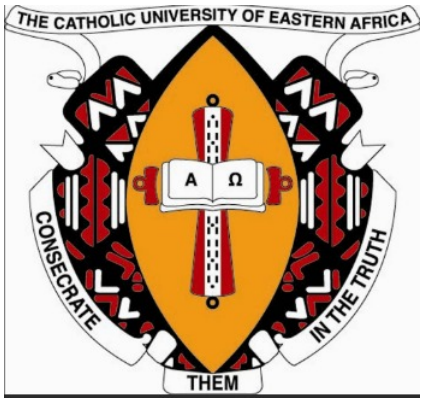


CATHOLIC UNIVERSITY OF EASTERN AFRICA

Mathematical Modelling of HIV/AIDS Dynamics in Relation to Drug Abuse

BY

DOLPHINE MOCHERE

A Thesis Submitted in Partial Fulfillment of the Requirements for the Award of Master of Science (MSc) in Applied Mathematics of the Catholic University of Eastern Africa.

DEPARTMENT OF MATHEMATICS AND ACTUARIAL SCIENCE

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Declaration

I hereby declare that this thesis is my original work and has not been presented in any other university for consideration. This thesis is specifically credited, and references are cited accordingly.

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Dedication

I dedicate this thesis to my parents, who have always encouraged me and believed in me. Their unwavering support throughout my academic journey has allowed me to pursue my dreams and reach this milestone. I would also like to thank other family members, including my brother and sisters, for their patience, for always being there to listen and provide advice, and for their unconditional encouragement and guidance throughout my research journey. Finally, I am deeply grateful to my supervisors, who have guided me with their expertise and wisdom. Their commitment, trust, and support have been instrumental in the success of this research thesis. This work is a testament to all who have been a part of my journey, and I sincerely thank them for their assistance and kindness.

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Abstract

This study has investigated the co-dynamics of HIV/AIDS and drug abuse, focusing on how protection strategies and rehabilitation programmes influence transmission. The problem addressed is the strong interaction between drug abuse and HIV spread, particularly in Kenya, where injection drug use and risky behaviours heighten vulnerability to infection. The primary objective of the study was to create and evaluate a mathematical model that integrates protection and rehabilitation as control strategies. Specifically, the study aimed to investigate the stability of disease-free and endemic equilibria, compute basic and control reproduction numbers, conduct sensitivity analysis, and evaluate the impact of interventions through numerical simulations. A deterministic model with seven compartments, founded on ordinary differential equations, was developed and analyzed. The next-generation matrix method was employed to calculate reproduction numbers, while equilibrium stability was evaluated using the Jacobian method, Routh–Hurwitz criteria, and Lyapunov functions. Sensitivity analysis was performed to determine the most significant parameters, and numerical simulations were executed with Maple software. The findings indicated that the disease-free equilibrium is both locally and globally stable when $R_C < 1$, suggesting that HIV/AIDS and drug misuse can be effectively managed with adequate measures. Simulations confirmed that higher protection and rehabilitation rates significantly reduce the prevalence of drug abusers, HIV-infected, and co-infected individuals. Sensitivity analysis highlighted protection and rehabilitation as the most critical parameters. The study concludes that integrated interventions, combining protection measures with effective rehabilitation, are essential for reducing the dual burden of HIV/AIDS and drug abuse. These findings offer significant insights for policymakers and health practitioners in formulating targeted public health interventions.

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Abbreviation

HIV	Human Immunodeficiency Virus
AIDS	Acquired Immunodeficiency Syndrome
RNA	Ribonucleic acid
WHO	World Health Organization
ARV	Antiretroviral drugs
R_0	Basic Reproduction number
E_0	Endemic Equilibrium point
$CD_4 + T$ cells	Cluster of Differentiation 4 Positive T Lymphocytes
SAHR	Susceptible, light-grade Abuse, Heavy-grade abuse, Treatment, Recover

Chapter 1

1 Introduction

1.1 Background of the Study

HIV, or Human Immunodeficiency Virus, is a type of retrovirus characterized by ribonucleic acid (RNA). It is the causative agent of AIDS, which stands for Acquired Immunodeficiency Syndrome [4]. The inaugural reported case of HIV/AIDS was identified in 1981, and as of 2023, more than 42.3 million individuals globally are afflicted with HIV/AIDS, and the condition continues to pose a significant global health challenge. [27]. The World Health Organisation (WHO) reported in 2024 that approximately 630,000 deaths were attributed to HIV/AIDS in 2023 [20], [25].

Kenya is among the most severely affected nations, ranking among the top 10 globally and third in Africa in terms of HIV burden[20]. Despite widespread awareness of HIV/AIDS in Kenya, many individuals do not seek testing [15]. HIV weakens the immune system, making the body more susceptible to infections. While there is no cure, an individual with the infection can survive for about 9-11 years with the infection since there is no possible way to eradicate the Antiretroviral drugs (ARVs) have been developed to suppress the virus, decelerate illness progression, and improve daily functioning for people living with the infection. These medications reduce viral replication, allowing HIV/AIDS to be managed as a chronic condition rather than an acute illness. HIV/AIDS leads to other health complications that damage body parts such as the heart, kidney, and brain [18].

The main way that HIV is spread is by contact with contaminated blood, which can happen through transfusions, contaminated needles, or blood products. It can also be transmitted from mother to child during delivery and via unprotected sexual contact with an infected partner. Additionally, sharing drug needles and syringes is a known transmission route [11]. Africa remains the most heavily affected region, with Kenya's adult HIV prevalence estimated at 4.5% in 2021.

Drug abuse encompasses the hazardous use of consciousness-altering chemicals, both legal, like alcohol, and illegal substances, that produce dependence and harmful results[30]. Society is adversely affected because drug addiction causes serious health problems in many individuals. Drug addiction is extremely costly to the individual, the family, and society as a whole through both direct costs and indirect costs, such as loss of productivity and social problems. Globally, as reported by the WHO, the number of drug users increased from 240 million people in 2011 to 296 million in 2021, with population growth being a partial contributing factor [12]. Drug abuse is highly correlated with poverty, political instability, social unrest, and refugee challenges, which are prevalent in developing countries, particularly in Africa and Asia. Most formal control measures put in place to address substance abuse do not involve the community and are, therefore, ineffective in curbing the problem. The effectiveness of control measures is hindered by barriers such as insufficient funding, political unwillingness, lack of efficient laboratory facilities, and inadequate treatment facilities. These challenges have allowed the drug abuse crisis to

persist in the population [19]. Examples of the drugs highly abused are cocaine, amphetamines, ecstasy, cannabis, Opioids, and some un-prescribed drugs.

Drug and substance abuse in Kenya places a social, economic, and health burden on society. Drug abuse in Kenya has become a pervasive social issue affecting people across all age groups, from adolescents to adults who mainly abuse alcohol, cigarettes, marijuana (bhang) smoking, khat chewing, kuber, shisha, tobacco, and cocaine consumption [13][24]. The policies placed to help curb drug abuse are not effective due to shortcomings such as a lack of community involvement. The stigmatization of individuals in recovery from drug abuse often causes them to relapse [16].

Protection consists of a system of measures, strategies, and interventions globally to prevent the likelihood of exposure, transmission, and other adverse outcomes related to a public health threat. Various strategies of prevention have been put in place to help fight against the spread of HIV/AIDS [29]. These include antiretroviral drugs to reduce mother-to-child transmission at birth, extensive sex education, mass sensitization on HIV/AIDS, and regular HIV testing to enable early detection and counseling for the affected individuals. Similarly, programs geared towards preventing drug abuse stress more on drug-use and drug-dealing laws; enlightening the public, youth especially, of the hazardous results of drug abuse, besides post-rehabilitation programs towards readmitting an individual to regular life and ways to reduce recidivism[8].

Substance and drug abuse often intersect with HIV/AIDS because substance use often promotes dangerous behaviours that substantially elevate HIV infection risk, such as multiple sex partners, injection of drug use, and inconsistent condom use. Protection and rehabilitation curb the spread of HIV/AIDS since rehabilitation tends to take control of the drug abusers' behaviors [21]. Rehabilitation centers play a vital role in addressing substance abuse and reducing the burden of HIV/AIDS among people who use drugs by providing comprehensive care and integrating HIV prevention, testing, and treatment services [22].

The dynamics between HIV transmission and substance abuse require analysis together with the role of protecting and rehabilitating institutions for the development of effective prevention strategies in countering this global health problem.

One fundamental method for resolving issues in the actual world is mathematical modeling, that utilizes mathematical structures as tools to model real-world problems. It gives a structured statistical expression of reality through dynamical system models or differential equations[7], [17]. Mathematical modeling, particularly via differential equations, has become an essential instrument in the analysis of infectious illnesses, including the dynamics of HIV/AIDS. The mathematical modeling of diseases through expressions, principles, and formulas allows for informed decisions on intervention strategies and enhances the understanding of disease transmission patterns [1].

Extensive research on effective control measures for HIV/AIDS and drug abuse has resulted in a variety of approaches aimed at containing the epidemic. Models formulated by mathematicians can help to control and contain the spread of HIV/AIDS infection and drug use behaviors.

This study primarily focuses on protection and rehabilitation as the main interventions to control the transmission of HIV/AIDS through the reduction of drug abuse. In this respect, using an SIR-like model, the target population will be categorized into different subgroups to model dynamics and consequences at various stages of disease progression for both HIV/AIDS and drug abuse. In addition, the proposed framework enhances insight into disease dynamics and effectiveness in reducing risks using rehabilitation.

1.2 Problem Statement

The complex interaction between HIV/AIDS transmission and drug abuse in Kenya represents a significant public health challenge that necessitates innovative and data-driven approaches for effective management.Although prior studies have examined the correlation between drug abuse and HIV/AIDS transmission, a need persists in research on the development of a thorough mathematical model that clearly incorporates the role of protection and rehabilitation centers in mitigating HIV/AIDS transmission among drug-dependent populations.

Rehabilitation centers serve a pivotal function in supporting individuals through addiction recovery and promoting safer behaviors, thereby reducing the likelihood of HIV/AIDS transmission. However, the extent to which protection and rehabilitation centers contribute to HIV/AIDS prevention remains insufficiently examined, underscoring the necessity for further research in this domain.

1.3 Objectives of the Study

1.3.1 General Objectives

To develop and analyse a mathematical model for HIV/AIDS and drug abuse, with rehabilitation and protection as a control measure.

1.3.2 Specific Objectives

1. To develop a mathematical model representing HIV/AIDS transmission related to drug abuse, including rehabilitation centers.
2. To determine the stability analysis of disease-free and endemic equilibrium.
3. To carry out the sensitivity analysis of the model.
4. To carry out numerical analysis of protection and rehabilitation centers in reducing the spread of HIV/AIDS

1.4 Significance of the Study

This study explores how different drug use behaviors, protection strategies, and rehabilitation efforts influence the spread of HIV/AIDS in Kenya. By incorporating elements such as behavioral interventions, education, and preventive measures into the mathematical model, the research demonstrates the importance of these strategies in reducing vulnerability to both drug abuse and HIV infection.

The findings add value to the effectiveness of HIV/AIDS control interventions in Kenya by providing a clear explanation of how drug abuse, protection strategies, and rehabilitation efforts interact to influence HIV/AIDS transmission. This study, therefore, helps policymakers and clinicians design appropriate interventions that address specific needs within the population.
The findings of this study significantly elucidate the dynamics related to the trend in HIV transmission. They consequently inform appropriate decisions on resource allocation and intervention strategies. With a mathematical model of drug abuse, protection, and rehabilitation effects on HIV/AIDS transmission dynamics, the critical gap that has hitherto existed in this area of literature now comes under active focus in the present study.

The research thus furthers the cause of developing an advanced, holistic, and exact model of analysis for transmission dynamics of HIV/AIDS, toward the effective enabling of targeted and integrated public health strategies.

1.5 Scope of the study

This study develops and analyses a deterministic, compartmental model to investigate the co-dynamics of HIV/AIDS and drug abuse and the impact of protection strategies and rehabilitation programmes on transmission. The model partitions the population into seven epidemiological classes: susceptible (S), protected (P), HIV-infected (I_H), drug abusers (I_D), dual-infected (I_{HD}), on HIV treatment (T_H), and in rehabilitation (R_D). The analysis focuses on deriving threshold quantities, stability properties, sensitivity indices, and illustrative simulations to understand how protection and rehabilitation influence both epidemics. The work is framed in the context of Kenya's HIV/drug-use burden and is intended for population-level insights relevant to Kenyan public health planning. It does not target a particular county or facility; rather, it considers a homogeneous, well-mixed population with emphasis on drug-using sub-populations that elevate HIV risk.

1.6 Organization of the Study

This work is presented in five chapters. Chapter one introduces the work with a critical review of HIV/AIDS, drug abuse, rehabilitation, and mathematical modelling, stating the problem statement, research objectives, and the scope of the study. Chapter two gives the literature review, summarises previous works on HIV/AIDS and substance abuse, states the research gap, and identifies what this study will seek to achieve.

Chapter three, methodology, lists the mathematical methods adopted to achieve the objectives developed in Chapter one. Chapter four, Results and Analysis, discusses the application of the methodologies that were introduced in chapter three, states the derived results, and explains what they mean.

The final chapter contains a Discussion, Conclusion, and Recommendations, where the graphical representation of findings is analyzed and their interpretation is also provided. Moreover, from the results of the study, recommendations for further research are suggested.

Chapter 2

2 Literature Review

2.1 Introduction

The literature review examines mathematical modeling approaches for HIV/AIDS transmission, drug abuse dynamics, and their intersection. Its organized into three key areas: HIV/AIDS models, focusing on population-level dynamics, within-host mechanisms, and treatment interventions; drug abuse models, examining rehabilitation strategies and control interventions; and the limited research combining both epidemics. This review reveals a critical gap: while extensive models exist for each area separately, few studies have developed integrated frameworks examining how drug abuse influences HIV/AIDS transmission dynamics and protection and rehabilitation effectiveness.

