}_{12}$, $\tilde{\alpha}^{t}_{21}$, $\tilde{\alpha}^{t}_{22}$	0.7, 0.8, 0.8, 0.7	$year^{-1}$	Estimated

Table 5.5: Estimates for positive County attitudes and high HIV/AIDS control rates

Parameter	Value	Unit	Source
ρ_{11}^{ht}, ρ_{12}^{ht}, ρ_{21}^{ht}, ρ_{22}^{ht}	0.9, 0.9, 0.9, 0.9	$year^{-1}$	Estimated
ρ_{c}^{11}, ρ_{12}^{c}, ρ_{21}^{c}, ρ_{22}^{c}	0.9, 0.9, 0.9, 0.9	$year^{-1}$	Estimated
ρ_{11}^{t}, ρ_{12}^{t}, ρ_{21}^{t}, ρ_{22}^{t}	0.9, 0.9, 0.9, 0.9	$year^{-1}$	Estimated
α_{11}^{ht}, α_{12}^{ht}, α_{21}^{ht}, α_{22}^{ht}	0.1, 0.1, 0.1, 0.1	$year^{-1}$	Estimated
$\tilde{\alpha}_{11}^{ht}$, $\tilde{\alpha}_{12}^{ht}$, $\tilde{\alpha}_{21}^{ht}$, $\tilde{\alpha}_{22}^{ht}$	0.9, 0.8, 0.8, 0.9	$year^{-1}$	Estimated
α_{11}^{c}, α_{12}^{c}, α_{21}^{c}, α_{22}^{c}	0.1, 0.1, 0.1, 0.1	$year^{-1}$	Estimated
$\tilde{\alpha}_{11}^{c}$, $\tilde{\alpha}_{12}^{c}$, $\tilde{\alpha}_{21}^{c}$, $\tilde{\alpha}_{22}^{c}$	0.9, 0.8, 0.8, 0.9	$year^{-1}$	Estimated
α_{11}^{t}, α_{12}^{t}, α_{21}^{t}, α_{22}^{t}	0.1, 0.1, 0.1, 0.1	$year^{-1}$	Estimated
$\tilde{\alpha}_{11}^{t}$, $\tilde{\alpha}_{12}^{t}$, $\tilde{\alpha}_{21}^{t}$, $\tilde{\alpha}_{22}^{t}$	0.9, 0.8, 0.8, 0.9	$year^{-1}$	Estimated

5.4.2 $\mathcal{R}_c$ Simulation Results

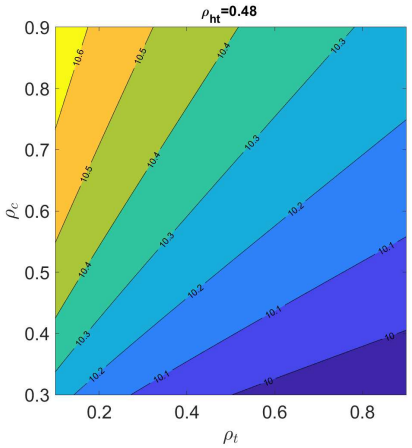
Using the parameter values given in section 5.4.1 we are able to determine the four largest spectral radii of $\mathcal{F}\mathcal{V}^{-1}$ given as

$$\rho\left(\mathcal{F}\mathcal{V}^{-1}\right) = \mathcal{R}_c^1, \mathcal{R}_c^2, \mathcal{R}_c^3, \mathcal{R}_c^4 \tag{5.14}$$

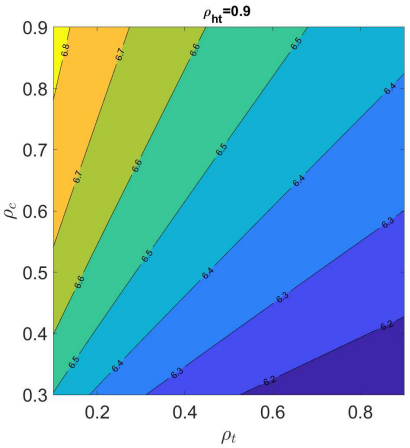
where $\mathcal{R}_c^1 = 12.4661$ is the local number of secondary infections resulting from interactions between migrant youth in Nairobi County with susceptible youth in Nairobi County who are not aware of their HIV/AIDS status, $\mathcal{R}_c^2 = 10.2644$ is the local number of secondary infections resulting from interactions between migrant youth in Homa Bay County with susceptible youth in Homa Bay County who are not aware of their HIV/AIDS status, $\mathcal{R}_c^3 = 8.5143$ is the local number of secondary infections resulting from interactions between migrant youth in Nairobi County with susceptible youth in Nairobi County who are aware of their HIV/AIDS status and $\mathcal{R}_c^4 = 6.8105$ is the local number of secondary infections resulting from interactions between migrant youth in Homa Bay County with susceptible youth in Homa Bay County who are aware of their HIV/AIDS status.

Figures 5.3 and 5.4 show the change in the local control reproduction number when HIV/AIDS testing rate, ρ_{ht} is fixed at low efficacy rate ($\rho_{ht} = 0.48$) and high efficacy rate ($\rho_{ht} = 0.9$) while condom use rate, ART usage and HIV/AIDS testing rate are varied from low efficacy rates of 0.3 and 0.1 respectively to high efficacy rates of 0.9 when interaction is between infected migrant youth and susceptible unaware/aware youth in County of origin. Figures 5.3 - 5.4 reflect the change in the control reproduction number among the migrating youth in Homa Bay and Nairobi Counties when departure rates are higher than the return rates. Figures 5.5, 5.6 and 5.7 show the change in the local control reproduction number when interaction is between the unaware/aware Nairobi youth and migrant Homa Bay youth with high return rates and low departure rates, high return and high departure rates and low return and low departure rates respectively .

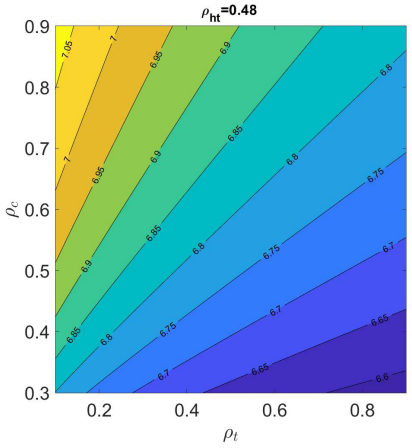
High departure rates and high return rates, high departure rates and low return rates and low departure rates and low return rates have little effect in increasing the control reproduction number among migrant Homa Bay youth and Nairobi youth unaware of their HIV/AIDS status (see figures 5.4, 5.6, 5.7) with slightly high new HIV/AIDS infection among the HIV/AIDS tested youth in Nairobi County who are aware of their HIV/AIDS status. This qualitative results is similar for interactions between migrating Nairobi youth and susceptible unaware/aware youth in Homa Bay County.



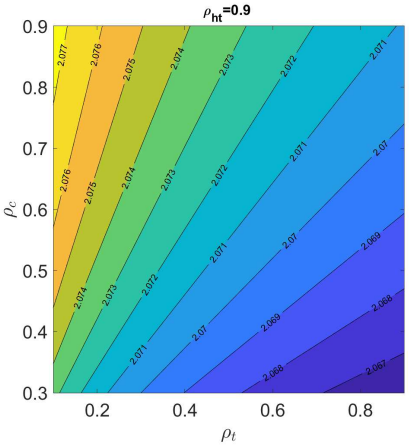
(a) Change in $\mathcal{R}_c$ with low ρ_{ht} and varying ρ_c and ρ_t in unaware Homa Bay County youth



(b) Change in $\mathcal{R}_c$ with high ρ_{ht} and varying ρ_c and ρ_t in unaware Homa Bay County youth

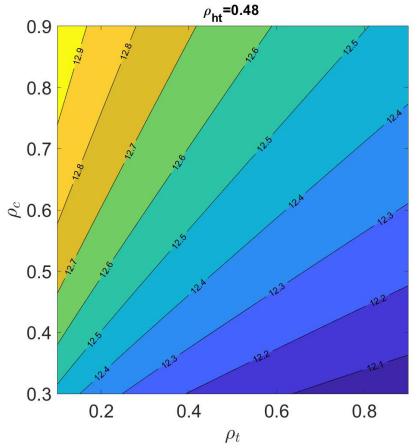


(c) Change in $\mathcal{R}_c$ with low ρ_{ht} and varying ρ_c and ρ_t in aware Homa Bay County youth

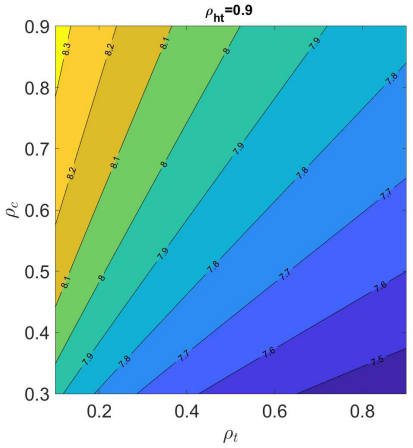


(d) Change in $\mathcal{R}_c$ with high ρ_{ht} and varying ρ_c and ρ_t in aware Homa Bay County youth

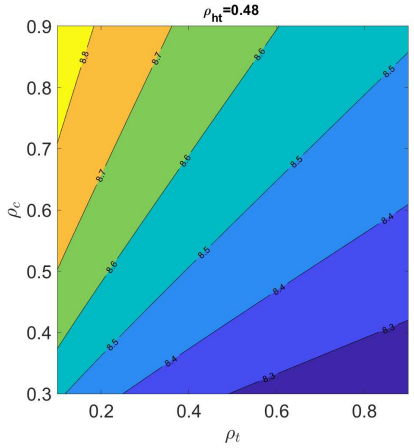
Figure 5.3: Change in the local control reproduction number $\mathcal{R}_c$ with fixed HIV/AIDS testing rate ρ_{ht}, varying condom use rate ρ_c and ART usage rate ρ_t when interaction is between unaware/aware Homa Bay youth and infected nairobi migrant youth in Homa Bay with high departure rate d_{21} and low return rate r_{21} given in table 5.3.



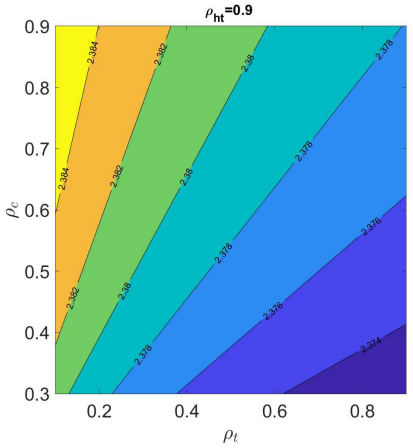
(a) Change in $\mathcal{R}_c$ with low ρ_{ht} and varying ρ_c and ρ_t in unaware Nairobi County youth



(b) Change in $\mathcal{R}_c$ with high ρ_{ht} and varying ρ_c and ρ_t in unaware Nairobi County youth

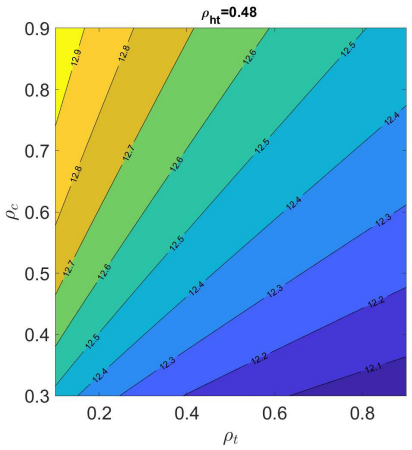


(c) Change in $\mathcal{R}_c$ with low ρ_{ht} and varying ρ_c and ρ_t in aware Nairobi County youth

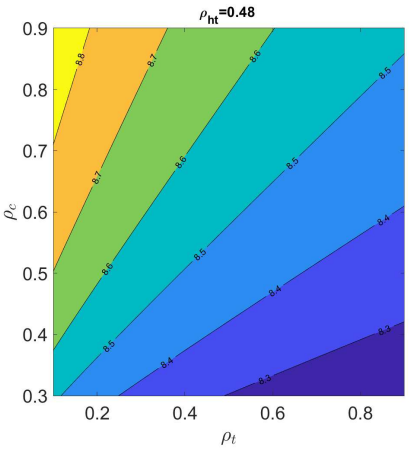


(d) Change in $\mathcal{R}_c$ with high ρ_{ht} and varying ρ_c and ρ_t in aware Nairobi County youth

Figure 5.4: Change in the local control reproduction number $\mathcal{R}_c$ with fixed HIV/AIDS testing rate ρ_{ht}, varying condom use rate ρ_c and ART usage rate ρ_t when interaction is between unaware/aware Nairobi youth and infected Homa Bay migrant youth in Nairobi with high departure rate d_{21} and low return rate r_{21} given in table 5.3.

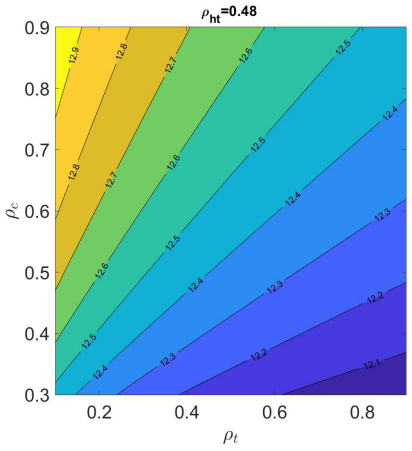


(a) Change in $\mathcal{R}_c$ with low ρ_{ht} and varying ρ_c and ρ_t in unaware Nairobi County youth

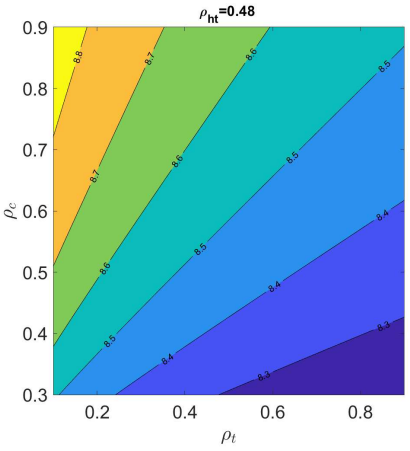


(b) Change in $\mathcal{R}_c$ with high ρ_{ht} and varying ρ_c and ρ_t in aware Nairobi County youth

Figure 5.5: Change in the local control reproduction number $\mathcal{R}_c$ with fixed HIV/AIDS testing rate ρ_{ht}, varying condom use rate ρ_c and ART usage rate ρ_t when interaction is between unaware/aware Nairobi youth and infected Homa Bay migrant youth in Nairobi with high departure rate d_{21} and low return rate r_{21} given in table 5.3.

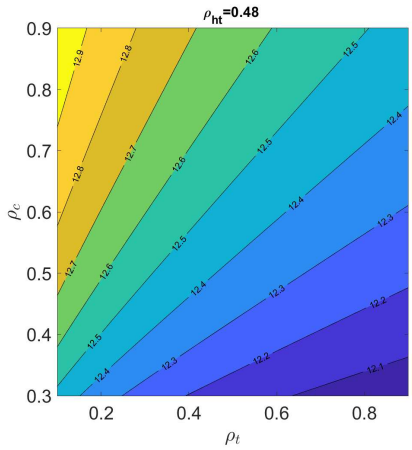


(a) Change in $\mathcal{R}_c$ with low ρ_{ht} and varying ρ_c and ρ_t in unaware Nairobi County youth

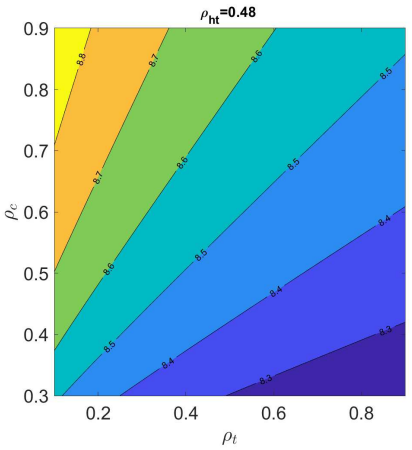


(b) Change in $\mathcal{R}_c$ with high ρ_{ht} and varying ρ_c and ρ_t in aware Nairobi County youth

Figure 5.6: Change in the local control reproduction number $\mathcal{R}_c$ with fixed HIV/AIDS testing rate ρ_{ht}, varying condom use rate ρ_c and ART usage rate ρ_t when interaction is between unaware/aware Nairobi youth and infected Homa Bay migrant youth in Nairobi with high departure rate $d_{21} = 0.1016$ and high return rate $r_{21} = 0.01016$.



(a) Change in $\mathcal{R}_c$ with low ρ_{ht} and varying ρ_c and ρ_t in unaware Nairobi County youth



(b) Change in $\mathcal{R}_c$ with high ρ_{ht} and varying ρ_c and ρ_t in aware Nairobi County youth

Figure 5.7: Change in the local control reproduction number $\mathcal{R}_c$ with fixed HIV/AIDS testing rate ρ_{ht}, varying condom use rate ρ_c and ART usage rate ρ_t when interaction is between unaware/aware Nairobi youth and infected Homa Bay migrant youth in Nairobi with low departure rate $d_{21} = 0.0001016$ and low return rate $r_{21} = 0.00001016$.

Low HIV/AIDS testing rate coupled with increased negative County attitudes negating the efficacy of HIV/AIDS controls and increasing condom use and ART usage rates reduces the control reproduction number slightly among the migrating youth. High HIV/AIDS testing rate, increased positive factors encouraging the efficacy of HIV/AIDS controls, increasing condom use and ART usage rates significantly reduces the control reproduction number (see figures 5.3 - 5.4). Interaction between HIV/AIDS infected migrant youth and susceptible youth in County of origin may still sustain the HIV/AIDS epidemic despite high efficacy in condom use, ART usage and HIV/AIDS testing rates and increased positive factors influencing the uptake of the said HIV/AIDS controls (see figures 5.3(d) and 5.4(d)). Varying departure rates and return rates have little effect in increasing new HIV/AIDS infections in the two high risk Counties. Kenya's HIV/AIDS reproduction number is not really clear but [60] study estimated it to be about 6.3 using antenatal clinic data. Simulations with migration present estimates the control reproduction numbers for Nairobi and Homa Bay Counties youth who are unaware of their HIV/AIDS status to be about 12.5 and 10.3 whereas youth who have tested and are aware of their HIV/AIDS status in the two high risk Counties are approximately 8.5 and 6.8 respectively. This reflects the HIV/AIDS burden among the youth in both Counties as the number of new HIV/AIDS infection is much greater in Nairobi County in comparison to Homa Bay County [2].

5.4.3 Model Simulation Results

Figures 5.8 - 5.10 show the time series solution for model systems (5.3) - (5.6) subject to low and high controls, low and high controls attitudes given in tables 5.4 - 5.5 and high departure rates and low return rates given in table 5.3 among the Homa Bay and Nairobi youth.

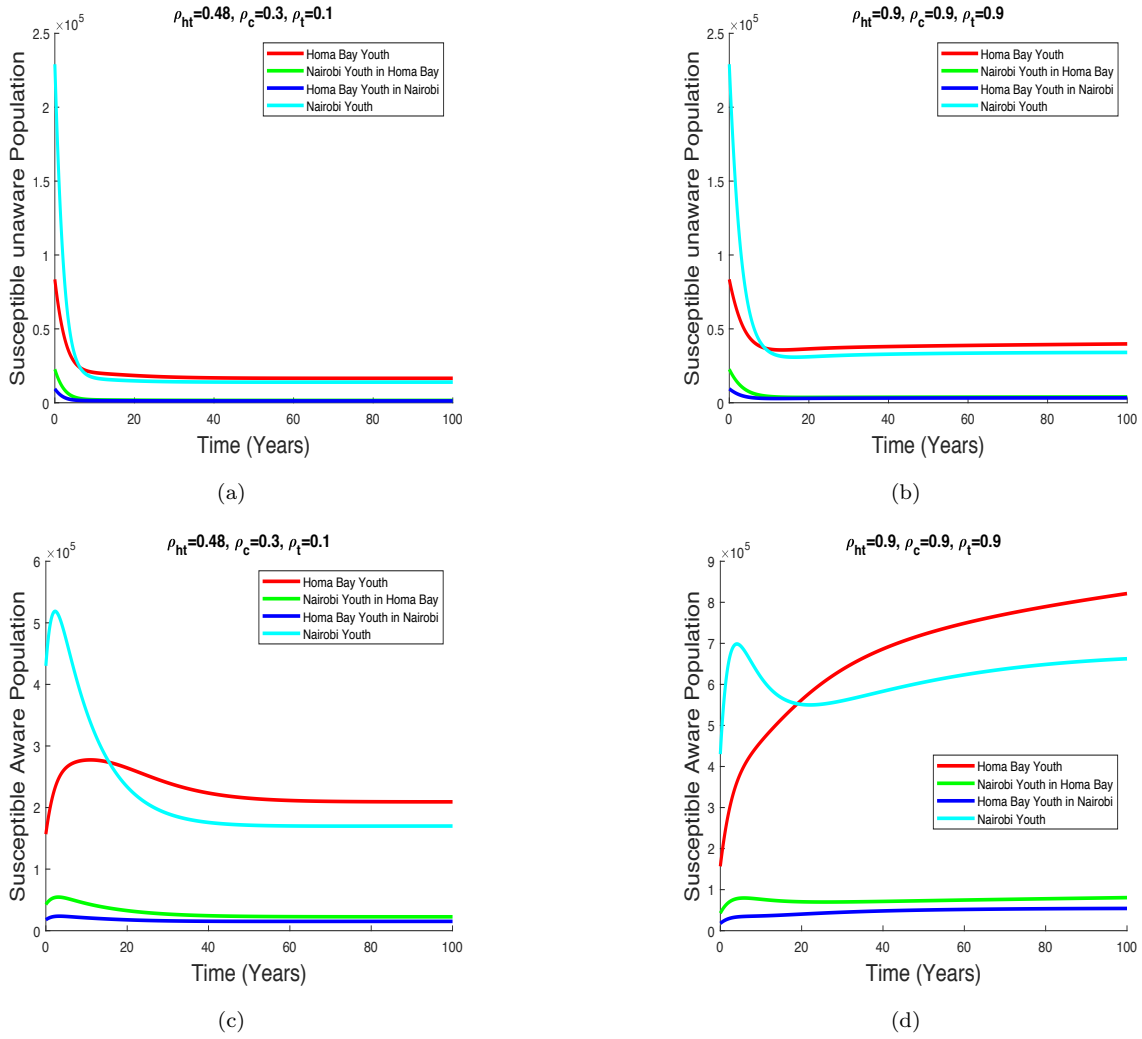
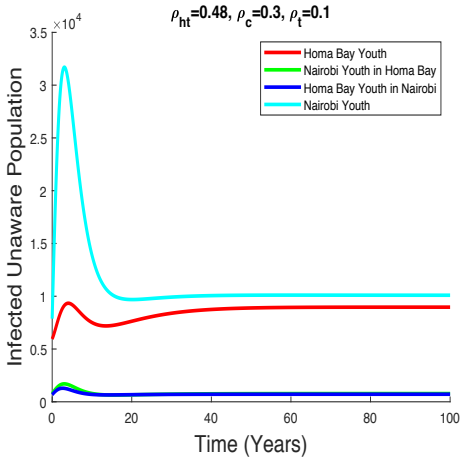
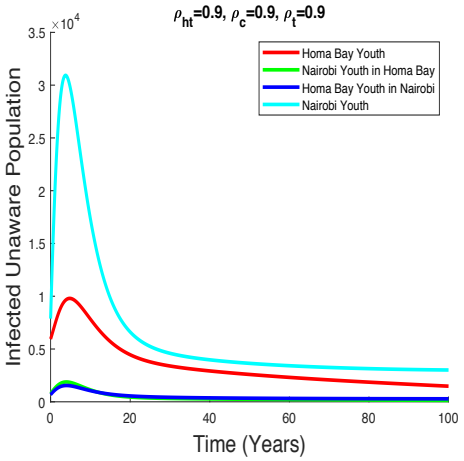


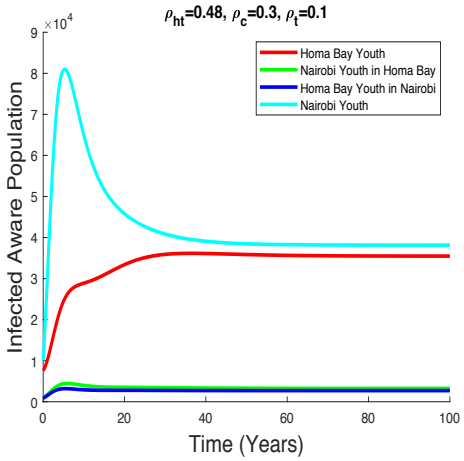
Figure 5.8: Transmission Dynamics of susceptible populations with varying control, high departure rates and low return rates .



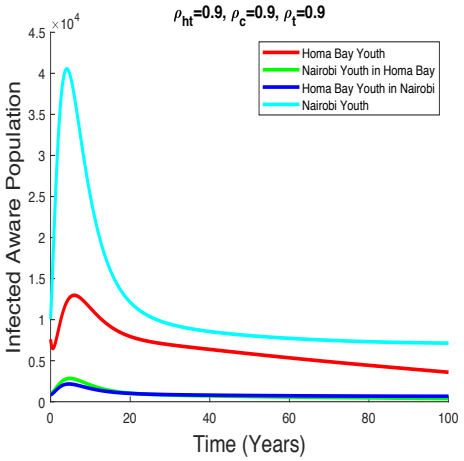
(a)



(b)



(c)



(d)

Figure 5.9: Transmission Dynamics of infected unaware and aware populations with varying control, high departure rates and low return rates .

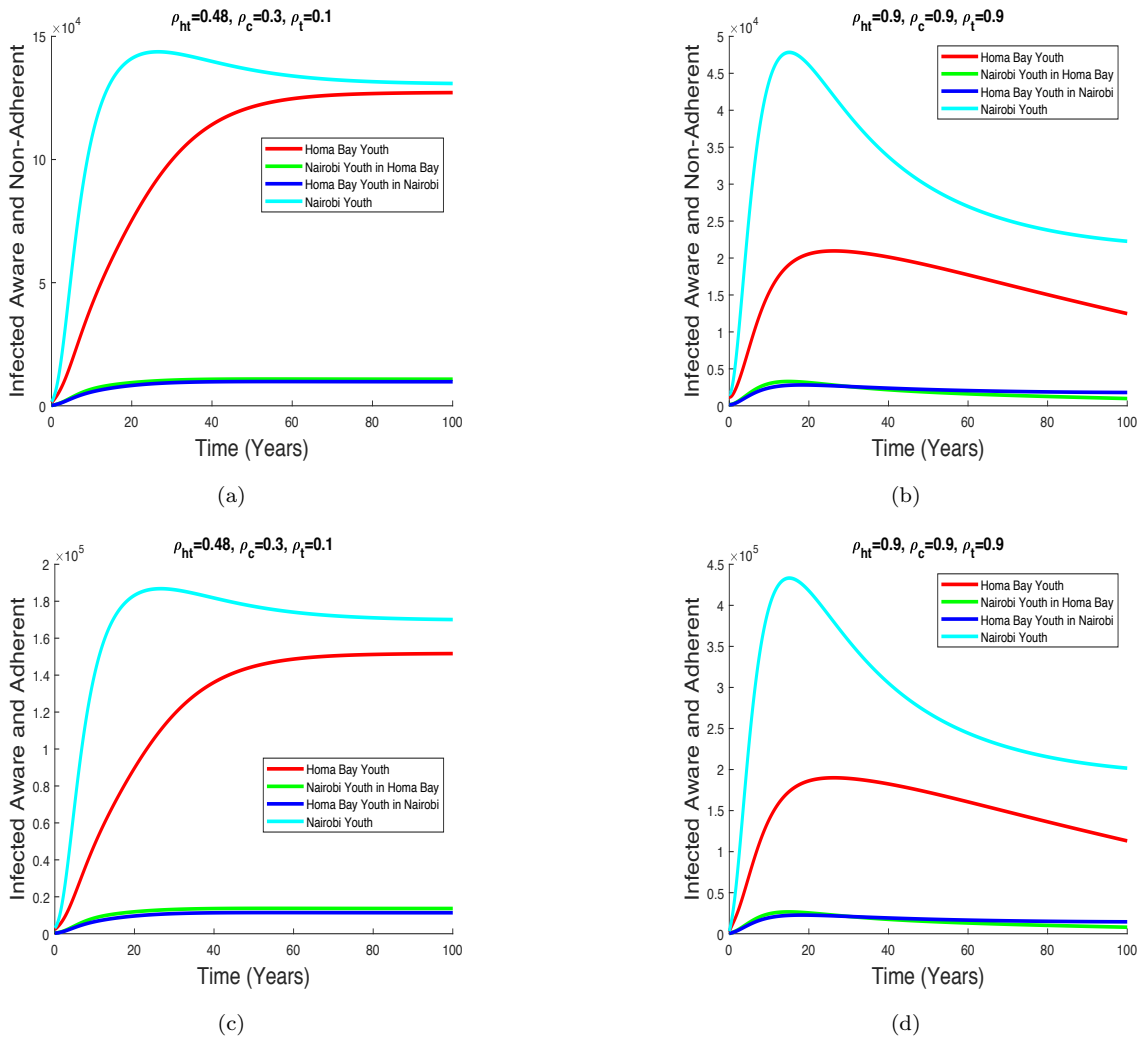


Figure 5.10: Transmission Dynamics of infected aware (non-adherent and adherent to condom use and ART respectively) populations with varying control, high departure rates and low return rates .

Susceptible unaware populations have the highest risk of new HIV/AIDS infections in both Counties and among the migrant youth with better results when HIV/AIDS controls are high. Increased HIV/AIDS controls such as HIV/AIDS testing, condom use and ART adherence among the infected populations with increased positive County attitudes promoting the efficacy of the said controls in both Counties while maintaining the present youth migration patterns offers hope for significant HIV/AIDS disease reduction in both high risk Counties.

5.5 Discussion

We studied the impact of migration on HIV/AIDS disease dynamics among the youth aged $15-24$ in Homa Bay County and Nairobi County in Kenya. This model formulation considered

combinatory HIV/AIDS controls such as HIV/AIDS testing, condom use and ART use paired with factors promoting or negating their efficacy such as poverty, health services accessibility, psycho-social conditions, community norms among others. We varied the HIV/AIDS testing rate, condom use rate and ART adherence rate from low efficacy rates of 0.48, 0.3 and 0.1 to high efficacy rates of 0.9 for each control respectively. Increased negative County attitudes reduced the efficacy of controls in low control simulations whereas increased positive County attitudes promoted the efficacy of the HIV/AIDS controls in high control simulations. This formulation considered a homogeneous population differentiated according to their HIV/AIDS status knowledge such as susceptible aware and unaware, infected aware and unaware, infected aware adherent to condom use and ART use and infected aware non-adherent to condom use and ART usage. Unaware populations could reverse their status through HIV/AIDS testing, condom use and ART use.

Low control simulations on the control reproduction number revealed that new HIV/AIDS infections in both Homa Bay County and Nairobi County could be significantly reduced when infected migrant youth interacted with HIV/AIDS tested and aware youth in County of origin who were willing to disclose their HIV/AIDS status. Otherwise interactions between infected migrant youth and youth in County of origin who are not aware of their HIV/AIDS status propagates the HIV/AIDS scourge. Fixed high HIV/AIDS testing rate with increasing condom use and ART usage under the current defining migration patterns among the youth in these Counties, that is, increased departure rates and reduced return rates, reduces new HIV/AIDS infection significantly. This strategy offers hope for HIV/AIDS disease reduction in both Counties. Migration has little effect in increasing new HIV/AIDS infections in both high risk Counties. Numerical simulations on our model system under the current defining migration patterns among the Homa Bay youth and Nairobi youth with increased disease transmission risk revealed that low HIV/AIDS controls are actually helpful in reducing the HIV/AIDS infected populations but better results are realized when HIV/AIDS controls are high. Further, uninfected unaware populations in both Counties and the migrant uninfected unaware youth are most susceptible to new HIV/AIDS infections.

Despite the modest progress in HIV/AIDS response in Kenya, implementation of HIV/AIDS controls among the youth continue to face numerous societal and governance challenges [47]. The current strategy seeks to upscale the HIV/AIDS control efforts in high risk regions while prioritizing high risk population groups such as the adolescents and young adults [2, 47]. Our model results adds to the ample evidence that effective HIV/AIDS response must have adequate cooperation of the entire society and the government. It is clear from our results that while interaction between the youth from these high-risk Counties sustains the HIV/AIDS pandemic under low targeted HIV/AIDS controls paired with increased negative societal attitudes, migration has little effect in increasing new HIV/AIDS infections in these Counties. Regardless of migration patterns among the youth in these Counties, it is necessary for each County to scale up HIV/AIDS testing rate, condom use and increase ART initiation among the infected populations with their adherence rates monitored. Further, the entire society and the County governments must work together to provide, promote and encourage the youth to consider effective use of HIV/AIDS controls.

To the best of our knowledge, there are no existing mathematical models that have considered the effects of multiple controls paired with either negative or positive factors influencing their efficacy among the mobile youth in Homa Bay and Nairobi Counties with differentiated HIV/AIDS status knowledge. Study done by [33] agree with our finding that migration is not quite significant in driving HIV/AIDS disease in Kenya. It will be interesting to further study the cost benefit analysis of multiple control strategies of the HIV/AIDS disease among the youth as well as study the individual based model for this formulation. Given the behavior heterogeneity among the youth, studying each individual behavior explicitly could offer greater insight in understanding the disease dynamics.

Chapter 6

Conclusion and Future Work

6.1 Conclusion

This thesis centered on constructing and analyzing epidemiological mathematical models to understand the transmission dynamics of HIV/AIDS among adolescents and young adults populations in Kenya. Considering the transmission dynamics of HIV/AIDS among the youth, various controls were incorporated into the models developed.

A detailed presentation of the background of HIV/AIDS disease was given in Chapter one. Particular information of the disease highlighted are background details, biology of HIV/AIDS immunology and transmission, global picture of the HIV/AIDS disease, current state of HIV/AIDS disease in Kenya, disease control measures, research motivation, research objectives, study outline and manuscripts that have built the thesis.

In Chapter 2, we considered the effects of increasing comprehensive knowledge of HIV/AIDS disease in the infected adolescents girls and young women and, adolescent boys and young men populations in Kenya. The infected populations were differentiated according to their comprehensive knowledge of HIV/AIDS. Simulations on the local control reproduction number with comprehensive knowledge of HIV/AIDS among the youth as a sole control estimated the control reproduction number to be 6 which was quite close to the Kenyan estimate of 6.3 provided in [100]. The model results suggest that protecting susceptible adolescent girls and young women against new HIV/AIDS infection required increasing adolescent boys and young men HIV/AIDS knowledge; also to protect susceptible adolescent boys and young men populations against new HIV/AIDS infections required increasing adolescent girls and young women HIV/AIDS knowledge. Considering increasing HIV/AIDS comprehensive knowledge as a sole control results in decreased infected populations without comprehensive knowledge of HIV/AIDS and increased infected populations with comprehensive knowledge of HIV/AIDS who stabilize at high population values posing a significant threat to susceptible populations. While effective dispensation of comprehensive knowledge of HIV/AIDS in all the newly infected populations significantly slowed

down infection spread, it did not eradicate the disease spread among the youth in Kenya. A similar conclusion was arrived at in [26] which used data from numerous countries where they considered a homogeneous population and integrated public education campaign. Our study further investigated the gender-wise effects of educating infectious Kenyan youth on HIV/AIDS. It is possible adolescent girls and young women in Kenya are receiving greater attention due to their vulnerability and that they are twice likely to be infected with HIV/AIDS when compared to their male peers. Our results suggest that such efforts work to protect the adolescent boys and young men ultimately.

In Chapter 3, we further investigated the effects of combinatory control given that comprehensive knowledge of HIV/AIDS alone was not sufficient in reducing the infected youth populations in Kenya. We looked at the effects of varying HIV/AIDS testing rates, condom use rates and adherence rates among the adolescent girls and young women and adolescent boys and young men populations. The susceptible and infected populations were each differentiated according to their HIV/AIDS status awareness. Also considered were attitudes affecting the efficacy of the HIV/AIDS controls both positively and negatively. With combined HIV/AIDS controls paired with constant negative/positive attitudes affecting the three considered controls, simulations on the control reproduction number dropped down to about 4.1003 against a high of 6 when comprehensive knowledge of HIV/AIDS was the only considered control. Our findings suggest that while highly efficacious combinatory control approach significantly reduces HIV/AIDS prevalence rates among the adolescent and young adult populations, the disease remains endemic provided adolescents and young adults who are yet to know their HIV/AIDS status continue to interact sexually. Study done by [25] considered multiple control strategies such as HIV/AIDS screening, ARV drug treatment and condom use in a homogeneous population to understand the potential impact on the current HIV/AIDS controls. Their results reflected the projections of HIV/AIDS epidemic trends when controls and risky sexual behavior varied. While study done by [25] considered a homogeneous population we further considered similar controls on a sex-structured youth model with differentiated populations according to HIV/AIDS status knowledge. Our study further revealed that disproportional gender-wise attitudes towards HIV/AIDS controls played a key role in reducing the Kenyan youth HIV/AIDS prevalence trends.

In Chapter 4, we investigated the role of migration on HIV/AIDS disease dynamics among the youth in Homa Bay and Nairobi Counties in Kenya. We considered targeted HIV/AIDS controls such as HIV/AIDS testing, condom use and ART use coupled with County attitudes promoting or negating their efficacy. The model results suggest that movement between the two high risk Counties has little effect in increasing new HIV/AIDS infections. Fewer HIV/AIDS tested youth who are aware of their HIV/AIDS status in Homa Bay and Nairobi Counties and are willing to disclose their HIV/AIDS status are reduced to new HIV/AIDS infection. While migration has little effect on the overall increase of new HIV/AIDS infection in the considered high risk Counties, increase in new HIV/AIDS infections is propagated by sexual interactions with youth who are unaware of their HIV/AIDS status. HIV/AIDS study by [59] considered movement between different regions in Kenya and they similarly observed that migration increases HIV/AIDS in

a relatively small way. Our study further revealed that the youth who are not aware of their HIV/AIDS status in both their home and host Counties, will sustain the HIV/AIDS pandemic.

6.2 Recommendations

These results have very important implications for HIV/AIDS control, hence we make the following recommendations:

1. Where public health education against HIV/AIDS is the sole control of reducing new HIV/AIDS infections among the youth, dispensation of HIV/AIDS knowledge should cut across both male and female youth populations. Should a particular gender be targeted, then HIV/AIDS knowledge should be increased in the opposite gender.