2.2 HIV/AIDS

Cassels' research examined the capacity of mathematical models to forecast population-level HIV/AIDS outcomes based on individual-level behavioral and biological inputs. Their comprehensive review examined various modelling approaches, including deterministic compartmental models, stochastic models, and network models, to understand HIV/AIDS transmission dynamics. The study emphasised how individual behaviours aggregate to create population-level disparities in HIV/AIDS prevalence and incidence. TThey illustrated that mathematical models are essential instruments for comprehending the counterintuitive impacts of behavioral interventions and for forecasting epidemic trajectories. The models reviewed incorporated factors such as sexual mixing patterns, transmission probabilities, and disease progression rates. Their findings showed that models help explain HIV/AIDS disparities across different populations and can evaluate potential intervention strategies. However, the study focused exclusively on sexual transmission networks and biological factors without considering the significant role of drug abuse in HIV/AIDS transmission and the behavioural disinhibition associated with substance abuse.[6]

Mwangi, in his research in 2018, developed a three-compartment mathematical model incorporating uninfected $CD4+T$ cells, infected $CD4+T$ cells, and free virus particles, with special consideration for weak $CD4+T$ cells that transition directly to the virus class. Using ordinary differential equations, they analyzed the model's equilibrium points and stability properties through the Routh-Hurwitz criterion and Lyapunov's method. The model exhibited two stable states: a disease-free equilibrium and an infected equilibrium state. Their study indicated that the fundamental reproduction number governs system dynamics, with the infection-free equilibrium being globally asymptotically stable when $R_0 < 1$. The research emphasized the significance of the spontaneous regeneration of $CD4+T$ cells in HIV dynamics. The model successfully explained the puzzling features of HIV infection, including the slow decline of $CD4+T$ cells. However, the research maintained a purely biological focus, examining only within-host dynamics without incorporating behavioral factors such as drug abuse that significantly influence HIV transmission patterns at the population level.[9]

In 2022, Espitia constructed a mathematical framework to study HIV/AIDS spread by incorporating sexual orientation preferences, establishing a triangular transmission pattern among heterosexual women, heterosexual men, and homosexual men. The model incorporated the effects of antiretroviral medication and utilized a set of eight-compartment nonlinear differential equations. The study entailed calculating the basic reproduction number using next-generation matrix methods and doing stability analyses of equilibrium states. The study found that the homosexual population has higher transmission rates ($\beta = 0.7$) compared to heterosexual transmission ($\beta = 0.3$), reducing homosexual partner rates crucial for reaching disease-free equilibrium. Local and global stability analyses confirmed that $R_0 < 1$ ensures disease elimination. Sensitivity analysis revealed that the departure rate for infected individuals was the most influential parameter. The model successfully captured sexual transmission dynamics, and the ART impact does not incorporate the impact of drug use in the HIV/AIDS transmission, missing the significant role of drug usage in HIV spread, particularly in populations where both sexual and injection risks overlap. [10]

Research in 2012 by Shiri constructed a nonlinear deterministic model examining HIV/AIDS dynamics, incorporating both antiretroviral treatment and vertical transmission from mother to child. Their model categorized the population into susceptible, infected, AIDS patients, and treated individuals, employing a system of ordinary differential equations. The research established the local stability of the disease-free equilibrium through the next-generation approach and illustrated that $R_0 < 1$ ensures disease elimination. Their numerical simulations revealed that antiretroviral therapy significantly reduces viral load and AIV/AIDS-related deaths, while controlling vertical transmission rates prevents new pediatric infections. The model showed forward bifurcation at $R_0 = 1$, indicating predictable disease dynamics. Sensitivity analysis identified treatment coverage and vertical transmission rates as critical parameters. The findings emphasized that combining ARV treatment with prevention of mother-to-child transmission creates synergistic effects in controlling HIV/AIDS. Despite comprehensive coverage of treatment dynamics, the model does not incorporate drug abuse as a major transmission route, failing to account for injection drug use or how substance abuse affects treatment adherence and disease progression.[28]

2.3 Drug Abuse

In 2023, Alfiniyah developed a comprehensive six-compartment mathematical model examining drug abuse dynamics with consideration for both indoor and outdoor treatment modalities. Their model divided the population into susceptible, light drug users, heavy drug users, indoor treatment, outdoor treatment, and recovered individuals, using a system of ordinary differential equations. The study incorporated anti-drug campaign effectiveness as a key parameter affecting transition rates between compartments. Through stability analysis and numerical simulations, they demonstrated that anti-drug campaigns significantly reduce relapse rates among individuals in rehabilitation, with campaign effectiveness parameter θ showing a strong negative correlation with relapse rates. The basic reproduction number R_0 calculated using the next-generation matrix approach indicates that $R_0 < 1$ ensures drug-free equilibrium stability. Their findings emphasized that combining treatment with continuous awareness campaigns creates synergistic effects in controlling drug epidemics. Nevertheless, despite addressing drug abuse comprehen-

sively, the model completely overlooked the critical intersection with HIV/AIDS, failing to account for increased HIV/AIDS transmission risks among drug users or how HIV/AIDS status might affect treatment outcomes.[2]

Muin constructed a five-compartment SAHR model (Susceptible, light-grade Abuse, Heavy-grade abuse, Treatment, Recovery) in his research with two optimal control interventions: anti-drug campaigns and psychological counseling. Their approach utilized Pontryagin's Minimum Principle to derive optimal control strategies minimizing drug abuse prevalence while considering intervention costs. The model incorporated progression from light to heavy drug use, treatment effectiveness, and relapse dynamics through eight coupled differential equations with time-varying controls. Numerical solutions employed the forward-backward sweep method, revealing that front-loaded interventions (high initial effort tapering over time) proved most cost-effective. The study found that combining both controls synergistically reduced the drug-using population more effectively than single interventions. Sensitivity analysis showed campaign effectiveness and counseling impact as critical parameters. Simulations demonstrated that without sustained intervention, drug abuse resurges even from low endemic levels. The optimal control formulation provided practical insights for resource allocation in drug abuse prevention. However, the model maintained exclusive focus on drug abuse dynamics without considering the substantial HIV/AIDS transmission risks associated with drug use, particularly injection drug use.[3]

2.4 Drug Abuse and HIV/AIDS

In 2020, Lokaran developed a deterministic mathematical model to evaluate the impact of drug and substance misuse on the transmission of HIV/AIDS among adults in Turkana County, Kenya. This deterministic model divided the population into six categories: healthy susceptible people, those with HIV/AIDS alone, drug users without HIV, individuals with both conditions, HIV-positive people receiving medical care, drug users in treatment programs, and those being treated for both conditions simultaneously. The study determined the fundamental reproduction number and analyzed the stability properties of both disease-free and endemic conditions. When the reproduction number decreased below one, the disease-free state exhibited global asymptotic stability. The work shown through Lyapunov analysis that the endemic equilibrium attained global asymptotic stability when the reproduction number surpassed one. Results indicated that drug misuse could amplify HIV/AIDS spread by affecting the basic reproduction number. Computer simulations investigated how different factors influenced the combined dynamics of drug abuse and HIV transmission in the region. Interestingly, the findings revealed an inverse relationship where rising HIV infections corresponded with declining drug abuse prevalence, suggesting drug use may have a reduced impact on HIV spread over the coming five years in Turkana County. However, Lokaran's research had limitations, as it failed to account for protective interventions like public awareness programs, educational initiatives, and harm reduction approaches that could mitigate both HIV transmission and substance abuse. This omission underscores the necessity for more comprehensive modeling that integrates rehabilitation services and preventive measures into the analysis of HIV transmission dynamics within drug-using communities.[14]

Bloomquist and Vaidya's research in 2021 created a mathematical model to assess

HIV/AIDS infection probability among substance users, with particular emphasis on how morphine affects HIV/AIDS transmission patterns. Their research examined the intricate biological changes induced by substances like morphine and how these modifications influence HIV/AIDS infection susceptibility and the potential for spread. The researchers employed a compartmental approach featuring two $CD4 + T$ cells: low susceptibility and high susceptibility cells. Their model additionally incorporated infected cellular components and circulating viral particles. The investigation revealed that morphine significantly modifies co-receptor presentation on target cells, resulting in a dramatically increased percentage of highly vulnerable ($CD4+T$ cells) cells in individuals with chronic morphine exposure. The fraction of highly susceptible cells rose substantially from 39% in morphine-naive individuals to 98% following extended morphine conditioning, demonstrating significantly elevated infection risk among substance users.[5]

Bloomquist and Vaidya developed a model to calculate infection probability for drug-free individuals following a single exposure to an HIV-positive partner who uses drugs. Their research demonstrated that chronic substance use conditioning significantly elevates transmission risk, showing approximately 2.5 times greater infection likelihood per sexual encounter(0.83% without drugs of abuse vs. 2.09% under drugs of abuse). The investigation revealed that infection risk among drug users correlates with the time interval between morphine consumption and viral exposure, where extended periods between drug use and exposure resulted in heightened infection vulnerability. Additionally, the researchers examined how prophylactic treatments (pre-exposure and post-exposure prophylaxis) could reduce transmission risk. Their model indicated that treatment timing plays a crucial role in both scenarios, whether or not substances are involved. The research also demonstrated that therapeutic effectiveness varies based on the treatment's potency and the critical time frame available for initiating intervention following viral exposure. While Bloomquist and Vaidya provide valuable insights into the impact of drug abuse on HIV/AIDS transmission dynamics, their model does not explicitly incorporate the role of rehabilitation centers in reducing HIV/AIDS transmission among drug-abusing populations. This literature void demonstrates the critical need for an all-encompassing mathematical model that considers the impact of protection and rehabilitation centers on HIV/AIDS transmission dynamics among drug abusers, which is the focus of this research proposal.[5]

The literature review reveals a significant gap: while numerous mathematical models exist for HIV/AIDS transmission and drug abuse separately, and some consider rehabilitation for drug abuse control, none have developed an integrated mathematical framework that examines how protection and rehabilitation programs for drug abuse specifically impact HIV/AIDS transmission dynamics. This gap justifies the research, which uniquely combines all three elements in a unified model.

Chapter 3

3 Model Formulation and Analysis

3.1 Model Formulation

In this chapter, we create a mathematical model that represents the dynamics of a population impacted by drug Abuse, HIV/AIDS, or both. The model's behavior will be examined through parameter estimation, and the effects of each parameter on the dynamics of the system will be examined through numerical simulations.

The population is divided into seven compartments. These compartments are Susceptible (S), Protected (P), Infected with HIV/AIDS(I_H), Drug Abuse population (I_D), dually infected with HIV/AIDS and also drug abusers (I_{HD}), Individuals under HIV/AIDS treatment (T_H) and finally individuals under rehabilitation for Drug abuse (R_D). The model applies some assumptions that are included in this chapter. Taking the population at time t, the total population is given by the expression.

$$N(t) = S + P + I_H + I_D + I_{HD} + T_D + R_D$$

The recruitment for the susceptible is at a rate π as a result of both birth and immigration. Those under treatment for HIV/AIDS (T_H) are assumed not to transmit the disease due to low viral load, while those under Rehabilitation for drug abuse can relapse at a rate α_2; those who recover from drug abuse do not leave the population but become susceptible at a rate σ_2. The susceptible are assumed to acquire drug abuse at the age of 15 due to peer influence at a rate β_2 while β_1 is the rate at which susceptibles become infected with HIV/ AIDS either by birth(children) or by sexual interaction with infected individuals. There are control measures that are put in place, such as sensitisation and educational campaigns, among others. The rate of susceptibility being protected is δ, and the ineffectiveness of the measures leads to individuals becoming infected with HIV/AIDS or Drug users at ω_1 and ω_2 respectively. The force of infection of HIV/AIDS is

$$\beta_1 = \frac{\Lambda_1(I_H + \lambda_1 I_{HD})}{N}$$

where Λ_1 is the rate of contact of HIV/AIDS and λ_1 accounts for the infectiousness of the infected individuals in I_{HD} compartment.The individuals in I_{HD} have a higher viral load than those in I_H, hence higher infectious capabilities.
The drug abuse increases at a rate

$$\beta_2 = \frac{\Lambda_2(I_D + (1-\lambda_1)I_{HD} + \lambda_2 R_D)}{N}$$

where Λ_2 is the influence of I_D, I_{HD} and R_D on the S while $1-\lambda_1$ and λ_2 are the parameters of modification I_{HD} and R_D which leads to increase in $I_D$and I_{HD}

The natural death occurs across all the compartments at a rate μ. There is induced death due to HIV/AIDS and drug abuse at γ and ρ respectively. Those infected with HIV/AIDS can become drug abusers as a result of stigmatisation, discrimination and depression at a rate of θ_1 while the drug users can get infected with HIV/AIDS as a

result of factors such as reckless immoral acts, sharing of needles among others at a rate θ_2 and therefore move from their respective compartment to I_{HD}. In I_{HD}, death can be a result of natural factors, drug abuse or HIV/AIDS. Individuals infected with HIV/AIDS receive ARV and transition to treatment compartments at τ. Those infected with both HIV/AIDS and are drug abusers can either transition to HIV/AIDS treatment or Rehabilitation (in rehabilitation, there is no treatment for HIV/AIDS) at the rates η_1 and η_2 respectively. the drug users transition to rehabilitation at α_1. Once those who have both infection and transition to rehabilitation are rehabilitated, they can go on with HIV/AIDS treatment at T_H at a rate σ_1.

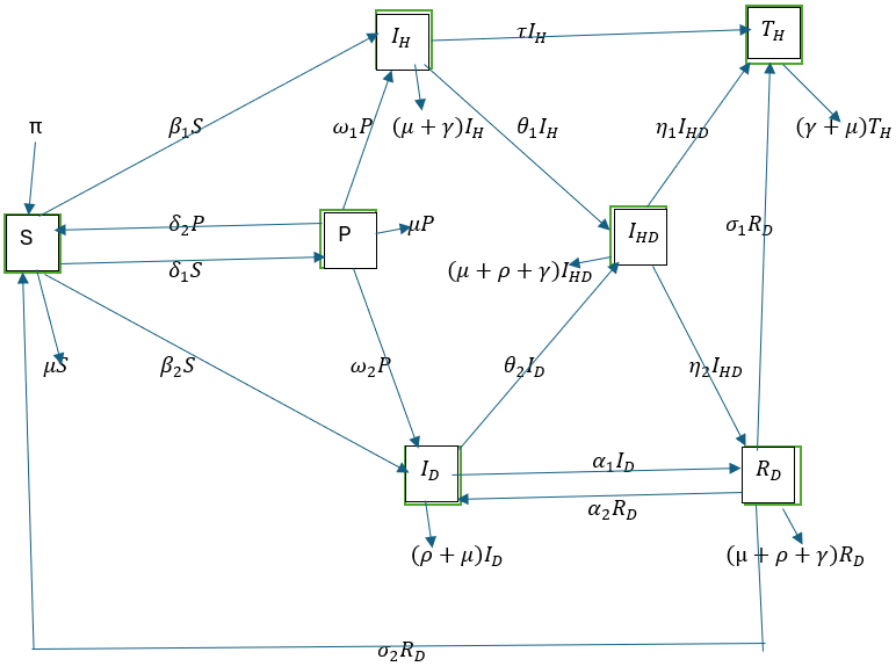


Figure 1: Flow of the model

The components used in the model are described in the table below.