2. Aside from scaling up HIV/AIDS testing, condom use and ART usage, societal negative attitudes towards the use of these controls by the sexually active youth should be discouraged. Greater attention should be given in educating the Kenyan society and the health sector to highly encourage the sexually active youth on responsible sexual behaviour.

3. There should be increased efforts in scaling up HIV/AIDS testing among the youth in Kenya given that youth who are yet to know their HIV/AIDS status are most at risk of new HIV/AIDS infections. HIV/AIDS tested youth who are aware of their HIV/AIDS status should be encouraged to disclose their status to their sex partner(s) and use protection as our model results showed that HIV/AIDS status disclosure and perfect adherence to condom use and antiretroviral therapy reduces new HIV/AIDS infections significantly.

4. Care should be given on the use of HIV/AIDS eradication or HIV/AIDS elimination in Kenya as our study showed that even with 90% HIV/AIDS testing , 90% condom use and 90% ART adherence the youth population who are not aware of their HIV/AIDS status may still sustain the HIV/AIDS pandemic. Eradication of HIV/AIDS among the youth in Kenya may not be possible unless the entire youth population is tested for HIV/AIDS and perfect adherence of condom use and ART maintained or for each sexual relationship established among the youth at least one partner ought to have tested for HIV/AIDS and should be willing to disclose his/her HIV/AIDS status and adhere perfectly to condom use and ART.

6.3 Suggestions for Future Work

This study has managed to provide significant details to the existing knowledge regarding the spread of HIV/AIDS among the youth. This research could further improve by considering the following :

1. The cost benefit analysis of the combination control strategies to ensure maximum benefits while minimizing costs. This will help in designing the best choice controls with the greatest impact while saving costs.

2. Dynamic attitudes towards HIV/AIDS controls: this research considered constant negative/positive attitudes affecting HIV/AIDS controls among the youth. It will be interesting to look at the impact of dynamic attitudes and its effect in HIV/AIDS controls among the youth.

3. Behavior heterogeneity among the adolescents and young adults: studying each individual behavior explicitly to population level could shed more light in understanding the social drivers of HIV/AIDS in this population.

Appendix

A.1 Definitions and Terminologies

Young Adult

We define young adults here to be person(s) in the $15-24$ age cohort according to the United Nations definition for young adults or youth [139].

Adolescents

Adolescents are defined as those persons between the ages of 10 and 19 years according to United Nations Children's Fund (UNICEF) definition for adolescents [139].

Youth

Simplified term for adolescents and young adults.

Reproduction Number

The reproduction number $\mathcal{R}_0$ is defined as the average number of secondary cases arising from an average primary case in an entirely susceptible population over the period of infection. The reproduction number is used to predict whether the epidemic will spread or die out. Any epidemiological model has a disease free equilibrium (DFE) at which the population remains in the absence of the disease. The basic reproduction number is such that if $\mathcal{R}_0 < 1$ then the DFE is locally asymptotically stable and the disease dies out but if $\mathcal{R}_0 > 1$ then the DFE is unstable and the epidemic spreads. At the endemic equilibrium, the average replacement number is one. If $\mathcal{R}_0 = 1$, and this is defined as the disease threshold, then one individual infects one more individual [140].

Stability

Consider the differential equation $\dot{x} = f(t, x), x \in R^n$ then a point x is **Lyapunov stable** if and only if for all $\epsilon > 0$ there exists $\delta > 0$ such that if $|x - y| < \delta$ then $|f(x,t) - f(y,t)| < \epsilon$ for all $t \geq 0$. A point x is **quasi-asymptotically stable** iff there exists $\delta > 0$ such that if $|x - y| < \delta$ then $|\varphi(x,t) - \varphi(y,t)| \to 0$ as $t \to \infty$. A point x is **asymptotically stable** if it is both Lyapunov stable and quasi-asymptotically stable [141].

Local Asymptotic Stability

A point x^* is an equilibrium point of the system if $f(x^*) = 0$. x^* is locally stable if all solutions which start near x^* (meaning that the initial conditions are in a neighborhood of x^*) remain near x^* for all time. The equilibrium point x^* is said to be **locally asymptotically stable** if x^* is locally stable and, furthermore, all solutions starting near x^* tend towards x^* as $t \to \infty$ [141].

Global Asymptotic Stability

The system $\dot{x} = f(t, x)$ is **globally asymptotically stable** if for every trajectory $x(t)$, we have $x(t) \to x^*$ as $t \to \infty$ (implies x^* is the unique equilibrium point) [141].

Compartmental Models

Compartmental models are often used to describe transport of material in biological systems. A compartment model contains a number of compartments, each containing well mixed material. They exchange material with each other following certain rules. Compartments are represented by boxes and the connections between the compartments are represented by arrows. Every compartment (that is every box) has a number of connections leading to the box (inflows) and a number of arrows leading from the box (outflows). Material can either flow from one compartment to another, it can be added from the outside through a source like birth or new infection, or it can be removed through a drain where the drain in our case is death. Modeling of dynamical systems play a very important role in applied science, and compartment models are among the most important tools used for analyzing dynamical systems [142]. A few examples of compartmental models are listed below:

- SIR Model: The SIR model labels these three compartments S = number susceptible, I = number infectious, and R = number recovered. This is a good and simple model for many infectious diseases.

$$\text{Birth} \longrightarrow \boxed{\text{S}} \longrightarrow \boxed{\text{I}} \longrightarrow \boxed{\text{R}} \longrightarrow \text{Death}$$

- SEIR Model: The SEIR model labels four compartments S = number susceptible, E = number exposed but not infectious, I = number infectious, and R = number recovered. Compartment E comprises of individuals with latent infections that have yet to become infectious. Some diseases take a certain time for the infective agent to multiply inside the host up to the critical level where the disease then manifests itself in the body of the host. This phase is known as the incubation period [143].

$$\text{Birth} \longrightarrow \boxed{\text{S}} \longrightarrow \boxed{\text{E}} \longrightarrow \boxed{\text{I}} \longrightarrow \boxed{\text{R}} \longrightarrow \text{Death}$$

- SIRS Model: The SIRS model labels these four compartments S = number susceptible, I = number infectious, R = number recovered and back to S, that is the recovered become susceptible.

A.2 Chapter 2 Appendices

A.2.1 $\mathcal{R}_c$ Expressions

Expressions for q_1, q_2, ..., q_4, k_1, k_2, ..., k_8, η_1, η_2, ..., η_8, ε_{11}, ε_{12}, ε_{13}, ..., ε_{26} in section 3.3.5.

$$
\begin{cases}
q_1 = (1-\rho_f)\beta_{\Theta}\gamma_g S_g^0,\; q_2 = \rho_f\beta_{\Theta}\gamma_g S_g^0,\; q_3 = \dfrac{(1-\rho_f)\beta_{♀}\gamma_f S_f^0}{S_{mv}^0},\; q_4 = \dfrac{\rho_f\beta_{♀}\gamma_f S_f^0}{S_{mv}^0}, \\
k_1 = (1-\rho_m)\beta'_{♂}\gamma_m S_m^0,\; k_2 = \rho_m\beta'_{♂}\gamma_m S_m^0,\; k_3 = \dfrac{(1-\rho_m)\beta_{♂}\gamma_m S_m^0}{S_f^0},\; k_4 = \dfrac{\rho_m\beta_{♂}\gamma_m S_m^0}{S_f^0}, \\
k_5 = (1-\rho_m)\beta'_{♂}\gamma_m S_v^0,\; k_6 = \rho_m\beta'_{♂}\gamma_m S_v^0,\; k_7 = \dfrac{(1-\rho_m)\beta_{♂}\gamma_m S_v^0}{S_f^0},\; k_8 = \dfrac{\rho_m\beta_{♂}\gamma_m S_v^0}{S_f^0}, \\
\eta_1 = \dfrac{\psi_1\,\lambda_3\,m_{gu}}{(\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1)\,\bar\mu_5} + \dfrac{\psi_1\,m_{bu}\,\lambda_3\,m_{gu}}{\bar\mu_5\,(\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1)\,\bar\mu_{11}} + \\
\quad \dfrac{\psi_2\,(\bar\mu_2\,\bar\mu_4\,m_{gu}\,\lambda_3\,\rho_2 + \bar\mu_2\,\bar\mu_5\,m_{gu}\,\lambda_4\,\rho_1 + \bar\mu_3\,\bar\mu_5\,m_{ga}\,\lambda_4\,\rho_1 - m_{ga}\,m_{gu}\,\lambda_2\,\lambda_3\,\rho_2)}{(\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2)\,(\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1)\,\bar\mu_5\,\bar\mu_6} + \\
\quad \dfrac{1}{\bar\mu_6\,(\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2)\,(\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1)\,\bar\mu_5\,\bar\mu_{11}\,\bar\mu_{12}}\{\psi_2\,(\bar\mu_2\,\bar\mu_4\,\bar\mu_6\,m_{bu}\,m_{gu}\,\lambda_3\,\rho_2 + \\
\quad \bar\mu_2\,\bar\mu_4\,m_{ba}\,m_{gu}\,\lambda_3\,\bar\mu_{11}\,\rho_2 + \bar\mu_2\,\bar\mu_5\,m_{ba}\,m_{gu}\,\lambda_4\,\bar\mu_{11}\,\rho_1 + \bar\mu_3\,\bar\mu_5\,m_{ba}\,m_{ga}\,\lambda_4\,\bar\mu_{11}\,\rho_1 \\
\quad -\bar\mu_6\,m_{bu}\,m_{ga}\,m_{gu}\,\lambda_2\,\lambda_3\,\rho_2 - m_{ba}\,m_{ga}\,m_{gu}\,\lambda_2\,\lambda_3\,\bar\mu_{11}\,\rho_2)\}, \\
\eta_2 = \dfrac{\psi_2\,\lambda_4\,m_{ga}}{\bar\mu_6\,(\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2)} + \dfrac{\psi_2\,m_{ba}\,\lambda_4\,m_{ga}}{\bar\mu_6\,(\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2)\,\bar\mu_{12}}, \\
\eta_3 = \dfrac{\psi_1\,\lambda_3\,\bar\mu_1}{(\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1)\,\bar\mu_5} + \dfrac{\psi_1\,m_{bu}\,\lambda_3\,\bar\mu_1}{\bar\mu_5\,(\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1)\,\bar\mu_{11}} + \\
\quad \dfrac{\psi_2\,(\bar\mu_1\,\bar\mu_2\,\bar\mu_4\,\lambda_3\,\rho_2 + \bar\mu_1\,\bar\mu_2\,\bar\mu_5\,\lambda_4\,\rho_1 - \bar\mu_1\,m_{ga}\,\lambda_2\,\lambda_3\,\rho_2 + \bar\mu_5\,m_{ga}\,\lambda_1\,\lambda_4\,\rho_1)}{(\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2)\,(\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1)\,\bar\mu_5\,\bar\mu_6} + \\
\quad \dfrac{1}{\bar\mu_6\,(\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2)\,(\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1)\,\bar\mu_5\,\bar\mu_{11}\,\bar\mu_{12}}\{\psi_2\,(\bar\mu_1\,\bar\mu_2\,\bar\mu_4\,\bar\mu_6\,m_{bu}\,\lambda_3\,\rho_2 + \\
\quad \bar\mu_1\,\bar\mu_2\,\bar\mu_4\,m_{ba}\,\lambda_3\,\bar\mu_{11}\,\rho_2 + \bar\mu_1\,\bar\mu_2\,\bar\mu_5\,m_{ba}\,\lambda_4\,\bar\mu_{11}\,\rho_1 + \bar\mu_5\,m_{ba}\,m_{ga}\,\lambda_1\,\lambda_4\,\bar\mu_{11}\,\rho_1 \\
\quad -\bar\mu_1\,\bar\mu_6\,m_{bu}\,m_{ga}\,\lambda_2\,\lambda_3\,\rho_2 - \bar\mu_1\,m_{ba}\,m_{ga}\,\lambda_2\,\lambda_3\,\bar\mu_{11}\,\rho_2)\}, \\
\eta_4 = \dfrac{\psi_2\,\lambda_4\,\bar\mu_2}{\bar\mu_6\,(\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2)} + \dfrac{\psi_2\,m_{ba}\,\lambda_4\,\bar\mu_2}{\bar\mu_6\,(\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2)\,\bar\mu_{12}}, \\
\eta_5 = \dfrac{\psi_1}{\bar\mu_5} + \dfrac{\psi_2\,\rho_2}{\bar\mu_5\,\bar\mu_6} + \dfrac{\psi_1\,m_{bu}}{\bar\mu_5\,\bar\mu_{11}} + \dfrac{\psi_2\,\rho_2\,(\bar\mu_6\,m_{bu} + m_{ba}\,\bar\mu_{11})}{\bar\mu_6\,\bar\mu_5\,\bar\mu_{11}\,\bar\mu_{12}}, \\
\eta_6 = \dfrac{\psi_2}{\bar\mu_6} + \dfrac{\psi_2\,m_{ba}}{\bar\mu_6\,\bar\mu_{12}}, \qquad \eta_7 = \dfrac{\psi_1}{\bar\mu_{11}} + \dfrac{\psi_2\,\rho_2}{\bar\mu_{11}\,\bar\mu_{12}}, \qquad \eta_8 = \dfrac{\psi_2}{\bar\mu_{12}}, \\
\varepsilon_{11} = \dfrac{\psi_1\,k_1\,\bar\mu_3}{\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1} + \dfrac{\psi_2\,k_1\,\rho_1\,(\bar\mu_3\,\bar\mu_4 + m_{gu}\,\lambda_2)}{(\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1)\,(\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2)} + \dfrac{\psi_1\,k_2\,m_{gu}}{\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1} + \\
\quad \dfrac{\psi_2\,k_2\,\rho_1\,(\bar\mu_2\,m_{gu} + \bar\mu_3\,m_{ga})}{(\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1)\,(\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2)}, \\
\varepsilon_{12} = \dfrac{\psi_2\,k_1\,\bar\mu_4}{\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2} + \dfrac{\psi_2\,k_2\,m_{ga}}{\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2}, \\
\varepsilon_{13} = \dfrac{\psi_1\,k_1\,\lambda_1}{\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1} + \dfrac{\psi_2\,k_1\,\rho_1\,(\bar\mu_1\,\lambda_2 + \bar\mu_4\,\lambda_1)}{(\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1)\,(\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2)} + \dfrac{\psi_1\,k_2\,\bar\mu_1}{\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1} + \\
\quad \dfrac{\psi_2\,k_2\,\rho_1\,(\bar\mu_1\,\bar\mu_2 + m_{ga}\,\lambda_1)}{(\bar\mu_1\,\bar\mu_3 - m_{gu}\,\lambda_1)\,(\bar\mu_2\,\bar\mu_4 - m_{ga}\,\lambda_2)},
\end{cases}
\tag{A.1}
$$

$$
\begin{cases}
\varepsilon_{14} = \dfrac{\psi_2\, k_1\, \lambda_2}{\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2} + \dfrac{\psi_2\, k_2\, \bar{\mu}_2}{\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2}, \\
\varepsilon_{15} = \dfrac{\psi_1\, k_3\, \bar{\mu}_3}{\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1} + \dfrac{\psi_2\, k_3\, \rho_1\ (\bar{\mu}_3\, \bar{\mu}_4 + m_{gu}\, \lambda_2)}{(\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1)\, (\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2)} + \dfrac{\psi_1\, k_4\, m_{gu}}{\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1} + \\
\qquad \dfrac{\psi_2\, k_4\, \rho_1\ (\bar{\mu}_2\, m_{gu} + \bar{\mu}_3\, m_{ga})}{(\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1)\, (\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2)}, \\
\varepsilon_{16} = \dfrac{\psi_2\, k_3\, \bar{\mu}_4}{\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2} + \dfrac{\psi_2\, k_4\, m_{ga}}{\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2}, \\
\varepsilon_{17} = \dfrac{\psi_1\, k_3\, \lambda_1}{\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1} + \dfrac{\psi_2\, k_3\, \rho_1\ (\bar{\mu}_1\, \lambda_2 + \bar{\mu}_4\, \lambda_1)}{(\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1)\, (\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2)} + \dfrac{\psi_1\, k_4\, \bar{\mu}_1}{\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1} + \\
\qquad \dfrac{\psi_2\, k_4\, \rho_1\ (\bar{\mu}_1\, \bar{\mu}_2 + m_{ga}\, \lambda_1)}{(\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1)\, (\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2)}, \\
\varepsilon_{18} = \dfrac{\psi_2\, k_3\, \lambda_2}{\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2} + \dfrac{\psi_2\, k_4\, \bar{\mu}_2}{\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2}, \\
\varepsilon_{19} = \dfrac{\psi_1\, k_5\, \bar{\mu}_3}{\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1} + \dfrac{\psi_2\, k_5\, \rho_1\ (\bar{\mu}_3\, \bar{\mu}_4 + m_{gu}\, \lambda_2)}{(\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1)\, (\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2)} + \dfrac{\psi_1\, k_6\, m_{gu}}{\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1} + \\
\qquad \dfrac{\psi_2\, k_6\, \rho_1\ (\bar{\mu}_2\, m_{gu} + \bar{\mu}_3\, m_{ga})}{(\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1)\, (\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2)}, \\
\varepsilon_{20} = \dfrac{\psi_2\, k_5\, \bar{\mu}_4}{\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2} + \dfrac{\psi_2\, k_6\, m_{ga}}{\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2}, \\
\varepsilon_{21} = \dfrac{\psi_1\, k_5\, \lambda_1}{\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1} + \dfrac{\psi_2\, k_5\, \rho_1\ (\bar{\mu}_1\, \lambda_2 + \bar{\mu}_4\, \lambda_1)}{(\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1)\, (\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2)} + \dfrac{\psi_1\, k_6\, \bar{\mu}_1}{\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1} + \\
\qquad \dfrac{\psi_2\, k_6\, \rho_1\ (\bar{\mu}_1\, \bar{\mu}_2 + m_{ga}\, \lambda_1)}{(\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1)\, (\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2)}, \\
\varepsilon_{22} = \dfrac{\psi_2\, k_5\, \lambda_2}{\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2} + \dfrac{\psi_2\, k_6\, \bar{\mu}_2}{\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2}, \\
\varepsilon_{23} = \dfrac{\psi_1\, k_7\, \bar{\mu}_3}{\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1} + \dfrac{\psi_2\, k_7\, \rho_1\ (\bar{\mu}_3\, \bar{\mu}_4 + m_{gu}\, \lambda_2)}{(\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1)\, (\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2)} + \dfrac{\psi_1\, k_8\, m_{gu}}{\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1} + \\
\qquad \dfrac{\psi_2\, k_8\, \rho_1\ (\bar{\mu}_2\, m_{gu} + \bar{\mu}_3\, m_{ga})}{(\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1)\, (\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2)}, \\
\varepsilon_{24} = \dfrac{\psi_2\, k_7\, \mu_4}{\mu_2\, \mu_4 - m_{ga}\, \lambda_2} + \dfrac{\psi_2\, k_8\, m_{ga}}{\mu_2\, \mu_4 - m_{ga}\, \lambda_2}, \\
\varepsilon_{25} = \dfrac{\psi_1\, k_7\, \lambda_1}{\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1} + \dfrac{\psi_2\, k_7\, \rho_1\ (\bar{\mu}_1\, \lambda_2 + \bar{\mu}_4\, \lambda_1)}{(\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1)\, (\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2)} + \dfrac{\psi_1\, k_8\, \bar{\mu}_1}{\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1} + \\
\qquad \dfrac{\psi_2\, k_8\, \rho_1\ (\bar{\mu}_1\, \bar{\mu}_2 + m_{ga}\, \lambda_1)}{(\bar{\mu}_1\, \bar{\mu}_3 - m_{gu}\, \lambda_1)\, (\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2)}, \\
\varepsilon_{26} = \dfrac{\psi_2\, k_7\, \lambda_2}{\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2} + \dfrac{\psi_2\, k_8\, \bar{\mu}_2}{\bar{\mu}_2\, \bar{\mu}_4 - m_{ga}\, \lambda_2}.
\end{cases}
\tag{A.2}
$$

Computation of the Control Reproduction Number, $\mathcal{R}_c$

Computing the characteristic polynomial $f(\tilde{x}) = det(\mathcal{F}\mathcal{V}^{-1} - \tilde{x}\, I_n)$ of the next generation matrix $(\mathcal{F}\mathcal{V}^{-1})$ given in section 3.3.5 yields

$$
\begin{aligned}
&f(\tilde{x}) = det(\mathcal{F}\mathcal{V}^{-1} - \tilde{x}\, I_n) = \\
&\tilde{x}^8 + (-q_1\,\eta_1 - q_2\,\eta_2 - q_3\,\eta_3 - q_4\,\eta_4)\,\tilde{x}^7 + \\
&(-q_1\,\eta_5\,\varepsilon_{11} - q_1\,\eta_6\,\varepsilon_{15} - q_1\,\eta_7\,\varepsilon_{19} - q_1\,\eta_8\,\varepsilon_{23} - q_2\,\eta_5\,\varepsilon_{12} - q_2\,\eta_6\,\varepsilon_{16} - q_2\,\eta_7\,\varepsilon_{20} - q_2\,\eta_8\,\varepsilon_{24} \\
&-q_3\,\eta_5\,\varepsilon_{13} - q_3\,\eta_6\,\varepsilon_{17} - q_3\,\eta_7\,\varepsilon_{21} - q_3\,\eta_8\,\varepsilon_{25} - q_4\,\eta_5\,\varepsilon_{14} - q_4\,\eta_6\,\varepsilon_{18} - q_4\,\eta_7\,\varepsilon_{22} - q_4\,\eta_8\,\varepsilon_{26})\,\tilde{x}^6
\end{aligned}
$$

Equating $f(\tilde{x}) = 0$ we have

$$
\begin{aligned}
&\tilde{x}^6\,[\tilde{x}^2 + (-q_1\,\eta_1 - q_2\,\eta_2 - q_3\,\eta_3 - q_4\,\eta_4)\,\tilde{x} + \\
&(-q_1\,\eta_5\,\varepsilon_{11} - q_1\,\eta_6\,\varepsilon_{15} - q_1\,\eta_7\,\varepsilon_{19} - q_1\,\eta_8\,\varepsilon_{23} - q_2\,\eta_5\,\varepsilon_{12} - q_2\,\eta_6\,\varepsilon_{16} - q_2\,\eta_7\,\varepsilon_{20} - q_2\,\eta_8\,\varepsilon_{24} \\
&-q_3\,\eta_5\,\varepsilon_{13} - q_3\,\eta_6\,\varepsilon_{17} - q_3\,\eta_7\,\varepsilon_{21} - q_3\,\eta_8\,\varepsilon_{25} - q_4\,\eta_5\,\varepsilon_{14} - q_4\,\eta_6\,\varepsilon_{18} - q_4\,\eta_7\,\varepsilon_{22} - q_4\,\eta_8\,\varepsilon_{26})] = 0
\end{aligned}
$$

Equating $f(\tilde{x}) = 0$ yields the eigenvalues

$$
\begin{aligned}
&\tilde{x} = 0 \text{ with multiplicity six (6) and} \\
&\tilde{x} = \frac{1}{2}\,(q_1\eta_1 + q_2\eta_2 + q_3\eta_3 + q_4\eta_4) - \frac{1}{2}\,\{{q_1}^2{\eta_1}^2 + {q_2}^2{\eta_2}^2 + {q_3}^2{\eta_3}^2 + {q_4}^2{\eta_4}^2 + \\
&\quad 2\,[q_1\,q_2\,\eta_1\,\eta_2 + q_1\,q_3\,\eta_1\,\eta_3 + q_1\,q_4\,\eta_1\,\eta_4 + q_2\,q_3\,\eta_2\,\eta_3 + q_2\,q_4\,\eta_2\,\eta_4 \\
&\quad + q_3\,q_4\,\eta_3\,\eta_4] + 4\,q_1\,[\eta_5\,\varepsilon_{11} + \eta_6\,\varepsilon_{15} + \eta_7\,\varepsilon_{19} + \eta_8\,\varepsilon_{23}] \\
&\quad + 4\,q_2\,[\eta_5\,\varepsilon_{12} + \eta_6\,\varepsilon_{16} + \eta_7\,\varepsilon_{20} + \eta_8\,\varepsilon_{24}] \\
&\quad + 4\,q_3\,[\eta_5\,\varepsilon_{13} + \eta_6\,\varepsilon_{17} + \eta_7\,\varepsilon_{21} + \eta_8\,\varepsilon_{25}] \\
&\quad + 4\,q_4\,[\eta_5\,\varepsilon_{14} + \eta_6\,\varepsilon_{18} + \eta_7\,\varepsilon_{22} + \eta_8\,\varepsilon_{26}]\}^{\frac{1}{2}}, \\
&\tilde{x} = \frac{1}{2}\,(q_1\eta_1 + q_2\eta_2 + q_3\eta_3 + q_4\eta_4) + \frac{1}{2}\,\{{q_1}^2{\eta_1}^2 + {q_2}^2{\eta_2}^2 + {q_3}^2{\eta_3}^2 + {q_4}^2{\eta_4}^2 + \\
&\quad 2\,[q_1\,q_2\,\eta_1\,\eta_2 + q_1\,q_3\,\eta_1\,\eta_3 + q_1\,q_4\,\eta_1\,\eta_4 + q_2\,q_3\,\eta_2\,\eta_3 + q_2\,q_4\,\eta_2\,\eta_4 \\
&\quad + q_3\,q_4\,\eta_3\,\eta_4] + 4\,q_1\,[\eta_5\,\varepsilon_{11} + \eta_6\,\varepsilon_{15} + \eta_7\,\varepsilon_{19} + \eta_8\,\varepsilon_{23}] \\
&\quad + 4\,q_2\,[\eta_5\,\varepsilon_{12} + \eta_6\,\varepsilon_{16} + \eta_7\,\varepsilon_{20} + \eta_8\,\varepsilon_{24}] \\
&\quad + 4\,q_3\,[\eta_5\,\varepsilon_{13} + \eta_6\,\varepsilon_{17} + \eta_7\,\varepsilon_{21} + \eta_8\,\varepsilon_{25}] \\
&\quad + 4\,q_4\,[\eta_5\,\varepsilon_{14} + \eta_6\,\varepsilon_{18} + \eta_7\,\varepsilon_{22} + \eta_8\,\varepsilon_{26}]\}^{\frac{1}{2}}.
\end{aligned}
$$

It follows that the spectral radius of $f(\tilde{x}) = 0$ which is the control reproduction number of the model system (5.3) is

$$\tilde{x} = \frac{1}{2}\left(q_1\eta_1 + q_2\eta_2 + q_3\eta_3 + q_4\eta_4\right) + \frac{1}{2}\left\{{q_1}^2{\eta_1}^2 + {q_2}^2{\eta_2}^2 + {q_3}^2{\eta_3}^2 + {q_4}^2{\eta_4}^2 + \right.$$
$$2\left[q_1\,q_2\,\eta_1\,\eta_2 + \; q_1\,q_3\,\eta_1\,\eta_3 + \; q_1\,q_4\,\eta_1\,\eta_4 + \; q_2\,q_3\,\eta_2\,\eta_3 + \; q_2\,q_4\,\eta_2\,\eta_4\right.$$
$$\left. + q_3\,q_4\,\eta_3\,\eta_4\right] + 4\,q_1\left[\eta_5\,\varepsilon_{11} + \eta_6\,\varepsilon_{15} + \eta_7\,\varepsilon_{19} + \eta_8\,\varepsilon_{23}\right]$$
$$+4\,q_2\left[\eta_5\,\varepsilon_{12} + \eta_6\,\varepsilon_{16} + \eta_7\,\varepsilon_{20} + \eta_8\,\varepsilon_{24}\right]$$
$$+4\,q_3\left[\eta_5\,\varepsilon_{13} + \eta_6\,\varepsilon_{17} + \eta_7\,\varepsilon_{21} + \eta_8\,\varepsilon_{25}\right]$$
$$\left.+4\,q_4\left[\eta_5\,\varepsilon_{14} + \eta_6\,\varepsilon_{18} + \eta_7\,\varepsilon_{22} + \eta_8\,\varepsilon_{26}\right]\right\}^{\frac{1}{2}}.$$

Jacobian matrix $\mathcal{F}$

$$\mathcal{F} = \left[\frac{\partial F_i(x_0)}{\partial x_j}\right] = \begin{bmatrix} 0 & 0 & 0 & 0 & \psi_1\,q_1 & \psi_2\,q_1 & \psi_1\,q_1 & \psi_2\,q_1 \\ 0 & 0 & 0 & 0 & \psi_1\,q_2 & \psi_2\,q_2 & \psi_1\,q_2 & \psi_2\,q_2 \\ 0 & 0 & 0 & 0 & \psi_1\,q_3 & \psi_2\,q_3 & \psi_1\,q_3 & \psi_2\,q_3 \\ 0 & 0 & 0 & 0 & \psi_1\,q_4 & \psi_2\,q_4 & \psi_1\,q_4 & \psi_2\,q_4 \\ \psi_1\,k_1 & \psi_2\,k_1 & \psi_1\,k_2 & \psi_2\,k_2 & 0 & 0 & 0 & 0 \\ \psi_1\,k_3 & \psi_2\,k_3 & \psi_1\,k_4 & \psi_2\,k_4 & 0 & 0 & 0 & 0 \\ \psi_1\,k_5 & \psi_2\,k_5 & \psi_1\,k_6 & \psi_2\,k_6 & 0 & 0 & 0 & 0 \\ \psi_1\,k_7 & \psi_2\,k_7 & \psi_1\,k_8 & \psi_2\,k_8 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

A.2.2 Endemic Equilibrium Expressions

Expressions for a_{11}, a_{12}, ..., a_{35}, g_{11}, g_{12}, ..., g_{14}, h_{01}, h_{02}, ..., h_{57} and C_0, C_1,...C_5 in section 3.3.4.

$$
\begin{cases}
a_{11} = \dfrac{\lambda_1}{\bar{\mu}_1},\ a_{12} = \dfrac{(1-\rho_f)\Lambda_1(N_f^* + C_w)}{\bar{\mu}_1},\ a_{13} = \dfrac{\lambda_2}{\bar{\mu}_2}, \\
a_{14} = \dfrac{\rho_f \Lambda_1(N_f^* + C_w)}{\bar{\mu}_2}, \quad a_{15} = \dfrac{\rho_1 a_{11}}{\bar{\mu}_2},\ a_{16} = \dfrac{\rho_1 a_{12}}{\bar{\mu}_2}, \\
a_{17} = \dfrac{m_{gu}}{\bar{\mu}_3},\ a_{18} = \dfrac{m_{gu}(1-\rho_f)\Lambda_1(N_f^* + C_w)}{\bar{\mu}_3},\ a_{19} = \dfrac{m_{ga}}{\bar{\mu}_4}, \\
a_{20} = \dfrac{\rho_f m_g \Lambda_1(N_f^* + C_w)}{\bar{\mu}_4},\ a_{21} = \dfrac{\rho_1}{\bar{\mu}_4},\ a_{22} = \dfrac{\lambda_3}{\bar{\mu}_5}, \quad a_{23} = \dfrac{(1-\rho_m)\Lambda_2(N_f^* + C_w)}{\bar{\mu}_5}, \\
a_{24} = \dfrac{\lambda_4}{\bar{\mu}_6}, \quad a_{25} = \dfrac{\rho_m \Lambda_2(N_f^* + C_w)}{\bar{\mu}_6}, \quad a_{26} = \dfrac{\rho_2}{\bar{\mu}_6}, \\
a_{27} = \dfrac{m_{bu}}{\bar{\mu}_{11}},\ a_{28} = \dfrac{m_{bu}(1-\rho_m)\Lambda_2(N_f^* + C_w)}{\bar{\mu}_{11}},\ a_{29} = \dfrac{m_{ba}}{\bar{\mu}_{12}},\ a_{30} = \dfrac{\rho_2}{\bar{\mu}_{12}}, \\
a_{31} = \dfrac{\rho_m m_{bu} \Lambda_2(N_f^* + C_w)}{\bar{\mu}_{12}},\ a_{32} = \dfrac{a_{12}a_{17}}{1 - a_{11}a_{17}},\ a_{11}a_{17} < 1, \\
a_{33} = \dfrac{a_{18}(N_f^* + C_w)}{1 - a_{11}a_{17}},\ a_{34} = \dfrac{a_{19}(a_{14} + a_{16}) + a_{19}a_{15}a_{32} + a_{21}a_{32}}{1 - a_{19}a_{13}}, \\
a_{19}a_{13} < 1,\ a_{35} = \dfrac{a_{19}a_{15}a_{33} + a_{21}a_{33} + a_{20}}{1 - a_{19}a_{13}}, \\
\bar{m}_g = min\left\{m_g\, m_{gu},\, m_{ga}\right\},\ \bar{m}_b = min\left\{m_b,\, m_{bu},\, m_{ba}\right\}, \\
\bar{\Lambda}_1 = min\left\{\lambda_1,\, \lambda_2\right\},\ \bar{\Lambda}_2 = min\left\{\lambda_3,\, \lambda_4\right\}, \\
g_{11} = \dfrac{\Lambda_1}{\bar{m}_g + \delta + \bar{\mu}_f},\ g_{12} = \dfrac{\bar{m}_g}{\bar{\mu}_f + \delta + \sigma},\ g_{13} = \dfrac{\Lambda_2 + \bar{\lambda}_2}{\bar{m}_b + \delta + \sigma + \bar{\mu}_m}, \\
g_{14} = \dfrac{\bar{m}_b}{\bar{\mu}_m + \delta},\ d_1 = \dfrac{\beta_\Theta \gamma_g}{\beta_\varphi \gamma_f}, \\
h_{01} = a_{11}a_{31} + a_{12},\ h_{02} = a_{14} + a_{16},\ h_{03} = a_{13}a_{34} + a_{15}a_{32} + h_{02}, \\
h_{04} = a_{13}a_{35} + a_{15}a_{33},\ h_{05} = a_{22}a_{31},\ h_{06} = a_{22}a_{32},\ h_{07} = a_{24}a_{33} + a_{26}h_{05}, \\
h_{08} = a_{24}a_{34} + a_{26}h_{06},\ h_{09} = a_{25} + a_{26}a_{23},\ h_{10} = a_{27}h_{05}, \\
h_{11} = a_{27}h_{06},\ h_{12} = a_{27}a_{23},\ h_{13} = a_{29}h_{07} + a_{30}h_{10}, \\
h_{14} = a_{29}h_{08} + a_{30}h_{11},\ h_{15} = a_{29}h_{09} + a_{30}h_{12},\ h_{16} = a_{30}a_{28} + a_{31}, \\
h_{17} = \beta_\Theta\, \gamma_g\, N_{mv},\ h_{18} = \dfrac{\gamma_m \beta_{\male}}{N_f}(\psi_1 a_{32} + \psi_2 a_{34}) + \beta'_{\male}\gamma_m(\psi_1 h_{01} + \psi_2 h_{03}), \\
h_{19} = \dfrac{\gamma_m \beta_{\male}}{N_f}(\psi_1 a_{33} + \psi_2 a_{35}) + \beta'_{\male}\gamma_m(\psi_1 a_{33} + \psi_2 h_{04}), \\
h_{20} = \dfrac{\gamma_f \beta_{\female}}{N_{mv}}(\psi_1 h_{05} + \psi_2 h_{07} + \psi_1 h_{10} + \psi_2 h_{13}),\ h_{21} = \dfrac{\gamma_f \beta_{\female}}{N_{mv}}(\psi_1 h_{06} + \psi_2 h_{08} + \psi_1 h_{11} + \psi_2 h_{14}), \\
h_{22} = \dfrac{\gamma_f \beta_{\female}}{N_{mv}}(\psi_1 a_{23} + \psi_2 h_{09} + \psi_1 h_{12} + \psi_2 h_{15}),\ h_{23} = \dfrac{\gamma_f \beta_{\female}}{N_{mv}}(\psi_1 a_{28} + \psi_2 h_{16}), \\
h_{24} = h_{18}^2 h_{20},\ h_{25} = h_{19}^2 h_{21},\ h_{26} = 2h_{18}h_{19}h_{20} + h_{18}^2 h_{21},
\end{cases}
\tag{A.3}
$$