VARIABLE	DESCRIPTION
S	Susceptible population
P	Protected population
I_H	population infected with HIV/AIDS
I_D	Drug Abuse population
I_{HD}	Population infected with HIV/AIDS and uses drugs
T_H	Population under HIV/AIDS treatment
R_D	Population under rehabilitation

Table 1: Description of the model Variables

The model applies the following assumptions

1. The population is homogeneous.
2. All individuals are susceptible to HIV/AIDS, but they become vulnerable to drug abuse from the age of 15 years due to peer influence.
3. There is a possibility of being HIV/AIDS positive, but not a drug abuser or a drug abuser who is not HIV/AIDS positive.
4. Those who undergo rehabilitation as a treatment method for Drug abuse can relapse.
5. Since there is no permanent cure for HIV/AIDS, individuals living with HIV/AIDS may be more susceptible to drug abuse. At the same time, individuals who recover from drug abuse rehabilitation without relapse may become susceptible to HIV/AIDS transmission..
6. Those who are infected by HIV/AIDS and Drug abuse and are in rehabilitation, once they recover from Drug abuse, continue with HIV/AIDS treatment.
7. Death in the model is a result of a natural course that all individuals are subject to; those with drug abuse problems can suffer Drug-attributable death, such as overdose, while those with HIV/AIDS die as a result of it. Those with both can succumb to either natural or HIV/AIDS-induced death or Drug-attributed death.
8. Those under treatment for HIV/AIDS are assumed to have a low viral load and therefore do not spread the HIV/AIDS infection.
9. Those infected with HIV/AIDS and are drug users, but are in denial of drug addiction, do not go for rehabilitation, but rather go for HIV/AIDS treatment only.

The parameters used in the model are described in the table below

PARAMETER	DESCRIPTION
π	Recruitment rate of the population
β_1	Rate of HIV&AIDS infection of the susceptible
β_2	Rate of Drug abuse of the Susceptible
δ_1	Rate of Protection of the susceptible
δ_2	Rate at which the protection is effective
ω_1	Rate of HIV&AIDS Infection of the protected
ω_2	Rate of Drug abuse of the protected
θ_1	Rate of Drug abuse of the Infected with HIV&AIDS
θ_2	Rate of HIV&AIDS infection of the Drug Abuse
τ	Rate of HIV&AIDS to undergo Treatment
η_1	Rate of HIV&AIDS treatment of infected with both HIV&AIDS and Drugs abuse
η_2	Rehabilitation rate of the infected with both HIV&AIDS and Drug abuse
σ_1	Rate of rehabilitated undergoing HIV&AIDS treatment
σ_2	Rate at which the rehabilitated become susceptible
α_1	Rehabilitation Rate of the Drug abusers
α_2	Relapse rate of the rehabilitated
μ	Natural Death
γ	Rate of HIV&AIDS induced death
ρ	Rate of Drug abuse induced death

Table 2: Parameters and Description

3.1.1 Model Equations

The following equations are derived from the model and used to analyse its dynamics.

$$\begin{aligned}
\frac{dS}{dt} &= \pi + \delta_2 P + \sigma_1 R_D - (\delta_1 + \beta_1 + \beta_2 + \mu)S \\
\frac{dP}{dt} &= \delta_1 S - (\delta_2 + \omega_1 + \omega_2 + \mu)P \\
\frac{dI_H}{dt} &= \beta_1 S + \omega_1 P - (\tau + \theta_1 + \mu + \gamma)I_H \\
\frac{dI_D}{dt} &= \beta_2 S + \omega_2 P + \alpha_2 R_D - (\theta_2 + \alpha_1 + \mu + \rho)I_D \\
\frac{dI_{HD}}{dt} &= \theta_2 I_D + \theta_1 I_H - (\eta_1 + \eta_2 + \mu + \rho + \gamma)I_{HD} \\
\frac{dT_H}{dt} &= \eta_1 I_{HD} + \tau I_H + \sigma_1 R_D - (\mu + \gamma)T_H \\
\frac{dR_D}{dt} &= \eta_2 I_{HD} + \alpha_1 I_D - (\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho)R_D
\end{aligned} \tag{1}$$

Chapter 4

4 Results and Analysis

4.1 Introduction

This chapter presents the results obtained from the mathematical model developed in Chapter 3. The aim is to analyze the co-dynamics of HIV/AIDS and drug abuse by establishing key epidemiological thresholds and assessing the effects of protection and rehabilitation strategies. The analysis begins by determining the invariant region and positivity conditions to ensure the model is mathematically and epidemiologically well-posed. Subsequently, the disease-free equilibrium and endemic equilibrium are derived, and their stability properties are examined using local and global stability techniques.

The basic and control reproduction numbers for HIV/AIDS and drug abuse are computed to quantify transmission potential under various intervention scenarios. Sensitivity analysis is then performed to identify the most influential parameters that affect the system's dynamics. Finally, numerical simulations are conducted to illustrate the theoretical results, assess the impact of protection and rehabilitation, and provide insight into the long-term behavior of the epidemics within the population.

Overall, this chapter provides a comprehensive analysis of the model outcomes, linking theoretical results with practical implications for public health interventions.

4.2 Invariant Region and Positivity

4.2.1 Invariant Region

The invariant region shows that the system equations' feasible solutions start and end in the region.[20] It is a subset space where all possible solutions of a dynamical system remain confined. Invariant regions set the boundaries in which the system's behavior is studied ***Theorem***: The model solutions are uniformly bounded for all time $t > 0$ if
$\bar{X} = \Big\{(S(t) + P(t) + I_H(t) + I_D(t) + I_{HD}(t) + T_H(t) + R_D(t) \in R_+^7)$
$: S(0), P(0), I_H(0), I_D(0), I_{HD}(0), T_H(0), R(0)\Big\}.$
$0 \leq N \leq Max\Big(N(0), \frac{\pi}{\mu}\Big)$

Proof : Taking the total population, as at time t as

$$N(t) = S(t) + P(t) + I_H(t) + I_D(t) + I_{HD}(t) + T_H(t) + R_D(t)$$

Using the initial conditions
$S(0) \geq 0, P(0) \geq 0, I_H \geq 0, I_D(0) \geq 0, I_{HD}(0) \geq 0, T_H(0) \geq 0$ and $R(0) \geq 0$ we Differentiate $N(t)$ with respect to t;

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dP}{dt} + \frac{dI_H}{dt} + \frac{dI_D}{dt} + \frac{dI_{HD}}{dt} + \frac{dR_D}{dt} \tag{2}$$

$$\frac{dN}{dt} = \pi + \delta_2 P + \sigma_2 R_D - \beta_1 S - \beta_2 S - \delta_1 S - \mu S + \delta_1 S - \mu P - \omega_1 P - \delta_2 P - \omega_2 P - \beta_1 S + \omega_1 P - \mu I_H - \gamma I_H -$$

$\tau I_H - \theta_1 I_H + \beta_2 S + \omega_2 P + \sigma_2 R_D - \mu I_D - \rho I_D - \theta_2 I_D - \alpha_1 I_D + \theta_2 I_D + \theta_1 I_H - \eta_1 I_{HD} - \eta_2 I_{HD} - \mu I_{HD} - \rho I_{HD}$
$-\gamma I_{HD} + \eta I_{HD} + \tau I_H + \sigma_1 R_D - \mu T_H - \gamma T_H + \eta_2 I_{HD} + \alpha_1 I_D - \sigma_1 R_D - \alpha_2 R_D - \sigma_2 R_D - \mu R_D - \gamma R_D - \rho R_D$

The equation can be simplified as

$$\frac{dN}{dt} = \pi - \mu(S + P + I_H + I_D + I_{HD} + T_H + R_D) - \gamma(I_H + I_{HD} + T_H + R_D) - \rho(I_D + I_{HD} + R_D)$$

given that $N = S + P + I_H + I_D + I_{HD} + T_H + R_D$ then the equation can be simplified to

$$\frac{dN}{dt} = \pi - \mu N - \gamma(I_H + I_{HD} + T_H + R_D) - \rho(I_D + I_{HD} + R_D) \tag{3}$$

In a state of no HIV/AIDS, then there are no HIV/AIDS-induced deaths and therefore $\gamma = 0$, which further reduces the equation to

$$\frac{dN}{dt} = \pi - \mu N - \rho(I_D + I_{HD} + R_D)$$

taking a situation where there is no drug abuse in the population, this translates to no drug abuse caused deaths, therefore $\rho = 0$. This further reduces the equation to

$$\frac{dN}{dt} \leq \pi - \mu N(t) \tag{4}$$

integrating the equation on both sides we have

$$\Rightarrow \int \frac{dN}{dt} = \int (\pi - \mu N(t)) \tag{5}$$

taking $v = \pi - \mu N(t)$

$$\frac{dv}{dt} = \frac{d(N)}{dt} - \mu$$

hence

$$dN = \int \frac{dV}{-\mu}$$
$$= \frac{-1}{\mu} \int \frac{dv}{\mu} = \frac{-1}{\mu} lnv$$

but

$$V = \pi - \mu(N(t))$$

therefore

$$\frac{-1}{\mu} ln((\pi - \mu N(t)) = t$$
$$ln((\pi - \mu N(t)) = -\mu t + c$$

Introducing exponential on both sides, we have

$$e^{(ln(\pi - \mu N))} = F e^{(-\mu t + c)}$$

This then becomes

$$\pi - \mu(N(t)) = e^{(-\mu t + c)}$$

$$F = e^c$$
$$\pi - \mu(N(t) = Fe^{(-\mu t)} \tag{6}$$

From the equation above, we have

$$\frac{\mu N(t)}{\mu} = \frac{\pi - Fe^{-\mu t}}{\mu}$$

$$N(t) = \frac{\pi}{\mu} - \frac{F}{\mu}e^{-\mu t}$$

With $t = 0$ as the initial condition, we have $t = 0$ we have

$$N(0) = \frac{\pi}{\mu} - \frac{F}{\mu}$$

hence

$$F = \pi - \mu N(0)$$

When substituted, we have

$$N(t) = \frac{\pi}{\mu} - \frac{\pi - \mu N}{\mu}e^{-\mu t}$$

as t goes to ∞ we have $t \to \infty$

$$\frac{F - \mu N(0))}{\mu} \to 0$$

This shows that

$$N(t) = \frac{\pi}{\mu} - 0(e^{-\mu t})$$

therefore

$$0 \leq N(t) \leq \frac{\pi}{\mu} \tag{7}$$

This proves that the model is bounded therefore

$$\bar{X} = \left\{(S(t) + P(t) + I_H(t) + I_D(t) + I_{HD}(t) + T_H(t) + R_D(t) \in R_+^7), 0 \leq N(t) \leq \frac{\pi}{\mu}\right\} \tag{8}$$

therefore $\bar{X}$ is invariant for all $S \geq 0, P \geq 0, I_H \geq 0, I_D \geq 0, I_{HD} \geq 0, T_H \geq 0, R \geq 0$ [23]

4.2.2 Positivity

For a model to be epidemiologically meaningful, it must have non-negative values for all time (t).
Theorem: For all initial conditions $S > 0, P > 0, I_H > 0, I_D > 0, I_{HD} > 0, T_H > 0, R_D > 0$ Then the solution $S, P, I_H, I_D, I_{HD}, T_H, R_D$ for all $t \geq 0$ are positive.
Considering the equations of the model

$$\frac{dS}{dt} = \pi + \delta_2 P + \sigma_1 R_D - (\delta_1 + \beta_1 + \beta_2 + \mu)S$$

$$\frac{dS}{dt} \geq -(\delta_1 + \beta_1 + \beta_2 + \mu)S$$
$$\frac{dS}{s} \geq -(\delta_1 + \beta_1 + \beta_2 + \mu)d(t)$$

$$\int \frac{dS}{s} \geq -\int (\delta_1 + \beta_1 + \beta_2 + \mu)d(t)$$

$$S(t) \geq S_0 e^{-(\delta_1+\beta_1+\beta_2+\mu)t} \geq 0 \tag{9}$$

From the second equation of the model, we have

$$\frac{dP}{dt} = \delta_1 S - (\delta_2 + \omega_1 + \omega_2 + \mu)P$$

$$\frac{dP}{dt} \geq -(\delta_2 + \omega_1 + \omega_2 + \mu)P$$

$$\frac{dP}{p} \geq -(\delta_2 + \omega_1 + \omega_2 + \mu)d(t)$$

$$\int \frac{dP}{p} \geq \int -(\delta_2 + \omega_1 + \omega_2 + \mu)d(t)$$

$$P(t) \geq P_0 e^{-(\delta_2+\omega_1+\omega_2+\mu)t} \geq 0 \tag{10}$$

From the third equation

$$\frac{dI_H}{dt} = \beta_1 S + \omega_1 P - (\tau + \theta_1 + \mu + \gamma)I_H$$

$$\frac{dI_H}{dt} \geq -(\tau + \theta_1 + \mu + \gamma)I_H$$

$$\frac{dI_H}{I_H} \geq -(\tau + \theta_1 + \mu + \gamma)d(t)$$

$$\int \frac{dI_H}{I_H} \geq \int -(\tau + \theta_1 + \mu + \gamma)d(t)$$

$$I_H(t) \geq I_{H_0} e^{-(\tau+\theta_1+\mu+\gamma)t} \geq 0 \tag{11}$$

From the fourth equation

$$\frac{dI_D}{dt} = \beta_2 S + \omega_2 P + \alpha_2 R_D - (\theta_2 + \alpha_1 + \mu + \rho)I_D$$

$$\frac{dI_D}{dt} \geq -(\theta_2 + \alpha_1 + \mu + \rho)I_D$$

$$\frac{dI_D}{I_D} \geq -(\theta_2 + \alpha_1 + \mu + \rho)d(t)$$

$$\int \frac{dI_D}{I_D} \geq \int -(\theta_2 + \alpha_1 + \mu + \rho)d(t)$$

$$I_D(t) \geq I_{D_0} e^{-(\theta_2+\alpha_1+\mu+\rho)t} \geq 0 \tag{12}$$

From the fifth equation, we have

$$\frac{dI_{HD}}{dt} = \theta_2 I_D + \theta_1 I_H - (\eta_1 + \eta_2 + \mu + \rho + \gamma)I_{HD}$$

$$\frac{dI_{HD}}{dt} \geq -(\eta_1 + \eta_2 + \mu + \rho + \gamma)I_{HD}$$

$$\frac{dI_{HD}}{I_{HD}} \geq -(\eta_1 + \eta_2 + \mu + \rho + \gamma)d(t)$$

$$\int \frac{dI_{HD}}{I_{HD}} \geq \int -(\eta_1 + \eta_2 + \mu + \rho + \gamma)d(t)$$

$$I_{HD}(t) \geq I_{HD}0e^{-(\eta_1+\eta_2+\mu+\rho+\gamma)t} \geq 0 \tag{13}$$

Taking the sixth equation of the model into account, we have

$$\frac{dT_H}{dt} = \eta_1 I_{HD} + \tau I_H + \sigma_1 R_D - (\mu + \gamma)T_H$$

$$\frac{dT_H}{dt} \geq -(\mu + \gamma)T_H$$

$$\frac{dT_H}{T_H} \geq -(\mu + \gamma)dt$$

$$\int \frac{dT_H}{T_H} \geq \int -(\mu + \gamma)dt$$

$$T_H(t) \geq T_{H_0}e^{-(\mu+\gamma)t} \geq 0 \tag{14}$$

From the last equation of the system, we have

$$\frac{dR_D}{dt} = \eta_2 I_{HD} + \alpha_1 I_D - (\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho)R_D$$

$$\frac{dR_D}{dt} \geq -(\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho)R_D$$

$$\frac{dR_D}{R_D} \geq -(\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho)d(t)$$

$$\int \frac{dR_D}{R_D} \geq \int -(\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho)d(t)$$

$$R_D(t) \geq R_{D_0}e^{-(\sigma_1+\alpha_2+\sigma_2+\mu+\gamma+\rho)t} \geq 0 \tag{15}$$