$$
\begin{cases}
h_{27} = 2h_{18}h_{19}h_{21} + h_{19}^2 h_{20},\ h_{28} = h_{19}h_{20}\bar{\mu}_{10} + h_{19}h_{20}\bar{\mu}_7 + h_{18}h_{21}\bar{\mu}_7 + h_{18}h_{21}\bar{\mu}_{10}, \\
h_{29} = h_{18}h_{20}\bar{\mu}_7 + h_{18}h_{20}\bar{\mu}_{10},\ h_{30} = h_{19}h_{21}\bar{\mu}_7 + h_{19}h_{21}\bar{\mu}_{10}, \\
h_{31} = h_{20}\bar{\mu}_7\bar{\mu}_{10} + h_{22}h_{18} + h_{23}h_{18},\ h_{32} = h_{21}\bar{\mu}_7\bar{\mu}_{10} + h_{22}h_{19} + h_{23}h_{19}, \\
h_{33} = 2h_{18}h_{19},\ h_{34} = h_{18}\bar{\mu}_7 + h_{18}\bar{\mu}_{10},\ h_{35} = h_{19}\bar{\mu}_7 + h_{19}\bar{\mu}_{10},\ h_{36} = \bar{\mu}_7\bar{\mu}_{10}, \\
h_{37} = h_{22}\bar{\mu}_{10},\ h_{38} = h_{18}^2,\ h_{39} = h_{19}^2, \\
h_{40} = {N_{mv}}^3 {d_1}^3 h_{24} + {N_{mv}}^3 {d_1}^3 h_{29} + {N_{mv}}^3 {d_1}^3 h_{31}, \\
h_{41} = 3\,\bar{\mu}_8\,{N_{mv}}^3 {d_1}^3 h_{24} + 3\,\bar{\mu}_8\,{N_{mv}}^3 {d_1}^3 h_{29} + 3\,\bar{\mu}_8\,{N_{mv}}^3 {d_1}^3 h_{31} + \bar{\mu}_9\,{N_{mv}}^2 {d_1}^2 h_{29} + \\
\qquad 2\,\bar{\mu}_9\,{N_{mv}}^2 {d_1}^2 h_{31} + {N_{mv}}^2 {d_1}^2 h_{26} + {N_{mv}}^2 {d_1}^2 h_{28} + {N_{mv}}^2 {d_1}^2 h_{32}, \\
h_{42} = 3\,\bar{\mu}_8^2 {N_{mv}}^3 {d_1}^3 h_{24} + 3\,\bar{\mu}_8^2 {N_{mv}}^3 {d_1}^3 h_{29} + 3\,\bar{\mu}_8^2 {N_{mv}}^3 {d_1}^3 h_{31} + \\
\qquad 3\,\bar{\mu}_8\,\bar{\mu}_9\,{N_{mv}}^2 {d_1}^2 h_{29} + 6\,\bar{\mu}_8\,\bar{\mu}_9\,{N_{mv}}^2 {d_1}^2 h_{31} + 2\,\bar{\mu}_8\,{N_{mv}}^2 {d_1}^2 h_{26} + \\
\qquad 2\,\bar{\mu}_8\,{N_{mv}}^2 {d_1}^2 h_{28} + 2\,\bar{\mu}_8\,{N_{mv}}^2 {d_1}^2 h_{32} + \bar{\mu}_9^2 N_{mv}\,d_1\,h_{31} \\
\qquad + \bar{\mu}_9\,N_{mv}\,d_1\,h_{28} + 2\,\bar{\mu}_9\,N_{mv}\,d_1\,h_{32} + N_{mv}\,d_1\,h_{27} + N_{mv}\,d_1\,h_{30}, \\
h_{43} = \bar{\mu}_8^3 {N_{mv}}^3 {d_1}^3 h_{24} + \bar{\mu}_8^3 {N_{mv}}^3 {d_1}^3 h_{29} + \bar{\mu}_8^3 {N_{mv}}^3 {d_1}^3 h_{31} + \\
\qquad 3\,v{\mu_8}^2\bar{\mu}_9\,{N_{mv}}^2 {d_1}^2 h_{29} + 6\,\bar{\mu}_8^2\bar{\mu}_9\,{N_{mv}}^2 {d_1}^2 h_{31} + \bar{\mu}_8^2 {N_{mv}}^2 {d_1}^2 h_{26} + \\
\qquad \bar{\mu}_8^2 {N_{mv}}^2 {d_1}^2 h_{28} + \bar{\mu}_8^2 {N_{mv}}^2 {d_1}^2 h_{32} + 3\,\bar{\mu}_8\,\bar{\mu}_9^2 N_{mv}\,d_1\,h_{31} + 2\,\bar{\mu}_8\,\bar{\mu}_9\,N_{mv}\,d_1\,h_{28} + \\
\qquad 4\,\bar{\mu}_8\,\bar{\mu}_9\,N_{mv}\,d_1\,h_{32} + \bar{\mu}_8\,N_{mv}\,d_1\,h_{27} + \bar{\mu}_8\,N_{mv}\,d_1\,h_{30} + \bar{\mu}_9^2 h_{32} + \bar{\mu}_9\,h_{30} + h_{25}, \\
h_{44} = \bar{\mu}_8^3\bar{\mu}_9\,{N_{mv}}^2 {d_1}^2 h_{29} + 2\,\bar{\mu}_8^3\bar{\mu}_9\,{N_{mv}}^2 {d_1}^2 h_{31} + 3\,\bar{\mu}_8^2\bar{\mu}_9^2 N_{mv}\,d_1\,h_{31} + \\
\qquad \bar{\mu}_8^2\bar{\mu}_9\,N_{mv}\,d_1\,h_{28} + 2\,\bar{\mu}_8^2\bar{\mu}_9\,N_{mv}\,d_1\,h_{32} + 2\,\bar{\mu}_8\,\bar{\mu}_9^2 h_{32} + \bar{\mu}_8\,\bar{\mu}_9\,h_{30}, \\
h_{45} = \bar{\mu}_8^3\bar{\mu}_9^2 N_{mv}\,d_1\,h_{31} + \bar{\mu}_8^2\bar{\mu}_9^2 h_{32}, \\
h_{46} = 2\,{N_{mv}}^2 {d_1}^2 h_{34} + {N_{mv}}^2 {d_1}^2 h_{36} + {N_{mv}}^2 {d_1}^2 h_{38}, \\
h_{47} = 4\,\bar{\mu}_8\,{N_{mv}}^2 {d_1}^2 h_{34} + 2\,\bar{\mu}_8\,{N_{mv}}^2 {d_1}^2 h_{36} + 2\,\bar{\mu}_8\,{N_{mv}}^2 {d_1}^2 h_{38} + \\
\qquad 2\,\bar{\mu}_9\,N_{mv}\,d_1\,h_{34} + 2\,\bar{\mu}_9\,N_{mv}\,d_1\,h_{36} + N_{mv}\,d_1\,h_{35}, \\
h_{48} = 2\,\bar{\mu}_8^2 {N_{mv}}^2 {d_1}^2 h_{34} + \bar{\mu}_8^2 {N_{mv}}^2 {d_1}^2 h_{36} + \bar{\mu}_8^2 {N_{mv}}^2 {d_1}^2 h_{38} + \\
\qquad 4\,\bar{\mu}_8\,\bar{\mu}_9\,N_{mv}\,d_1\,h_{34} + 4\,\bar{\mu}_8\,\bar{\mu}_9\,N_{mv}\,d_1\,h_{36} + \bar{\mu}_8\,N_{mv}\,d_1\,h_{35} + \\
\qquad \bar{\mu}_9^2 h_{36} + \bar{\mu}_9\,h_{35} + h_{39}, \\
h_{49} = 2\,\bar{\mu}_8^2\bar{\mu}_9\,N_{mv}\,d_1\,h_{34} + 2\,\bar{\mu}_8^2\bar{\mu}_9\,N_{mv}\,d_1\,h_{36} + 2\,\bar{\mu}_8\,\bar{\mu}_9^2 h_{36} + \bar{\mu}_8\,\bar{\mu}_9\,h_{35}, \\
h_{50} = \bar{\mu}_8^2\bar{\mu}_9^2 h_{36},\ h_{51} = N_{mv}\,d_1\,h_{46},\ h_{52} = \bar{\mu}_8\,N_{mv}\,d_1\,h_{46} + N_{mv}\,d_1\,h_{47} + \mu_9\,h_{46}, \\
h_{53} = \bar{\mu}_8\,N_{mv}\,d_1\,h_{47} + \bar{\mu}_8\,\bar{\mu}_9\,h_{46} + N_{mv}\,d_1\,h_{48} + \bar{\mu}_9\,h_{47}, \\
h_{54} = \bar{\mu}_8\,N_{mv}\,d_1\,h_{48} + \bar{\mu}_8\,\mu_9\,h_{47} + N_{mv}\,d_1\,h_{49} + \mu_9\,h_{48}, \\
h_{55} = \bar{\mu}_8\,N_{mv}\,d_1\,h_{49} + \mu_8\,\mu_9\,h_{48} + N_{mv}\,d_1\,h_{50} + \bar{\mu}_9\,h_{49}, \\
h_{56} = \bar{\mu}_8\,N_{mv}\,d_1\,h_{50} + \bar{\mu}_8\,\mu_9\,h_{49} + \bar{\mu}_9\,h_{50},\ h_{57} = \bar{\mu}_8\,v\mu_9\,h_{50}, \\
C_0 = h_{45} - h_{47},\ h_{45} > h_{47},\ C_1 = \dfrac{h_{56} - h_{44}}{h_{51}},\ C_2 = \dfrac{h_{55} - h_{43}}{h_{51}}, \\
C_3 = \dfrac{h_{54} - h_{42}}{h_{51}},\ C_4 = \dfrac{h_{53} - h_{41}}{h_{51}},\ C_5 = \dfrac{h_{52} - h_{40}}{h_{51}}.
\end{cases}
\tag{A.4}
$$

A.2.3 Stability Analysis

Global Stability Analysis of the DFE

In this section, we prove the global stability of the DFE. We use a matrix-theoretic method defined by [144] to construct a Lyapunov function L involving the Perron eigenvector. We let $l^T \geq 0$ be the left eigenvector corresponding to the eigenvalue $\rho(\mathcal{V}^{-1}\mathcal{F}) = \rho(\mathcal{F}\mathcal{V}^{-1}) = \mathcal{R}_c$ and $f(x) := (\mathcal{F} - \mathcal{V})x - F(x) + V(x)$ where $x = (I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va})$. Model system (**??**) can be written as $\dot{x} = (\mathcal{F} - \mathcal{V})x - f(x)$. Equation (3.19) defines $\mathcal{F}$ and $\mathcal{V}$.

Theorem A.2.1. *If $f(x) \geq 0$ in Ω, $\mathcal{F} \geq 0$, $\mathcal{V}^{-1} \geq 0$ and $\mathcal{R}_c \leq 1$ then the function $L = l^T\mathcal{V}^{-1}x$ is a Lyapunov function for model (**??**) on Ω.*

Proof. Differentiating L with respect to time along the model solutions of (**??**) yields

$$\dot{L} = l^T\mathcal{V}^{-1}\dot{x} = l^T\mathcal{V}^{-1}(\mathcal{F} - \mathcal{V})x - l^T\mathcal{V}^{-1}f(x) = (\mathcal{R}_c - 1)l^T x - l^T\mathcal{V}^{-1}f(x). \tag{A.5}$$

Given that l, $\mathcal{V}^{-1}$, and $f(x)$ are non-negative in Ω, the last term of equation (A.5), $l^T\mathcal{V}^{-1}f(x)$ will be negative. If $\mathcal{R}_c \leq 1$, *then* $\dot{L} \leq 0$ *in* Ω hence L is the Lyapunov function for the model system (**??**).

$$l^T = [l_1,\, l_2,\, l_3,\, l_4,\, l_5,\, l_6,\, l_7,\, l_8] = l^T\mathcal{F}\mathcal{V}^{-1} = \mathcal{R}_c\, l^T =$$

$$[q_{52} \quad q_{48} \quad q_{51} \quad q_{47} \quad q_{46} \quad q_{45} \quad q_{44} \quad 1]^T$$

where

$$\begin{cases}
q_{11} = \dfrac{\bar{\mu}_3}{\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1},\ q_{12} = \dfrac{\rho_1\ (\mu_3\,\mu_4 + m_{gu}\,\lambda_2)}{(\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1)\,(\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)},\ q_{13} = \dfrac{m_{gu}}{\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1}, \\
q_{14} = \dfrac{\rho_1\ (\bar{\mu}_2\,m_{gu} + \mu_3\,m_{ga})}{(\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1)\,(\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)},\ q_{15} = \dfrac{\lambda_3\,m_{gu}}{(\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1)\,\bar{\mu}_5}, \\
q_{16} = \dfrac{\bar{\mu}_2\,\bar{\mu}_4\,m_{gu}\,\lambda_3\,\rho_2 + \bar{\mu}_2\,\bar{\mu}_5\,m_{gu}\,\lambda_4\,\rho_1 + \bar{\mu}_3\,\bar{\mu}_5\,m_{ga}\,\lambda_4\,\rho_1 - m_{ga}\,m_{gu}\,\lambda_2\,\lambda_3\,\rho_2}{(\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)\,(\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1)\,\bar{\mu}_5\,\bar{\mu}_6}, \\
q_{17} = \dfrac{m_{bu}\,\lambda_3\,m_{gu}}{\bar{\mu}_5\ (\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1)\,\bar{\mu}_{11}}, \\
q_{18} = \dfrac{1}{\bar{\mu}_6\ (\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)\,(\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1)\,\bar{\mu}_5\,\bar{\mu}_{11}\,\bar{\mu}_{12}}\,(\bar{\mu}_2\,\bar{\mu}_4\,\bar{\mu}_6\,m_{bu}\,m_{gu}\,\lambda_3\,\rho_2 + \\
\quad \bar{\mu}_2\,\bar{\mu}_4\,m_{ba}\,m_{gu}\,\lambda_3\,\bar{\mu}_{11}\,\rho_2 + \bar{\mu}_2\,\bar{\mu}_5\,m_{ba}\,m_{gu}\,\lambda_4\,\bar{\mu}_{11}\,\rho_1 + \bar{\mu}_3\,\bar{\mu}_5\,m_{ba}\,m_{ga}\,\lambda_4\,\bar{\mu}_{11}\,\rho_1 \\
\quad -\bar{\mu}_6\,m_{bu}\,m_{ga}\,m_{gu}\,\lambda_2\,\lambda_3\,\rho_2 - m_{ba}\,m_{ga}\,m_{gu}\,\lambda_2\,\lambda_3\,\bar{\mu}_{11}\,\rho_2)\,, \\
q_{19} = \dfrac{\bar{\mu}_4}{\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2},\ q_{20} = \dfrac{m_{ga}}{(\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)},\ q_{21} = \dfrac{\lambda_4 m_{ga}}{(\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)\,\bar{\mu}_6}, \\
q_{22} = \dfrac{\lambda_4 m_{ba} m_{ga}}{(\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)\,\bar{\mu}_6\bar{\mu}_{12}}, \\
.
\end{cases} \tag{A.6}$$

$$
\begin{cases}
q_{23} = \dfrac{\lambda_1}{\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1},\ q_{24} = \dfrac{\rho_1\ (\bar{\mu}_1\,\lambda_2 + \bar{\mu}_4\,\lambda_1)}{(\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1)\,(\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)},\ q_{25} = \dfrac{\bar{\mu}_1}{\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1}, \\
q_{26} = \dfrac{\rho_1\ (\bar{\mu}_1\bar{\mu}_2\ + \lambda_1\,m_{ga})}{(\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1)\,(\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)},\ q_{27} = \dfrac{\lambda_3\,\bar{\mu}_1}{(\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1)\,\bar{\mu}_5}, \\
q_{28} = \dfrac{\bar{\mu}_1\,\bar{\mu}_2\,\bar{\mu}_4\,\lambda_3\,\rho_2 + \bar{\mu}_1\,\bar{\mu}_2\,\bar{\mu}_5\,\lambda_4\,\rho_1 - \bar{\mu}_1\,m_{ga}\,\lambda_2\,\lambda_3\,\rho_2 + \bar{\mu}_5\,m_{ga}\,\lambda_1\,\lambda_4\,\rho_1}{(\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)\,(\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1)\,\bar{\mu}_5\,\bar{\mu}_6}, \\
q_{29} = \dfrac{m_{bu}\,\lambda_3\,\bar{\mu}_1}{\bar{\mu}_5\ (\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1)\,\bar{\mu}_{11}}, \\
q_{30} = \dfrac{1}{\bar{\mu}_6\ (\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)\,(\bar{\mu}_1\,\bar{\mu}_3 - m_{gu}\,\lambda_1)\,\bar{\mu}_5\,\bar{\mu}_{11}\,\bar{\mu}_{12}}\,(\bar{\mu}_1\bar{\mu}_2\,\bar{\mu}_4\,\bar{\mu}_6\,m_{bu}\,\lambda_3\,\rho_2 + \\
\qquad \bar{\mu}_1\bar{\mu}_2\,\bar{\mu}_4\,m_{ba}\,\lambda_3\,\bar{\mu}_{11}\,\rho_2 + \bar{\mu}_1\bar{\mu}_2\,\bar{\mu}_5\,m_{ba}\,\lambda_4\,\bar{\mu}_{11}\,\rho_1 \\
\qquad -\bar{\mu}_1\bar{\mu}_6\,m_{bu}\,m_{ga}\,\lambda_2\,\lambda_3\,\rho_2 - \bar{\mu}_1 m_{ba}\,m_{ga}\,\lambda_2\,\lambda_3\,\bar{\mu}_{11}\,\rho_2 + \bar{\mu}_5\,m_{ba}\,m_{ga}\,\lambda_1\,\lambda_4\,\bar{\mu}_{11}\,\rho_1)\,, \\
q_{31} = \dfrac{\lambda_2}{\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2},\ q_{32} = \dfrac{\bar{\mu}_2}{\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2},\ q_{33} = \dfrac{\lambda_4\,\bar{\mu}_2}{\bar{\mu}_6\ (\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)}, \\
q_{34} = \dfrac{\lambda_4\,m_{ba}\bar{\mu}_2}{\bar{\mu}_6\ (\bar{\mu}_2\,\bar{\mu}_4 - m_{ga}\,\lambda_2)\,\bar{\mu}_{12}},\ q_{35} = \dfrac{1}{\bar{\mu}_5},\ q_{36} = \dfrac{\rho_2}{\bar{\mu}_5\bar{\mu}_6},\ q_{37} = \dfrac{m_{bu}}{\bar{\mu}_5\bar{\mu}_{11}}, \\
q_{38} = \dfrac{\rho_2\ (\bar{\mu}_6\,m_{bu} + m_{ba}\,\bar{\mu}_{11})}{\bar{\mu}_6\,\bar{\mu}_5\,\bar{\mu}_{11}\,\bar{\mu}_{12}},\ q_{39} = \dfrac{1}{\bar{\mu}_6},\ q_{40} = \dfrac{m_{ba}}{\bar{\mu}_6\bar{\mu}_{12}},\ q_{41} = \dfrac{1}{\bar{\mu}_{11}}, \\
q_{42} = \dfrac{\rho_2}{\bar{\mu}_{11}\bar{\mu}_{12}},\ q_{43} = \dfrac{1}{\bar{\mu}_{12}},\ q_{44} = \dfrac{q_{42}}{\mathcal{R}_c - q_{41}},\ \mathcal{R}_c > q_{41}, \\
q_{45} = \dfrac{q_{40}}{\mathcal{R}_c - q_{39}},\ \mathcal{R}_c > q_{39},\ q_{46} = \dfrac{q_{45}q_{36} + q_{44}q_{37} + q_{38}}{\mathcal{R}_c - q_{35}},\ \mathcal{R}_c > q_{35}, \\
q_{47} = \dfrac{(\mathcal{R}_c - q_{19})(q_{45}q_{33} + q_{34}) + q_{31}(q_{45}q_{21} + q_{22})}{(\mathcal{R}_c - q_{19})(\mathcal{R}_c - q_{32}) - q_{31}q_{20}},\ \mathcal{R}_c > q_{19},\ \mathcal{R}_c > q_{32}, \\
\qquad (\mathcal{R}_c - q_{19})(\mathcal{R}_c - q_{32}) > q_{31}q_{20},\ q_{48} = \dfrac{q_{47} + q_{45}q_{21} + q_{22}}{\mathcal{R}_c - q_{19}}, \\
q_{49} = q_{12}q_{48} + q_{14}q_{47} + q_{15}q_{46} + q_{16}q_{45} + q_{17}q_{44} + q_{18}, \\
q_{50} = q_{24}q_{48} + q_{26}q_{47} + q_{27}q_{46} + q_{28}q_{45} + q_{29}q_{44} + q_{30}, \\
q_{51} = \dfrac{q_{33}q_{48} + (\mathcal{R}_c - q_{11})q_{50}}{(\mathcal{R}_c - q_{25})(\mathcal{R}_c - q_{11}) - q_{33}q_{13}},\ \mathcal{R}_c > q_{11},\ \mathcal{R}_c > q_{25},\ (\mathcal{R}_c - q_{25})(\mathcal{R}_c - q_{11}) > q_{33}q_{13}, \\
q_{52} = \dfrac{q_{13}q_{51} + q_{12}q_{48} + q_{14}q_{47} + q_{15}q_{46} + q_{16}q_{45} + q_{17}q_{44} + q_{18}}{\mathcal{R}_c - q_{11}}.
\end{cases}
\tag{A.7}
$$

The Lyapunov function for the model system (**??**) is given as

$$
L = \omega^T \mathcal{V}^{-1} x = \ \omega_1\,I_{gu} + \omega_2\,I_{ga} + \omega_3\,I_{fu} + \omega_4\,I_{fa} + \omega_5\,I_{mu} + \omega_6\,I_{ma} +
$$

$$
\omega_7\,I_{vu} + \omega_8\,I_{va}
$$

where

$$\begin{cases} \omega_1 = q_{52}q_{11} + q_{48}q_{12} + q_{51}q_{13} + q_{47}q_{14} + q_{46}q_{15} + q_{45}q_{16} + q_{44}q_{17} + q_{18}, \\ \omega_2 = q_{48}q_{19} + q_{47}q_{20} + q_{45}q_{21} + q_{22}, \\ \omega_3 = q_{52}q_{23} + q_{48}q_{24} + q_{51}q_{25} + q_{47}q_{26} + q_{46}q_{27} + q_{45}q_{28} + q_{44}q_{29} + q_{30}, \\ \omega_4 = q_{48}q_{31} + q_{47}q_{32} + q_{45}q_{33} + q_{34}, \\ \omega_5 = q_{46}q_{35} + q_{45}q_{36} + q_{44}q_{37} + q_{38},, \\ \omega_6 = q_{45}q_{39} + q_{40},\ \omega_7 = q_{44}q_{41} + q_{42},\ \omega_8 = q_{43}. \end{cases} \tag{A.8}$$

□

Theorem A.2.2. *Let $\Omega \in \mathbb{R}^{12}_+$ be compact and positively invariant such that $x_0 \in \Omega$. Suppose that $f(x) \geq 0$ with $f(x_0) = 0 \in \Omega$, $\mathcal{F} \geq 0$, $\mathcal{V}^{-1} \geq 0$ and $\mathcal{V}^{-1}\mathcal{F}$ is irreducible. Let the disease free system $\dot{x} = g(0)$ have a unique equilibrium $x_0 > 0$ that is globally asymptotically stable in $\mathbb{R}^{12}_+$. If $\mathcal{R}_c < 1$ then the DFE, E_0, is globally asymptotically stable on Ω and if $\mathcal{R}_c > 1$ then E_0 is unstable and there exists at least one endemic equilibrium.*

Proof. By Perron-Frobenius theory, $l > 0$ since L is a Lyapunov function and $\mathcal{V}^{-1}\mathcal{F}$ is irreducible and non-negative. Hence by equation (A.5), $\dot{L} = 0$ implies that $l^T x = 0$ and thus $x = 0$. It can be seen that the only invariant set in $\mathbb{R}^{12}_+$ where $x = 0$ is the set $\{E_0\}$ since $f(x) = 0$ and by LaSalle's invariance principle, E_0 is globally asymptotically stable on Ω. This concludes the proof. □

Global Stability Analysis of the EE

Theorem A.2.3. *The unique endemic equilibrium E^*_e presented in equation 3.17 is globally asymptotically stable if and only if $\mathcal{R}_c > 1$.*

Proof. Let

$$L_1 = \sum_{i=1}^{12} D_i L_i, \quad D_i > 0, \tag{A.9}$$

be a Lyapunov function with

$$L_i = x_i - x_i^* - x_i^* \ln\left(\frac{x_i}{x_i^*}\right),$$
$$x_i \in \{S_g, S_f, S_m, S_v, I_{gu}, I_{ga}, I_{fu}, I_{fa}, I_{mu}, I_{ma}, I_{vu}, I_{va}\}. \tag{A.10}$$

Next, we equate the partial derivatives of L_1 w.r.t x_i, such that

$$\frac{\partial L_i}{\partial x_i} = D_i\left(1 - \frac{x_i^*}{x_i}\right) = 0 \tag{A.11}$$

which yields $x_i = x_i^*$ and this implies that $S_g = S_g^*$, $S_f = S_f^*$, $S_m = S_m^*$, $S_v = S_v^*$, $I_{gu} = I_{gu}^*$, $I_{ga} = I_{ga}^*$, $I_{fu} = I_{fu}^*$, $I_{fa} = I_{fa}^*$, $I_{mu} = I_{mu}^*$, $I_{ma} = I_{ma}^*$, $I_{vu} = I_{vu}^*$, $I_{va} = I_{va}^*$. Thus E_e^* is the only critical point for L_1. Also, the endemic equilibrium point is the global minimum for L_1 since

$$\frac{\partial^2 L_i}{\partial x_i^2} = D_i \frac{x_i^*}{x_i^2} > 0. \tag{A.12}$$

Differentiating L_1 with respect to time yields

$$\frac{dL_1}{dt} = \sum_{i=1}^{12} D_i \left(1 - \frac{x_i^*}{x_i}\right) \frac{dx_i}{dt}. \tag{A.13}$$

We present proof for the following cases:

1. $x_i < x_i^*, \qquad \frac{x_i^*}{x_i} > 1$

2. $x_i > x_i^*, \qquad \frac{x_i^*}{x_i} < 1$

CASE 1: $x_i < x_i^*, \qquad \frac{x_i^*}{x_i} > 1$

Given that $\frac{dx_i}{dt} \leq \frac{dN}{dt}$ for $i = 1, 2, ..., 12$, we have

$$\frac{dL_1}{dt} \leq \sum_{i=1}^{12} D_i \left(1 - \frac{x_i^*}{x_i}\right) \frac{dN}{dt} \tag{A.14}$$

Since

$$N \leq \frac{\Lambda}{\bar{\mu}_{min}} + \left(N(0) - \frac{\Lambda}{\bar{\mu}_{min}}\right) e^{-\bar{\mu}_{min} t}. \tag{A.15}$$

The time derivative of equation(A.15) yields

$$\frac{dN}{dt} \leq \bar{\mu}_{min} \left(\frac{\Lambda}{\bar{\mu}_{min}} - N_h(0)\right) e^{-\bar{\mu}_{min} t}. \tag{A.16}$$

Thus, from equation (A.16) we deduce that

$$\frac{dL_1}{dt} \leq \sum_{i=1}^{12} D_i \left(1 - \frac{x_i^*}{x_i}\right) \bar{\mu}_{min} \left(\frac{\Lambda}{\bar{\mu}_{min}} - N(0)\right) e^{-\bar{\mu}_{min} t} \tag{A.17}$$

It is obvious from equation (A.17) that if $N(0) \leq \frac{\Lambda}{\bar{\mu}_{min}}$ then $\frac{dL_1}{dt} \leq 0$ as $t \to \infty$. Also, if $N(0) > \frac{\Lambda}{\bar{\mu}_{min}}$ then $\frac{dL_1}{dt} \leq 0$ as $t \to \infty$. In addition, $\frac{dL_1}{dt} = 0$ if and only if $x_i < x_i^*$, that is, $S_g = S_g^*$, $S_f = S_f^*$, $S_m = S_m^*$, $S_v = S_v^*$, $I_{gu} = I_{gu}^*$, $I_{ga} = I_{ga}^*$, $I_{fu} = I_{fu}^*$, $I_{fa} = I_{fa}^*$, $I_{mu} =$

I^*_{mu}, $I_{ma} = I^*_{ma}$, $I_{vu} = I^*_{vu}$, $I_{va} = I^*_{va}$. Hence, the largest invariant set in Ω is the singleton $\{E^*_e\}$.

CASE 2: $x_i > x^*_i, \qquad \dfrac{x^*_i}{x_i} < 1$

From the construction of L_1 we have

$$L_i = x_i - x^*_i - x^*_i \ln\left(\frac{x_i}{x^*_i}\right) = x_i(1 - \frac{x^*_i}{x_i}) + x^*_i \ln\left(\frac{x^*_i}{x_i}\right),$$

which implies that $x_i(1 - \dfrac{x^*_i}{x_i}) \leq L_i \ \forall i = 1, 2, ..., 12,$ and dividing by x_i we get

$$(1 - \frac{x^*_i}{x_i}) \leq \frac{L_i}{x_i} \text{ by positivity of } x_i.$$

It follows that

$$D_i\,(1 - \frac{x^*_i}{x_i}) \leq \frac{D_i\,L_i}{x_i} \text{ since } D_i > 0. \tag{A.18}$$

$$\text{Thus, } \sum_{i=1}^{12} D_i \left(1 - \frac{x^*_i}{x_i}\right) \leq \sum_{i=1}^{12} \frac{D_i\,L_i}{x_i}, \tag{A.19}$$

and since

$$\frac{dx_i}{dt} \leq \frac{dN}{dt}, \quad \forall\ i = 1, 2, ..., 12 \tag{A.20}$$

we have

$$\frac{dL_1}{dt} \quad \leq \quad \sum_{i=1}^{12} \frac{D_i\,L_i}{x_i} \frac{dN}{dt}. \tag{A.21}$$

Since

$$N \leq \frac{\Lambda}{\bar{\mu}_{min}} + \left(N(0) - \frac{\Lambda}{\bar{\mu}_{min}}\right) e^{-\bar{\mu}_{min} t}. \tag{A.22}$$

The time derivative of equation(A.22) yields

$$\frac{dN}{dt} \leq \bar{\mu}_{min} \left(\frac{\Lambda}{\bar{\mu}_{min}} - N(0)\right) e^{-\bar{\mu}_{min} t}. \tag{A.23}$$

Thus, from equation (A.23) we deduce that

$$\frac{dL_1}{dt} \leq \sum_{i=1}^{12} \frac{D_i\,L_i}{x_i} \bar{\mu}_{min} \left(\frac{\Lambda}{\bar{\mu}_{min}} - N(0)\right) e^{-\bar{\mu}_{min} t}. \tag{A.24}$$

It is obvious from equation (A.24) that if $N(0) \leq \frac{\Lambda}{\bar{\mu}_{min}}$ then $\frac{dL_1}{dt} \leq 0$ as $t \to \infty$. Also, if $N(0) > \frac{\Lambda}{\bar{\mu}_{min}}$ then $\frac{dL_1}{dt} \leq 0$ as $t \to \infty$. In addition, $\frac{dL_1}{dt} = 0$ if and only if $L_i = 0$. This is possible if and only if $x_i = x_i^*$ implying $S_g = S_g^*$, $S_f = S_f^*$, $S_m = S_m^*$, $S_v = S_v^*$, $I_{gu} = I_{gu}^*$, $I_{ga} = I_{ga}^*$, $I_{fu} = I_{fu}^*$, $I_{fa} = I_{fa}^*$, $I_{mu} = I_{mu}^*$, $I_{ma} = I_{ma}^*$, $I_{vu} = I_{vu}^*$, $I_{va} = I_{va}^*$. Hence, the largest invariant set in Ω is the singleton $\{E_e^*\}$.

From case 1 and case 2 we conclude that by LaSalle's invariance principle E_e^* is globally asymptotically stable.

□

A.3 Chapter 3 Appendices

A.3.1 Endemic Equilibrium Expressions

Expressions for $g_{00}, g_{01}, ..., g_{11}$, $q_{01}, q_{02}, ..., q_{20}$, $h_{01}, h_{02}, ..., h_{20}$, $C_1, C_2, ..., C_5$ and $C_{11}, C_{21}, ..., C_{51}$ in section 4.2.4.

$$
\begin{cases}
g_{00} = \dfrac{\rho_{ct}^{f1}\,\mu_{f5} + \rho_{ct}^{f1}\,\rho_{ct}^{f}}{\mu_{f5}\mu_{f6} - \rho_{ct}^{f1}\,\rho_{ct}^{f}},\ g_{01} = \dfrac{\rho_{ct}^{f}(1+g_{00})}{\mu_{f5}},\ g_{02} = 1 + \rho_{ht}^{m},\ g_{03} = \dfrac{\Lambda_{fa}\,\rho_{ht}^{m1}}{\mu_{f4}},\ g_{04} = \dfrac{\Lambda_{fu}\,\rho_{ht}^{m1}\rho_{ht}^{f}}{\mu_{f4}}, \\
g_{05} = \dfrac{\Lambda_{fu}\,\rho_{ht}^{m}}{\mu_{f4}} + \dfrac{\Lambda_{fu}\,\rho_{ht}^{f}}{\mu_{f3}\,\mu_{f4}}, \\
g_{06} = \dfrac{\rho_{ct}^{m1}\,\mu_{m5} + \rho_{ct}^{m1}\,\rho_{ct}^{m}}{\mu_{m5}\mu_{m6} - \rho_{ct}^{m1}\,\rho_{ct}^{m}},\ g_{07} = \dfrac{\rho_{ct}^{m}(1+g_{06})}{\mu_{m5}},\ g_{08} = 1 + \rho_{ht}^{f},\ g_{09} = \dfrac{\Lambda_{ma}\,\rho_{ht}^{f1}}{\mu_{m4}},\ g_{10} = \dfrac{\Lambda_{mu}\,\rho_{ht}^{f1}\rho_{ht}^{m}}{\mu_{m4}}, \\
g_{11} = \dfrac{\Lambda_{mu}\,\rho_{ht}^{f}}{\mu_{m4}} + \dfrac{\Lambda_{mu}\,\rho_{ht}^{m}}{\mu_{m3}\,\mu_{m4}}, \\
q_{01} = N_f^*(\bar{\mu}_f + \delta_f) - (\Lambda_{fu} + \Lambda_{fa}),\ q_{02} = q_{01}\,g_{02}\,\rho_{ht}^{f}, \\
q_{03} = (g_{02}\,q_{01}\,\mu_{f2} + q_{01}\,\mu_{f1}\rho_{ht}^{f}) - (\Lambda_{fu}\,\rho_{ht}^{f1}\,\delta_f + \Lambda_{fa}\,g_{02}\,\delta_f), \\
q_{04} = q_{01}\,\mu_{f1}\,\mu_{f2} - (\Lambda_{fu}\,\mu_{f2}\,\delta_f + \Lambda_{fa}\,\mu_{f1}\,\delta_f + \rho_{ht}^{f}\,\Lambda_{fu}\,\delta_f),\ q_{05} = \delta_f\,\rho_{ht}^{f}\,g_{00}\,g_{02}, \\
q_{06} = g_{00}\,g_{02}\,\delta_f\,\mu_{f2} + g_{00}\,\delta_f\,\mu_{f1}\,\rho_{ht}^{f},\ q_{07} = g_{00}\,\delta_f\,\mu_{f1}\,\mu_{f2}, \\
q_{08} = g_{02}\,g_{03}\,\rho_{ht}^{m1} + g_{05}\,\rho_{ht}^{m}\,\rho_{ht}^{m1}, \\
q_{09} = g_{02}\,g_{03}\,\mu_{f2} + g_{03}\,\mu_{f1}\,\rho_{ht}^{m1} + g_{05}\,\mu_{f1}\,\rho_{ht}^{m1} + g_{05}\,\mu_{f2}\,\rho_{ht}^{m} + g_{04}\,\rho_{ht}^{m},
\end{cases}
\tag{A.25}
$$

$$
\begin{cases}
q_{10} = g_{03}\,\mu_{f1}\,\mu_{f2} + g_{05}\,\mu_{f1}\,\mu_{f2} + g_{04}\,\mu_{f2},\ q_{11} = g_{02}\,\rho_{ht}^{m}\,\rho_{ht}^{m1},\\
q_{12} = g_{02}\,\mu_{f2}\,\rho_{ht}^{m} + g_{02}\,\mu_{f2}\,\rho_{ht}^{m1} + \mu_{f1}\,\rho_{ht}^{m}\,\rho_{ht}^{m1},\\
q_{13} = g_{02}\,\mu_{f2}^{2} + \mu_{f1}\,\mu_{f2}\rho_{ht}^{m} + \mu_{f1}\,\mu_{f2}\rho_{ht}^{m1},\ q_{14} = \mu_{f1}\,\mu_{f2}^{2},\\
q_{15} = q_{08}\,q_{05} - q_{02}\,q_{11},\ \ q_{16} = q_{06}\,q_{08} + q_{05}\,q_{09} - (q_{02}\,q_{12} + q_{03}\,q_{11}),\\
q_{17} = q_{05}\,q_{10} + q_{06}\,q_{09} + q_{07}\,q_{08} - (q_{02}\,q_{13} + q_{03}\,q_{12} + q_{04}\,q_{11}),\\
q_{18} = q_{06}\,q_{10} + q_{07}\,q_{09} - (q_{02}\,q_{14} + q_{03}\,q_{13} + q_{04}\,q_{12}),\\
q_{19} = q_{07}\,q_{10} - (q_{03}\,q_{14} + q_{04}\,q_{13}),\ \ q_{20} = q_{04}\,q_{14},\\
h_{01} = N_{m}^{*}(\bar{\mu}_{m} + \delta_{m}) - (\Lambda_{mu} + \Lambda_{ma}),\ h_{02} = h_{01}\,g_{08}\,\rho_{ht}^{m},\\
h_{03} = (g_{08}\,h_{01}\,\mu_{m2} + h_{01}\,\mu_{m1}\rho_{ht}^{m}) - (\Lambda_{mu}\,\rho_{ht}^{m1}\,\delta_{m} + \Lambda_{ma}\,g_{08}\,\delta_{m}),\\
h_{04} = h_{01}\,\mu_{m1}\,\mu_{m2} - (\Lambda_{mu}\,\mu_{m2}\,\delta_{m} + \Lambda_{ma}\,\mu_{m1}\,\delta_{m} + \rho_{ht}^{m}\,\Lambda_{mu}\,\delta_{m}),\ h_{05} = \delta_{m}\,\rho_{ht}^{m}\,g_{06}\,g_{08},\\
h_{06} = g_{06}\,g_{08}\,\delta_{m}\,\mu_{m2} + g_{06}\,\delta_{m}\,\mu_{m1}\,\rho_{ht}^{m},\ \ h_{07} = g_{06}\,\delta_{m}\,\mu_{m1}\,\mu_{m2},\\
h_{08} = g_{08}\,g_{09}\,\rho_{ht}^{f1} + g_{11}\,\rho_{ht}^{f}\,\rho_{ht}^{f1},\\
h_{09} = g_{08}\,g_{09}\,\mu_{m2} + g_{09}\,\mu_{m1}\,\rho_{ht}^{f1} + g_{11}\,\mu_{m1}\,\rho_{ht}^{f1} + g_{11}\,\mu_{m2}\,\rho_{ht}^{f} + g_{10}\,\rho_{ht}^{f},\\
h_{10} = g_{09}\,\mu_{m1}\,\mu_{m2} + g_{11}\,\mu_{m1}\,\mu_{m2} + g_{10}\,\mu_{m2},\ h_{11} = g_{08}\,\rho_{ht}^{f}\,\rho_{ht}^{f1},\\
h_{12} = g_{08}\,\mu_{m2}\,\rho_{ht}^{f} + g_{08}\,\mu_{m2}\,\rho_{ht}^{f1} + \mu_{m1}\,\rho_{ht}^{f}\,\rho_{ht}^{f1},\\
h_{13} = g_{08}\,\mu_{m2}^{2} + \mu_{m1}\,\mu_{m2}\rho_{ht}^{f} + \mu_{m1}\,\mu_{m2}\rho_{ht}^{f1},\ h_{14} = \mu_{m1}\,\mu_{m2}^{2},\\
h_{15} = h_{08}\,h_{05} - h_{02}\,h_{11},\ \ h_{16} = h_{06}\,h_{08} + h_{05}\,h_{09} - (h_{02}\,h_{12} + h_{03}\,h_{11}),\\
h_{17} = h_{05}\,h_{10} + h_{06}\,h_{09} + h_{07}\,h_{08} - (h_{02}\,h_{13} + h_{03}\,h_{12} + h_{04}\,h_{11}),\\
h_{18} = h_{06}\,h_{10} + h_{07}\,h_{09} - (h_{02}\,h_{14} + h_{03}\,h_{13} + h_{04}\,h_{12}),\\
h_{19} = h_{07}\,h_{10} - (h_{03}\,h_{14} + h_{04}\,h_{13}),\ \ h_{20} = h_{04}\,h_{14},\\
C_{1} = \dfrac{q_{16}}{q_{15}},\ \ C_{2} = \dfrac{q_{17}}{q_{15}},\ \ C_{3} = \dfrac{q_{18}}{q_{15}},\ \ C_{4} = \dfrac{q_{19}}{q_{15}},\ \ C_{5} = \dfrac{q_{20}}{q_{15}},\\
\\
C_{11} = \dfrac{h_{16}}{h_{15}},\ \ C_{21} = \dfrac{h_{17}}{h_{15}},\ \ C_{31} = \dfrac{h_{18}}{h_{15}},\ \ C_{41} = \dfrac{h_{19}}{h_{15}},\ \ C_{51} = \dfrac{h_{20}}{h_{15}}.
\end{cases}
\tag{A.26}
$$