From the equation as proven from 9, 10, 11, 12, 13, 14 and 15 that given the initial condition that $S(T) \geq 0, P(T) \geq 0, I_D(t) \geq 0, I_H(t) \geq 0, I_{HD}(t) \geq 0, T_H(t) \geq 0$ and $R_D(t) \geq 0$ are non-negative at all $t > 0$ the equations system is positive

4.3 Diseases Free Equilibrium(DFE)

In the disease-free equilibrium state, the system of differential equations is evaluated at zero, reflecting a condition in which the population is entirely free from infection. Under this equilibrium, there are no individuals infected with HIV/AIDS, no cases of drug abuse, no co-infections, no individuals undergoing HIV/AIDS treatment, and no individuals recovering from drug addiction. The DFE is given by $E^* = (S^*, P^*, I_H^*, I_D^*, I_{H_D}^*, T_H^*, R_D^*)$

$$\frac{dS}{dt} = \pi + \delta_2 P + \sigma_2 R_D - (\beta_1 + \beta_2 + \delta_1 + \mu)S \tag{16}$$

$$\frac{dP}{dt} = \delta_1 S - (\delta_2 + \omega_1 + \omega_2 + \mu)P \tag{17}$$

with no disease then $R_D = 0$ and $\beta_1 = \beta_2 = \omega_1 = \omega_2 = 0$ then the equation become

$$\frac{dS}{dt} = \pi + \delta_2 P - (\delta_1 + \mu)S$$

and

$$\frac{dP}{dt} = \delta_1 S - (\delta_2 + \mu)P$$

Considering the second equation, we have

$$\frac{dP}{dt} = \delta_1 S - (\delta_2 + \mu)P$$

$$0 = \delta_1 S - (\delta_2 + \mu)P$$

hence

$$P^* = \frac{\delta_1 S}{\delta_2 + \mu} \tag{18}$$

accounting for equation(16) we have

$$\frac{dS}{dt} = \pi + \delta_2 P - (\delta_1 + \mu)S$$

but

$$P^* = \frac{\delta_1 S}{\delta_2 + \mu}$$

therefore we have

$$\frac{dS}{dt} = \pi + \delta_2\left(\frac{\delta_1 S}{\delta_2 + \mu}\right) - (\delta_1 + \mu)S$$

$$0 = \pi(\delta_2 + \mu) + \delta_2(\delta_1 S) - (\delta_2 + \mu)(\delta_1 + \mu)S \tag{19}$$

therefore

$$-\pi(\delta_2 + \mu) = \delta_2(\delta_1 S) - (\delta_2 + \mu)(\delta_1 + \mu)S$$

The right side of the equation is reduced to

$$-\mu(\delta_1 + \delta_2 + \mu)S$$

the equation(19) becomes

$$-\pi(\delta_2 + \mu) = -\mu(\delta_1 + \delta_2 + \mu)S$$

Then we have

$$S^* = \frac{\pi}{\mu} \cdot \frac{\delta_2 + \mu}{\delta_1 + \delta_2 + \mu} \tag{20}$$

now substituting S^* in P^* we have

$$P^* = \frac{\pi}{\mu} \cdot \frac{\delta_1}{\delta_1 + \delta_2 + \mu} \tag{21}$$

at Disease free Equilibrium then $I_H = 0, I_D = 0, I_{HD} = 0, T_D = 0, R_D$ Therefore the Disease Free Equilibrium is given by $E^* = (S^*, P^*, I_H^*, I_D^*, I_{HD}^*, T_H^*, R_D^*)$

$$E^* = \left(\frac{\pi}{\mu} \cdot \frac{\delta_2 + \mu}{\delta_1 + \delta_2 + \mu}, \frac{\pi}{\mu} \cdot \frac{\delta_1}{\delta_1 + \delta_2 + \mu}, 0, 0, 0, 0, 0\right)$$

4.4 Basic Reproduction Number (R_0)

The reproduction number is the number of secondary infections originating from one primary infection. With control measures such as protection, we have control reproduction number. In this research, there are two control reproduction numbers for HIV and AIDS, and the other for Drug Abuse. Taking the equations of the model,

$$\frac{dS}{dt} = \pi + \delta_2 P + \sigma_1 R_D - (\delta_1 + \beta_1 + \beta_2 + \mu)S$$
$$\frac{dP}{dt} = \delta_1 S - (\delta_2 + \omega_1 + \omega_2 + \mu)P$$
$$\frac{dI_H}{dt} = \beta_1 S + \omega_1 P - (\tau + \theta_1 + \mu + \gamma)I_H$$
$$\frac{dI_D}{dt} = \beta_2 S + \omega_2 P + \alpha_2 R_D - (\theta_2 + \alpha_1 + \mu + \rho)I_D$$
$$\frac{dI_{HD}}{dt} = \theta_2 I_D + \theta_1 I_H - (\eta_1 + \eta_2 + \mu + \rho + \gamma)I_{HD}$$
$$\frac{dT_H}{dt} = \eta_1 I_{H_D} + \tau I_H + \sigma_1 R_D - (\mu + \gamma)T_H$$
$$\frac{dR_D}{dt} = \eta_2 I_{H_D} + \alpha_1 I_D - (\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho)R_D$$

Taking the infectious compartments as I_H, I_D, I_{H_D}

4.4.1 Basic Reproduction Number of HIV&AIDS

Therefore, when considering HIV & AIDS the infectious compartments are I_H and I_{H_D}

$$\frac{dI_H}{dt} = \beta_1 S + \omega_1 P - (\tau + \theta_1 + \mu + \gamma)I_H$$

and

$$\frac{dI_{HD}}{dt} = \theta_2 I_D + \theta_1 I_H - (\eta_1 + \eta_2 + \mu + \rho + \gamma)I_{HD}$$

Taking Q to represent the new infection and L to represent the transmission of HIV &AIDS in the infectious compartment. The reproduction number will be given by QL^{-1} therefore

$$Q = \begin{bmatrix} \frac{\partial q_1}{\partial I_H} & \frac{\partial q_1}{\partial I_{HD}} \\ \frac{\partial q_2}{\partial I_H} & \frac{\partial q_2}{\partial I_{HD}} \end{bmatrix}$$

and

$$L = \begin{bmatrix} \frac{\partial l_1}{\partial I_H} & \frac{\partial l_1}{\partial I_{HD}} \\ \frac{\partial l_2}{\partial I_H} & \frac{\partial l_2}{\partial I_{HD}} \end{bmatrix}$$

we have

$$Q = \begin{bmatrix} \beta_1 S & 0 \\ 0 & 0 \end{bmatrix}$$

$$L = \begin{bmatrix} \tau + \theta_1 + \mu + \gamma & 0 \\ \theta_2 & \eta_1 + \eta_2 + \mu + \rho + \gamma \end{bmatrix}$$

The inverse of L is

$$L^{-1} = \begin{bmatrix} \frac{1}{\tau+\theta_1+\mu+\gamma} & 0 \\ \frac{-\theta_2}{(\tau+\theta_1+\mu+\gamma)(\eta_1+\eta_2+\mu+\rho+\gamma)} & \frac{1}{\eta_1+\eta_2+\mu+\rho+\gamma} \end{bmatrix}$$

The reproduction is given byQL^{-1} therefore

$$\begin{bmatrix} \beta_1 S & 0 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\tau+\theta_1+\mu+\gamma} & 0 \\ \frac{-\theta_2}{(\tau+\theta_1+\mu+\gamma)(\eta_1+\eta_2+\mu+\rho+\gamma)} & \frac{1}{\eta_1+\eta_2+\mu+\rho+\gamma} \end{bmatrix}$$

=

$$\begin{bmatrix} \frac{\beta_1 S}{\tau+\theta_1+\mu+\gamma} & 0 \\ 0 & 0 \end{bmatrix}$$

hence, the Reproduction number for HIV& AIDS is

$$R_0 = \frac{\beta_1 S}{\tau + \theta_1 + \mu + \gamma} \tag{22}$$

The reproduction is given byQL^{-1} therefore

$$\begin{bmatrix} \beta_1 S + \omega_1 P & 0 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\tau+\theta_1+\mu+\gamma} & 0 \\ \frac{-\theta_2}{(\tau+\theta_1+\mu+\gamma)(\eta_1+\eta_2+\mu+\rho+\gamma)} & \frac{1}{\eta_1+\eta_2+\mu+\rho+\gamma} \end{bmatrix}$$

=

$$\begin{bmatrix} \frac{\beta_1 S+\omega_1 P}{\tau+\theta_1+\mu+\gamma} & 0 \\ 0 & 0 \end{bmatrix}$$

Therefore, the control reproduction number of HIV& AIDS is

$$R_c = \frac{\beta_1 S + \omega_1 P}{\tau + \theta_1 + \mu + \gamma} \tag{23}$$

4.4.2 Basic Reproduction Number of Drug Abuse

The reproduction number of Drug abuse is calculated considering the I_D and I_{HD}. The equations of the compartments are

$$\frac{dI_D}{dt} = \beta_2 S + \omega_2 P + \alpha_2 R_D - (\theta_2 + \alpha_1 + \mu + \rho) I_D$$
$$\frac{dI_{HD}}{dt} = \theta_2 I_D + \theta_1 I_H - (\eta_1 + \eta_2 + \mu + \rho + \gamma) I_{HD}$$

Taking Q to represent the new infection and L to represent the transmission of Drug abuse in the infectious compartment. The reproduction number will be given by QL^{-1} therefore

$$Q = \begin{bmatrix} \frac{\partial q_1}{\partial I_D} & \frac{\partial q_1}{\partial I_{HD}} \\ \frac{\partial q_2}{\partial I_D} & \frac{\partial q_2}{\partial I_{HD}} \end{bmatrix}$$

and

$$L = \begin{bmatrix} \frac{\partial l_1}{\partial I_D} & \frac{\partial l_1}{\partial I_{HD}} \\ \frac{\partial l_2}{\partial I_D} & \frac{\partial l_2}{\partial I_{HD}} \end{bmatrix}$$

we have

$$Q = \begin{bmatrix} \beta_2 S & 0 \\ 0 & 0 \end{bmatrix}$$

while

$$L = \begin{bmatrix} \theta_2 + \alpha_1 + \mu + \rho & 0 \\ \theta_2 & \eta_1 + \eta_2 + \mu + \rho + \gamma \end{bmatrix}$$

Taking the inverse of R, we have

$$L^{-1} = \begin{bmatrix} \frac{1}{\theta_2+\alpha_1+\mu+\rho} & 0 \\ \frac{-\theta_2}{(\theta_2+\alpha_1+\mu+\rho)(\eta_1+\eta_2+\mu+\rho+\gamma)} & \frac{1}{\eta_1+\eta_2+\mu+\rho+\gamma} \end{bmatrix}$$

QL^{-1} is therefore given by

$$QL^{-1} = \begin{bmatrix} \beta_2 S & 0 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\theta_2+\alpha_1+\mu+\rho} & 0 \\ \frac{-\theta_2}{(\theta_2+\alpha_1+\mu+\rho)(\eta_1+\eta_2+\mu+\rho+\gamma)} & \frac{1}{\eta_1+\eta_2+\mu+\rho+\gamma} \end{bmatrix}$$

=

$$\begin{bmatrix} \frac{\beta_2 S}{\theta_2+\alpha_1+\mu+\rho} & 0 \\ 0 & 0 \end{bmatrix}$$

Therefore, the reproduction number of Drug Abuse is

$$R_0 = \frac{\beta_2 S}{\theta_2 + \alpha_1 + \mu + \rho} \tag{24}$$

The control reproduction number for Drug Abuse is QL^{-1}, therefore

$$QL^{-1} = \begin{bmatrix} \beta_2 S + \omega_2 P & 0 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\theta_2+\alpha_1+\mu+\rho} & 0 \\ \frac{-\theta_2}{(\theta_2+\alpha_1+\mu+\rho)(\eta_1+\eta_2+\mu+\rho+\gamma)} & \frac{1}{\eta_1+\eta_2+\mu+\rho+\gamma} \end{bmatrix}$$

=

$$\begin{bmatrix} \frac{\beta_2 S+\omega_2 P}{\theta_2+\alpha_1+\mu+\rho} & 0 \\ 0 & 0 \end{bmatrix}$$

$$R_c = \frac{\beta_2 S + \omega_2 P}{\theta_2 + \alpha_1 + \mu + \rho} \tag{25}$$

4.4.3 Local Stability of the Disease Free Equilibrium

This is how the illness acts as it gets closer to the equilibrium point. The disease can be eliminated from the population if the reproduction number is less than one, $R_0 < 1$, but in a situation where the reproduction number is greater than one, then the disease persist$R_0 > 1$. The Jacobian matrix is used to determine the stability of the disease

$$J = \begin{bmatrix} \frac{\partial m_1}{\partial S} & \frac{\partial m_1}{\partial P} & \frac{\partial m_1}{\partial I_H} & \frac{\partial m_1}{\partial I_D} & \frac{\partial m_1}{\partial I_{HD}} & \frac{\partial m_1}{\partial T_H} & \frac{\partial m_1}{\partial R_D} \\ \frac{\partial m_2}{\partial S} & \frac{\partial m_2}{\partial P} & \frac{\partial m_2}{\partial I_H} & \frac{\partial m_2}{\partial I_D} & \frac{\partial m_2}{\partial I_{HD}} & \frac{\partial m_2}{\partial T_H} & \frac{\partial m_2}{\partial R_D} \\ \frac{\partial m_3}{\partial S} & \frac{\partial m_3}{\partial P} & \frac{\partial m_3}{\partial I_H} & \frac{\partial m_3}{\partial I_D} & \frac{\partial m_3}{\partial I_{HD}} & \frac{\partial m_3}{\partial T_H} & \frac{\partial m_3}{\partial R_D} \\ \frac{\partial m_4}{\partial S} & \frac{\partial m_4}{\partial P} & \frac{\partial m_4}{\partial I_H} & \frac{\partial m_4}{\partial I_D} & \frac{\partial m_4}{\partial I_{HD}} & \frac{\partial m_4}{\partial T_H} & \frac{\partial m_4}{\partial R_D} \\ \frac{\partial m_5}{\partial S} & \frac{\partial m_5}{\partial P} & \frac{\partial m_5}{\partial I_H} & \frac{\partial m_5}{\partial I_D} & \frac{\partial m_5}{\partial I_{HD}} & \frac{\partial m_5}{\partial T_H} & \frac{\partial m_5}{\partial R_D} \\ \frac{\partial m_6}{\partial S} & \frac{\partial m_6}{\partial P} & \frac{\partial m_6}{\partial I_H} & \frac{\partial m_6}{\partial I_D} & \frac{\partial m_6}{\partial I_{HD}} & \frac{\partial m_6}{\partial T_H} & \frac{\partial m_6}{\partial R_D} \\ \frac{\partial m_7}{\partial S} & \frac{\partial m_7}{\partial P} & \frac{\partial m_7}{\partial I_H} & \frac{\partial m_7}{\partial I_D} & \frac{\partial m_7}{\partial I_{HD}} & \frac{\partial m_7}{\partial T_H} & \frac{\partial m_7}{\partial R_D} \end{bmatrix}$$

where

$$\begin{aligned} m_1 &= \pi + \delta_2 P + \sigma_1 R_D - (\delta_1 + \beta_1 + \beta_2 + \mu)S \\ m_2 &= \delta_1 S - (\delta_2 + \omega_1 + \omega_2 + \mu)P \\ m_3 &= \beta_1 S + \omega_1 P - (\tau + \theta_1 + \mu + \gamma)I_H \\ m_4 &= \beta_2 S + \omega_2 P + \alpha_2 R_D - (\theta_2 + \alpha_1 + \mu + \rho)I_D \\ m_5 &= \theta_2 I_D + \theta_1 I_H - (\eta_1 + \eta_2 + \mu + \rho + \gamma)I_{HD} \\ m_6 &= \eta_1 I_{HD} + \tau I_H + \sigma_1 R_D - (\mu + \gamma)T_H \\ m_7 &= \eta_2 I_{HD} + \alpha_1 I_D - (\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho)R_D \end{aligned} \tag{26}$$

$$J = \begin{bmatrix} -Z_1 & \delta_2 P^* & 0 & 0 & 0 & 0 & \sigma_2 \\ \delta_1 & -Z_2 & 0 & 0 & 0 & 0 & 0 \\ \beta_1 S^* & \omega_1 P^* & -Z_3 & 0 & 0 & 0 & 0 \\ \beta_2 S^* & \omega_2 P^* & 0 & --Z_4 & 0 & 0 & 0 \\ 0 & 0 & \theta_1 & \theta_1 & -Z_5 & 0 & 0 \\ 0 & 0 & \tau & 0 & \eta_1 & -Z_6 & \sigma_2 \\ 0 & 0 & 0 & \alpha_1 & \eta_2 & 0 & -Z_7 \end{bmatrix} \tag{27}$$

Where $Z_1 = \delta_1 + \beta_1 + \beta_2 + \mu, Z_2 = \delta_2 + \omega_1 + \omega_2 + \mu, Z_3 = \tau + \theta_1 + \mu + \gamma, Z_4 = \theta_2 + \alpha_1 + \mu + \rho, Z_5 = \mu + \gamma + \rho + \eta_1 + \eta_2, Z_6 = \mu + \gamma$ and $Z_7 = \sigma_1 + \sigma_2 + \mu + \gamma + \rho$

At disease-free Equilibrium

$$E^* = \left(\frac{\pi}{\mu} \cdot \frac{\delta_2 + \mu}{\delta_1 + \delta_2 + \mu}, \frac{\pi}{\mu} \cdot \frac{\delta_1}{\delta_1 + \delta_2 + \mu}, 0, 0, 0, 0, 0\right)$$

$$J_E = \begin{bmatrix} -Z_1 & \frac{\pi\delta_2}{\mu} \cdot \frac{\delta_1}{\delta_1+\delta_2+\mu} & 0 & 0 & 0 & 0 & \sigma_2 \\ \delta_1 & -Z_2 & 0 & 0 & 0 & 0 & 0 \\ \frac{\pi\beta_1}{\mu} \cdot \frac{\delta_2+\mu}{\delta_1+\delta_2+\mu} & \frac{\pi\omega_1}{\mu} \cdot \frac{\delta_1}{\delta_1+\delta_2+\mu} & -Z_3 & 0 & 0 & 0 & 0 \\ \frac{\pi\beta_2}{\mu} \cdot \frac{\delta_2+\mu}{\delta_1+\delta_2+\mu} & \frac{\pi\omega_2}{\mu} \cdot \frac{\delta_1}{\delta_1+\delta_2+\mu} & 0 & --Z_4 & 0 & 0 & 0 \\ 0 & 0 & \theta_1 & \theta_1 & -Z_5 & 0 & 0 \\ 0 & 0 & \tau & 0 & \eta_1 & -Z_6 & \sigma_2 \\ 0 & 0 & 0 & \alpha_1 & \eta_2 & 0 & -Z_7 \end{bmatrix}$$

Then

$$J_E - \Lambda = \begin{bmatrix} -Z_1 - \lambda & \frac{\pi\delta_2}{\mu} \cdot \frac{\delta_1}{\delta_1+\delta_2+\mu} & 0 & 0 & 0 & 0 & \sigma_2 \\ \delta_1 & -Z_2 - \lambda & 0 & 0 & 0 & 0 & 0 \\ \frac{\pi\beta_1}{\mu} \cdot \frac{\delta_2+\mu}{\delta_1+\delta_2+\mu} & \frac{\pi\omega_1}{\mu} \cdot \frac{\delta_1}{\delta_1+\delta_2+\mu} & -Z_3 - \lambda & 0 & 0 & 0 & 0 \\ \frac{\pi\beta_2}{\mu} \cdot \frac{\delta_2+\mu}{\delta_1+\delta_2+\mu} & \frac{\pi\omega_2}{\mu} \cdot \frac{\delta_1}{\delta_1+\delta_2+\mu} & 0 & -Z_4 - \lambda & 0 & 0 & 0 \\ 0 & 0 & \theta_1 & \theta_1 & -Z_5 - \lambda & 0 & 0 \\ 0 & 0 & \tau & 0 & \eta_1 & -Z_6 - \lambda & \sigma_2 \\ 0 & 0 & 0 & \alpha_1 & \eta_2 & 0 & -Z_7 - \lambda \end{bmatrix}$$

The eigenvalues of the matrix are

$$\Lambda_1 = -\delta_1 - \beta_1 - \beta_2 - \mu < 0$$

$$\Lambda_2 = -\delta_2 - \omega_1 - \omega_2 - \mu < 0$$

$$\Lambda_3 = -\tau - \theta_1 - \mu - \gamma < 0$$

$$\Lambda_4 = -\theta_2 - \alpha_1 - \mu - \rho < 0$$
$$\Lambda_5 = -\mu - \gamma - \rho - \eta_1 - \eta_2 < 0$$
$$\Lambda_6 = -\sigma_1 - \sigma_2 - \mu - \gamma - \rho < 0$$
$$\Lambda_7 = -\mu - \gamma < 0$$

Following the principle in mathematical epidemiology, "the disease-free equilibrium is locally asymptotically stable if and only if $R_0 < 1$, which corresponds to all eigenvalues of the Jacobian evaluated at the DFE having negative real parts." [26]
The system is asymptotically locally stable since the eigenvalues are negative or smaller than zero.

4.4.4 Global Stability of Disease Free Equilibrium

Theorem; If the $R_0 < 1$, then the Disease Free Equilibrium is globally stable asymptotically.

Proof: using the Lyapunov function technique

- Global stability of HIV/AIDS

$$P_1 = \frac{1}{(\tau + \theta_1 + \mu + \gamma)} I_H + \frac{1}{(\eta_1 + \eta_2 + \mu + \rho + \gamma)} I_{HD}$$
$$\frac{1}{(\tau + \theta_1 + \mu + \gamma)} (\beta_1 S + \omega_1 P - (\tau + \theta_1 + \mu + \gamma))$$
$$+ \frac{1}{(\eta_1 + \eta_2 + \mu + \rho + \gamma)} (\theta_2 I_D + \theta_1 I_H - (\eta_1 + \eta_2 + \mu + \rho + \gamma))$$

Taking the negatives on one side we have

$$\frac{1}{(\tau + \theta_1 + \mu + \gamma)} \beta_1 S + \omega_1 P + \frac{1}{(\eta_1 + \eta_2 + \mu + \rho + \gamma)} \theta_2 I_D + \theta_1 I_H$$
$$\leq \frac{1}{(\tau + \theta_1 + \mu + \gamma)} (\tau + \theta_1 + \mu + \gamma) + \frac{1}{(\eta_1 + \eta_2 + \mu + \rho + \gamma)} (\eta_1 + \eta_2 + \mu + \rho + \gamma)$$
$$\frac{\beta_1 S + \omega_1 P}{(\tau + \theta_1 + \mu + \gamma)} + \frac{\theta_2 I_D + \theta_1 I_H}{(\eta_1 + \eta_2 + \mu + \rho + \gamma)} \leq 2$$

The controlled reproduction number of HIV/AIDS is $\frac{\beta_1 S + \omega_1 P}{(\tau + \theta_1 + \mu + \gamma)}$ then

$$R_C + \frac{\theta_2 I_D + \theta_1 I_H}{(\eta_1 + \eta_2 + \mu + \rho + \gamma)} \leq 2$$

Therefore

$$R_C \leq 2 - \frac{\theta_2 I_D + \theta_1 I_H}{(\eta_1 + \eta_2 + \mu + \rho + \gamma)}$$

this shows that $R_C \leq 1$ then $\frac{dP_1}{dt} \leq 0$ hence the disease-free equilibrium for HIV/AIDS is globally asymptotically stable.

- Global stability for Drug Abuse

$$P_2 = \frac{1}{(\theta_2+\alpha_1+\mu+\rho)} I_D + \frac{1}{(\eta_1+\eta_2+\mu+\rho+\gamma)} I_{HD}$$

$$\frac{1}{(\theta_2+\alpha_1+\mu+\rho)}(\beta_2 S + \omega_2 P + \alpha_2 R_D - (\theta_2+\alpha_1+\mu+\rho))$$

$$+\frac{1}{(\eta_1+\eta_2+\mu+\rho+\gamma)}(\theta_2 I_D + \theta_1 I_H - (\eta_1+\eta_2+\mu+\rho+\gamma))$$

$$\frac{\beta_2 S+\omega_2 P}{\theta_2+\alpha_1+\mu+\rho} + \frac{\theta_2 I_D+\theta_1 I_H}{\eta_1+\eta_2+\mu+\rho+\gamma}$$

$$\leq \frac{1}{\theta_2+\alpha_1+\mu+\rho}(\theta_2+\alpha_1+\mu+\rho)+\frac{1}{\eta_1+\eta_2+\mu+\rho+\gamma}(\eta_1+\eta_2+\mu+\rho+\gamma)-\frac{\alpha_2 R_D}{\theta_2+\alpha_1+\mu+\rho}$$

$$\frac{\beta_2 S+\omega_2 P}{\theta_2+\alpha_1+\mu+\rho} + \frac{\theta_2 I_D+\theta_1 I_H}{\eta_1+\eta_2+\mu+\rho+\gamma} \leq 2 - \frac{\alpha_2 R_D}{\theta_2+\alpha_1+\mu+\rho}$$

then

$$\frac{\beta_2 S+\omega_2 P}{\theta_2+\alpha_1+\mu+\rho} \leq 2 - \frac{\alpha_2 R_D}{\theta_2+\alpha_1+\mu+\rho} - \frac{\theta_2 I_D+\theta_1 I_H}{\eta_1+\eta_2+\mu+\rho+\gamma}$$

since $\frac{\beta_2 S+\omega_2 P}{\theta_2+\alpha_1+\mu+\rho} = R_C$ then

$$R_C \leq 2 - \frac{\alpha_2 R_D}{\theta_2+\alpha_1+\mu+\rho} - \frac{\theta_2 I_D+\theta_1 I_H}{\eta_1+\eta_2+\mu+\rho+\gamma}$$

Therefore $R_C \leq 1$ hence $\frac{dP_1}{dt} \leq 0$ hence the disease-free equilibrium for Drug Abuse is globally asymptotically stable.