A.3.2 Single-Sex Model Description, Parameter Description and Parameter Values

Equation A.27 gives the single-sex model incidence rates and exit rates presented in equation 4.20.

$$
\begin{cases}
\beta_u = \dfrac{c\,\gamma}{N_y}\left[I_u + \alpha_c\rho_c I_a + (\alpha_c\rho_c + \alpha_t\rho_t)T_u\right], \\
\beta_a = \dfrac{c\,\gamma}{N_y}\left[I_u + \alpha_c\rho_c I_a + (\alpha_c\rho_c + \alpha_t\rho_t)T_u\right]\alpha_{ht}\,\rho_{ht}, \\
\tilde{\beta}_a = \dfrac{c\,\gamma}{N_y}\left[I_u + \alpha_c\rho_c I_a + (\alpha_c\rho_c + \alpha_t\rho_t)T_u\right]\alpha^1_{ht}\,\rho_{ht}, \\
\mu_1 = \rho_{ht} + \mu + \sigma,\ \mu_2 = \mu + \sigma,\ \mu_3 = \rho_{ht} + \mu + \sigma + \delta,\ \mu_4 = \rho_{ct} + \rho^1_{ct} + \mu + \sigma + \delta, \\
\mu_5 = \rho^1_{ct} + \bar{\mu} + \delta,\ \mu_6 = \rho_{ct} + \mu + \sigma.
\end{cases}
\tag{A.27}
$$

Table A.1: Description of Single-Sex Model State variables

Variable	Description
S_u	Susceptible youth who have never tested for HIV/AIDS
S_a	Susceptible youth who have ever tested for HIV/AIDS
I_u	Infected youth who have never tested for HIV/AIDS
I_a	Infected youth who have ever tested for HIV/AIDS
T_u	Infected aware youth who are not adherent to ART or consistent condom use
T_a	Infected aware youth who are adherent to ART and use condoms consistently

Table A.2: Description of Single-Sex Model Parameters

Parameter	Description
Λ_u	Natural birth and maturity rate of susceptible youth unaware of their HIV status
Λ_a	Natural birth and maturity rate of susceptible youth aware of their HIV status
ρ_{ht}	Youth HIV/AIDS testing rates
ρ_t	Youth adherence rate to anti-retroviral therapy treatment
ρ_c	Youth condom use rate
μ	Natural death rate of youth respectively
γ	Probability of youth transmission risk
δ	Disease induced deaths in youth
c_m	Youth sexual contact rate
α_{ht}, α^1_{ht}	Factors negatively and positively influencing HIV/AIDS testing rate among the youth
α_c, α^1_c	Factors negatively and positively influencing condom use rate among the youth
α_t, α^1_t	Factors negatively and positively influencing ART adherence rate among the youth
σ	Exit rate of youth upon turning 24 years

Table A.3: Parameter Values for the Single-Sex Model, $\tilde{\gamma} = c\,\gamma$

Parameter	Value	Unit	Source
Λ_u, Λ_a	60.476325, 100.55365	$year^{-1}$	Data Estimated
μ	0.0095859	$year^{-1}$	Data Estimated
$\tilde{\gamma}$	3.17245525	$year^{-1}$	Data Estimated
δ	0.0095	$year^{-1}$	Data Estimated
σ	0.041667	$year^{-1}$	Calculated
ρ_{ht}	0.48	$year^{-1}$	Data Estimated
ρ_c	0.3	$year^{-1}$	Data Estimated
ρ_t	0.1	$year^{-1}$	Data Estimated
α_{ht}, α_c, α_t	0.4, 0.27, 0.1	$year^{-1}$	Estimated
α^1_{ht}, α^1_c, α^1_t	0.78, 0.8, 0.75	$year^{-1}$	Estimated

Table A.4: Adjusted Parameter Values for the Single-Sex Model

Parameter	Value	Unit	Source
Λ_u, Λ_a	60.476325, 100.55365	$year^{-1}$	Data Estimated
μ	0.0095859	$year^{-1}$	Data Estimated
$\tilde{\gamma}$	0.03022869	$year^{-1}$	Data Estimated
δ	0.0095	$year^{-1}$	Data Estimated
σ	0.041667	$year^{-1}$	Calculated
ρ_{ht}	0.48	$year^{-1}$	Data Estimated
ρ_c	0.3	$year^{-1}$	Data Estimated
ρ_t	0.1	$year^{-1}$	Data Estimated
α_{ht}, α_c, α_t	0.4, 0.27, 0.1	$year^{-1}$	Estimated
α^1_{ht}, α^1_c, α^1_t	0.78, 0.8, 0.75	$year^{-1}$	Estimated

A.4 Chapter 4 Appendices

A.4.1 Matrices $\mathcal{F}$, $\mathcal{V}$ and $\mathcal{F}\mathcal{V}^{-1}$ entries expressions

$$
\begin{cases}
\mathcal{F}_{11} = \begin{bmatrix} q_1 & 0 & 0 & 0 \\ 0 & q_2 & 0 & 0 \\ 0 & 0 & q_3 & 0 \\ 0 & 0 & 0 & q_4 \end{bmatrix}, \qquad \mathcal{F}_{12} = \begin{bmatrix} p_1 & 0 & 0 & 0 \\ 0 & p_2 & 0 & 0 \\ 0 & 0 & p_3 & 0 \\ 0 & 0 & 0 & p_4 \end{bmatrix}, \qquad \mathcal{F}_{13} = \begin{bmatrix} \omega_1 & 0 & 0 & 0 \\ 0 & \omega_2 & 0 & 0 \\ 0 & 0 & \omega_3 & 0 \\ 0 & 0 & 0 & \omega_4 \end{bmatrix}, \\
\mathcal{F}_{21} = \begin{bmatrix} q_{11} & 0 & 0 & 0 \\ 0 & q_{21} & 0 & 0 \\ 0 & 0 & q_{31} & 0 \\ 0 & 0 & 0 & q_{41} \end{bmatrix}, \qquad \mathcal{F}_{22} = \begin{bmatrix} p_{11} & 0 & 0 & 0 \\ 0 & p_{21} & 0 & 0 \\ 0 & 0 & p_{31} & 0 \\ 0 & 0 & 0 & p_{41} \end{bmatrix}, \qquad \mathcal{F}_{23} = \begin{bmatrix} \omega_{11} & 0 & 0 & 0 \\ 0 & \omega_{21} & 0 & 0 \\ 0 & 0 & \omega_{31} & 0 \\ 0 & 0 & 0 & \omega_{41} \end{bmatrix}, \\
\mathcal{F}_0 = \mathcal{F}_{31} = \mathcal{F}_{32} = \mathcal{F}_{33} = \mathcal{V}_{12} = \mathcal{V}_{13} = \mathcal{V}_{23} = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \\
\mathcal{V}_{11}^{-1} = \begin{bmatrix} a_1 & 0 & a_2 & 0 \\ 0 & a_3 & 0 & a_4 \\ a_5 & 0 & a_6 & 0 \\ 0 & a_7 & 0 & a_8 \end{bmatrix}, \qquad \mathcal{V}_{21}^{-1} = \begin{bmatrix} b_1 & 0 & b_2 & 0 \\ 0 & b_3 & 0 & b_4 \\ b_5 & 0 & b_6 & 0 \\ 0 & b_7 & 0 & b_8 \end{bmatrix}, \qquad \mathcal{V}_{22}^{-1} = \begin{bmatrix} c_1 & 0 & c_2 & 0 \\ 0 & c_3 & 0 & c_4 \\ c_5 & 0 & c_6 & 0 \\ 0 & c_7 & 0 & c_8 \end{bmatrix}, \\
\mathcal{V}_{31}^{-1} = \begin{bmatrix} d_1 & 0 & d_2 & 0 \\ 0 & d_3 & 0 & d_4 \\ d_5 & 0 & d_6 & 0 \\ 0 & d_7 & 0 & d_8 \end{bmatrix}, \qquad \mathcal{V}_{32}^{-1} = \begin{bmatrix} e_1 & 0 & e_2 & 0 \\ 0 & e_3 & 0 & e_4 \\ e_5 & 0 & e_6 & 0 \\ 0 & e_7 & 0 & e_8 \end{bmatrix}, \qquad \mathcal{V}_{33}^{-1} = \begin{bmatrix} g_1 & 0 & g_2 & 0 \\ 0 & g_3 & 0 & g_4 \\ g_5 & 0 & g_6 & 0 \\ 0 & g_7 & 0 & g_8 \end{bmatrix}, \\
q_1 = \dfrac{c_{12}\,\gamma_{12}\;S_{11}^{u0}}{\tilde{N}_{12}}, \qquad q_2 = \dfrac{c_{12}\,\gamma_{12}\;S_{12}^{u0}}{\tilde{N}_{12}}, \qquad q_3 = \dfrac{c_{21}\,\gamma_{21}\;S_{21}^{u0}}{\tilde{N}_{21}}, \qquad q_4 = \dfrac{c_{21}\,\gamma_{21}\;S_{22}^{u0}}{\tilde{N}_{21}}, \\
p_1 = \dfrac{\alpha_{12}^c\,\rho_{12}^c\,c_{12}\,\gamma_{12}\;S_{11}^{u0}}{\tilde{N}_{12}}, \qquad p_2 = \dfrac{\alpha_{12}^c\,\rho_{12}^c\,c_{12}\,\gamma_{12}\;S_{12}^{u0}}{\tilde{N}_{12}}, \qquad p_3 = \dfrac{\alpha_{21}^c\,\rho_{21}^c\,c_{21}\,\gamma_{21}\;S_{21}^{u0}}{\tilde{N}_{21}}, \\
p_4 = \dfrac{\alpha_{21}^c\,\rho_{21}^c\,c_{21}\,\gamma_{21}\;S_{22}^{u0}}{\tilde{N}_{21}}, \\
\omega_1 = \dfrac{\rho_{12}^{ct}\,c_{12}\,\gamma_{12}\;S_{11}^{u0}}{\tilde{N}_{12}}, \qquad \omega_2 = \dfrac{\rho_{12}^{ct}\,c_{12}\,\gamma_{12}\;S_{12}^{u0}}{\tilde{N}_{12}}, \qquad \omega_3 = \dfrac{\rho_{21}^{ct}\,c_{21}\,\gamma_{21}\;S_{21}^{u0}}{\tilde{N}_{21}}, \qquad \omega_4 = \dfrac{\rho_{21}^{ct}\,c_{21}\,\gamma_{21}\;S_{22}^{u0}}{\tilde{N}_{21}},
\end{cases}
\tag{A.28}
$$

$$
\begin{cases}
q_{11} = \dfrac{\tilde{\rho}_{12}^{ht}\, c_{12}\, \gamma_{12}\, \left(S_{11}^{u0} + \alpha_{12}^{ht}\, S_{11}^{a0}\right)}{\tilde{N}_{12}}, \qquad q_{21} = \dfrac{\tilde{\rho}_{12}^{ht}\, c_{12}\, \gamma_{12}\, \left(S_{12}^{u0} + \alpha_{12}^{ht}\, S_{12}^{a0}\right)}{\tilde{N}_{12}}, \\
q_{31} = \dfrac{\tilde{\rho}_{21}^{ht}\, c_{21}\, \gamma_{21}\, \left(S_{21}^{u0} + \alpha_{21}^{ht}\, S_{21}^{a0}\right)}{\tilde{N}_{21}}, \qquad q_{41} = \dfrac{\tilde{\rho}_{21}^{ht}\, c_{21}\, \gamma_{21}\, \left(S_{22}^{u0} + \alpha_{21}^{ht}\, S_{22}^{a0}\right)}{\tilde{N}_{21}}, \\
p_{11} = \dfrac{\alpha_{12}^{c}\, \rho_{12}^{c}\, \tilde{\rho}_{12}^{ht}\, c_{12}\, \gamma_{12}\, \left(S_{11}^{u0} + \alpha_{12}^{ht}\, S_{11}^{a0}\right)}{\tilde{N}_{12}}, \qquad p_{21} = \dfrac{\alpha_{12}^{c}\, \rho_{12}^{c}\, \tilde{\rho}_{12}^{ht}\, c_{12}\, \gamma_{12}\, \left(S_{12}^{u0} + \alpha_{12}^{ht}\, S_{12}^{a0}\right)}{\tilde{N}_{12}}, \\
p_{31} = \dfrac{\alpha_{21}^{c}\, \rho_{21}^{c}\, \tilde{\rho}_{21}^{ht}\, c_{21}\, \gamma_{21}\, \left(S_{21}^{u0} + \alpha_{21}^{ht}\, S_{21}^{a0}\right)}{\tilde{N}_{21}}, \qquad p_{41} = \dfrac{\alpha_{21}^{c}\, \rho_{21}^{c}\, \tilde{\rho}_{21}^{ht}\, c_{21}\, \gamma_{21}\, \left(S_{22}^{u0} + \alpha_{21}^{ht}\, S_{22}^{a0}\right)}{\tilde{N}_{21}}, \\
\omega_{11} = \dfrac{\rho_{12}^{ct}\, \tilde{\rho}_{12}^{ht}\, c_{12}\, \gamma_{12}\, \left(S_{11}^{u0} + \alpha_{12}^{ht}\, S_{11}^{a0}\right)}{\tilde{N}_{12}}, \qquad \omega_{21} = \dfrac{\rho_{12}^{ct}\, \tilde{\rho}_{12}^{ht}\, c_{12}\, \gamma_{12}\, \left(S_{12}^{u0} + \alpha_{12}^{ht}\, S_{12}^{a0}\right)}{\tilde{N}_{12}}, \\
\omega_{31} = \dfrac{\rho_{21}^{ct}\, \tilde{\rho}_{21}^{ht}\, c_{21}\, \gamma_{21}\, \left(S_{21}^{u0} + \alpha_{21}^{ht}\, S_{21}^{a0}\right)}{\tilde{N}_{21}}, \qquad \omega_{41} = \dfrac{\rho_{21}^{ct}\, \tilde{\rho}_{21}^{ht}\, c_{21}\, \gamma_{21}\, \left(S_{22}^{u0} + \alpha_{21}^{ht}\, S_{22}^{a0}\right)}{\tilde{N}_{21}}, \\
a_1 = \dfrac{\mu_{21}^{3}}{d_{21}\, r_{21} + \mu_{11}^{3}\, \mu_{21}^{3}}, \quad a_2 = \dfrac{r_{21}}{d_{21}\, r_{21} + \mu_{11}^{3}\, \mu_{21}^{3}} \quad a_3 = \dfrac{\mu_{22}^{3}}{d_{12}\, r_{12} + \mu_{12}^{3}\, \mu_{22}^{3}}, \\
a_4 = -\dfrac{d_{12}}{d_{12}\, r_{12} + \mu_{12}^{3}\, \mu_{22}^{3}}, \quad a_5 = -\dfrac{d_{21}}{d_{21}\, r_{21} + \mu_{11}^{3}\, \mu_{21}^{3}}, \quad a_6 = \dfrac{\mu_{11}^{3}}{d_{21}\, r_{21} + \mu_{11}^{3}\, \mu_{21}^{3}}, \\
a_7 = \dfrac{r_{12}}{d_{12}\, r_{12} + \mu_{12}^{3}\, \mu_{22}^{3}}, \quad a_8 = \dfrac{\mu_{12}^{3}}{d_{12}\, r_{12} + \mu_{12}^{3}\, \mu_{22}^{3}}, \\
b_1 = -\dfrac{d_{21}\, r_{21}\, \bar{\alpha}_{21} - \bar{\alpha}_{11}\, \mu_{21}^{3}\, \mu_{21}^{4}}{\left(d_{21}\, r_{21} + \mu_{11}^{4}\, \mu_{21}^{4}\right)\left(d_{21}\, r_{21} + \mu_{11}^{3}\, \mu_{21}^{3}\right)}, \quad b_2 = \dfrac{r_{21}\, \left(\bar{\alpha}_{11}\, \mu_{21}^{4} + \bar{\alpha}_{21}\, \mu_{11}^{3}\right)}{\left(d_{21}\, r_{21} + \mu_{11}^{4}\, \mu_{21}^{4}\right)\left(d_{21}\, r_{21} + \mu_{11}^{3}\, \mu_{21}^{3}\right)}, \\
b_3 = -\dfrac{d_{12}\, r_{12}\, \bar{\alpha}_{22} - \bar{\alpha}_{12}\, \mu_{22}^{3}\, \mu_{22}^{4}}{\left(d_{12}\, r_{12} + \mu_{12}^{4}\, \mu_{22}^{4}\right)\left(d_{12}\, r_{12} + \mu_{12}^{3}\, \mu_{22}^{3}\right)}, \quad b_4 = -\dfrac{d_{12}\, \left(\bar{\alpha}_{12}\, \mu_{22}^{4} + \bar{\alpha}_{22}\, \mu_{12}^{3}\right)}{\left(d_{12}\, r_{12} + \mu_{12}^{4}\, \mu_{22}^{4}\right)\left(d_{12}\, r_{12} + \mu_{12}^{3}\, \mu_{22}^{3}\right)}, \\
b_5 = -\dfrac{d_{21}\, \left(\bar{\alpha}_{11}\, \mu_{21}^{3} + \bar{\alpha}_{21}\, \mu_{11}^{4}\right)}{\left(d_{21}\, r_{21} + \mu_{11}^{3}\, \mu_{21}^{3}\right)\left(d_{21}\, r_{21} + \mu_{11}^{4}\, \mu_{21}^{4}\right)}, \quad b_6 = -\dfrac{d_{21}\, r_{21}\, \bar{\alpha}_{11} - \bar{\alpha}_{21}\, \mu_{11}^{3}\, \mu_{11}^{4}}{\left(d_{21}\, r_{21} + \mu_{11}^{3}\, \mu_{21}^{3}\right)\left(d_{21}\, r_{21} + \mu_{11}^{4}\, \mu_{21}^{4}\right)}, \\
b_7 = \dfrac{r_{12}\, \left(\bar{\alpha}_{12}\, \mu_{22}^{3} + \bar{\alpha}_{22}\, \mu_{12}^{4}\right)}{\left(d_{12}\, r_{12} + \mu_{12}^{3}\, \mu_{22}^{3}\right)\left(d_{12}\, r_{12} + \mu_{12}^{4}\, \mu_{22}^{4}\right)}, \quad b_8 = -\dfrac{d_{12}\, r_{12}\, \bar{\alpha}_{12} - \bar{\alpha}_{22}\, \mu_{12}^{3}\, \mu_{12}^{4}}{\left(d_{12}\, r_{12} + \mu_{12}^{3}\, \mu_{22}^{3}\right)\left(d_{12}\, r_{12} + \mu_{12}^{4}\, \mu_{22}^{4}\right)}, \\
c_1 = \dfrac{\mu_{21}^{4}}{d_{21}\, r_{21} + \mu_{11}^{4}\, \mu_{21}^{4}}, \quad c_2 = \dfrac{r_{21}}{d_{21}\, r_{21} + \mu_{11}^{4}\, \mu_{21}^{4}}, \quad c_3 = \dfrac{\mu_{22}^{4}}{d_{12}\, r_{12} + \mu_{12}^{4}\, \mu_{22}^{4}}, \\
c_4 = -\dfrac{d_{12}}{d_{12}\, r_{12} + \mu_{12}^{4}\, \mu_{22}^{4}}, \quad c_5 = -\dfrac{d_{21}}{d_{21}\, r_{21} + \mu_{11}^{4}\, \mu_{21}^{4}}, \quad c_6 = \dfrac{\mu_{11}^{4}}{d_{21}\, r_{21} + \mu_{11}^{4}\, \mu_{21}^{4}}, \\
c_7 = \dfrac{r_{12}}{d_{12}\, r_{12} + \mu_{12}^{4}\, \mu_{22}^{4}}, \quad c_8 = \dfrac{\mu_{12}^{4}}{d_{12}\, r_{12} + \mu_{12}^{4}\, \mu_{22}^{4}}, \\
d_1 = -\dfrac{d_{21}\, r_{21}\, \bar{\alpha}_{11}\, \mu_{21}^{3}\, \rho_{21} + d_{21}\, r_{21}\, \bar{\alpha}_{21}\, \mu_{11}^{4}\, \rho_{21} + d_{21}\, r_{21}\, \bar{\alpha}_{21}\, \mu_{21}^{5}\, \rho_{11} - \bar{\alpha}_{11}\, \mu_{21}^{3}\, \mu_{21}^{4}\, \mu_{21}^{5}\, \rho_{11}}{\left(d_{21}\, r_{21} + \mu_{11}^{4}\, \mu_{21}^{4}\right)\left(d_{21}\, r_{21} + \mu_{11}^{3}\, \mu_{21}^{3}\right)\left(d_{21}\, r_{21} + \mu_{11}^{5}\, \mu_{21}^{5}\right)}, \\
d_2 = -\dfrac{r_{21}\, \left(d_{21}\, r_{21}\, \bar{\alpha}_{11}\, \rho_{21} - \bar{\alpha}_{11}\, \mu_{21}^{4}\, \mu_{21}^{5}\, \rho_{11} - \bar{\alpha}_{21}\, \mu_{11}^{3}\, \mu_{11}^{4}\, \rho_{21} - \bar{\alpha}_{21}\, \mu_{11}^{3}\, \mu_{21}^{5}\, \rho_{11}\right)}{\left(d_{21}\, r_{21} + \mu_{11}^{4}\, \mu_{21}^{4}\right)\left(d_{21}\, r_{21} + \mu_{11}^{3}\, \mu_{21}^{3}\right)\left(d_{21}\, r_{21} + \mu_{11}^{5}\, \mu_{21}^{5}\right)}, \\
d_3 = -\dfrac{d_{12}\, r_{12}\, \bar{\alpha}_{12}\, \mu_{22}^{3}\, \rho_{22} + d_{12}\, r_{12}\, \bar{\alpha}_{22}\, \mu_{12}^{4}\, \rho_{22} + d_{12}\, r_{12}\, \bar{\alpha}_{22}\, \mu_{22}^{5}\, \rho_{12} - \bar{\alpha}_{12}\, \mu_{22}^{3}\, \mu_{22}^{4}\, \mu_{22}^{5}\, \rho_{12}}{\left(d_{12}\, r_{12} + \mu_{12}^{4}\, \mu_{22}^{4}\right)\left(d_{12}\, r_{12} + \mu_{12}^{3}\, \mu_{22}^{3}\right)\left(d_{12}\, r_{12} + \mu_{12}^{5}\, \mu_{22}^{5}\right)}, \\
d_4 = \dfrac{d_{12}\, \left(d_{12}\, r_{12}\, \bar{\alpha}_{12}\, \rho_{22} - \bar{\alpha}_{12}\, \mu_{22}^{4}\, \mu_{22}^{5}\, \rho_{12} - \bar{\alpha}_{22}\, \mu_{12}^{3}\, \mu_{12}^{4}\, \rho_{22} - \bar{\alpha}_{22}\, \mu_{12}^{3}\, \mu_{22}^{5}\, \rho_{12}\right)}{\left(d_{12}\, r_{12} + \mu_{12}^{4}\, \mu_{22}^{4}\right)\left(d_{12}\, r_{12} + \mu_{12}^{3}\, \mu_{22}^{3}\right)\left(d_{12}\, r_{12} + \mu_{12}^{5}\, \mu_{22}^{5}\right)},
\end{cases}
\tag{A.29}
$$