4.5 Endemic Equilibrium

When at least one of the infectious compartments is not equal to zero, the disease will persist in the population. At equilibrium, the rate of change in the compartments is Zero, and the system is written as follows.

$$\begin{aligned}
0 &= \pi + \delta_2 P + \sigma_1 R_D - (\delta_1+\beta_1+\beta_2+\mu)S \\
0 &= \delta_1 S - (\delta_2+\omega_1+\omega_2+\mu)P \\
0 &= \beta_1 S + \omega_1 P - (\tau+\theta_1+\mu+\gamma)I_H \\
0 &= \beta_2 S + \omega_2 P + \alpha_2 R_D - (\theta_2+\alpha_1+\mu+\rho)I_D \\
0 &= \theta_2 I_D + \theta_1 I_H - (\eta_1+\eta_2+\mu+\rho+\gamma)I_{HD} \\
0 &= \eta_1 I_{HD} + \tau I_H + \sigma_1 R_D - (\mu+\gamma)T_H \\
0 &= \eta_2 I_{HD} + \alpha_1 I_D - (\sigma_1+\alpha_2+\sigma_2+\mu+\gamma+\rho)R_D
\end{aligned} \tag{28}$$

Taking the endemic equilibrium point as E_0. Then

$$E_0 = (S^0, P^0, I_H^0, I_D^0, I_{HD}^0, T_H^0, R_D^0)$$

Accounting for the first equation in the system, we have,

$$0 = \pi + \delta_2 P + \sigma_1 R_D - (\delta_1+\beta_1+\beta_2+\mu)S$$

$$(\delta_1 + \beta_1 + \beta_2 + \mu)S = \pi + \delta_2 P + \sigma_1 R_D^0$$

$$S^0 = \frac{\pi + \delta_2 P^0 + \sigma_1 R_D^0}{(\delta_1 + \beta_1 + \beta_2 + \mu)} \tag{29}$$

from the second equation

$$0 = \delta_1 S - (\delta_2 + \omega_1 + \omega_2 + \mu)P$$

$$(\delta_2 + \omega_1 + \omega_2 + \mu)P = \delta_1 S^0$$

$$P^0 = \frac{\delta_1 S^0}{(\delta_2 + \omega_1 + \omega_2 + \mu)} \tag{30}$$

From the third equation, we have

$$0 = \beta_1 S + \omega_1 P - (\tau + \theta_1 + \mu + \gamma)I_H$$

$$(\tau + \theta_1 + \mu + \gamma)I_H = \beta_1 S^0 + \omega_1 P^0$$

$$I_H = \frac{\beta_1 S^0 + \omega_1 P^0}{(\tau + \theta_1 + \mu + \gamma)} \tag{31}$$

The fourth equation

$$0 = \beta_2 S + \omega_2 P + \alpha_2 R_D - (\theta_2 + \alpha_1 + \mu + \rho)I_D$$

$$(\theta_2 + \alpha_1 + \mu + \rho)I_D = \beta_2 S^0 + \omega_2 P^0 + \alpha_2 R_D^0$$

$$I_D^0 = \frac{\beta_2 S^0 + \omega_2 P^0 + \alpha_2 R_D^0}{\theta_2 + \alpha_1 + \mu + \rho} \tag{32}$$

The fifth equation we have

$$0 = \theta_2 I_D + \theta_1 I_H - (\eta_1 + \eta_2 + \mu + \rho + \gamma)I_{HD}$$

$$(\eta_1 + \eta_2 + \mu + \rho + \gamma)I_{H_D} = \theta_2 I_D^0 + \theta_1 I_H^0$$

$$I_{HD}^0 = \frac{\theta_2 I_D^0 + \theta_1 I_H^0}{\eta_1 + \eta_2 + \mu + \rho + \gamma} \tag{33}$$

Taking the sixth equation, we have

$$0 = \eta_1 I_{HD} + \tau I_H + \sigma_1 R_D - (\mu + \gamma)T_H$$

$$(\mu + \gamma)T_H = \eta_1 I_{HD}^0 + \tau I_H^0 + \sigma_1 R_D^0$$

$$T_H^0 = \frac{\eta_1 I_{HD}^0 + \tau I_H^0 + \sigma_1 R_D^0}{\mu + \gamma} \tag{34}$$

From the seventh equation, we have

$$0 = \eta_2 I_{HD} + \alpha_1 I_D - (\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho)R_D$$

$$(\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho)R_D = \eta_2 I_{HD}^0 + \alpha_1 I_D^0$$

$$R_D^0 = \frac{\eta_2 I_{H_D}^0 + \alpha_1 I_D^0}{\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho} \tag{35}$$

Therefore, the endemic equilibrium point is given by

$$E_0 = (S^0, P^0, I_H^0, I_D^0, I_{HD}^0, T_H^0, R_D^0)$$

$$E_0 = \left(\frac{\pi + \sigma_1 R_D^0 + \delta_2 P^0}{(\delta_1 + \beta_1 + \beta_2 + \mu)}, \frac{\delta_1 S^0}{(\delta_2 + \omega_1 + \omega_2 + \mu)}, \frac{\beta_1 S^0 + \omega_1 P^0}{(\tau + \theta_1 + \mu + \gamma)}, \frac{\beta_2 S^0 + \omega_2 P^0 + \alpha_2 R_D^0}{\theta_2 + \alpha_1 + \mu + \rho}, \right.$$
$$\left. \frac{\theta_2 I_D^0 + \theta_1 I_H^0}{\eta_1 + \eta_2 + \mu + \rho + \gamma}, \frac{\eta_1 I_{HD}^0 + \tau I_H^0 + \sigma_1 R_D^0}{\mu + \gamma}, \frac{\eta_2 I_{HD}^0 + \alpha_1 I_D^0}{\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho}\right)$$

4.5.1 Stability of Endemic Equilibrium Point

The stability of the Endemic Equilibrium is investigated using the Lyapunov function. The function is viewed as

$$Q = (S_*, P_*, I_{H_*}, I_{D_*}, I_{HD_*}, T_{H_*}, R_{D_*}) = (S - S_* - S_* lnS) + (P - P_* - P_* lnP)$$
$$+(I_H - I_{H*} - I_{H*} lnI_H) + (I_D - I_{D*} - I_{D_*} lnI_D) + (I_{HD} - I_{HD*} - I_{HD*} lnI_{HD})$$
$$+(T_H - T_{H_*} - T_{H_*} lnT_H + (R_D - R_{D_*} - R_{D_*} lnR_D)$$

Taking the differentiation of both sides we have

$$\frac{dQ}{dt} = \frac{dS}{dt}\left(\frac{S - S_*}{S}\right) + \frac{dP}{dt}\left(\frac{P - P_*}{P}\right) + \frac{dI_H}{dt}\left(\frac{I_H - I_{H_*}}{I_H}\right) +$$
$$\frac{dI_D}{dt}\left(\frac{I_D - I_{D*}}{I_D}\right) + \frac{dI_{HD}}{dt}\left(\frac{I_{HD} - I_{HD*}}{I_{HD}}\right) + \frac{dT_H}{dt}$$
$$\left(\frac{T_H - T_{H*}}{T_H}\right) + \frac{dR_D}{dt}\left(\frac{R_D - R_{D*}}{R_D}\right)$$

$$\frac{dQ}{dt} = \left(1 - \frac{S_*}{S}\right)[\pi + \delta_2 P + \sigma_1 R_D - (\delta_1 + \beta_1 + \beta_2 + \mu)S] + \left(1 - \frac{P_*}{P}\right)[\delta_1 S - (\delta_2 + \omega_1 + \omega_2 + \mu)P] + \tag{36}$$
$$\left(1 - \frac{I_{H*}}{I_H}\right)[\beta_1 S + \omega_1 P - (\tau + \theta_1 + \mu + \gamma)I_H] + \left(1 - \frac{I_{D*}}{I_D}\right)[\beta_2 S + \omega_2 P + \alpha_2 R_D - (\theta_2 + \alpha_1 + \mu + \rho)I_D] +$$
$$\left(1 - \frac{I_{HD*}}{I_{HD}}\right)[\theta_2 I_D + \theta_1 I_H - (\eta_1 + \eta_2 + \mu + \rho + \gamma)I_{HD}] + \left(1 - \frac{T_{H*}}{T_H}\right)[\eta_1 I_{HD} + \tau I_H + \sigma_1 R_D - (\mu + \gamma)T_H]$$
$$+\left(1 - \frac{R_{D*}}{R_D}\right)[\eta_2 I_{HD} + \alpha_1 I_D - (\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho)R_D]$$

The equation 20 above can be simplified

$$\frac{dQ}{dt} = \pi + \delta_2 P + \sigma_1 R_D - (\delta_1 + \beta_1 + \beta_2 + \mu)S - \frac{\pi S_*}{S} - \frac{\sigma_1 R_D S_*}{S} + (\delta_1 + \beta_1 + \beta_2 + \mu)S_*$$
$$+\delta_1 S - (\delta_2 + \omega_1 + \omega_2 - \mu)P - \frac{\delta_1 S P_*}{P} + (\delta_2 + \omega_1 + \omega_2 + \mu)P_* + \beta_1 S + \omega_1 P - (\tau + \theta_1 + \mu + \gamma)I_H$$

$$-\frac{\beta_1 S I_{H_*}}{I_H} - \frac{\omega_1 P I_{H_*}}{I_H} + (\tau+\theta_1+\mu+\gamma)I_{H_*} + \beta_2 S + \omega_2 P + \alpha_2 R_D$$

$$-(\theta_2+\alpha_1+\mu+\rho)I_D - \frac{\beta_2 S I_{D_*}}{I_D} - \frac{\omega_2 P I_{D_*}}{I_D} - \frac{\alpha_2 R_D I_{D_*}}{I_D} + (\theta_2+\alpha_1+\mu+\rho)I_{D_*}$$

$$+\theta_2 I_D + \theta_1 I_H - (\eta_1+\eta_2+\mu+\rho+\gamma)I_{HD} - \frac{\theta_2 I_D I_{HD*}}{I_{HD}} + \frac{\theta_1 I_H I_{HD_*}}{I_{HD}} + (\eta_1+\eta_2+\mu+\rho+\gamma)I_{HD_*} + \eta_1 I_{HD}$$

$$+\tau I_H + \sigma_1 R_D - (\mu+\gamma)T_H - \frac{\eta_1 I_{HD} T_{H_*}}{T_H} - \frac{\tau I_H T_{H_*}}{T_H} - \frac{\sigma_1 R_D T_{H_*}}{T_H} + (\mu+\gamma)T_{H_*} + \eta_2 I_{HD} + \alpha_1 I_D$$

$$-(\sigma_1+\alpha_2+\sigma_2+\mu+\gamma+\rho)R_D - \frac{\eta_2 I_{HD} R_{D_*}}{R_D} - \frac{\alpha_1 I_D R_{D_*}}{R_D} + (\sigma_1+\alpha_2+\sigma_2+\mu+\gamma+\rho)R_{D_*} \quad (37)$$

Rearranging the equations, we have

$$\frac{dQ}{dt} = \pi+\delta_2 P+\sigma_1 R_D+(\delta_1+\beta_1+\beta_2+\mu)S_*+\delta_1 S+(\delta_2+\omega_1+\omega_2+\mu)P_*+\beta_1 S+(\delta_2+\omega_1+\omega_2+\mu)P_*$$

$$+\beta_1 S + \omega_1 P(\tau+\theta_1+\mu+\gamma)I_{H_*} + \beta_2 S + \omega_2 P + \alpha_2 R_D$$

$$+(\theta_2+\alpha_1+\mu+\rho)I_{D_*} + \theta_2 I_D + \theta_1 I_H + (\eta_1+\eta_2+\mu+\rho$$

$$+\gamma)I_{HD_*} + \eta_1 I_{HD} + \tau I_H + \sigma_1 R_D + \eta_2 I_{HD} + \alpha_1 I_D + (\sigma_1+\alpha_2+\sigma_2+\mu+\gamma+\rho)R_{D_*}$$

$$-\Bigg((\delta_1+\beta_1+\beta_2+\mu)S + \frac{\pi S_*}{S} + \frac{\sigma_1 R_D S_*}{S} + (\delta_2+\omega_1+\omega_2+\mu)P + \frac{\delta S P_*}{P}$$

$$+(\tau+\theta_1+\mu+\gamma)I_H + \frac{\beta_1 S I_{H_*}}{I_H} + \frac{\omega_1 P I_{H_*}}{I_H} + (\theta_2+\alpha_1+\mu+\rho)I_D$$

$$+\frac{\beta_2 S I_{D_*}}{I_D} + \frac{\omega_2 P I_{D_*}}{I_D} + \frac{\alpha_2 R_D I_{D_*}}{I_D} + (\eta_1+\eta_2+\mu+\rho+\gamma)I_{HD} + \frac{\theta_2 I_D I_{HD_*}}{I_{HD}}$$

$$+\frac{\theta_1 I_H I_{HD_*}}{I_{HD}} + (\mu+\gamma)T_H + \frac{\eta_1 I_{HD} T_{H_*}}{T_H} + \frac{\tau I_H T_{H_*}}{T_H} + \frac{\sigma_1 R_D T_{H_*}}{T_H}$$

$$+(\sigma_1+\alpha_2+\sigma_2+\mu+\gamma+\rho)R_D + \frac{\eta_2 I_{HD} R_{D_*}}{R_D} + \frac{\alpha_1 I_D R_{D_*}}{R_D}\Bigg) \quad (38)$$

Using W and R to represent the positive and negative terms respectively, we have

$$W = \frac{dQ}{dt} = \pi+\delta_2 P+\sigma_1 R_D+(\delta_1+\beta_1+\beta_2+\mu)S_*+\delta_1 S+(\delta_2+\omega_1+\omega_2+\mu)P_*+\beta_1 S+\omega_1 P+$$

$$(\delta_2+\omega_1+\omega_2+\mu)P_* + \beta_1 S + \omega_1 P(\tau+\theta_1+\mu+\gamma)I_{H_*} + \beta_2 S + \omega_2 P + \alpha_2 R_D +$$

$$(\theta_2+\alpha_1+\mu+\rho)I_{D_*}+\theta_2 I_D+\theta_1 I_H+(\eta_1+\eta_2+\mu+\rho+\gamma)I_{HD_*}+\eta_1 I_{HD}+\tau I_H+\sigma_1 R_D+\eta_2 I_{H_D}+\alpha_1 I_D+$$

$$(\sigma_1+\alpha_2+\sigma_2+\mu+\gamma+\rho)R_{D_*}$$

And

$$R = (\delta_1+\beta_1+\beta_2+\mu)S+\frac{\pi S_*}{S}+\frac{\sigma_1 R_D S_*}{S}+(\delta_2+\omega_1+\omega_2+\mu)P+\frac{\delta_1 S P_*}{P}+(\tau+\theta_1+\mu+\gamma)I_H+$$

$$\frac{\beta_1 S I_{H_*}}{I_H}+\frac{\omega_1 P I_{H_*}}{I_H}+(\theta_2+\alpha_1+\mu+\rho)I_D+\frac{\beta_2 S I_{D_*}}{I_D}+\frac{\omega_2 P I_{D_*}}{I_D}+\frac{\alpha_2 R_D I_{D_*}}{I_D}+(\eta_1+\eta_2+\mu+\rho+\gamma)I_{HD}+$$

$$\frac{\theta_2 I_D I_{HD_*}}{I_{HD}} + \frac{\theta_1 I_H I_{HD*}}{I_{HD}} + (\mu+\gamma)T_H + \frac{\eta_1 I_{HD} T_{H_*}}{T_H} +$$
$$\frac{\tau I_H T_{H_*}}{T_H} + \frac{\sigma_1 R_D T_{H_*}}{T_H} + (\sigma_1 + \alpha_2 + \sigma_2 + \mu + \gamma + \rho)R_D + \frac{\eta_2 I_{H_D} R_{D_*}}{R_D} + \frac{\alpha_1 I_D R_{D_*}}{R_D}$$

Therefore

$$\frac{dQ}{dt} = W - R$$

Using the conditions if $W < R$, then $\frac{dQ}{dt} \leq 0$ If and only if $S = S_*, P = P_*, I_H = I_{H_*}, I_D = I_{D_*}, I_{HD} = I_{HD_*}, T_H = T_{H_*}, R_D = R_{D_*}$ Then

$$\frac{dQ}{dt} = 0$$

this indicates that the highest invariant set in

$$\left(S_*, P_*, I_{H_*}, I_{D_*}, I_{HD_*}, T_{H_*}, R_{D_*}\right)\epsilon\Psi : \frac{dQ}{dt} = 0$$

The Endemic Equilibrium Point E_0 is globally asymptotically stable in the invariant set if $W < R$

4.6 Sensitvity Analysis

In this subsection, we focus on the importance of each parameter on HIV/AIDS and drug abuse by looking at the effectiveness of the control reproduction number. We use the variable normalized forward sensitivity, which is differentiable and relies on parameters. The Reproduction number sensitivity analysis index as $H_W^{R_C}$, is given by

$$H_W^{R_C} = \frac{W}{R_C} * \frac{\partial R_C}{\partial W} \tag{39}$$

where W is Taking the $R_C = \frac{\beta_2 S + \omega_2 P}{\theta_2 + \alpha_1 + \mu + \rho}$ for drug Abuse and

$R_C = \frac{\beta_1 S + \omega_1 P}{\tau + \theta_1 + \mu + \gamma}$ for HIV/AIDS and the their respective R_C at DFE as

$R_C = \frac{\pi\beta_1(\delta_2+\mu)+\omega_1(\pi\delta_1)}{\mu(\delta_1+\delta_2+\mu)(\tau+\theta_1+\mu+\gamma)}$ and

$R_c = \frac{\pi\beta_2(\delta_2+\mu)+\omega_2(\delta_1\pi)}{\mu(\delta_1+\delta_2+\mu)(\theta_2+\alpha_1+\mu+\rho)}$

Finding the sensitivity of π we have

$$H_W^{R_C} = \frac{\pi}{R_C} * \frac{\partial R_C}{\partial \pi} \tag{40}$$

But the terms in the brackets are the same as R_C, then 40 is reduced to

$$\frac{R_C}{\pi} * \frac{\pi}{R_C} = 1$$

and therefore the sensitivity of $\pi = 1$ when the other parameters are calculated, the following are their respective sensitivities.

PARAMETER	SENSITIVITY
π	+ 1.00
μ	-1.145
γ	-0.252
θ_1	-0.700
θ_2	-0.729
τ	-0.032
α_1	-0.1212
α_2	-0.0285
ρ	-0.0643
ω_1	+0.899
ω_2	+0.996
β_1	+0.0245
β_2	+0.051
δ_1	+0.179

Table 3: Sensitivity indices of parameters on R_C

From table 3, it can be deduced that when the infectious rate of the susceptible (β_1 and β_2) increases, the HIV/AIDS and drug abuse rates increase while other factors are held constant. When $\mu.\gamma, \theta_1, \theta_2, \tau, \alpha_1$ α_2 and ρ are increased R_C decreases but when π, ω_1 δ and ω_2 are increased the R_C also increase hence they are the most delicate parameters.

4.7 Numerical Simulation

In this section, a quantitative analysis of the model equations is conducted to examine the dynamics of HIV/AIDS and drug abuse within a population. The simulations were carried out using Maple software, which numerically solved the system of differential equations and generated the corresponding graphs. The analysis is based on the assumed initial conditions: $S(0) = 800000$, $P(0) = 50000$, $I_D(0) = 30000$, $I_H(0) = 50000$, $I_{HD}(0) = 30000$, $T_H(0) = 20000$, and $R_D(0) = 20000$. These conditions represent the population distribution across the seven compartments at time zero and serve as the basis for exploring disease transmission and control strategies.

4.7.1 HIV/AIDS and Drug Abuse dynamics on the population

The figure 2 presents the interrelationship among the various epidemiological compartments, namely: the susceptible population (S), protected individuals (P), those infected with HIV/AIDS (I_H), individuals engaged in drug abuse (I_D), those co-infected with both HIV/AIDS and drug abuse (I_{HD}), individuals undergoing treatment for HIV/AIDS (T_H), and those in rehabilitation for drug abuse (R_D). The graphical trends demonstrate that the susceptible population dominates initially but experiences a consistent decline over time as individuals transition into other compartments. The protected population remains relatively stable at low levels throughout the simulation period. Both the HIV-infected and drug-abusing populations show gradual increases during the early phase before stabilizing at moderate levels. The treatment compartments (T_H and R_D) exhibit growth over time, with T_H showing more substantial increases compared to R_D, indicating expanding treatment coverage. Moreover, the size of the co-infected population (I_{HD}) remains consistently low and exhibits a direct dependence on the magnitudes of

PARAMETERS	VALUE	SOURCE
π	0.0397	World Bank 2017
β_1	0.04	Kenya HIV estimate 2015
β_2	0.06	NACADA 2017
δ_1	0.1200	Estimated
δ_1	0.05	Estimated
ω_1	1.1100	Estimated
ω_2	0.0800	Estimated
θ_1	0.8333	[14]
θ_2	0.1700	[14]
τ	0.0390	[14]
η_1	0.9008	[14]
η_2	0.0962	[14]
σ_1	0.5200	Estimated
σ_2	0.3142	[2]
α_1	0.02827	[2]
α_2	0.0005	[14]
μ	0.0200	WHO 2015
γ	0.300	[4]
ρ	0.015	[14]

Table 4: Parameters, values and source

both I_H and I_D; as treatment interventions expand and protective measures take effect, the number of individuals simultaneously affected by both conditions remains minimal, demonstrating the effectiveness of comprehensive intervention strategies, specifically protection and rehabilitation programs, in managing dual epidemics.

4.7.2 Effect of protection on HIV/AIDS and Drug abuse

Graph 3 illustrates how varying the protection rate ω_1 influences the population of HIV-infected individuals. As $\omega_1$increases from 0.5 to 1.0 and then to 1.5, the HIV-infected population exhibits distinctly different dynamics throughout the epidemic progression. Lower protection rates ($\omega_1 = 0.5$) result in a more gradual rise to peak infection levels and a slower subsequent decline, while higher protection rates ($\omega_1 = 1.5$) demonstrate both a steeper initial increase and a more rapid decline in the infected population after reaching the peak. The intermediate protection level ($\omega_1 = 1.0$) shows moderate behavior between these extremes. A higher protection rate leads to more pronounced epidemic curves with faster resolution, demonstrating that effective protective measures, such as condom use, education programs, and behavioral interventions, significantly influence HIV/AIDS transmission dynamics. This emphasizes the importance of strong preventive strategies in not only slowing the spread of HIV/AIDS but also in accelerating the decline of the infected population, ultimately reducing the overall burden and duration of the epidemic within the community.

Graph 4 shows how varying the protection parameter ω_2 influences the population of drug users over time. As ω_2 increases from 0.5 to 1.0 and then to 1.5, the drug-using population exhibits similar growth patterns initially, with all scenarios reaching comparable

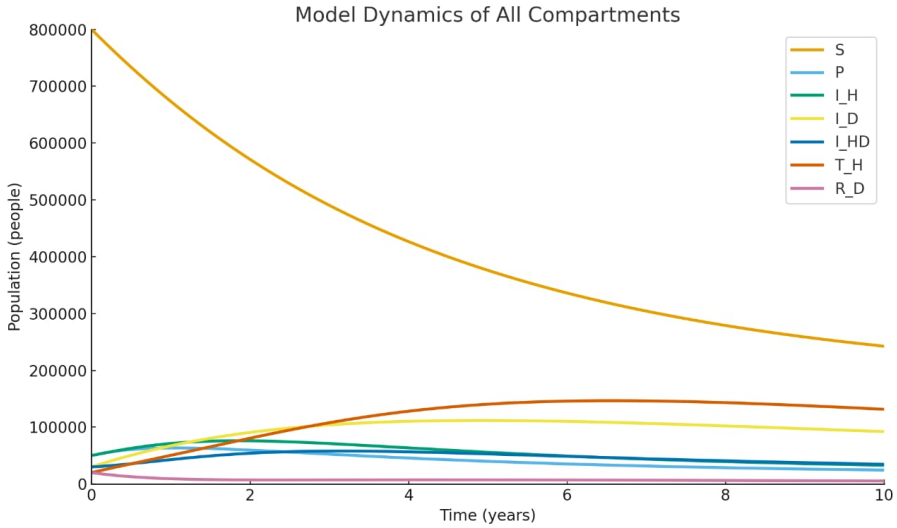


Figure 2: graph of the model

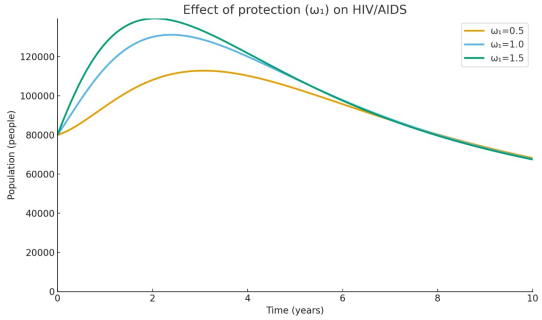


Figure 3: effect of protection on HIV/AIDS

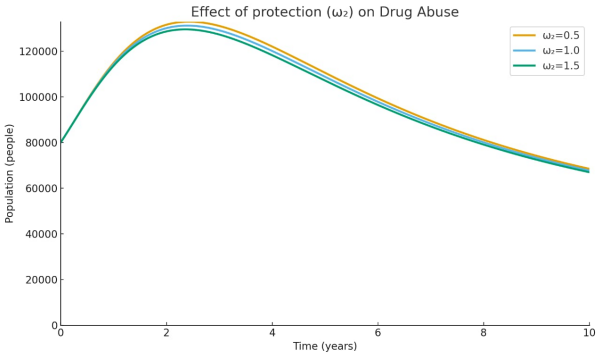


Figure 4: Effect of Protection on Drug Abuse

peak levels. However, the most significant differences emerge during the decline phase, where higher protection rates lead to marginally steeper decreases in the drug-using population. The $\omega_2 = 1.5$ scenario demonstrates the most rapid decline, followed by $\omega_2 = 1.0$, while $\omega_2 = 0.5$ shows the most gradual reduction. This demonstrates that effective protective measures, such as prevention programs, treatment accessibility, harm reduction strategies, and community education, can accelerate recovery rates and reduce the sustained burden of drug abuse within the population. However, all scenarios eventually converge to similar equilibrium levels, emphasizing that while protection measures influence the speed of population decline, comprehensive and sustained intervention strategies are necessary for achieving significant long-term reductions in drug abuse prevalence.

4.7.3 Effect of α_1 on Drug Abuse and Rehabilitation Compartments

Figure 5 illustrates the impact of varying the rehabilitation rate α_1 on the number of rehabilitated individuals (RD) over time. With parameters $\alpha_1 = 0.01000$, 0.02827, and 0.5000, it is evident that higher rehabilitation rates lead to a sharper rise in RD, reaching higher peaks more quickly. The curve for $\alpha_1 = 0.5$ shows the most significant increase, indicating that more drug abusers are successfully transitioned into rehabilitation. This

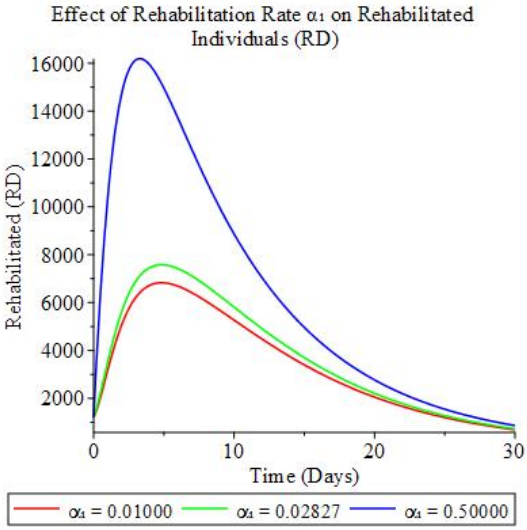


Figure 5: effect of α_1 on drug abuse

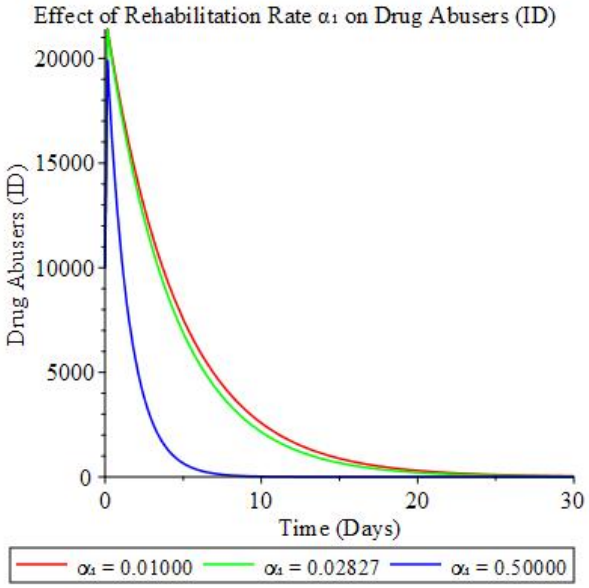


Figure 6: effect of α_1 on Rehabilitation

demonstrates that increasing α_1, the rate at which drug users receive help, significantly boosts the rehabilitation process and reduces prolonged addiction.

Figure 6 demonstrates how different rehabilitation rates $\alpha_1 = 0.0100$, 0.028270, and 0.50000influence the decline of drug abusers (ID) over time. Higher values of α_1 result in a faster and more pronounced reduction in the ID population. The curve for $\alpha_1 = 0.50000$ shows the sharpest decline, indicating highly effective rehabilitation. This reduction in drug abuse not only curbs addiction but also significantly lowers the risk of HIV transmission via drug-related behaviors. Therefore, enhancing rehabilitation efforts plays a critical dual role in managing drug abuse and preventing HIV/AIDS.

Chapter 5

5 Discussion, Conclusion, and Recommendation

5.1 Discussion

The results of this study showed that the disease-free equilibrium is locally and globally stable when the control reproduction number R_C is less than one. This means that under effective interventions such as protection and rehabilitation, both HIV/AIDS and drug abuse can be controlled and potentially eliminated from the population. These findings are in line with Mwangi [9], who also found that when $R_0 < 1$, the infection-free equilibrium is globally asymptotically stable in their HIV model, though their study focused only on within-host dynamics.

The study further demonstrated that rehabilitation and protection significantly reduce the prevalence of drug abusers, HIV-infected individuals, and dual infections. This is consistent with Alfiniyah [2], who showed that combining treatment with awareness campaigns reduced relapse and drug use prevalence, and Muin [3], who highlighted the effectiveness of psychological counselling alongside campaigns. However, this study extends their conclusions by showing that rehabilitation has an additional effect on reducing HIV transmission, thereby demonstrating a dual benefit.