$$
\begin{cases}
d_5 = \dfrac{d_{21}\,\left(d_{21}\,r_{21}\,\bar{\alpha}_{21}\,\rho_{11} - \bar{\alpha}_{11}\,\mu_{21}^3\,\mu_{21}^4\,\rho_{11} - \bar{\alpha}_{11}\,\mu_{21}^3\,\mu_{11}^5\,\rho_{21} - \bar{\alpha}_{21}\,\mu_{11}^4\,\mu_{11}^5\,\rho_{21}\right)}{\left(d_{21}\,r_{21} + \mu_{11}^4\,\mu_{21}^4\right)\left(d_{21}\,r_{21} + \mu_{11}^3\,\mu_{21}^3\right)\left(d_{21}\,r_{21} + \mu_{11}^5\,\mu_{21}^5\right)}, \\
d_6 = -\dfrac{d_{21}\,r_{21}\,\bar{\alpha}_{11}\,\mu_{21}^4\,\rho_{11} + d_{21}\,r_{21}\,\bar{\alpha}_{11}\,\mu_{11}^5\,\rho_{21} + d_{21}\,r_{21}\,\bar{\alpha}_{21}\,\mu_{11}^3\,\rho_{11} - \bar{\alpha}_{21}\,\mu_{11}^3\,\mu_{11}^4\,\mu_{11}^5\,\rho_{21}}{\left(d_{21}\,r_{21} + \mu_{11}^4\,\mu_{21}^4\right)\left(d_{21}\,r_{21} + \mu_{11}^3\,\mu_{21}^3\right)\left(d_{21}\,r_{21} + \mu_{11}^5\,\mu_{21}^5\right)}, \\
d_7 = -\dfrac{r_{12}\,\left(d_{12}\,r_{12}\,\bar{\alpha}_{22}\,\rho_{12} - \bar{\alpha}_{12}\,\mu_{22}^3\,\mu_{22}^4\,\rho_{12} - \bar{\alpha}_{12}\,\mu_{22}^3\,\mu_{12}^5\,\rho_{22} - \bar{\alpha}_{22}\,\mu_{12}^4\,\mu_{512}\,\rho_{22}\right)}{\left(d_{12}\,r_{12} + \mu_{12}^4\,\mu_{22}^4\right)\left(d_{12}\,r_{12} + \mu_{12}^3\,\mu_{22}^3\right)\left(d_{12}\,r_{12} + \mu_{12}^5\,\mu_{22}^5\right)}, \\
d_8 = -\dfrac{d_{12}\,r_{12}\,\bar{\alpha}_{12}\,\mu_{22}^4\,\rho_{12} + d_{12}\,r_{12}\,\bar{\alpha}_{12}\,\mu_{12}^5\,\rho_{22} + d_{12}\,r_{12}\,\bar{\alpha}_{22}\,\mu_{12}^3\,\rho_{12} - \bar{\alpha}_{22}\,\mu_{12}^3\,\mu_{12}^4\,\mu_{12}^5\,\rho_{22}}{\left(d_{12}\,r_{12} + \mu_{12}^4\,\mu_{22}^4\right)\left(d_{12}\,r_{12} + \mu_{12}^3\,\mu_{22}^3\right)\left(d_{12}\,r_{12} + \mu_{12}^5\,\mu_{22}^5\right)}, \\
e_1 = -\dfrac{d_{21}\,r_{21}\,\rho_{21} - \mu_{21}^4\,\mu_{21}^5\,\rho_{11}}{\left(d_{21}\,r_{21} + \mu_{11}^4\,\mu_{21}^4\right)\left(d_{21}\,r_{21} + \mu_{11}^5\,\mu_{21}^5\right)}, \quad e_2 = \dfrac{r_{21}\,\left(\mu_{11}^4\,\rho_{21} + \mu_{21}^5\,\rho_{11}\right)}{\left(d_{21}\,r_{21} + \mu_{11}^4\,\mu_{21}^4\right)\left(d_{21}\,r_{21} + \mu_{11}^5\,\mu_{21}^5\right)}, \\
e_3 = -\dfrac{d_{12}\,r_{12}\,\rho_{22} - \mu_{22}^4\,\mu_{22}^5\,\rho_{12}}{\left(d_{12}\,r_{12} + \mu_{12}^4\,\mu_{22}^4\right)\left(d_{12}\,r_{12} + \mu_{12}^5\,\mu_{22}^5\right)}, \quad e_4 = -\dfrac{d_{12}\,\left(\mu_{12}^4\,\rho_{22} + \mu_{22}^5\,\rho_{12}\right)}{\left(d_{12}\,r_{12} + \mu_{12}^4\,\mu_{22}^4\right)\left(d_{12}\,r_{12} + \mu_{12}^5\,\mu_{22}^5\right)}, \\
e_5 = -\dfrac{d_{21}\,\left(\mu_{21}^4\,\rho_{11} + \mu_{11}^5\,\rho_{21}\right)}{\left(d_{21}\,r_{21} + \mu_{11}^4\,\mu_{21}^4\right)\left(d_{21}\,r_{21} + \mu_{11}^5\,\mu_{21}^5\right)}, \quad e_6 = -\dfrac{d_{21}\,r_{21}\,\rho_{11} - \mu_{11}^4\,\mu_{11}^5\,\rho_{21}}{\left(d_{21}\,r_{21} + \mu_{11}^4\,\mu_{21}^4\right)\left(d_{21}\,r_{21} + \mu_{11}^5\,\mu_{21}^5\right)}, \\
e_7 = \dfrac{r_{12}\,\left(\mu_{22}^4\,\rho_{12} + \mu_{12}^5\,\rho_{22}\right)}{\left(d_{12}\,r_{12} + \mu_{12}^4\,\mu_{22}^4\right)\left(d_{12}\,r_{12} + \mu_{12}^5\,\mu_{22}^5\right)}, \quad e_8 = -\dfrac{d_{12}\,r_{12}\,\rho_{12} - \mu_{12}^4\,\mu_{12}^5\,\rho_{22}}{\left(d_{12}\,r_{12} + \mu_{12}^4\,\mu_{22}^4\right)\left(d_{12}\,r_{12} + \mu_{12}^5\,\mu_{22}^5\right)}, \\
g_1 = \dfrac{\mu_{21}^5}{d_{21}\,r_{21} + \mu_{11}^5\,\mu_{21}^5}, \quad g_2 = \dfrac{r_{21}}{d_{21}\,r_{21} + \mu_{11}^5\,\mu_{21}^5}, \quad g_3 = \dfrac{\mu_{22}^5}{d_{12}\,r_{12} + \mu_{12}^5\,\mu_{22}^5}, \\
g_4 = -\dfrac{d_{12}}{d_{12}\,r_{12} + \mu_{12}^5\,\mu_{22}^5}, \quad g_5 = -\dfrac{d_{21}}{d_{21}\,r_{21} + \mu_{11}^5\,\mu_{21}^5}, \quad g_6 = \dfrac{\mu_{11}^5}{d_{21}\,r_{21} + \mu_{11}^5\,\mu_{21}^5}, \\
g_7 = \dfrac{r_{12}}{d_{12}\,r_{12} + \mu_{12}^5\,\mu_{22}^5}, \quad g_8 = \dfrac{\mu_{12}^5}{d_{12}\,r_{12} + \mu_{12}^5\,\mu_{22}^5}, \\
\bar{\alpha}_{11} = \alpha_{11}^c\,\rho_{11}^c, \quad \bar{\alpha}_{12} = \alpha_{12}^c\,\rho_{12}^c, \quad \bar{\alpha}_{21} = \alpha_{21}^c\,\rho_{21}^c, \quad \bar{\alpha}_{22} = \alpha_{22}^c\,\rho_{22}^c, \\
\rho_{11} = \rho_{11}^{ct}, \quad \rho_{12} = \rho_{12}^{ct}, \quad \rho_{21} = \rho_{21}^{ct}, \quad \rho_{22} = \rho_{22}^{ct}, \\
\tilde{\rho}_{11}^{ht} = \alpha_{11}^{ht}\,\rho_{11}^{ht}, \quad \tilde{\rho}_{12}^{ht} = \alpha_{12}^{ht}\,\rho_{12}^{ht}, \quad \tilde{\rho}_{21}^{ht} = \alpha_{21}^{ht}\,\rho_{21}^{ht}, \quad \tilde{\rho}_{22}^{ht} = \alpha_{22}^{ht}\,\rho_{22}^{ht}, \\
\psi_1 = a_1\,q_1 + b_1\,p_1 + d_1\,\omega_1, \quad \psi_2 = a_2\,q_1 + b_2\,p_1 + d_2\,\omega_1, \quad \psi_3 = a_3\,q_2 + b_3\,p_2 + d_3\,\omega_2, \\
\psi_4 = a_4\,q_2 + b_4\,p_2 + d_4\,\omega_2, \quad \psi_5 = a_5\,q_3 + b_5\,p_3 + d_5\,\omega_3, \quad \psi_6 = a_6\,q_3 + b_6\,p_3 + d_6\,\omega_3, \\
\psi_7 = a_7\,q_4 + b_7\,p_4 + d_7\,\omega_4, \quad \psi_8 = a_8\,q_4 + b_8\,p_4 + d_8\,\omega_4, \\
\psi_{11} = a_1\,q_{11} + b_1\,p_1 + d_{11}\,\omega_1, \quad \psi_{21} = a_2\,q_{11} + b_2\,p_{11} + d_2\,\omega_{11}, \quad \psi_{31} = a_3\,q_{21} + b_3\,p_{21} + d_3\,\omega_{21}, \\
\psi_{41} = a_4\,q_{21} + b_4\,p_{21} + d_4\,\omega_{21}, \quad \psi_{51} = a_5\,q_{31} + b_5\,p_{31} + d_5\,\omega_{31}, \quad \psi_{61} = a_6\,q_{31} + b_6\,p_{31} + d_6\,\omega_{31}, \\
\psi_{71} = a_7\,q_{41} + b_7\,p_{41} + d_7\,\omega_{41}, \quad \psi_{81} = a_8\,q_{41} + b_8\,p_{41} + d_8\,\omega_{41}, \\
\varepsilon_1 = c_1\,p_1 + e_1\,\omega_1, \quad \varepsilon_2 = c_2\,p_1 + e_2\,\omega_1, \quad \varepsilon_3 = c_3\,p_2 + e_3\,\omega_2, \\
\varepsilon_4 = c_4\,p_2 + e_4\,\omega_2, \quad \varepsilon_5 = c_5\,p_3 + e_5\,\omega_3, \quad \varepsilon_6 = c_6\,p_3 + e_6\,\omega_3, \\
\varepsilon_7 = c_7\,p_4 + e_7\,\omega_4, \quad \varepsilon_8 = c_8\,p_4 + e_8\,\omega_4, \\
\varepsilon_{11} = c_1\,p_{11} + e_1\,\omega_{11}, \quad \varepsilon_{21} = c_2\,p_{11} + e_2\,\omega_{11}, \quad \varepsilon_{31} = c_3\,p_{21} + e_3\,\omega_{21}, \\
\varepsilon_{41} = c_4\,p_{21} + e_4\,\omega_{21}, \quad \varepsilon_{51} = c_5\,p_{31} + e_5\,\omega_{31}, \quad \varepsilon_{61} = c_6\,p_{31} + e_6\,\omega_{31}, \\
\varepsilon_{71} = c_7\,p_{41} + e_7\,\omega_{41}, \quad \varepsilon_{81} = c_8\,p_{41} + e_8\,\omega_{41},
\end{cases}
\tag{A.30}
$$

A.5 Expressions for $\mathcal{R}_c^1$, $\mathcal{R}_c^2$, ..., $\mathcal{R}_c^{12}$

$$
\begin{cases}
\mathcal{R}_c^1 = \dfrac{1}{2}\left(\psi_3 + \psi_8\right) + \dfrac{1}{2}\sqrt{(\psi_3 + \psi_8)^2 + 4\,\psi_4\,\psi_7}, \\
\mathcal{R}_c^2 = \dfrac{1}{2}\left(\psi_1 + \psi_6\right) + \dfrac{1}{2}\sqrt{\left(\psi_1 + \psi_6\right)^2 + 4\,\psi_2\,\psi_5}, \\
\mathcal{R}_c^3 = \dfrac{1}{2}\left(\psi_{31} + \psi_{81}\right) + \dfrac{1}{2}\sqrt{\left(\psi_{31} + \psi_{81}\right)^2 + 4\,\psi_{41}\,\psi_{71}}, \\
\mathcal{R}_c^4 = \dfrac{1}{2}\left(\psi_{11} + \psi_{61}\right) + \dfrac{1}{2}\sqrt{\left(\psi_{11} + \psi_{61}\right)^2 + 4\,\psi_{21}\,\psi_{51}}, \\
\mathcal{R}_c^5 = \dfrac{1}{2}\left(\varepsilon_3 + \varepsilon_8\right) + \dfrac{1}{2}\sqrt{\left(\varepsilon_3 + \varepsilon_8\right)^2 + 4\,\varepsilon_4\,\varepsilon_7}, \\
\mathcal{R}_c^6 = \dfrac{1}{2}\left(\varepsilon_1 + \varepsilon_6\right) + \dfrac{1}{2}\sqrt{\left(\varepsilon_1 + \varepsilon_6\right)^2 + 4\,\varepsilon_2\,\varepsilon_5}, \\
\mathcal{R}_c^7 = \dfrac{1}{2}\left(\varepsilon_{31} + \varepsilon_{81}\right) + \dfrac{1}{2}\sqrt{\left(\varepsilon_{31} + \varepsilon_{81}\right)^2 + 4\,\varepsilon_{41}\,\varepsilon_{71}}, \\
\mathcal{R}_c^8 = \dfrac{1}{2}\left(\varepsilon_{11} + \varepsilon_{61}\right) + \dfrac{1}{2}\sqrt{\left(\varepsilon_{11} + \varepsilon_{61}\right)^2 + 4\,\varepsilon_{21}\,\varepsilon_{51}}, \\
\mathcal{R}_c^9 = \dfrac{1}{2}\left(\omega_2\,g_3 + \omega_4\,g_8\right) + \dfrac{1}{2}\sqrt{\left(\omega_2\,g_3 + \omega_4\,g_8\right)^2 + 4\,g_4\,g_7\,\omega_2\,\omega_4}, \\
\mathcal{R}_c^{10} = \dfrac{1}{2}\left(\omega_1\,g_1 + \omega_3\,g_6\right) + \dfrac{1}{2}\sqrt{\left(\omega_1\,g_1 + \omega_3\,g_6\right)^2 + 4\,g_2\,g_5\,\omega_1\,\omega_3}, \\
\mathcal{R}_c^{11} = \dfrac{1}{2}\left(\omega_{21}\,g_3 + \omega_{41}\,g_8\right) + \dfrac{1}{2}\sqrt{\left(\omega_{21}\,g_3 + \omega_{41}\,g_8\right)^2 + 4\,g_4\,g_7\,\omega_{21}\,\omega_{41}}, \\
\mathcal{R}_c^{12} = \dfrac{1}{2}\left(\omega_{11}\,g_1 + \omega_{31}\,g_6\right) + \dfrac{1}{2}\sqrt{\left(\omega_{11}\,g_1 + \omega_{31}\,g_6\right)^2 + 4\,g_2\,g_5\,\omega_{11}\,\omega_{31}}.
\end{cases}
\tag{A.31}
$$

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