The numerical simulations confirmed that increasing protection rates substantially lowers the number of HIV/AIDS cases. This aligns with Espitia [10], who emphasized behavioural interventions (e.g., reducing risky sexual contacts) as crucial in controlling HIV transmission. However, Espitia did not include drug abuse, whereas our model shows that protection becomes even more impactful in contexts where substance abuse exacerbates risky behaviour.

Additionally, the analysis indicated that higher rehabilitation rates lead to a decline in drug abuse and co-infections, while lower rates allow both epidemics to persist. These findings parallel Lokaran [14], who found that drug misuse contributes to the spread of HIV/AIDS, but their model did not explicitly incorporate rehabilitation or protection. Our study, therefore, advances this line of research by quantifying the specific role of rehabilitation in reducing co-infections.

The sensitivity analysis revealed that parameters associated with protection (δ) and rehabilitation (ω_1) had the most influence on the control reproduction number. Similar observations were made by Shiri[28], whose model showed that treatment coverage and prevention strategies were highly influential in HIV/AIDS control. By combining protection and rehabilitation, our model demonstrates a synergistic effect similar to the synergy Shiri observed between ART and prevention of mother-to-child transmission.

Finally, Bloomquist and Vaidya [5] demonstrated that morphine use biologically increases susceptibility to HIV infection. Our findings complement theirs by confirming that drug abuse not only elevates biological vulnerability but also significantly increases HIV transmission potential at the population level.

Overall, this study supports existing literature while extending it by explicitly integrating protection and rehabilitation into a unified mathematical framework. The results reinforce the argument that sustainable reductions in HIV/AIDS transmission require not only treatment but also strong behavioural interventions targeting drug abuse.

5.2 Conclusion

This study developed and analyzed a mathematical model to investigate the transmission dynamics of HIV/AIDS in the context of drug abuse, incorporating key compartments such as susceptible individuals, protected individuals, drug abusers, HIV-infected, dual-infected, treated, and rehabilitated populations. The model successfully demonstrated how drug abuse significantly contributes to the spread of HIV and how intervention strategies such as protection and rehabilitation influence disease dynamics. Simulation results revealed that an increase in protection rate reduces the spread of both HIV and drug abuse, while a higher rehabilitation rate substantially decreases the number of drug abusers and enhances recovery. The reproduction number R_0 was shown to be a critical threshold parameter; reducing it below one effectively controls the epidemic. Overall, the study affirms that targeted public health measures—particularly those aimed at enhancing protection and expanding rehabilitation services—are essential in mitigating the dual burden of HIV/AIDS and drug abuse within affected populations.

5.3 Recommendation

Based on the findings of this study, several recommendations can be made for public health policy and future research. First, programs that promote behavioral change through education and increase access to protective measures should be prioritized, as higher protection rates have been shown to significantly reduce HIV transmission. Second, the study highlights the importance of investing in comprehensive and accessible rehabilitation services, which not only reduce drug abuse but also indirectly lower HIV/AIDS co-infection rates. Policymakers should therefore integrate HIV/AIDS and substance abuse strategies to achieve more impactful health outcomes. Third, future mathematical models could incorporate more real-time data, gender-specific parameters, or age-based stratifications to enhance realism and applicability. Lastly, implementing this model within specific regions or communities could help tailor interventions that are context-specific and data-driven. These combined efforts would support a more effective and sustainable response to the intertwined challenges of HIV/AIDS and drug addiction.

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I HEREBY CONFIRM THAT **DOLPHINE MOCHERE** REG NO: **1046417**, SUBMITTED A CHECK OF ANTIPLAGIARISM ON CRITICAL EVALUATION OF **MATHEMATICAL MODELLING OF HIV/AIDS DYNAMICS IN RELATION TO DRUG ABUSE. A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE AWARD OF MASTER OF SCIENCE (MSC) IN APPLIED MATHEMATICS OF THE CATHOLIC UNIVERSITY OF EASTERN AFRICA.**

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Name: HARUN MAINA Signature: ..... Date: 15/9/2025

CamScanner

Numerical simulation Code

```
pi := 0.0397;
beta1 := 0.04;
beta2 := 0.06;
delta := 0.1200;
omega1 := 1.1;
omega2 := 0.08;
theta1 := 0.8333;
theta2 := 0.1700;
tau := 0.0390;
eta1 := 0.9008;
eta2 := 0.0962;
sigma1 := 0.5200;
sigma2 := 0.3142;
alpha2 := 0.0005;
mu := 0.0200;
gamma_ := 0.0200;
rho := 0.0150;
alpha1_vals := [0.01, 0.02827, 0.5];
colors := [red, green, blue];
plots_RD := [];
for i to nops(alpha1_vals) do
    alpha1 := alpha1_vals[i];
    eq1 := diff(S(t), t) = pi - beta1*S(t)*IH(t) - beta2*S(t)*ID(t) - mu*S(
    eq2 := diff(P(t), t) = theta1*S(t) - omega1*beta1*P(t)*IH(t) - omega2*b
    eq3 := diff(IH(t), t) = beta1*S(t)*IH(t) + omega1*beta1*P(t)*IH(t) - (t
    eq4 := diff(ID(t), t) = beta2*S(t)*ID(t) + omega2*beta2*P(t)*ID(t) - (t
    eq5 := diff(IHD(t), t) = theta2*ID(t) + theta1*IH(t) - (sigma1 + sigma2
    eq6 := diff(TH(t), t) = tau*IH(t) + sigma1*IHD(t) - (eta1 + mu)*TH(t);
    eq7 := diff(RD(t), t) = alpha1*ID(t) + sigma2*IHD(t) - (eta2 + alpha2 +
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    de_system := [eq1, eq2, eq3, eq4, eq5, eq6, eq7];
    sol := dsolve({ics, op(de_system)}, numeric, output = listprocedure, m
    plots_RD := [op(plots_RD), odeplot(sol, [t, RD(t)/3], 0 .. 30, color =
end do;
display(plots_RD, title = "Effect of Rehabilitation Rate &alpha;    on Reh

    with(plots);
with(DEtools);
pi := 0.0397;
beta1 := 0.04;
beta2 := 0.06;
delta := 0.1200;
omega1 := 1.1;
omega2 := 0.08;
theta1 := 0.8333;
theta2 := 0.1700;
```

```
tau := 0.0390;
eta1 := 0.9008;
eta2 := 0.0962;
sigma1 := 0.5200;
sigma2 := 0.3142;
alpha2 := 0.0005;
mu := 0.0200;
gamma_ := 0.0200;
rho := 0.0150;
alpha1_vals := [0.01, 0.02827, 0.5];
colors := [red, green, blue];
plots_ID := [];
for i to nops(alpha1_vals) do
    alpha1 := alpha1_vals[i];
    eq1 := diff(S(t), t) = pi - beta1*S(t)*IH(t) - beta2*S(t)*ID(t) - mu*S(
    eq2 := diff(P(t), t) = theta1*S(t) - omega1*beta1*P(t)*IH(t) - omega2*b
    eq3 := diff(IH(t), t) = beta1*S(t)*IH(t) + omega1*beta1*P(t)*IH(t) - (t
    eq4 := diff(ID(t), t) = beta2*S(t)*ID(t) + omega2*beta2*P(t)*ID(t) - (t
    eq5 := diff(IHD(t), t) = theta2*ID(t) + theta1*IH(t) - (sigma1 + sigma2
    eq6 := diff(TH(t), t) = tau*IH(t) + sigma1*IHD(t) - (eta1 + mu)*TH(t);
    eq7 := diff(RD(t), t) = alpha1*ID(t) + sigma2*IHD(t) - (eta2 + alpha2 +
    ics := S(0) = 50000, P(0) = 5000, ID(0) = 30000, IH(0) = 20396, IHD(0)
    de_system := [eq1, eq2, eq3, eq4, eq5, eq6, eq7];
    sol := dsolve({ics, op(de_system)}, numeric, output = listprocedure, m
    plots_ID := [op(plots_ID), odeplot(sol, [t, ID(t)/3], 0 .. 30, color =
end do;
display(plots_ID, title = "Effect of Rehabilitation Rate &alpha;    on Drug

    pi := 0.0397;
beta1 := 0.04;
beta2 := 0.06;
delta := 0.1200;
omega1 := 1.1;
omega2 := 0.08;
theta1 := 0.8333;
theta2 := 0.1700;
tau := 0.0390;
eta1 := 0.9008;
eta2 := 0.0962;
sigma1 := 0.5200;
sigma2 := 0.3142;
alpha1 := 0.02827;
alpha2 := 0.0005;
mu := 0.0200;
gamma_ := 0.0200;
rho := 0.0150;
eq1 := diff(S(t), t) = pi - beta1*S(t)*IH(t) - beta2*S(t)*ID(t) - mu*S(t);
eq2 := diff(P(t), t) = theta1*S(t) - omega1*beta1*P(t)*IH(t) - omega2*beta2
```

```
eq3 := diff(IH(t), t) = beta1*S(t)*IH(t) + omega1*beta1*P(t)*IH(t) - (tau +
eq4 := diff(ID(t), t) = beta2*S(t)*ID(t) + omega2*beta2*P(t)*ID(t) - (theta
eq5 := diff(IHD(t), t) = theta2*ID(t) + theta1*IH(t) - (sigma1 + sigma2 + m
eq6 := diff(TH(t), t) = tau*IH(t) + sigma1*IHD(t) - (eta1 + mu)*TH(t);
eq7 := diff(RD(t), t) = alpha1*ID(t) + sigma2*IHD(t) - (eta2 + alpha2 + mu)
ics := S(0) = 50000, P(0) = 5000, ID(0) = 30000, IH(0) = 20396, IHD(0) = 10
de_system := [eq1, eq2, eq3, eq4, eq5, eq6, eq7];
sol := dsolve({ics, op(de_system)}, numeric, output = listprocedure, maxfun
plot1 := odeplot(sol, [t, S(t)/3], 0 .. 30, color = blue, legend = "Suscep
plot2 := odeplot(sol, [t, P(t)/3], 0 .. 30, color = navy, legend = "Protect
plot3 := odeplot(sol, [t, IH(t)/3], 0 .. 30, color = red, legend = "HIV-Inf
plot4 := odeplot(sol, [t, ID(t)/3], 0 .. 30, color = green, legend = "Drug
plot5 := odeplot(sol, [t, IHD(t)/3], 0 .. 30, color = brown, legend = "Dua
plot6 := odeplot(sol, [t, TH(t)/3], 0 .. 30, color = purple, legend = "Trea
plot7 := odeplot(sol, [t, RD(t)/3], 0 .. 30, color = orange, legend = "Reh
display([plot1, plot2, plot3, plot4, plot5, plot6, plot7], title = "Simulat

    with(plots);
with(DEtools);
pi := 0.0397;
beta2 := 0.06;
delta := 0.1200;
omega1 := 1.1;
omega2 := 0.08;
theta1 := 0.8333;
theta2 := 0.1700;
tau := 0.0390;
eta1 := 0.9008;
eta2 := 0.0962;
sigma1 := 0.5200;
sigma2 := 0.3142;
alpha1 := 0.02827;
alpha2 := 0.0005;
mu := 0.0200;
rho := 0.0150;
scenarios := [[" R   > 1", 0.06, 0.01, 0.01], [" R   < 1", 0.02, 0.08, 0.0
colors := [red, green];
plots_IH := [];
plots_TH := [];
for i to nops(scenarios) do
    lbl := scenarios[i][1];
    b1 := scenarios[i][2];
    treat := scenarios[i][3];
    gamma_val := scenarios[i][4];
    eq1 := diff(S(t), t) = pi - b1*S(t)*IH(t) - mu*S(t);
    eq2 := diff(P(t), t) = theta1*S(t) - omega1*b1*P(t)*IH(t) - mu*P(t);
    eq3 := diff(IH(t), t) = b1*S(t)*IH(t) + omega1*b1*P(t)*IH(t) - (treat +
    eq4 := diff(TH(t), t) = treat*IH(t) - (eta1 + mu)*TH(t);
```

```
    ics := S(0) = 50000, P(0) = 5000, IH(0) = 20396, TH(0) = 32000;
    de_system := [eq1, eq2, eq3, eq4];
    sol := dsolve({ics, op(de_system)}, numeric, output = listprocedure, m
    plots_IH := [op(plots_IH), odeplot(sol, [t, IH(t)/3], 0 .. 30, color =
    plots_TH := [op(plots_TH), odeplot(sol, [t, TH(t)/3], 0 .. 30, color =
end do;
display([op(plots_IH), op(plots_TH)], title = "Effect of  R   on HIV/AIDS C

    with(plots);
with(DEtools);
pi := 0.0397;
beta1 := 0.04;
beta2 := 0.06;
delta := 0.1200;
omega2 := 0.08;
theta1 := 0.8333;
theta2 := 0.1700;
tau := 0.0390;
eta1 := 0.9008;
eta2 := 0.0962;
sigma1 := 0.5200;
sigma2 := 0.3142;
alpha1 := 0.02827;
alpha2 := 0.0005;
mu := 0.0200;
gamma_ := 0.0200;
rho := 0.0150;
omega1_vals := [0.019, 1.0, 0.05];
colors := [blue, red, green];
plots_IH := [];
for i to nops(omega1_vals) do
    omega1 := omega1_vals[i];
    eq1 := diff(S(t), t) = pi - beta1*S(t)*IH(t) - beta2*S(t)*ID(t) - mu*S(
    eq2 := diff(P(t), t) = theta1*S(t) - omega1*beta1*P(t)*IH(t) - omega2*b
    eq3 := diff(IH(t), t) = beta1*S(t)*IH(t) + omega1*beta1*P(t)*IH(t) - (t
    eq4 := diff(ID(t), t) = beta2*S(t)*ID(t) + omega2*beta2*P(t)*ID(t) - (t
    eq5 := diff(IHD(t), t) = theta2*ID(t) + theta1*IH(t) - (sigma1 + sigma2
    eq6 := diff(TH(t), t) = tau*IH(t) + sigma1*IHD(t) - (eta1 + mu)*TH(t);
    eq7 := diff(RD(t), t) = alpha1*ID(t) + sigma2*IHD(t) - (eta2 + alpha2 +
    ics := S(0) = 50000, P(0) = 5000, ID(0) = 30000, IH(0) = 20396, IHD(0)
    de_system := [eq1, eq2, eq3, eq4, eq5, eq6, eq7];
    sol := dsolve({ics, op(de_system)}, numeric, output = listprocedure, m
    plots_IH := [op(plots_IH), odeplot(sol, [t, IH(t)/3], 0 .. 30, color =
end do;
display(plots_IH, title = "Effect of Protection Rate (&omega;   ) on HIV/AI
```